THE APPLICATION OF RESPIRATORY MUSCLE TRAINING TO COMPETITIVE ROWING

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by
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ABSTRACT

Respiratory muscle training (RMT) has been shown to improve exercise tolerance during a wide range of exercise modalities and durations of activity (McConnell & Romer, 2004b). However, there is a limited amount of research characterising the influence of RMT in specific athletic populations, or examining any sport-specific factors that may influence the benefits of RMT. Hence, the purpose of this dissertation was to evaluate the application of RMT in competitive rowers and to explore methods of optimising this to rowing. *Results:* Inspiratory muscle training (IMT) increased inspiratory muscle strength (~20-29%; $p < 0.05$) and attenuated inspiratory muscle fatigue (~8-28%; $p < 0.05$) during time trial performance in club-level and elite rowers. However, only in the club-level oarsmen was IMT associated with a measurable improvement in rowing performance (2.7% increase in mean power; $p < 0.05$). Expiratory muscle training (EMT) provided no ergogenic effect, and concurrent EMT and IMT did not enhance performance above that seen with IMT alone. IMT loads performed at 60-70% of maximal inspiratory mouth pressure (P_{Imax}) were equivalent to the widely used 30 repetition maximum, which is higher than reported for non-rowers (Caine & McConnell, 1998a); further, a load of 60% P_{Imax} was sufficient to activate the inspiratory muscle metaboreflex, as evidenced by a time-dependent rise in heart rate (70.1 ± 13.2 to 98.0 ± 22.8 bpm; $p < 0.05$) and mean arterial blood pressure (92.4 ± 8.5 to 99.7 ± 10.1 mmHg; $p < 0.05$). Higher and lower inspiratory loads did not activate the metaboreflex. Assessments of flow, pressure and volume in rowing relevant postures revealed no significant impairments, but optimal function occurred in the most upright postures. *Conclusions*: These data support the application of IMT, but not EMT, in elite and sub-elite rowers, and suggest that a load of 60-70% of P_{Imax} provides metaboreflex activation during loading. Further, the data do not support a requirement to undertake IMT in rowing relevant postures.

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LIST OF SYMBOLS AND ABBREVIATIONS

ANOVA	analysis of variance
ATS	American Thoracic Society
BIRO	British International Rowing Organisation
bpm	beats per minute
breaths·min^{-1}	breaths per minute
CI	confidence interval
cm H_2O	centimetres of water
cm H_2O·L	centimetres of water per litre
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
DBP	diastolic blood pressure
EELV	end expiratory lung volume
EIAH	exercise induced arterial hypoxemia
EILV	end inspiratory lung volume
EMF	expiratory muscle fatigue
EMG	electromyography
EMT	expiratory muscle training
ERS	European Respiratory Society
f_c	heart rate
f_R	respiratory frequency
FEV_1	forced expiratory volume in one second
FVC	forced vital capacity
Hz	Hertz
IFRL	inspiratory flow resistive loading
IMF	inspiratory muscle fatigue
IMT	inspiratory muscle training
IRV	inspiratory reserve volume
kg	kilogram
km	kilometre
L	litres
$[La^-]_B$	blood lactate concentration
LoA	limits of agreement
L·min^{-1}	litres per minute
L·sec^{-1}	litres per second
m	metres
min	minutes
MAP	mean arterial pressure
MIFR	mean inspiratory flow rate
MFVL	maximal flow volume loop
MIV	maximal isocapnic ventilation
ml·kg^{-1}·min^{-1}	millitres per kilogram per minute
mmHg	millimetres of mercury
mmol·L^{-1}	millimoles per litre
MSNA	muscle sympathetic nerve activity
MSVC	maximal sustainable ventilatory capacity
MVV	maximal voluntary ventilation
1RM	one repetition maximum
O_2	oxygen
SBP	systolic blood pressure

PAV	proportional assist ventilation
PCO_2	partial pressure of carbon dioxide
PEF	peak expiratory flow
P_{Emax}	maximal expiratory mouth pressure
P_{ETCO2}	pressure of end tidal carbon dioxide
PIF	peak inspiratory flow
P_{Imax}	maximal inspiratory mouth pressure
P_{di}	diaphragmatic pressure
$P_{di}tw$	twitch diaphragmatic pressure
$P_{ga}tw$	twitch gastric pressure
P_{OES}	oesophageal pressure
PTL	pressure threshold loading
$\dot{Q}_L$	limb cardiac output
$\dot{Q}_T$	total cardiac output
RM	repetition maximum
RMF	respiratory muscle fatigue
RMT	respiratory muscle training
RMS	respiratory muscle strength
RPE	rating of perceived exertion
RRE	rating of respiratory effort
RV	residual volume
s	seconds
SD	standard deviation
SE	standard error
SV	stroke volume
TLC	total lung capacity
T_{lim}	time to exhaustion
VC	vital capacity
$\dot{V}_E$	minute ventilation
VIH	voluntary isocapnic hyperpnoea
$\dot{V}O_2$	oxygen consumption
$\dot{V}O_{2max}$	maximal oxygen consumption
V_T	tidal volume
W	watts
W_b	work of breathing
6MAO	six minute all out effort

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PLANNED PUBLICATIONS

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OTHER DELIVERABLES

Consultancy reports provided to the British International Rowing Organisation. All projects were performed in collaboration with Professor Alison McConnell.

1) 'The effect inspiratory muscle training has on rowing ergometer performance in 'World Class' heavyweight oarsmen.' Research grant for project: £6,000. Project completed in 2003.

2) 'The influence of inspiratory muscle training upon rowing ergometer performance in adolescent rowers.' Project completed in 2005.

3) 'The influence of inspiratory muscle training upon rowing ergometer performance in adaptive (disabled) rowers.' Project completed in 2007.

CHAPTER ONE

GENERAL INTRODUCTION

1.0: OVERVIEW

The topic of respiratory muscle training (RMT) has been well described and many researchers have examined the effectiveness and usefulness of RMT in healthy (Gigliotti, Binazzi & Scano, 2006; McConnell & Romer, 2004b; Sapienza, 2008; Sheel, 2002) and clinical (McConnell & Romer, 2004a; Weiner & McConnell, 2005) populations. Inspiratory muscle training (IMT) has been shown to improve exercise tolerance during short term high intensity cycling and rowing (Caine & McConnell, 1998a; Volianitis, McConnell, Koutedakis, McNaughton, Backyx & Jones, 2001c), prolonged submaximal cycling (Caine & McConnell, 1998b; Guenette, Martins, Lee, Tyler, Richards, Foster, Warburton & Sheel, 2006; Romer, McConnell & Jones, 2002a) and repeated sprint exercise (Romer, McConnell & Jones, 2002c; Nicks, Morgan, Fuller & Caputo, 2009; Tong, Fu, Chung, Eston, Lu, Quach, Nie & So, 2008). However, there is a limited amount of research that has characterised the effect of IMT in specific athletic populations or examined any sport-specific factors that may influence the potential benefits of RMT.

Elite oarsmen have overwhelming physiological challenges associated with the high-intensity aerobic and anaerobic demands of the sport. Rowers experience hyperpnoea, an increased breathing frequency and tidal volume (V_T), during maximal exercise and racing conditions (Donnelly, Ellis, Keating, Keena, Woolcock & Bye, 1991). It has been shown that during high intensity exercise (>85% $\dot{V}O_{2max}$) there is an increase in the perceptions of dyspnoea (Harms, Wetter, McClaren, Pegelow, Nickele, Nelson, Hanson & Dempsey, 1998) and activation of a respiratory muscle metaboreflex (Harms et al., 1998; Harms, Wetter, St. Croix, Pegelow & Dempsey, 2000), both of which may limit exercise tolerance. In addition, the posture specific demands of rowing may have an even greater impact on both the ability to ventilate the lungs and the RMF experienced by these athletes. Inspiratory muscle training (IMT) has been shown to minimise the detrimental effects of RMF on rowing performance

during time trials in male and female rowers (Riganas, Vrabas, Benaxides, Papadopoulou, Vamvakoudis & Mandroukas, 2007; Volianitis et al., 2001c; Vrabas, Riganas, Benaxides & Mandroukas, 2007). However, a recent study investigating the benefits of IMT on time trial performance in highly-trained male and female rowers observed an improvement in inspiratory muscle strength and a decrease in exercise-induced IMF, but these changes did not translate to an improvement in 2 km rowing performance (Riganas, Vrabas, Christoulas & Mandroukas, 2008). Thus, the role of IMT on rowing performance remains uncertain and warrants further investigation.

At the elite level, minute improvements in rowing time trial performance can be the difference between a gold medal winner and last place. For example, 0.4% was the average margin at the rowing finals at the 2004 Athens Olympics. In fact, the spectacular finish of the GB heavyweight men's coxless four in which Matthew Pinsent's crew beat the Canadians was decided by a winning margin of 0.08 s (0.04%). Accordingly, national sport organisations and coaches alike, realise that top rankings in world championship sport provide an excellent opportunity for international recognition and increased monetary support for their sport. Accordingly, in 2002, the British International Rowing Organisation (BIRO) requested more research into RMT for competitive rowers in order to identify appropriate training protocols and outline the specific ergogenic benefits they might reasonably expect from this training in rowers across the age and disability spectra.

More research into the physiological and specific exercise performance benefits associated with RMT is necessary in order to explore methods of optimising the benefits of RMT for rowing. Additionally, a greater understanding of IMT and expiratory muscle training (EMT) needs to be explored to discern what, if any, performance enhancement can be gained from EMT, and its combination with IMT.

This chapter will introduce the physiological factors that underpin the application of RMT to rowing. It will also provide an overview of respiratory muscle function, define RMF, and discuss the various methods of RMT. This chapter will also present an overview of the physiological demands of elite rowing and discuss the respiratory and postural challenges associated with this sport.

1.1: RESPIRATORY MECHANICS AND FUNCTION

1.1.1: ANATOMY AND NON-RESPIRATORY FUNCTIONS OF THE RESPIRATORY MUSCLES

The principal function of the respiratory system is the regulation of gas exchange. During low to moderate exercise, the challenge is to facilitate alveolar ventilation to meet increasing metabolic demands, whilst simultaneously maintaining acid-base balance and arterial blood-gas tensions. Heavy or severe exercise may lead to metabolic acidosis, a fall in arterial pH due to the increase in free H^+ ions from aerobic metabolism, thus producing a hyperventilatory response to regain pH balance and prevent hypoxemia (reduced O_2 in the blood) (Romer & Polkey, 2008). These extensive demands require a well-coordinated interaction of the lungs with the central nervous system, the diaphragm and chest wall musculature, and the circulatory system. Healthy respiratory systems are anatomically well equipped to meet the O_2 requirements at rest and during increasing exercise intensities. Nonetheless, it is the specific challenges faced by the respiratory muscles in meeting the metabolic requirements of high intensity rowing that is the focus of this dissertation.

Quiet respiration requires minimal work, as it is regulated by pressure differences within the lung created by the coordinated movement of the lung and chest wall to inflate (inspiration) and deflate (expiration) the lungs. The lungs and chest wall are not attached, but rather joined together through surface tension by the intrapleural fluid; they act co-dependently in the

sense that they have opposing forces working together. The lung has no muscle to move or change its shape and is susceptible to collapse, whereas the chest wall is comprised of strong inspiratory muscles forcing it to spring open. It is this combination of the outward elasticity generated by the chest wall working in opposition to the inward elasticity of the lungs that keeps the lungs from collapsing. Both the lung and chest wall have elastic properties, which allow them to return to their original shape when the distorting force is removed, creating the 'elastic recoil' effect (West, 2005).

To provide optimal and efficient blood-gas exchange, the lung is composed of millions of small air sacs called alveoli. The alveolus is the space in the lungs where O_2 and carbon dioxide (CO_2) diffuse with the capillaries allowing for gas exchange. Thus, the primary goal of ventilation, the exchange of air between the lungs and the atmosphere, is to supply the alveoli with O_2 and remove CO_2 from the blood. At the base of the lung, the alveoli are smaller, hence denser, due to the lower intrapleural pressure. This is advantageous for gas exchange, as the larger number of alveoli maximises the surface area creating a capillary rich environment. The blood-gas barrier between the alveoli and the capillaries is extremely thin allowing for an enormous rate of gas exchange (West, 2005). However, during severe exercise the completeness of gas exchange may be affected by the rapid pulmonary transit time of the blood, which decreases the time available for oxygenation (Dempsey, Hanson & Henderson, 1984).

The respiratory muscles, necessary for ventilatory pump function, are utilised for both inspiration and expiration. The inspiratory muscles, which are responsible for elevating the rib cage upwards and outwards, include the diaphragm, serratus anterior, scalenes, sternocleidomastoids and external intercostal muscles (see fig. 1.1). These muscles are composed of both slow (type 1) and fast-twitch (type 2a and 2b) muscle fibres. The primary

inspiratory muscle is the diaphragm, a dome shaped muscle which creates the base of the thoracic cavity separating the heart and lungs from the abdominal cavity. The diaphragm is richly innervated by the phrenic nerves from cervical segments 3, 4 and 5 (West, 2005).

During inspiration, the inspiratory muscles contract expanding the thoracic cavity along the craniocaudal axis (De Troyer, Kirkwood & Wilson, 2005) allowing for an increased volume of air to enter the lungs. Thus, a pressure gradient is created between the atmosphere and the alveoli. As the negative pressure in the lung increases and becomes more negative, it draws air into the lungs. During expiration, the inspiratory muscles relax (displacement of the ribs in the caudal direction) and the elastic recoil of the lung returns it to its resting volume; thereby reducing the volume of the thoracic cavity in which tidal air is exhaled into the atmosphere.

At rest, expiration is usually passive and is achieved by the elastic recoil of the lungs, so little effort is required to exhale. The expiratory muscles, which include the rectus and transversus abdominis, external and internal obliques, triangularis sterni and the internal interosseous intercostals, take on a more active role in ventilation during heavy exercise to assist in rapid expiration (see fig. 1.1). With increased ventilation, there seems to be an orderly recruitment and activation of the expiratory muscles from the deep to the more superficial, in which the tranversus abdominis and internal oblique are activated first, followed by the external oblique and rectus abdominis (Abe, Kusuhara, Yoshimura, Tomita & Easton, 1996; De Troyer, Estenne, Ninane, Van Gansbeke & Gorini, 1990). When these muscles are contracted it raises intra-abdominal and intrathoracic (within the pleural cavity) pressures moving air out of the lungs. The addition of the intercostals interosseous muscles and triangularis sterni during exercise are responsible for enhanced caudal displacement (deflation) of the ribs increasing the expiratory effect (De Troyer et al., 1990, 2005; West, 2005).

During exercise, specific accessory muscles are necessary to accommodate increased airflow and meet increased ventilatory demands. The external intercostal muscles, small muscles joining adjacent ribs which slope downward and forward, are responsible for cranial and ventral displacement of the ribs. This movement of lifting the rib cage upwards and forwards increases the intrathoracic cavity allowing an even greater volume of air to enter the lungs during inspiration. Additionally, the sternocleidomastoids and the scalenes are activated to elevate the sternum and first pair of ribs further enlarging the thoracic cavity (West, 2005).

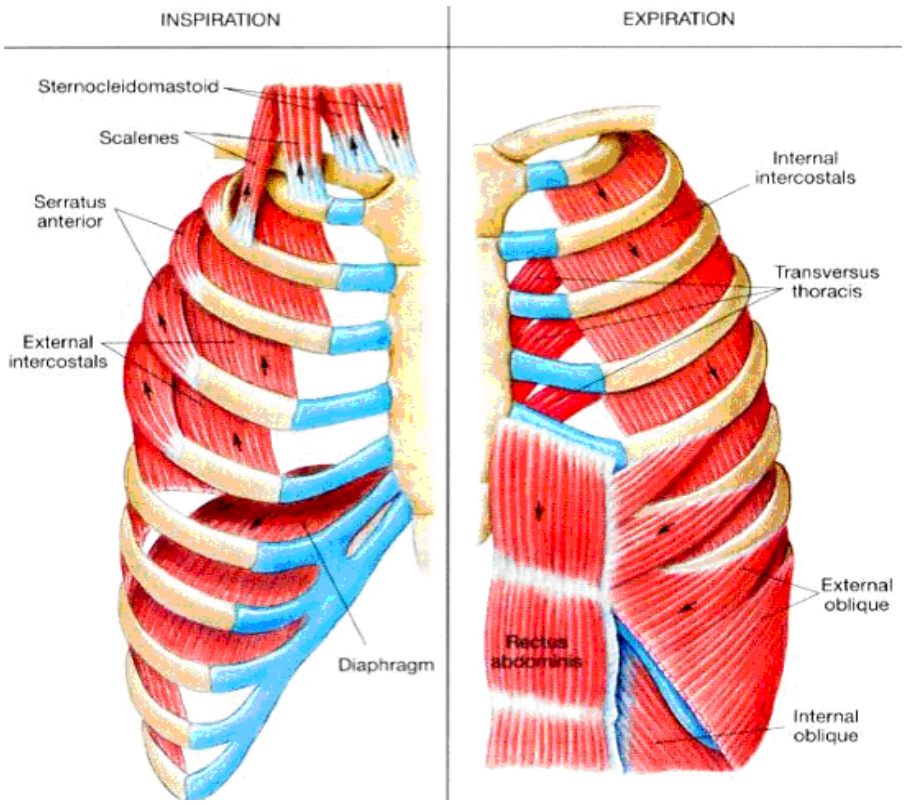


Figure 1.1 Illustration of the inspiratory and expiratory muscles (Adapted from Currie, 2003).

During exercise, greater contractions of both inspiratory and expiratory muscles are required to increase the airflow via increases in tidal volume (V_T) and respiratory frequency (f_R), thereby increasing minute ventilation ($\dot{V}_E$). This increase in $\dot{V}_E$ is a normal response to

increasing exercise intensity as a means to meet the escalating demand for gas exchange. The mechanisms driving exercise hyperpnoea (deep and rapid respiration) remain poorly understood, but include both feed-forward and feedback inputs to the respiratory controller (Tipton, Sawka, Tate & Terjung, 2006). The central respiratory controller, regulated by the brainstem (involuntary) and cerebral cortex (voluntary) group are responsible for the control of breathing by processing information from the receptors in the lung and chest wall to regulate rhythmic respiratory drive (Tipton et al., 2006).

1.1.2: FORCE PRODUCTION AND POSTURAL DEMANDS UPON THE RESPIRATORY MUSCULATURE

Similar to skeletal muscles, the force generating capacity of the respiratory muscles is influenced by their length-tension relationship. The length-tension relationship describes the amount of force a muscle is capable of generating at a discrete length. With respect to the respiratory muscles, the maximal pressure generated is inversely proportional to the starting lung volume (Agostini & Fenn, 1960; Braun, Arora & Rochester, 1982). When considering the muscle groups inclusively, the inspiratory muscles are at their maximum force generating capacity at residual volume (RV); conversely, the expiratory muscles reach their maximum potential at total lung capacity (TLC). However, the optimal length for force generation of the diaphragm is slightly below functional residual capacity (FRC), whereas the optimal length for most of the accessory inspiratory muscles is just above FRC (McKenzie, Allen & Gandevia, 1996). The diaphragm shortens by 30-40% when increasing lung volume from RV to TLC (Braun et al., 1982). With an increase in muscle length there is a decrease in the pressure-generating ability of the respiratory muscles, such that the diaphragm ceases to generate any pressure at TLC (Smith & Bellemare, 1987).

The load against which the inspiratory muscles contract (i.e. inspiratory impedance) increases throughout the contraction as the muscle shortens. In addition, the force developed during a muscle contraction is inversely related to its shortening velocity. For the inspiratory muscles, this relationship has been characterised functionally as the relationship between maximal inspiratory flow rate (MIFR) and inspiratory impedance, using externally applied loads (Romer & McConnell, 2003).

With heavy intensity endurance exercise, there is a progressive and time dependent increase in f_R, $\dot{V}_E$ and work of breathing (W_b) (Johnson, Babcock, Suman & Dempsey, 1993). Progressive exercise elicits a hyperventilatory response requiring greater recruitment of both the inspiratory and expiratory muscles to meet the elevated ventilatory requirements. Along with an increase in ventilatory demands there is also a time dependent increase in the perception of respiratory effort and limb discomfort (Harms et al., 2000; Johnson, Aaron, Babcock & Dempsey, 1996). The activation of the expiratory muscles at high workloads assists ventilation by reducing end expiratory lung volume (EELV; Henke, Sharratt, Pegelow & Dempsey, 1988). This reduction in EELV assists by increasing V_T, lengthening the diaphragm, thereby optimising its force generating capacity (Smith & Bellemare, 1987), as well as by increasing the elastic stored energy within the chest and abdominal walls to assist with inspiration. Generally, in untrained healthy subjects, the respiratory system is sufficiently capable of meeting the pressure generation capacity required at heavy intensity exercise; however in highly trained athletes, working at higher ventilations these demands may meet or exceed the capacity of the respiratory system to generate ventilation (Guenette & Sheel, 2007; Johnson, Saupe & Dempsey, 1992).

In addition to pulmonary ventilation, the respiratory muscles are also active in a number of other vital roles, including: trunk stabilisation, postural control, locomotion, speech,

parturition, coughing and regulation of airway calibre (Dempsey, 1986; Hodges, Butler, McKenzie & Gandevia, 1997; Hodges, Gandevia & Richardson, 1997). The activation of the diaphragm is related to postural and trunk control and its activation increases the mechanical stabilisation of the trunk muscles (Cresswell, Oddsson, & Thorstensson, 1994; Grillner, Nillson & Thorstensson, 1978). The demands upon the respiratory muscles are compounded when required to perform a multitude of functions simultaneously. In addition to maintaining ventilation, the tonic contraction of the diaphragm aids postural control by increasing the intra-abdominal pressure prior to the onset of limb movement (Hodges et al., 1997). The diaphragm is therefore challenged when there is a simultaneous increase in respiratory demand during exercise as it must meet both the demand for increased ventilation, as well as the demand to stabilise the trunk. Sports such as rowing, which involve the trunk in locomotor force generation/transmission, require the respiratory muscles to forcefully expand and contract to maintain a high level of ventilation, whilst simultaneously stabilising the trunk and performing locomotor work with both the upper and lower limbs (Steinacker, Both & Whipp, 1993). At high levels of ventilation, the central respiratory drive prioritises the need for respiration above postural control, and appears to decrease the component of diaphragm activation that assists in posture (Hodges, Heijnen & Gandevia, 2001). During high levels of ventilation with simultaneous locomotor movement, other respiratory muscles such as the scalenes and parasternal muscles are activated to assist with rib cage motion (Gandevia, Gorman, McKenzie & De Troyer, 1999). Hence, as the diaphragm reduces its contribution, the abdominal muscles become more active in maintaining the intra-abdominal pressure (Hodges & Gandevia, 2000a, 2000b). The competing demands placed upon the respiratory muscles during rowing may increase their susceptibility to fatigue, thus creating a potent argument in favour of RMT. On the other hand, it might be argued that the extreme demands of rowing may enhance respiratory muscle function sufficiently to render such training obsolete.

Research has demonstrated that the influence of body position and posture affects lung volume (Allen, Hunt & Green, 1985; Appel, Childs, Healey, Markowitz, Wong & Mead, 1986; Black & Hyatt, 1969; Meysman & Vincken, 1998; Talwar, Sood & Sethi, 2002; Tsubaki, Deguchi & Yoneda, 2009; Vilke, Chan, Neuman & Clausen, 2000; Watson & Pride, 2005) and respiratory muscle function (Druz & Sharp, 1981; Kera & Maruyama, 2001a, 2001b; Koulouris, Mulvey, LaRoche, Goldstone, Moxham & Green, 1989; Meysman & Vincken, 1998; Ogiwara & Miyachi, 2002; Tsubaki et al., 2009). Vital capacity and other lung volumes have been shown to significantly affect respiratory muscle strength (RMS) (Black & Hyatt, 1969). Respiratory muscle pressure generation is influenced by changes in intra-abdominal pressure shifts and the length-tension relationship of the respiratory muscles. For instance, both P_{Imax} and P_{Emax} are highest in the more erect supported postures, such as sitting and the orthopneic position (sitting down with elbows on knees) compared to standing, recumbent or supine postures (Kera & Maruyama, 2001a, 2001b; Ogiwara & Miyachi, 2002; Tsubaki et al., 2009). While standing, the abdominal muscles, particularly the rectus and transverse abdominis, assume a postural role in which they are unable to maximally contribute to expiratory muscle force generation; thus resulting in lower test values of P_{Emax}. However, during trunk flexion while standing the abdominal wall increases thoracic space and causes a positive shift in intra-abdominal pressure. This increase in pressure places the abdominal muscles in a more advantageous position in the length-tension relationship which allows for an increased P_{Emax} while also reinforcing P_{Imax} (Kera & Maruyama, 2001b). In the supine position, it is the influence of gravity which causes a shift in the intra-abdominal contents (the weight of the visceral organs pushes up the diaphragm) which benefits the length-tension relationship. These factors suggest that the primary influence of posture and body position on respiratory muscle function is gravity and its effect on the length-tension relationship.

1.1.3: ASSESSMENT OF RESPIRATORY MUSCLE FUNCTION

The evaluation of respiratory muscle function in the context of exercise limitation has primarily focused on respiratory muscle endurance (RME) and strength (RMS). Decreases in both RME and RMS have been shown to be limiting factors with increasing breathing loads particularly during sustained, heavy-intensity exercise (Johnson et al., 1993, 1996). Since it is not possible to access the respiratory muscle properties directly, a number of indirect methods of assessment have been developed.

In general, muscle endurance is defined as the muscle's ability to sustain a specific task over time. More specifically, RME was defined more than 30 years ago as the 'capacity for sustaining high levels of $\dot{V}_E$ for relatively long periods' (Leith & Bradley, 1976). Different tasks require varying levels of recruitment in motor units and differing interactions between muscles, hence, respiratory endurance is necessarily specific to the task performed (Clanton, Calverly & Celli, 2002). Typically, measurements of RME are performed using resistive or threshold inspiratory loads, which include tests based on: the percentage of maximum voluntary ventilation (MVV), limit of tolerance during a breathing endurance task (T_{lim}), maximum sustained ventilatory capacity (MSVC), or sustained maximal inspiratory pressure (SMIP). Measures of RME are usually expressed as the amount of time a particular load is tolerated or as the maximum load tolerated for a specified time period (Fiz, Romero, Gomez, Hernandez, Ruiz, Izquierdo, Coll & Morera, 1998). These types of measurements can be plotted as task intensity vs. time sustained, providing an index of the endurance properties of the respiratory muscles (Clanton et al., 2002).

The maximal force generating capacity of the respiratory muscles is assessed by measuring the amount of pressure generated during a maximal contraction. Two common methods of determining respiratory muscle pressures are the measurement of transdiaphragmatic pressure

(P_{di}) or maximal mouth or nasal pressures. These surrogate measures of RMS are performed using several techniques that can be divided into effort dependent manoeuvres, which are reliant on participant motivation (volitional effort), or methods that use magnetic or electrical stimulation to avoid these factors (non-volitional efforts) (Green, Road, Sieck & Similowski, 2002). A detailed description of each of these methods is outside the scope of this review (see American Thoracic Society/European Respiratory Society {ATS/ERS}, 2002); however a brief overview is required.

Transdiaphragmatic pressure (P_{di}) is defined as the difference between the pleural pressure (P_{pl}) and abdominal pressure (P_{ab}) (Green et al., 2002). The pressure differences between these structures, generated during a maximal contraction, represent P_{di}. Air filled balloon catheters, which are linked to pressure transducers, are used to assess pressure differences during the contraction. However, as the pleural and abdominal spaces are inaccessible to balloon catheters, oesophageal and gastric pressures (P_{OES} and P_{ga}, respectively) are commonly employed as surrogate measures of P_{pl} and P_{ab}. The assessment of P_{di} is accomplished by measuring pressure changes during a maximal contraction. A maximal contraction can be obtained by either having the participant perform a volitional respiratory effort or by electrically or magnetically stimulating the phrenic nerves (Green et al., 2002), as well as by using a combination of these techniques.

Since the diaphragm is solely innervated by the phrenic nerves, electrical or magnetic stimulation of the phrenic nerves can be used to isolate diaphragm contraction independent of central factors, to determine if a true maximal contraction has occurred. Bilateral phrenic nerve stimulation (BPNS) can be performed simultaneously while measuring P_{di} to determine pressure changes during a voluntary or superimposed contraction. The assessment of P_{di}

obtained via voluntary contractions and those superimposed by PNS have been shown to be valid and reliable methods for the estimation of RMS (Green et al., 2002).

An alternative assessment for measuring RMS is maximal static inspiratory and expiratory mouth pressures (P_{Imax} and $P_{Emax,}$ respectively). Mouth pressure measurements assess the pressure generated by the respiratory muscles, as well as the elastic recoil pressure within the respiratory system, providing a reflection of global RMS (Green et al., 2002). Respiratory mouth pressures can be measured using a mouth pressure meter, and participants are required to perform either a maximal inspiratory effort (Mueller manoeuvre) at or near residual volume (RV) or a maximal expiratory effort (Valsalva manoeuvre) at or near total lung capacity (TLC). Normal values for P_{Imax} in adult healthy subjects range from ~105 to 130 cm H_2O for males and ~70 to 100 cm H_2O for females; whereas P_{Emax} values are usually much higher with values ranging from ~140 to 240 cm H_2O for males and ~90 to 160 cm H_2O for females (Green et al., 2002). Although these measurements are effort dependent they have been shown to be valid and reliable measures of RMS in healthy, motivated subjects (Hamnegard, Wragg, Kyroussis, Daskos, Bake, Moxham & Green, 1994).

In addition to measures of RMS and RME, peak inspiratory flow rate (PIFR) is also influenced by IMT (Romer, McConnell & Jones, 2002a, 2002c; Romer & McConnell, 2003). Peak inspiratory flow rate (PIFR) is the fastest flow rate achieved during a maximum inspiration and provides a measure of the rate of volume change (i.e. volume acceleration) in the lungs. Thus, PIFR can be used as an expression of the maximal shortening velocity of the inspiratory muscles (Agostini & Fenn, 1960). As peak expiratory flow (PEF) is influenced by airway characteristics (e.g., airway dimensions, force generated by expiratory muscles) (Quanjer, Tammeling, Cotes, Pederson, Peslin & Yernault, 1993) it cannot be used to assess the expiratory muscles in the same way.

Muscle fatigue is defined ‘as a loss in the capacity to exert force and or velocity resulting from muscle activity under load, which was reversible by rest’ (National Heart, Lung and Blood Institute {NHLBI} Workshop, 1990). Therefore, exercise-induced respiratory muscle fatigue (RMF) can be estimated by comparing an index of pre-exercise to post-exercise muscle function. Exercise-induced diaphragmatic fatigue can be estimated by measuring the reduction of P_{di} using supramaximal BPNS following exercise (Aubier, Farkas, De Troyer, Mozes & Roussos, 1981; Polkey, Duguet, Luo, Hughes, Hart, Hamnegard, Green, Similowski & Moxham, 2000; Similowski, Fleury, Launois, Cathala, Bouche & Derenne, 1989). Similarly, assessment of abdominal fatigue can be quantified by measuring changes in P_{ga} following magnetic stimulation of the thoracic nerve root (Kyroussis, Mills, Polkey, Hamnegard, Koulouris, Green & Moxham, 1996). Tests using maximal mouth pressures provide a non-invasive index of the fatigue of the inspiratory or expiratory muscles; however maximal static efforts are coupled with high neuronal firing rates (~50 to 100 Hz) and may not therefore provide a reflection of long lasting low frequency fatigue (i.e. frequencies of 1 to 20 Hz) (Supinski, Fitting & Bellemare, 2002). However, it is impossible to differentiate the relative contribution of central and peripheral processes to fatigue with volitional measures. Advanced assessment techniques using nerve stimulation allows for maximal contraction of the affected nerve independent of participant motivation, providing objective evidence that RMF is due to physiological changes occurring within the respiratory muscles.

Although these advanced research techniques using electrical or magnetic stimulation are useful in objectively evaluating measures of respiratory muscle force output and exercise-induced RMF they are not without their limitations. For instance, measurements using P_{di} and BPNS stimulation are specific to diaphragm muscle contraction and cannot assess fatigue of the extra-diaphragmatic muscles (Supinski et al., 2002); however it has been suggested that cervical magnetic stimulation can be used to detect both diaphragm and rib cage fatigue

(Similowski, Straus, Attali, Duguet & Derenne., 1998). This is an important consideration as research has shown that there is a decrease in diaphragm contribution to respiratory output with increasing exercise duration; suggesting that accessory respiratory muscles may compensate for the increased respiratory muscle work (Babcock, Pegelow, McClaran, Suman & Dempsey, 1995; Babcock, Pegelow, Taha & Dempsey, 1998; Johnson et al., 1993). In addition, measurements of internal pressures have a high degree of test-retest variation when performed on separate days potentially making it unreliable for comparisons of strength or fatigue measurements in studies using a repeated-measures design (Maillard, Burdet, van Melle & Fitting, 1998). As these tests are also highly technical in nature and require a clinical or laboratory setting, applicability to applied testing in the field is limited.

Alternatively, maximal mouth pressure measurements are reflective of global respiratory muscle activation and therefore may be used to determine exercise-induced RMF. Post-exercise measures of P_{Imax} and P_{Emax} can be used to estimate the transient fall in pressure generation by comparing them to prior baseline values. However, the prime limitation of all volitional tests of muscle function is the ability of the individual to perform maximal neuromuscular activation; meaning that it is unclear whether any reduction in pressure is due to a reduction in muscle strength or neural activation (Supinski et al., 2002). As maximal mouth pressure measurements are volitional, effort dependent manoeuvres, they have been criticised as a means of assessing RMS; particularly, for those subjects who may have poor motivation, or have difficulty producing a maximal effort (Aldrich & Spiro, 1995; Polkey, Green & Moxham, 1995). This limitation may be particularly important for measures of exercise-induced RMF in which subjects are less likely to be able to perform an effort dependent maximal manoeuvre immediately following exhaustive exercise (Fuller, Sullivan & Fregosi, 1996). However, an inability to access the central factors required to perform a maximal manoeuvre may also be physiologically relevant for assessing post-exercise RMF.

Although these types of volitional tests have their limitations, P_{Imax} and P_{Emax} are considered to be reliable measures of RMF and exercise-induced IMF and EMF in highly motivated, healthy volunteers (Green et al., 2002; Supinski et al., 2002).

1.2: RESPIRATORY MUSCLE TRAINING

Respiratory muscle training (RMT) has been identified as a means of overcoming respiratory limitations that may occur in healthy subjects during sustained high intensity exercise. Most RMT studies have used one of three principal methods to train the respiratory muscles: (1) voluntary isocapnic hyperpnoea (VIH) to improve RME; (2) inspiratory flow resistive loading (IFRL) using variable flow resistive devices to improve both RME and RMS; and (3) pressure threshold loading (PTL), which can improve both RMS and RME. Depending on the type of training employed, RMT in healthy subjects has been shown to increase one or more of the following functional parameters: the maximal force production, the maximal velocity of shortening, the maximal power output and the endurance of the respiratory muscles (McConnell & Romer, 2004b; Sheel, 2002). Table 1.1 provides an overview of the research that has demonstrated significant changes in respiratory muscle function following RMT in healthy individuals.

The same training principles, i.e. specificity, overload, progression and reversibility, used in skeletal muscle training apply to RMT. Specificity refers to the type of change in muscle structure and function, and is dependent upon the type of demands placed upon the muscle (i.e. strength or endurance). For instance, increases in muscle force production are subject to the force-velocity specificity of the training. High force-low velocity contractions increase maximal force, but not maximal shortening velocity, whereas low force-high velocity training will increase maximal shortening velocity of the muscle but not maximal force (Romer &

McConnell, 2003; Tzelepsis, Vega, Cohen & McCool, 1994). Hence, higher forces produce greater strength.

For muscle adaptation to occur, resistance training programmes must be designed to overload the muscle in a methodical progression. 'Overloading' the muscle requires a sufficient training demand (resistance) to elicit a physiological adaptation whereas 'progression' is the stepwise programme to systematically induce overload. Initially, muscle will respond positively to training, however after a period of time, the muscle will adapt to the imposed demand and muscle development will plateau. By steadily increasing the frequency, intensity or duration of the training or a combination of these factors, the muscle will continue to adapt and develop (Kraemer & Ratamess, 2004). When exercise ceases, fitness and muscular adaptations will gradually be lost; this is known as 'reversibility'.

1.2.1: COMPARISON OF TRAINING METHODS

Voluntary isocapnic hyperpnoea (VIH) is primarily used to increase RME and has been shown to improve breathing endurance at a given $\dot{V}_E$, MSVC, MVV and vital capacity (VC) (see table 1.1). This form of training requires the participant to breathe into a rebreathing circuit at, or near, maximal ventilation for up to 30 min. During normal breathing, the partial pressure of carbon dioxide (PCO_2) remains relatively unchanged (~40 mmHg); however any change in $\dot{V}_E$ at the same metabolic rate (such as hyperventilation at rest) induces a change in PCO_2. Therefore, a complex rebreathing circuit is used to maintain isocapnia (arterial CO_2 remains unchanged) during this type of training (see fig. 1.2). Training sessions have typically been performed in a laboratory to achieve continual monitoring of PCO_2 to ensure isocapnic conditions. This method of training is physically demanding, as it requires high levels of ventilation for ~15-30 min per session, 2-3 times daily for a minimum of 4-5 times per wk (McConnell & Romer, 2004b). Training intensity is usually set between ~60-90% of

MVV. Normally, this type of training would be limited to the laboratory, but more recently commercially available devices are available (i.e., Spirotiger®, Idiag AG, Volketswil, Switzerland) that allows for individuals to perform this type of training independently.

Figure 1.2 Voluntary isocapnic hyperpnoea respiratory muscle endurance training device (Spirotiger®, Idiag AG, Volketswil, Switzerland).

Inspiratory flow resistive loading (IFRL) has been shown to improve breathing endurance and RMS (see table 1.1). This type of training requires the participant to inspire against a fixed flow resistance; ostensibly, resistance is altered by increasing or decreasing the diameter of the orifice (the smaller the diameter the greater the resistance). During training, a maximum flow is set during the inspiratory effort proportional to the pressure achieved. However, the participant may alter their breathing pattern to decrease the sensation of effort, thereby reducing the training load with an inevitable impact upon the training response (Smith, Cook, Guyatt, Madhavan & Oxman, 1992). Therefore, this type of training requires careful monitoring to regulate inspiratory flow in order to elicit a training effect.

An alternative IFRL training program, based on the Test of Incremental Respiratory Endurance (TIRE), also uses a flow resistive load set at a training intensity of 80% sustained maximal inspiratory pressure (SMIP). Sustained maximal inspiratory pressure (SMIP) is a

measurement of the highest pressure a subject can generate in each breath for 10 min; this measurement is used to develop a baseline pressure-time profile. This measurement has been shown to be independent of the resistance or flow rate (Chatham, Baldwin, Griffiths, Summers & Enright, 1999; Chatham, Baldwin, Oliver, Summers & Griffiths, 1996). Participants are required to perform a series of inspiratory efforts across a pressure profile typically set to 80% SMIP. The manoeuvre is performed for six inspiratory efforts with the initial rest period of 60 s. Following each rest period, the participant performs another six efforts but with a diminishing rest period (45, 30, 15, 10 and 5 s) until they can no longer sustain the set target on the pressure profile or they successfully complete the training range (Chatham et al., 1996).

Figure 1.3 Inspiratory flow resistive loading device (Pflex® Inspiratory Muscle Trainer, Respironics Ltd., NJ, USA).

In contrast to the variable flow resistive devices, a PTL device only permits air to flow through it once the user achieves a sufficient negative pressure or overcomes a critical threshold, which thereby initiates inspiration. Threshold loading differs from traditional flow resistive loading in that PTL provides a quantifiable and adjustable resistance using near flow

independent loading. Typical threshold devices employ either a weighted plunger or spring loaded valve in which flow is initiated once sufficient pressure is generated to meet or exceed the selected threshold pressure (Caine & McConnell, 2000). Pressure threshold loading (PTL) devices using spring loaded valves, such as the POWERbreathe® Inspiratory Muscle Trainer (Gaiam Ltd., Southam, UK; see fig 1.4*A*) and Powerlung® (PowerLung Inc., Houston, TX, USA; see fig 1.4*B*) (a combined inspiratory and expiratory muscle trainer) are ideal for individual training purposes as they can be performed independently and are easy to use.

Inspiratory PTL has been shown to be effective in improving RME, RMS, muscle shortening velocity and muscle power output (Caine & McConnell, 1998a; Romer & McConnell, 2003) making this device a versatile training method. Pressure threshold training is typically performed using loads of 30-65% of $P_{Imax,}$ depending upon the focus of the training programme (Caine & McConnell, 1998a; see table 1.1). Training using moderate loads and number of repetitions (i.e. 30 repetitions maximum) has been shown to increase both RMS and RME (31.2% and 27.8% respectively), whereas training with low loads will provide more of an endurance benefit (29.7%; Caine & McConnell, 1998a).

A

B

Figure 1.4 Pressure threshold inspiratory muscle trainer (*A*) and combined inspiratory and expiratory muscle trainer (*B*). Images taken from powerbreathe.com and powerlung.com.

Table 1.1 Summary of reported improvements in pulmonary function indices following respiratory muscle training in healthy individuals.

Training Modality	**References**	**Changes in pulmonary function indices following RMT**
Voluntary Isocapnic Hyperpnoea	Leith & Bradley, 1976	↑ MSVC, MVV and breathing endurance
	Keens et al., 1977	↑ MSVC
	Morgan et al., 1987	↑ MVV and breathing endurance
	Fairbarn et al., 1991	↑ breathing endurance
	Boutellier et al., 1992	↑ breathing endurance, ↓ exercise $\dot{V}_E$ and respiratory effort
	Boutellier & Piwko, 1992	↑ breathing endurance, ↓ exercise $\dot{V}_E$
	O'Kroy & Coast, 1993	↑ MSVC and MVV
	Kohl et al.,1997	↑ breathing endurance and exercise $\dot{V}_E$
	Boutellier, 1998	↑ breathing endurance, ↓ exercise $\dot{V}_E$ and respiratory effort
	Belman & Gaesser, 1999	↑ MSVC, VC, MVV
	Spengler et al.,1999	↑ breathing endurance
	Stuessi et al., 2001	↑ breathing endurance
	Markov et al., 2001	↑ MSVC and breathing endurance
	McMahon et al., 2002	↑ VC, MVV and breathing endurance
	Holm et al., 2004	↑ breathing endurance and exercise $\dot{V}_E$
	Verges et al., 2007a	↑ breathing endurance, P_{Imax} and P_{Emax}
	Verges et al., 2007b	↑ breathing endurance, ↓ breathlessness and respiratory effort
	Wyegala et al., 2007	↑ breathing endurance, P_{Imax} and P_{Emax}
	Leddy et al., 2007	↑ MVV and breathing endurance, ↓exercise breathing frequency and $\dot{V}_E$
	Verges et al., 2008	↑ breathing endurance

Note: MSVC, maximum sustained ventilatory capacity; MVV, maximum voluntary ventilation; $\dot{V}_E$, minute ventilation; VC, vital capacity; TLC, total lung capacity; P_{Imax}, maximal inspiratory pressure; P_{Emax}, maximal expiratory pressure.

Table 1.1 ***cont***. Summary of reported improvements in pulmonary indices following respiratory muscle training in healthy individuals.

Training Modality	References	Changes in pulmonary function indices following RMT
Inspiratory Resistive Loading	Leith & Bradley, 1976	↑ P_{Imax}, TLC, MSVC and MVV
	Hanel & Secher, 1991	↑ P_{Imax}
	O'Kroy & Coast, 1993	↑ P_{Imax}, ↓IMF
	Chatham et al., 1996	↑ P_{Imax} and breathing endurance
	Chatham et al.,1999	↑ P_{Imax} and breathing endurance, ↓ exertional dyspnoea
	Enright et al., 2000	↑ P_{Imax}, SMIP and breathing endurance
	Gething et al., 2004a	↑ P_{Imax} and breathing endurance
	Gething et al., 2004b	↑ P_{Imax}, SMIP and breathing endurance, ↓ exercising $\dot{V}_E$
	Enright et al., 2006	↑ P_{Imax}, SMIP, VC, TLC, T_{di}
	Mickleborough et al., 2008	↑ P_{Imax}, breathing endurance
	Mickleborough et al., 2009	↑ P_{Imax}, SMIP, maximal inspiratory muscle power output, inspiratory muscle work capacity, $\dot{V}_E$
Mixed Voluntary Isocapnic Hyperpnoea and Inspiratory Resistive Loading	Sonnetti et al., 2001	↑ P_{Imax}
Pressure Threshold Loading	Clanton et al., 1985	↑ P_{Imax} and breathing endurance
	Redline et al. 1991	↑ P_{Imax}
	Suzuki et al., 1993	↑ P_{Imax} and MVV
	Caine & McConnell, 1998b	↑ P_{Imax}, ↓ IMF
	Inbar et al., 2000	↑ P_{Imax} and breathing endurance
	Kellerman et al.,2000	↑ P_{Imax}
	Hart et al., 2001	↑ P_{Imax}
	Volianitis et al., 2001c	↑ P_{Imax},↓ IMF

Note: MSVC, maximum sustained ventilatory capacity; MVV, maximum voluntary ventilation; SMIP, sustained maximal inspiratory pressure; $\dot{V}_E$, minute ventilation; VC, vital capacity; TLC, total lung capacity; T_{di}, diaphragm thickness; IMF, inspiratory muscle fatigue; P_{Imax}, maximal inspiratory pressure.

Table 1.1 ***cont***. Summary of reported improvements in pulmonary indices following respiratory muscle training in healthy individuals.

Training Modality	References	Changes in pulmonary function indices following RMT
Pressure Threshold Loading	Sonnetti et al., 2001	↑ P_{Imax}
	Akiyoshi et al., 2001	↑ P_{Imax} and P_{Emax}
	Romer et al., 2002a, 2002b, 2002c	↑ P_{Imax}, maximal velocity and shortening of inspiratory muscles; ↓ IMF
	Williams et al., 2002	↑ P_{Imax} and breathing endurance
	Amonette & Dupler, 2002	↑ maximal $\dot{V}_E$ and V_T
	Huang et al., 2003	↑ P_{Imax} and $P_{O.1}$
	Romer & McConnell, 2003	↑ P_{Imax} and MRPD
	Edwards & Cooke, 2004	↑ P_{Imax}
	Johnson & Sharpe, 2004	↑ P_{Imax}
	McConnell & Sharpe, 2005	↑ P_{Imax}
	Sasaki et al., 2005	↑ P_{Imax} and P_{Emax}
	Butts et al., 2005	↑ P_{Imax} and breathing endurance
	Baker et al., 2005	↑ P_{Emax}
	Brilla & Feutz, 2006	↑ P_{Imax}
	Guenette et al., 2006	↑ P_{Imax}
	Wyegala et al., 2006	↑ $P_{Imax,}$ P_{Emax} and breathing endurance
	Wyegala et al., 2007	↑ $P_{Imax,}$ P_{Emax} and breathing endurance
	McConnell & Lomax, 2006	↑ $P_{Imax,}$ delays RMF and onset of metaboreflex
	Sasaki, 2007	↑ P_{Emax}
	Lindholm et al., 2007	↑ $P_{Imax,}$ P_{Emax} and breathing endurance

Note: P_{Imax}, maximal inspiratory pressure; P_{Emax}, maximal expiratory pressure; IMF, inspiratory muscle fatigue; $\dot{V}_E$, minute ventilation; V_T, tidal vital; $P_{O.1,}$ mouth occlusion pressure; MRPD, maximal rate of pressure development; RMF, respiratory muscle fatigue.

Table 1.1 ***cont***. Summary of reported improvements in pulmonary indices following respiratory muscle training in healthy individuals.

Training Modality	**References**	**Changes in pulmonary function indices following RMT**
Pressure Threshold Loading	Johnson et al., 2007	↑ P_{Imax}
	Downey et al., 2007	↑ P_{Imax} and ↑ $T_{di,}$ ↓ IMF
	Witt et al., 2007	↑ $P_{Imax,}$ delays RMF and onset of blood flow competition
	Edwards et al. 2008	↑ P_{Imax}
	Klusiewicz et al., 2008	↑ P_{Imax}
	Riganas et al., 2008	↑ P_{Imax} and breathing endurance, ↓ IMF
	Tong et al., 2008	↑ $P_{Imax,}$ MRPD and Vmax
	Brown et al., 2008	↑ P_{Imax}
	Lomax & McConnell, 2009	↑ P_{Imax}
	Huang et al., 2009	↑ P_{Imax}
	Nicks et al., 2009	↑ P_{Imax}

Note: P_{Imax}, maximal inspiratory pressure; IMF, inspiratory muscle fatigue; $P_{O.1}$, mouth occlusion pressure; RMF, respiratory muscle fatigue; P_{Emax}, maximal expiratory pressure; T_{di}, diaphragm thickness; MRPD, maximal rate of pressure development, Vmax, maximal inspiratory flow.

1.3: VENTILATORY DEMANDS DURING ROWING

Rowing is a highly challenging and physically demanding sport requiring tremendous aerobic and anaerobic capabilities to perform at the elite level (Shephard, 1998). A direct relationship has been shown between a high absolute $\dot{V}O_{2max}$ and international rowing performance (Yoshiga & Higuchi, 2003), with elite open-class rowers having some of the highest maximal aerobic capacities recorded (Clark, Hagerman & Gelfand, 1983; McKenzie & Rhodes, 1982; Secher, 1990). This is most likely due to the large muscle mass required, more so than compared to other sports, in that all four extremities and the trunk muscles work near maximally throughout the entire event. Hence, a large body size, stature and body mass have been shown to be indicators of potential rowing performance (Secher, 1993; Yoshiga & Higuchi, 2003). Given that the boat supports body weight, the heavyweight male rower has a distinct advantage over his lightweight and female counterparts. It is not only the increase in size that creates an edge, but also the additional benefits that increased size offers, including higher absolute $\dot{V}O_{2,}$ increase in limb length and greater overall strength of the individual. It is common for elite open class rowers to reach a $\dot{V}O_2$ of greater than 6 $L \cdot min^{-1}$ in males and 4 $L \cdot min^{-1}$ in females during a simulated 2 km ergometer race (Hagerman, Hagerman & Mickelson, 1979; Secher, 1993; Shephard, 1998), thus demonstrating the importance of high aerobic capacity to performance.

Rowing requires extremely high levels of $\dot{V}_E$ (> 200 $ml \cdot min^{-1}$) in elite male rowers (Hagerman, Connors, Gault, Hagerman & Polonski, 1978; McKenzie & Rhodes, 1982); these athletes have very large TLC, VC (Donnelly et al., 1991), and peak expiratory flows (PEF) (Steinacker et al., 1993). Ventilatory responses, including the entrainment of breathing, vary among rowers depending upon the level of training and experience of the athlete (Mahler, Shuhart, Brew & Stukel, 1991b; Siegmund, Edwards, Moore, Tiessen, Sanderson & McKenzie, 1999). In addition to meeting the high ventilatory demand, the respiratory

muscles aid in stabilising and stiffening the trunk during the rowing stroke (Bierstaker, Bierstacker & Schreurs, 1986; Mahler et al., 1991b). These competing demands are even greater in 'World-Class' athletes as they work at considerably higher ventilatory workloads than their novice counterparts (Shephard, 1998). As such, when the respiratory muscles fatigue this may result in a decline in the ability to maintain proper rowing posture and technique.

1.3.1: ROWING STROKE AND ENTRAINMENT OF BREATHING

There are two different types of rowing: sweep and sculling. In sweep (or oar sweep) rowing, each rower holds onto one oar with both hands; in sculling, each rower has two oars, one in each hand. In the boat, each rower is positioned on a seat, which sits on runners (known as a slide); the slide allows the rower to position themselves in the most advantageous position to place the oars in the water. Regardless of the style of rowing, the mechanical components of the rowing stroke cycle are relatively the same and are divided into two phases: the power phase and the recovery phase. The power phase is when the oar is in the water and physical force is used to propel the boat forward. The recovery phase is when the oars are out of the water and the athlete is resting whilst preparing for the next power phase (Nolte, 2005). Rowers organise the two phases of the stroke cycle into a single motion allowing them to push the boat through the water, whilst minimising hydrodynamic drag.

The rowing stroke can be further sub-divided into four distinct phases: the catch, the drive, the finish and the recovery (Mahler, Nelson & Hagerman, 1984; Nolte, 2005). Rowers begin the stroke in the 'catch' (or start) position in which the athlete is in a seated position, hips flexed with chest pivoted forward touching the thighs, shoulders held high with back and arms straight (see fig. 1.5*A*). This is probably the most challenging element of the stroke cycle as it is performed in a 'compressed' position (Nolte, 2005). It has been speculated that

this compressed position increases abdominal pressure thereby limiting the downward excursion of the diaphragm, potentially constricting diaphragmatic and abdominal muscle movement (Cunningham, Goode & Critz, 1975); thus impairing the ability to generate maximal pressures and flows.

During the 'drive' phase, the rower drives their legs and holds their back firm while pulling the oar through the water (see fig 1.5B). All the limb musculature and the torso must work together to accelerate the boat (Nolte, 2005). In particular, the expiratory muscles are recruited to assist with ventilation, aid in trunk flexion and stiffen the trunk in order to transfer force to the upper torso (Siegmund et al., 1999). During this phase of the rowing stroke the hip angle is > 90°, the competing demand of the abdominal muscles to counteract gravity to maintain rowing posture may impair their respiratory role. Once the 'drive' is completed, the torso should be in an upright position (hip angle = 90º to 120º) with legs straight (known as the 'finish'; see fig 1.5C) (Mahler et al., 1984).

As the athlete moves forward on the slide returning to the 'catch' position, there is a brief period of 'recovery'; this is important to the overall stroke, as it is the only time when the rower is not actively 'working'. Elite rowers tend to spend a majority of their rowing stroke cycle in the recovery phase (Mahler et al., 1991b).

A

B

C

Figure 1.5 Different phases of the rowing stroke: *A*, the catch; *B*, the drive and/or recovery; *C*, the finish.

Ventilation and locomotion coupling (entrainment) is the linking of breathing frequency with the rhythmic pattern of locomotion. It has been suggested that linking respiration with a movement task is one strategy used to assist the diaphragm in maintaining respiration and assist with postural control during exercise (Hodges et al., 2001). By synchronising ventilation and movement, the recruitment of the diaphragm can be coordinated to specific phases of the movement (Hodges et al., 2001). This entrainment has been shown to assist in the complex role of ventilatory muscles in various rhythmic sports such as running, cycling and rowing (Bechbache & Duffin, 1977; Berry, Puntenney & Sandt, 1989; Bramble & Carrier, 1983; Mahler, Hunter, Lentine & Ward, 1991a; Mahler et al., 1991b; Siegmund et al., 1999; Steinacker et al., 1993; Szal & Schoene, 1989).

Usually, after around eight months of training, rowers adopt an entrainment of ventilation in which a consistent breathing pattern develops in rhythm to their rowing stroke (Mahler et al., 1991a; Siegmund et al., 1999). Typically, breathing becomes entrained to the rowing stroke with breathing ratios of 1:1, 2:1 and 3:1 (Mahler et al., 1991a, 1991b; Steinacker et al., 1993). Most elite rowers use either the 1:1 or 2:1 breathing pattern. The 1:1 breathing pattern consists of one expiration during the drive phase and one inspiration during the recovery phase. Some athletes adopt a 2:1 breathing pattern (2 breaths per stroke), in which the rower inspires just before the catch and at the finish and expires at later points during the drive and recovery (Mahler et al., 1991b; Siegmund et al., 1999; Steinacker et al., 1993).

Research into entrainment of breathing during rowing has identified that experienced rowers take a smaller breath, which decreases their V_T at particular points during the drive and recovery phase of the stroke. Some research has suggested that highly trained rowers (i.e. elite rowers) predominantly increase V_T while maintaining f_R to facilitate increases in exercise $\dot{V}_E$ (Mahler et al., 1991b; Steinacker et al., 1993). However, Szal & Schoene (1989)

suggested that rowing induces hyperventilation, in which rowers adopt a higher breathing frequency with a lower V_T. The authors speculated that the hyperventilatory response may be due to a change in respiratory mechanics attributable to the variable seated position, or possibly a reduced lung volume at the catch. It has been suggested that rowers 'develop a larger negative intrapleural pressure than is needed to achieve their required V_T'; this alteration in breathing pattern may be due to 'rowers stabilising their thorax by taking an inspiration at the catch phase of their stroke' (Shephard, 1998). Mahler et al. (1991b) stated that the inspiration at the catch is essential to optimise the power output, particularly during the drive phase, in order to maximise the transmission of force between the upper limbs to the blade handle (oar). This pre-catch breath is essential for force transmission from the blade through the trunk as it increases the internal pressures within the chest and abdomen to stiffen the trunk. However, the respiratory muscles that are responsible for maintaining high levels of ventilation during exercise also play a role in maintaining trunk posture and intra-abdominal pressure during the rowing stroke (Manning, Plowman, Drake, Looney & Ball, 2000). A decrease in lung volume at the catch may decrease internal pressures potentially leading to an increased risk of rib stress fractures and low back injury (Rumball, Lebrun, Di Ciacca & Orlando, 2005). When entrained breathing pattern during the stroke breaks down due to fatigue, this increases the risk of injury and potentially decreases rowing performance.

During rowing, the ventilatory demands are exacerbated by the simultaneous and rhythmical movement of the upper and lower limbs, making the respiratory muscles more susceptible to RMF. An increase in RMF may impair rowing performance. Previous research has shown that RMT attenuates IMF (Caine & McConnell, 1998b; McConnell & Lomax, 2006; O'Kroy & Coast, 1993; Romer et al., 2002b; Volianitis et al., 2001c), and may attenuate the respiratory muscle metaboreflex (McConnell & Lomax, 2006; Witt et al., 2007), thus providing a potential ergogenic effect on rowing time trial performance (see table 1.1). The

following chapter will highlight the primary limitations of the ventilatory muscles during exercise, including RMF and the respiratory muscle metaboreflex response, on locomotor muscles and exercise tolerance. It will also review the current literature on RMT and its effects, if any, on exercise and sport performance and other physiological variables.

CHAPTER TWO

REVIEW OF LITERATURE

2.0: RESPIRATORY MUSCLE LIMITATIONS DURING EXERCISE

Historically, research investigating exercise performance limitations has focused principally on cardiac and skeletal muscle constraints to exercise (Harms, Babcock, McClaren, Pegelow, Nickele, Nelson & Dempsey, 1997; Saltin, Nazar, Costill, Stein, Jansson, Essen & Gollnick, 1976; Saltin & Strange, 1992) in which maximal oxygen consumption ($\dot{V}O_{2max}$) is acknowledged as the prime limitation to exercise performance/tolerance (Bassett & Howley, 1997, 2000). In 1986, Dempsey, in his article 'Is the lung built for exercise?', questioned this traditional view that oxygen (O_2) transport to the muscle cells and utilisation of O_2 were the only limiting factors to exercise. Wherein, he speculated that chronic exercise adaptations occur in both the musculoskeletal and cardiovascular system to meet the increasing demands, but the respiratory system fails to compensate and is 'left behind'; hence the lungs end up becoming the limiting factor. At higher exercise intensities, the respiratory muscles are susceptible to exercise-induced fatigue arising from an imbalance between the increasing metabolic demands of locomotor muscles and the physiological capacity of the respiratory muscles to meet the associated demand for gas exchange (Harms et al., 1997, 1998, 2000). Consequently an increase in respiratory muscle work induces changes that result in working limb blood flow being reduced (Harms et al., 1997; Sheel et al., 2001, 2002) and/or an accelerated rate of muscular fatigue (McConnell & Lomax, 2006), thus potentially limiting exercise tolerance (Harms et al., 2000).

2.0.1: OXYGEN COST OF BREATHING

The work of the respiratory muscles is significantly higher during exercise compared to that at rest. The O_2 cost of respiratory muscle work during eupnoea (quiet breathing) is usually < 5% of the total cardiac output ($\dot{Q}_T$); however with an increase in exercise intensity, the O_2 cost rises and the respiratory muscles demand a greater proportion (up to 16%) of $\dot{Q}_T$ (Aaron, Johnson, Seow & Dempsey, 1992; Harms et al., 1998). This response during exercise is due

to an increase in the total work of breathing (W_b) imposed by the respiratory muscles performing more work to move the lung and the chest wall and overcoming flow resistance to meet increasing ventilatory demands and metabolic requirements (Harms et al., 1998). Hence, an increase in the O_2 cost of breathing reduces the amount of available O_2 for non-ventilatory work (i.e. working locomotor muscles).

At maximal intensity exercise, the W_b requires an increase in O_2 consumption up to 10% of $\dot{V}O_{2max}$ in moderately fit subjects and up to 15% in highly fit subjects (300-600 $ml \cdot min^{-1}$ absolute $\dot{V}O_{2max}$) (Aaron et al., 1992; Harms et al., 1998). Harms et al. (1998) investigated the changes in W_b on $\dot{Q}_T$ during maximal exercise. Eight male cyclists performed repeated maximal intensity bouts of cycling (2.5 min) whilst oesophageal pressure (P_{OES}) was measured during normal, unloaded and loaded breathing. During unloaded breathing (using PAV) there were reductions in stroke volume (SV), $\dot{Q}_T$ and pulmonary O_2 consumption at $\dot{V}O_{2max}$ compared to normal breathing. The reduction in SV, at least in part, can be explained by a decrease in $\dot{V}O_2$ demonstrating a decrease in reduction of work required by the ventilatory musculature. However, no differences were observed during loaded breathing (increased using resistive loads) compared to the control group as the increased metabolic requirement caused a reduction in the available $\dot{Q}$ to the working limbs. The authors concluded that the increase in $\dot{Q}_T$ (14-16%) to the contracting respiratory muscles was due to a local reflex vasoconstriction which compromised leg blood flow.

Thus, as respiratory muscle work increases the respiratory muscles demand a greater portion of the $\dot{Q}_T$. Previous animal studies have shown an increase in blood flow to the respiratory muscles during submaximal and maximal exercise (Laughlin, Klabunde, Delp & Armstrong, 1989; Manohar, 1986, 1988; Musch, Friedman, Pitetti, Haidet, Stray-Gunderson, Mitchell & Ordway, 1987) by which cardiac output was redistributed from the active limb locomotor

muscles ($\dot{Q}_L$). The concept of 'competition' for blood flow between active muscle groups in humans was previously unclear, as initial investigations of this phenomenon using limb muscles during submaximal exercise had generated contradictory results. One study showed that blood pressure remained constant during a cycle ergometry test, even when arm exercise was added (Secher, Clausen, Klaussen, Noer & Trap-Jensen, 1977), suggesting that vasoconstriction must have occurred in the leg muscle to maintain the constant blood pressure (Harms et al., 2000). However, other investigators were not able to substantiate these findings when they performed similar studies at submaximal intensities (Savard, Richter, Strange, Kiens, Christensen & Saltin, 1989; Richardson, Kennedy, Knight & Wagner, 1995; Richter, Kiens, Hargreaves & Kaejer, 1992). Even though they found an increase of noradrenaline spillover due to sympathetic activation, no change was evident in the $\dot{Q}_L$. It seems at submaximal exercise intensities, although the sympathetic response still creates a systemic vasoconstriction, unless there is an increase in respiratory muscle work (sufficient to increase local metabolites) there is no physiological rationale for a redistribution away from the working limb musculature.

Harms et al. (1997, 1998) measured the SV, $\dot{Q}_L$ and $\dot{V}O_2$ of the legs of healthy trained subjects during an incremental $\dot{V}O_{2max}$ cycle test, and manipulated inspiratory muscle work. The authors observed that an increase in inspiratory muscle work (loading inhalation using resistive loads) during maximal exercise caused localised vasoconstriction in the active limb muscle with a subsequent increase in $\dot{Q}$ to the respiratory muscles (up to 14-16% of $\dot{Q}_T$). The authors speculated that this was due to an increase in blood flow directed to the respiratory muscles in order to maintain their increased O_2 demand. Thus, suggesting at maximal levels of cardiac output and with fatiguing respiratory muscle work, arterial ischemia at the respiratory muscles causes an increase in local metabolites and chemical afferents to signal the central nervous system to elicit a sympathetic response for general vasoconstriction.

Although this has not been measured directly, it has been assumed that blood is redistributed to the respiratory muscles to meet increasing O_2 demand (see 2.0.3: Respiratory Muscle Metaboreflex). Consequently, any decrease in available blood flow to the limb musculature accelerates limb fatigue leading to a decrease in exercise tolerance (Harms et al., 2000).

2.0.2: EXERCISE-INDUCED RESPIRATORY MUSCLE FATIGUE

Respiratory muscle fatigue (RMF) has been shown to occur in healthy adults during both short-term high intensity (Bye, Esau, Walley & Macklem, 1984; Coast, Clifford, Henrich, Stray-Gundersen & Johnson, 1990; Johnson et al., 1993, 1996; Mador, Magalang, Rodis & Kufel, 1993; Roussos & Macklem, 1977) and prolonged submaximal exercise (Johnson et al., 1993; Loke, Mahler & Virgulto, 1982). The occurrence of RMF during exercise leads to alterations in breathing pattern to facilitate and maintain the force generating capacity of the inspiratory muscles. Similar to skeletal muscle, the diaphragm and other respiratory muscles react to any modification in physical activity patterns by increasing or decreasing $\dot{V}_E$ (Powers & Shanely, 2002). With increasing exercise intensity there is an increase in ventilatory demand; any increase in $\dot{V}_E$ raises the W_b, which increases the propensity for RMF (Aubier, 1989; Harms, Wetter, St. Croix, Pegelow & Dempsey, 2000). Research has demonstrated that both the inspiratory and expiratory muscles are susceptible to fatigue which may impose a limitation to exercise tolerance in healthy adults during heavy-intensity sustained exercise (Boutellier, Buchel, Kundert & Spengler, 1992; Boutellier & Piwko, 1992; Johnson et al., 1996; Taylor and Romer, 2009).

It has been suggested that exercise-induced RMF occurs when the requirements imposed upon the respiratory muscles are increased substantially, such as during heavy endurance exercise (Babcock et al., 1995, 2002; Johnson et al., 1993: Mador et al., 1993). Studies have shown that whole body exercise elicits global IMF and EMF, as assessed using voluntary

evoked pressures measured at the mouth, and/or changes in spirometry before and after exercise (Bye, Farkas & Roussos, 1983; Bye et al., 1984; Coast et al., 1990; Fuller et al., 1996; Hill, Jacoby & Faber, 1991; Loke et al., 1982; McConnell, Caine & Sharpe, 1997; Ozkaplan, Rhodes, Sheel & Taunton, 2005; Romer et al., 2002a, 2002b; Taylor, How & Romer, 2006; Verges, Schulz, Perret & Spengler, 2006; Volianitis et al., 2001c). Similarly, the existence of exercise-induced diaphragm fatigue using oesophageal balloons/electrodes (Bye et al., 1984; Roussos & Macklem, 1977; Roussos, Fixley, Gross & Macklem, 1979), or electrically evoked transdiaphragmatic pressures is well documented (Aubier et al., 1981; Bellemare & Bigland-Ritchie, 1984, 1987; Johnson et al., 1993; Mador et al., 1993; McKenzie, Bigland-Ritchie, Gorman & Gandevia, 1992; Moxham, Morris, Spiro, Edwards & Green, 1981).

Using nerve stimulation techniques to quantify changes in muscle force activation, research has shown that progressive exercise at intensities exceeding 80% of VO_{2max} induces significant fatigue in both the diaphragm (Babcock et al., 1995, 1998; Babcock, Pegelow, Harms & Dempsey, 2002; Johnson et al., 1993; Mador et al., 1993) and abdominal muscles (Cordain, Rode, Gotshall & Tucker, 1994; Fuller et al., 1996; Taylor et al., 2006; Verges et al., 2006). Using the BPNS technique, Johnson et al. (1993) assessed diaphragm fatigue at various lung volumes (ranging from RV to TLC) before and after exercise. The authors observed a 32% reduction in peak twitch transdiaphragmatic pressure ($P_{di}tw$) and a mean reduction in volitional P_{di} following constant-load exercise to exhaustion. Studies using healthy adults showed a consistent reduction in P_{di} of ~15-30% (assessed using supramaximal stimulation of the phrenic nerve) following exhaustive exercise (> 80-85% VO_{2max}) (Babcock et al., 1995, 1996, 1998, 2002). Similarly, the abdominal muscles, responsible for expiration and assisting the inspiratory muscles during high levels of $\dot{V}_E$, have been shown to exhibit similar levels of fatigue to the inspiratory muscles, following sustained high intensity

exercise (Fuller et al., 1996; Taylor et al., 2006; Verges et al., 2006). Taylor et al. (2006) measured abdominal muscle fatigue following dynamic lower limb exercise to exhaustion (> 90% $\dot{V}O_2$ peak), following which they observed a 33% reduction in twitch gastric pressure ($P_{ga}tw$) and a 28% mean reduction in P_{ga} response to stimulations at 1-25 Hz. The potential mechanisms to account for the decrease in respiratory muscle pressures post-exercise have been attributed to both peripheral (i.e. muscle contractile failure, level of respiratory muscle work) (Babcock et al., 2002; Bellemare & Bigland-Ritchie, 1987) and/ or central fatigue (i.e. reduced central motor drive) (Bellemare & Bigland-Ritchie, 1987). The functional consequences of exercise-induced diaphragm and abdominal muscle fatigue may increase perceptions of dyspnoea or limb discomfort and/or potentially activate a muscle metaboreflex, all of which may potentially limit exercise tolerance.

Recently, a novel experiment set out to determine the 'time-point of manifestation' of diaphragmatic fatigue during exercise (Kabitz, Walker, Schwoerer, Sonntag, Walterspacher, Roecker & Windisch, 2007). Unlike previous studies which have only measured diaphragm fatigue pre and post-exercise (Mador & Acevedo, 1991; Johnson et al, 1993; Mador et al., 1993; Babcock et al., 1996; Hamnegard et al., 1996; Spengler & Boutellier, 2000; Dempsey et al., 2006), this was the first study to assess $P_{di}tw$ before, during and after an incremental workload test (85% $\dot{V}O_{2max}$). Instead of a decrease in $P_{di}tw$ with increasing intensity and duration, as was previously speculated, the authors observed that diaphragmatic strength progressively increased during exercise followed by a measureable decrease immediately at the termination of exercise. The authors also observed a correlation with increases in $\dot{V}O_2$, $\dot{V}_E$ and dyspnoea with the increase in $P_{di}tw$ during exercise ($P < 0.05$) suggesting that diaphragm strength increases and is not susceptible to fatiguing failure during high intensity exercise. Some authors have criticised this study as there was no record of a controlled stimulus or control for other measures of fatigue (e.g., change in diaphragm muscle length) (Amann,

Romer & Dempsey, 2007) both of which could be a potential source of error. Even so, these controversial findings present a conflicting argument that maximal exercise improves diaphragm contractility, not impairs respiratory muscle function as previously thought. More studies are required to understand why $P_{di}tw$ immediately decreased after exercise; however the authors have speculated that this may be due to a neural mechanism.

A follow-up study was performed to determine whether the changes in diaphragmatic strength during and after exercise were due to changes within the diaphragm or a response to whole body exercise (Kabitz, Walker, Walterspacher, Sonntag, Schwoerer, Roecker & Windisch, 2008). To test this, subjects performed a bout of progressive whole body exercise and a 'sham-training' session in which the participants performed isocapnic hyperventilation to mimic breathing rate and duration during the exercise bout. Interestingly, they found that both forms of exercise elicited progressive increases in diaphragmatic strength during the training session (as measured by $P_{di}tw$; $p < 0.05$), however only the whole-body exercise session led to an immediate decrease in $P_{di}tw$ at termination. Similar findings were reported by Babcock et al. (1995) in which the authors observed no diaphragmatic fatigue at rest when the duration and W_b incurred during exercise was mimicked. However, they reported a consistent and significant decrease in P_{di} response (measured using supramaximal BPNS) following whole body exercise to exhaustion (86-93% of $\dot{V}O_{2max}$; $P < 0.05$). The authors concluded that the competition for blood flow and/or extracellular fluid acidosis by the working locomotor muscle, along with an increase in respiratory muscle work led to the exercise-induced diaphragm fatigue in the exercise trial (Babcock et al., 1995). It seems that respiratory muscle work alone is not sufficient to induce RMF; rather the RMF expressed post-exercise occurs only when there is simultaneous involvement of other large muscle groups such as during exercise (Kabitz et al., 2008). The severity of exercise-induced RMF seems to be governed by the intensity of exercise relative to $\dot{V}O_{2max}$ (usually > 80%)

(Babcock et al., 1995; Johnson et al., 1993), as well as the simultaneous competition for blood flow from working locomotor muscles (Babcock et al., 1995; Harms et al., 1997, 1998; Sheel et al., 2001).

ROLE OF INSPIRATORY MUSCLE WORK ON EXERCISE LIMITATION

The inspiratory muscles have been the main focus of research on RMF to date. Inspiratory muscle fatigue (IMF) has been documented after short-duration high intensity exercise (Johnson et al., 1993; Lomax & McConnell, 2003; Mador et al., 1993; Riganas et al., 2008; Volianitis et al., 2001c), repetitive sprint (Romer et al., 2002c; Tong et al., 2008) and prolonged submaximal exercise (Guenette et al., 2006; Loke et al., 1982; McConnell et al., 1997; Romer et al., 2002b). The question of whether inspiratory muscle fatigue impacts exercise performance has been the subject of investigation for a number of years (Babcock et al., 2002; Johnson et al., 1996; Romer & Polkey, 2008). Two main methods have been used in an attempt to isolate the role of inspiratory muscle work during exercise 1) by pre-fatiguing them or 2) by reducing their workload.

Pre-fatigue of the inspiratory muscles prior to exercise can be done by using either sustained maximal isocapnic hyperpnoea (rapid breathing) or resistive loading methods. Both methods have been shown to decrease T_{lim} in short term, high intensity exercise (Mador & Acevedo, 1991; Martin, Heintzelman & Chen, 1982) and resistance exercise (McConnell & Lomax, 2006). For example, Mador & Acevedo (1991) demonstrated a reduction in cycling time in a group of ten healthy cyclists who performed exercise to maximal capacity. Inspiratory muscle fatigue (IMF) was achieved by having the subjects breathe against an inspiratory threshold load until they could no longer sustain the target pressure (~80% P_{Imax}). Their results showed a decrease in exercise time with prior IMF, and an increase in perceived exertion, suggesting that IMF impairs subsequent high-intensity (submaximal) exercise performance. However,

not all studies have demonstrated a change in subsequent exercise performance following ventilatory muscle work (Dodd, Powers, Thompson, Landry & Lawler, 1989; Sliwinski et al., 1996; Spengler, Knopfli-Lenzin, Birchler, Trapletti & Boutellier, 2000). Dodd et al. (1989) compared pulmonary function, gas exchange variables and time to exhaustion following 10 min of volitional hyperpnoea prior to a constant load exercise test (85% $\dot{V}O_{2max}$ to exhaustion). No difference in either the physiological or performance variables was evident following isocapnic hyperpnoea ($P > 0.05$). However, the authors did not objectively assess RMF following hyperpnoea; as such the participants may not have reached a sufficient level of RMF to induce changes in exercise performance. Hence, the lack of consistent findings in the pre-fatiguing literature may be due to several factors, including: differences in (or the lack of) the assessment of RMF prior to exercise, participant motivation and expectations between trials (fatiguing vs. non-fatigue trial) and/or an altered breathing pattern due to increased intensity of dyspnoea (Mador & Acevedo, 1991).

Alternatively, inspiratory muscle work can be reduced during exercise by mechanically unloading the muscles using a proportional assist ventilator (PAV) (Babcock et al., 2002; Gallagher & Younes, 1989; Harms et al., 1997, 1998, 2000; Romer, Haverkamp, Lovering, Pegelow & Dempsey, 2006; Romer, Miller, Haverkamp, Pegelow & Dempsey, 2007), or low density gas mixtures (Aaron, Henke, Pegelow, Dempsey & Rankin, 1985; Hussain, Pardy & Dempsey, 1985; Maio & Farhi, 1967). Harms et al. (2000) investigated the effects of respiratory muscle work following T_{lim} in 11 randomised cycling trials (n = 7 male cyclists). Using PAV, they compared the influences of loaded or unloaded breathing to a control condition. They found that unloaded breathing (reduction of W_b to ~37-45% of control) increased cycling T_{lim} by 1.3 ± 0.4 min ($P < 0.05$); whereas loaded breathing (increase in W_b to 128-157% of control) decreased performance 1.0 ± 0.6 min ($P > 0.05$). By reducing respiratory work and limiting IMF, unloaded breathing created a substantial decrease in $\dot{V}O_2$

and reduced the perceptual effort of respiratory and limb discomfort. However, the authors also observed a significant correlation with changes in T_{lim} and that of both respiratory and limb discomfort experienced during loaded and unloaded breathing trials. Not all studies have found a performance benefit following respiratory muscle unloading (Gallagher & Younes, 1989; Krishnan, Zintel, McParland & Gallagher, 1996; Marciniuk, McKim, Sanii & Younes, 1994). None of these studies showed an improvement in endurance time or ventilation even though they showed a reduction in respiratory muscle work compared to control. The lack of improvement in performance may have been due to the lower exercise intensity used during constant-load exercise (~70-80% $\dot{V}O_{2max}$), or possibly that there subjects were moderately fit and would use less of their total $\dot{V}O_{2max}$ compared to more highly fit subjects (15% $\dot{V}O_{2max}$) (Aaron et al., 1982).

Interestingly, a study performed by Romer et al. (2007) using PAV to investigate whether IMF had the same impact on submaximal and maximal incremental exercise performance observed no significant changes in performance for maximal exercise. Participants performed six cycle trials, during three of which the subjects' inspiratory muscles were unloaded using PAV; sham unloading was used for the remaining sessions. The authors observed a reduction in $\dot{V}O_2$, limb discomfort, and the perception of dyspnoea with PAV at submaximal intensities, but no significant difference in peak power output were found at maximal exercise intensities between PAV and sham unloading. Collectively, these studies suggest that the prevailing condition of the inspiratory muscles influences performance and perception during high-intensity, submaximal exercise, but does not seem to limit maximal incremental exercise performance.

Similarly, studies unloading the respiratory muscles using a helium oxygen gas mixture has shown an increase in $\dot{V}_E$, $\dot{V}O_{2max}$ and exercise tolerance during both incremental (Powers,

Jacques, Richard & Beadle, 1986) and high-intensity constant load exercise (> 90% $\dot{V}O_{2max}$) (Aaron et al., 1985; Power et al., 1986). It has been suggested that the helium oxygen mixture may work to unload the respiratory muscles by decreasing the expiratory flow limitation and the relative hyperinflation caused during heavy exercise (McClaren, Wetter, Pegelow & Dempsey, 1999). However, studies performing exercise at lower intensities (< 80-85% $\dot{V}O_2max$) did not elicit significant improvements in exercise time, regardless of the method used for unloading (Aaron et al., 1985; Gallagher & Younes, 1989; Krishnan, Zintel, McParland & Gallagher, 1996; Marciniuk, McKim, Sanii & Younes, 1994). Nonetheless, it is difficult to ascertain whether the physiological and performance changes which occurred during unloaded breathing were due to a change in RMF or whether the effects of PAV or helium provided a global decrease in the perception of respiratory and muscular effort (Spengler & Boutellier, 2000). Harms et al. (2000) highlighted three respiratory influences which may have led to the improved exercise performance: those due to, 1) O_2 and CO_2 transport, 2) IMF, and 3) perceived respiratory and muscle exertion.

Some studies have suggested that high intensity cardiovascular exercise training protects athletes from the effects of RMF (Coast et al., 1990; Martin & Chen, 1982; Robinson & Kjeldgaard, 1982; Mickleborough, Stager, Chatham, Lindley & Ionescu, 2008). It seems rational to conclude that a certain specific respiratory muscle performance benefit would occur with whole body endurance training for long periods. However previous studies have demonstrated that even highly trained cyclists, swimmers and rowers experience IMF after short-term high intensity exercise (Harms et al., 2000; Lomax & McConnell, 2003; Mador & Acevedo, 1991; McConnell et al., 1997; Ozkaplan et al., 2005; Riganas et al., 2008; Romer et al., 2002a, 2002b; Volianitis et al., 2001c) and prolonged submaximal exercise (Loke et al., 1982). In particular, highly-trained rowers (i.e. elite) have greater respiratory muscle pressures compared to normal healthy subjects of similar age and height (Shephard, 1998); it

has been suggested that their augmented P_{Imax} and P_{Emax} may protect rowers from IMF (Donnelly et al., 1991). Whilst these athletes may have more conditioned inspiratory muscles, IMF may be attributed to the relative increase in the demand for breathing during competitive racing conditions, as well as the role of these muscles in trunk stabilisation. Regardless of the level of the athlete, the severity of IMF seems to be related to the baseline absolute strength of the inspiratory muscles (McConnell et al., 1997), as well as the intensity of the exercise (Johnson et al., 1993; Babcock et al., 1995), not solely due to whole body training.

ROLE OF EXPIRATORY MUSCLE WORK ON EXERCISE LIMITATION

Although the expiratory muscles are primarily passive at rest, these muscles become vigorously active during high intensity exercise in order to achieve very high levels of ventilation and to facilitate inspiration (De Troyer, 1983; Fuller et al., 1996). The strong contraction of the abdominal muscles following expiration at high ventilations assists inspiration by placing the diaphragm in a more favourable region of its length-tension curve (Gandevia, 1992; Grassino, Goldman, Mead & Sears, 1978). Thus, the expiratory muscles facilitate ventilation by increasing expiratory flow rates, reducing FRC and by increasing stored elastic energy (Aliverti, Cala, Duranti, Ferrigno, Kenyon, Pedotti, Scano, Sliwinski, Macklem & Yan, 1997; De Troyer, 1991). Notwithstanding this, the early phase of expiration is assisted by stored elastic energy that has been generated by the inspiratory muscles, rendering inspiration the predominant phase of breathing from a muscle work perspective. However, the shared action of the inspiratory and expiratory muscles is a pre-requisite to high flow rates, and may render both muscle groups susceptible to fatigue during heavy exercise.

As exercise intensity increases, it seems that the expiratory muscles are recruited proportionally to inspiratory muscles to meet the increased ventilatory demand (Bye et al., 1984). Research has demonstrated that EMF occurs in healthy subjects following dynamic

exercise (Cordain et al., 1994; Fuller et al., 1996; Loke et al., 1982; Suzuki, Tanaka, Yan, Chen, Macklem & Kayser, 1999; Taylor et al., 2006; Taylor & Romer, 2008; Verges et al., 2006, 2007b), and expiratory resistive loaded breathing (Haverkamp, Metelits, Hartnett, Olsson & Coast, 2001; Suzuki, Suzuki & Okubo, 1991; Suzuki, Suzuki, Ishii, Akahori & Okubo, 1992; 2001; Taylor & Romer, 2009; Verges, Sager, Erni & Spengler, 2007b). Previous research has measured EMF in a similar manner to the inspiratory muscles, by using both submaximal and supramaximal nerve stimulation of the abdominal muscles under various conditions (Kyroussis et al., 1996; Suzuki et al., 1991, 1999; Taylor et al., 2006). Kyroussis et al. (1996) submaximally stimulated the abdominal muscles while assessing $P_{ga}tw$ following 2 min of maximal isocapnic ventilation (MIV) in the seated, supine and prone positions. Abdominal muscle fatigue was evident, as demonstrated by a 17 ± 9% (P = 0.03) decrease in mean $P_{ga}tw$ (as measured in the prone posture) in all six subjects, 20 min after MIV. Suzuki et al. (1999) used electrically evoked supramaximal stimulation of the abdominal muscles to measure P_{ga} following 2 min bouts of sit-ups to task failure. They observed a decrease in mean P_{ga} of 25% at 1 min post-exercise, and 37% at 30 min, with a decrease in P_{Emax}. A recent study by Taylor et al. (2006) used direct nerve stimulation to assess EMF following dynamic lower limb exercise, in which they also observed reductions in $P_{ga}tw$ following sustained, high intensity cycling exercise. Similar to the inspiratory muscles, these studies demonstrate that the abdominal muscles are susceptible to fatigue following high intensity breathing tasks, as well as localised resistance exercise and very high intensity cycling undertaken to the T_{lim}.

There is limited research on the impact of EMF on pulmonary function and exercise performance. Some studies have shown no change in pulmonary function (FVC, FEV_1, $FEF_{25\%}$, or $FEF_{25-75\%}$) following exercise (Fuller et al., 1996) and expiratory muscle loading (Haverkamp et al., 2001). Fuller et al. (1996) studied the fatigability of the expiratory

muscles during and following a progressive high intensity exercise test to volitional fatigue. The electromyographic (EMG) activity of the rectus abdominus and the external oblique, as well as voluntary efforts to generate P_{Emax}, showed a significant decline during and following exercise; however they found no impairment in pulmonary ventilation or exercise performance. The findings of Haverkamp et al. (2001) were in agreement with these observations; following a trial in which subjects performed expiratory threshold loading at 80% P_{Emax} to volitional fatigue, they found significant EMF following the trial, but they did not observe any changes in pulmonary function. Combined, these results suggest that EMF does not induce changes to pulmonary function. This is not entirely surprising, given that expiratory flows are primarily a function of airway physiology, and not of expiratory pressure generating capacity.

As is the case for pre-fatigue of the inspiratory muscles, there is emerging evidence that pre-fatigue of the expiratory muscles impairs subsequent exercise performance (Suzuki et al., 1991; Taylor & Romer, 2009; Verges et al., 2007b). Verges et al. (2007b) compared the effects of prior EMF on running distance achieved during a 12 min running test in which pre-fatigue of the expiratory muscles elicited a consistent decrease in running speed and the distance achieved. Taylor & Romer (2008) also reported a 33 ± 10% decrease in cycle exercise time following resistive breathing suggesting this was due to an increased limb fatigue and perception of leg discomfort during exercise. However, an important factor to bear in mind in the interpretation of these studies is that these authors, and others, have demonstrated that it was impossible to induce EMF without also eliciting some degree of IMF (Suzuki et al., 1991; Taylor & Romer, 2009; Verges et al., 2007b). This being the case, it is impossible to differentiate the effect of IMF and EMF upon subsequent exercise performance. The research on the physiological impact of EMF on exercise tolerance is limited and the findings remain unclear as to the relevance EMF has on exercise and sport

performance in healthy adults. Even so, these recent findings demonstrate that the expiratory muscles are susceptible to fatigue following exercise, which may potentially activate a respiratory muscle metaboreflex response (Derchak, Sheel, Morgan & Dempsey, 2002; Sheel et al., 2001), thus demonstrating a potential role for both the inspiratory and expiratory muscles in limiting human exercise tolerance.

In summary, there is good evidence for the existence of both IMF and EMF under conditions of high respiratory muscle work. The primary consequence of RMF is a decrease in O_2 supply to the working muscles, via metaboreflex mediated vasoconstriction (see below), thereby limiting exercise tolerance. Other factors, including RMF-induced increase in effort perception, and increasing haemodynamic challenges induced by higher mechanical loads with high ventilation, may also have a negative effect upon overall exercise performance (Dempsey, Romer, Rodman, Miller & Smith, 2006; Romer & Polkey, 2008). Although there is substantial research demonstrating the negative influence of IMF upon exercise performance, there is limited research on the limitations imposed by EMF, or of any exercise limitation due to a combination of IMF and EMF. More research investigating the role of EMF and the attenuation of EMF on exercise performance is required.

2.0.3: RESPIRATORY MUSCLE METABOREFLEX

Questions still remain as to the underlying mechanisms initiating the vasoconstriction that results in limb blood flow reduction during maximal exercise and the implications for exercise performance. Romer & Dempsey (2002) postulated that during high intensity exercise, the $\dot{Q}_T$ may be insufficient to meet the metabolic requirements of both the respiratory and limb musculature, thus eliciting a 'respiratory muscle metaboreflex'. A 'muscle metaboreflex' is the reflex arc associated with the biochemical (chemoreflex) or mechanical (mechanoreflex) pressor response to the contraction of that skeletal muscle

(Seals, 2001). The primary stimulus for activation of the muscle metaboreflex is inadequate blood flow (e.g., arterial ischemia) to the contracting muscle leading to an accumulation of metabolites and stimulation of chemical afferents. During muscular contractions, afferent nerves are stimulated leading to an increase in $\dot{V}_E$ and an increased efferent sympathetic nerve activity. The metaboreflex response leads to a substantial rise in arterial blood pressure, heart rate (f_c), $\dot{Q}_T$ and cardiac contractility (Rowell & Sheriff, 1988; Rowell & O'Leary, 1990; Sheel et al., 2002), thereby decreasing limb blood flow and intensifying effort perception (O'Leary & Joyner, 2006; Sheel et al., 2001, 2002). This decrease in limb blood flow hastens skeletal muscle fatigue ultimately resulting in a decrease in exercise performance (Babcock et al., 2000; Harms et al., 2000).

In 2000, St. Croix, Morgan, Wetter & Dempsey tested whether increases in fatiguing inspiratory muscle work would limit available blood flow to the locomotor muscle. The participants performed a series of high-resistance, prolonged duty cycle breathing at rest to elicit RMF; during the fatiguing trials the authors observed an increase in leg muscle sympathetic nerve activity (MSNA) which was independent of central motor output. This time dependent rise of MSNA (after 1-2 min of resistive breathing) was characteristic of a chemical reflex response (metaboreflex), rather than a force generated response (mechanoreceptor); hence the authors concluded that the rise in MSNA was mediated by a muscle metaboreflex. Later studies revealed that this gradual increase in limb muscle MSNA was associated with an increase in leg vascular resistance and decrease in limb blood flow (Sheel et al., 2001, 2002). Furthermore, Sheel et al. (2002) demonstrated that during fatiguing inspiratory muscle work, there is a similar increase (to that of skeletal muscles) in local metabolites which activates Group III/IV phrenic nerve afferents to discharge. Hence, demonstrating the existence of a muscle metaboreflex originating from the diaphragm in which the threshold for activation is fatiguing respiratory muscle work (Sheel et al., 2002).

Similar to that of the inspiratory muscles, Derchak et al. (2002) demonstrated the same increase in MSNA, mediated by an expiratory muscle metaboreflex, during expiratory loading to task failure. This activation of the respiratory muscle metaboreflex (see fig. 2.1) increases sympathetic vasoconstrictor outflow to all vascular beds (including the respiratory muscles), albeit the diaphragm vasculature appears to be less sensitive to sympathetic activity (Aaker & Laughlin, 2002). Thus it seems this metaboreflex is responsible for maintaining adequate blood flow to the respiratory musculature, thus ensuring the body's ability to maintain pulmonary ventilation during high intensity exercise.

Recent studies have demonstrated the effects of an inspiratory muscle metaboreflex upon leg vasoconstriction following IMF (McConnell & Lomax, 2006; Witt et al., 2007). In 2006, McConnell & Lomax used a series of test protocols to determine whether pre-fatiguing of the inspiratory muscles, as well as mechanical restriction of blood flow to the calf muscle would influence plantar flexion T_{lim}. They found that both conditions decreased plantar flexion T_{lim} compared to control; thus providing some evidence that mechanical occlusion, or potentially the activation of an inspiratory muscle metaboreflex, accelerates the rate of calf fatigue. Arguably, the stressful breathing challenge itself may have been sufficient to blunt the central drive to breath, thus leading to a decrease in calf endurance. However following 4 wk IMT, the authors observed a reduction in the rate of fatigue in the lower limb suggestive of an increase in the threshold for activation of the inspiratory muscle metaboreflex.

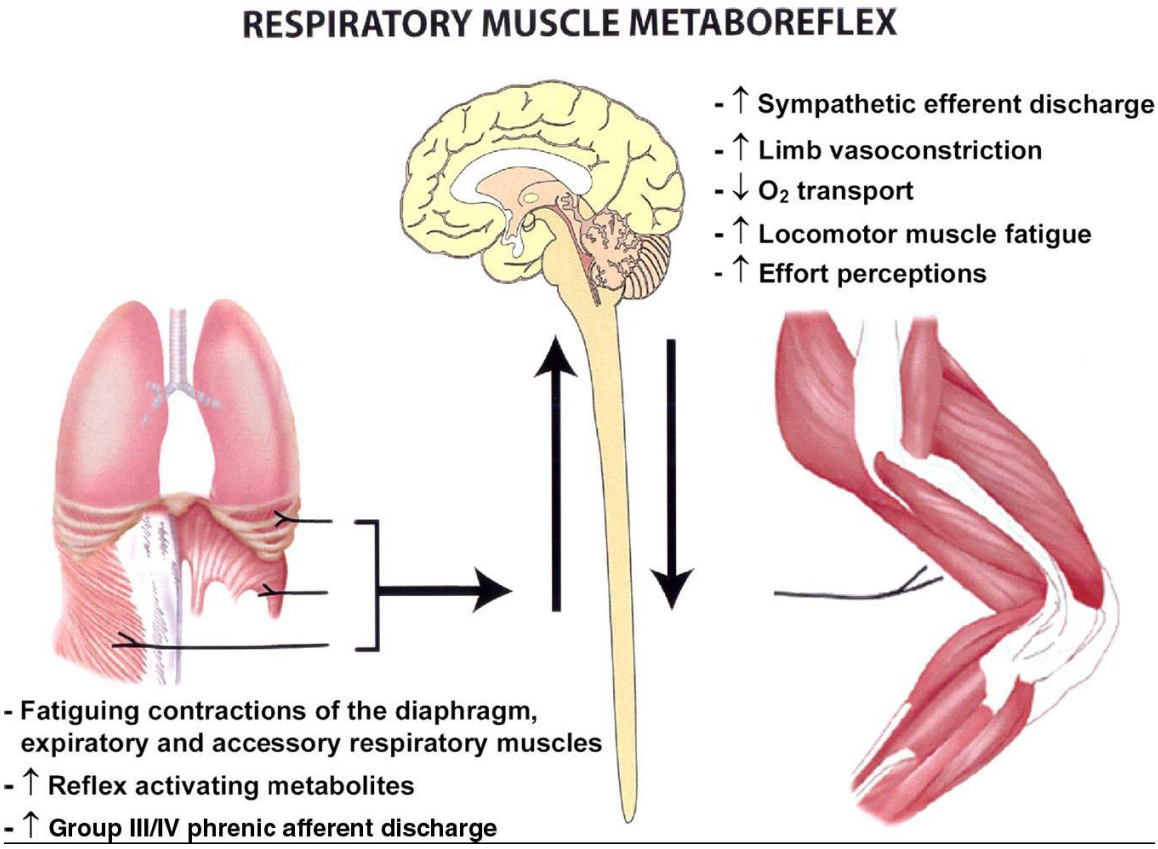


Figure 2.1 Schematic of the proposed respiratory muscle metaboreflex and its effects. (Adapted from Dempsey et al., 2002)

More recently, Witt et al. (2007) investigated the cardiovascular responses associated with the respiratory muscle metaboreflex following IMF. During a bout of inspiratory resistive loading at 60% P_{Imax}, participants showed a sustained increase in f_c and mean arterial blood pressure (MAP) within 2-3 min of the start of resistive breathing. Similar to the McConnell & Lomax (2006) study, this increase in cardiovascular response to RMF was attenuated following 5 wk of IMT (at ~50% P_{Imax}), an effect that was not seen in the sham training group. Witt et al. (2007) suggest that the decrease in cardiovascular response was most likely due to a reduced activation of chemosensitive afferents within the respiratory muscles. These studies seem to suggest that by increasing inspiratory muscle strength (through IMT) metabolite accumulation is attenuated, thereby delaying inspiratory muscle metaboreflex

activation. Hence, it seems there may be a threshold for the inspiratory muscle work required to elicit the metaboreflex response and that this threshold may potentially be increased by increasing inspiratory muscle strength.

Verges et al. (2007) conducted a RMT study in which participants performed either 4-5 wk of normocapnic hyperpnoea training or sham-training (n = 12 healthy males). The authors reported a reduction in pre to post-exercise $P_{di}tw$ in both groups following a constant-load cycling test to exhaustion (85% maximal power output). Neither group showed significant improvements in either IMF, EMF or cycling endurance; however a sub-group of the normocapnic hyperpnoea training group, those with > 10% fall in $P_{di}tw$ post-exercise at baseline, showed an improvement in both RMF (IMF and EMF; $P = 0.038$, respectively) and cycling endurance ($P = 0.017$) following training. The authors did find significant relationships between improved cycling endurance and a decrease in $[La^-]_B$ concentrations and ventilatory drive ($P < 0.05$) (Verges et al., 2007). A reduction in $[La^-]_B$ concentrations has previously been observed following both normocapnic hyperpnoea (Boutellier & Piwko, 1992; Boutellier et al., 1992; Spengler et al., 1999) and PTL training (McConnell & Sharpe, 2005; Romer et al., 2002c). Romer et al. (2002c) suggested that up to 52% of the total variance in sprint recovery time was due to changes in $[La^-]_B$ concentrations. Although no authors have suggested that a decrease in $[La^-]_B$ concentrations is the mechanism which improves exercise tolerance, it seems that $[La^-]_B$ is somehow associated with changes in performance following RMT.

It is interesting that the changes in P_{di} did not correlate to improvements in performance, as fatiguing contractions of the diaphragm during exercise at maximal cardiac output have been shown to elicit a respiratory muscle metaboreflex (Sheel et al., 2001; Witt et al., 2007). However, improvements in performance following RMT appear to have at least two

underlying mechanisms (McConnell, 2009), 1) alterations to the activation of a respiratory muscle metaboreflex originating from inspiratory muscles, and perhaps also expiratory muscles; 2) attenuation of effort perceptions. Given this multifactoral mechanism, and the potential involvement of both inspiratory and expiratory muscles, it would be surprising if RMT induced changes in diaphragm fatigue were correlated with changes in performance, since this would necessitate a proportional relationship between the two.

2.1: EFFECTS OF RESPIRATORY MUSCLE TRAINING UPON EXERCISE PERFORMANCE

As was discussed in the previous section, IMT has been shown to be an effective method for delaying or attenuating the detrimental effect of RMF (Sheel, 2002), and the associated inspiratory muscle metaboreflex (McConnell & Lomax, 2006; Witt et al., 2007) upon exercise performance. This evidence provides a rationale for the use of IMT as an ergogenic aid to improve exercise and sport performance.

As discussed in Ch.1, research has demonstrated improvements in both RMS and RME following RMT (see table 1.1), which increases the fatigue resistance of the respiratory muscles and their mechanical efficiency during dynamic whole body exercise (Romer & Polkey, 2008; Sheel, 2002). This section will highlight the specific physiological and ergogenic effects of IMT, expiratory muscle training (EMT) and concurrent IMT and EMT, discussing some of the physiological variables affected by RMT.

2.1.2: ERGOGENIC EFFECT OF IMT ON EXERCISE PERFORMANCE

By comparison to other training methods, IMT has been shown to provide consistent results in improving time trial performance in both healthy untrained and trained subjects (see table 2.1). Table 2.1 summarises the studies measuring the impact of IMT on exercise

performance in healthy individuals following IMT. The majority of studies using pressure threshold loading IMT have observed an improvement in performance during short-term high intensity exercise, prolonged submaximal and time trial cycling and high intensity, repetitive sprint performance (see table 2.1). Despite this, there remains contradiction in the literature relating to the exercise performance benefits associated with RMT in general. It has been suggested that the discrepancies in performance related outcomes following RMT may be attributed to differences in research design including inappropriate performance outcome variables, ineffective RMT protocols, small sample sizes and/or the lack of carefully matched experimental and placebo groups (McConnell & Romer, 2004b). These differences in research design may be a plausible explanation for the inconsistency in determining the effect RMT, or more specifically IMT, has on exercise performance.

For instance, studies investigating the effects of IMT on endurance performance, particularly running or cycling, have suggested that even with an increase in RMS or RME, no change is evident in endurance exercise capacity (Downey, Chenoweth, Townsend, Ferguson, Ranum & Harms, 2005; Downey, Chenoweth, Townsend, Ranum, Ferguson & Harms, 2007; Hanel & Secher, 1991; Sonnetti, Wetter, Pegelow & Dempsey, 20001; Williams, Wongsathikum, Boon & Acevedo, 2002). In Hanel & Secher's (1991) study, both the training and control groups increased 5 km run distance (8% and 6%, respectively), suggesting a lack of reliability within the key outcome measure of running performance. Williams et al. (2002) investigated the effects of 4 wk IMT on endurance run time at 85% $\dot{V}O_{2max}$ in 7 healthy subjects. Although subjects improved both RMS and RME, the authors observed no difference in $\dot{V}O_{2max}$ or endurance run time. Both of these running performance studies used T_{lim} as a performance variable; but without reliability data and/or a control group, these studies remain inconclusive.

A more recent study investigating the influence of 6 wk of IFRL using the TIRE training protocol on running time to exhaustion employed an IFRL group (80% SMIP), sham IFRL group (30% SMIP) and a control group (no training) (Mickleborough, Nichols, Lindley, Chatham & Ionescu, 2009). Although both the IRFL and sham IRFL group increased forced inspiratory volume in 1 s (FIV_1) following training, only the IFRL group increased P_{Imax}, SMIP, maximal inspiratory muscle power output, inspiratory muscle work capacity and time to fatigue during the endurance test compared to the other groups. In addition, the IRFL group experienced reductions in $\dot{V}O_{2max}$, $\dot{V}_E$, HR and $[La^-]_B$ and perceptual responses during constant workload exercise. Some researchers have argued that the lack of performance benefit shown in the earlier studies may be due to the use of constant work rate tests, which are open-ended, have subjective limits of exhaustion and are highly variable making the results difficult to interpret (Hopkins, 2000; Holm, Sattler & Fregosi, 2004). However, more recently Hopkins (2004) has revised this position, concluding that the sensitivity of T_{lim} tests of performance is at least as good as that of time trials (Amann, Hopkins & Marcora, 2008). Collectively, these studies suggest that more important than just the outcome variable, the use of a well controlled rigorous study design, with an appropriate RMT protocol which elicits significant improvements in respiratory muscle strength, may improve exercise tolerance.

Table 2.1 Research investigating the benefits of specific inspiratory muscle training on sport performance.

Intervention	Sample Size (N)	Control Sham Training Group	Exercise Test	Increase in P_{Imax}	Modality /Exercise	Improved Performance	References
50-65% P_{Imax}, ~10 min daily, 4-5 d·wk^{-1}, 4 wk	20	Y	5 km time trial	32%	Running	N	Hanel & Secher, 1991
>50% P_{Imax}, 30 breaths (b) x 2 sets, 6 d·wk^{-1}, 4 wk	8	Y	Submaximal T_{lim}	45%	Cycling	Y	Caine & McConnell, 1998b
30', 6 d·wk^{-1}, 10 wk	20	Y	$\dot{V}O_{2max}$	25%	Running	N	Inbar et al., 2000
30-35 min, 5 d·wk^{-1}, 5 wk	17	Y	Incremental & constant load test toT_{lim}	8%	Cycling	N	Sonnetti et al., 2001
>50% P_{Imax}, 30 b x 2 sets, 6 d·wk^{-1}, 11 wk	14	Y	6MAO 5 km time trial	45 ± 30%	Rowing (females)	Y	Volianitis et al., 2001c
>50% P_{Imax}, 30 b x 2 sets, 6 wk	16	Y	20 km & 40 km time trial	28 ± 7%	Cycling	Y	Romer et al., 2002a
>50% P_{Imax}, 30 b x 2 sets, 6 wk	24	Y	Repetitive sprint test	31 ± 2%	Repetitive Sprints	Y	Romer et al., 2002c
>50-65% $P_{Imax,}$ 25' daily, 4-5 d·wk^{-1}, 4 wk	7	N	85% $\dot{V}O_{2max}$, $\dot{V}O_{2max}$	31%	Running	N	Williams et al., 2002
>50% P_{Imax}, 30 b x 2 sets, 6 d·wk^{-1}, 4 wk	18	Y	Constant load to T_{lim}	20.3%	Running	Y	Edwards & Cooke, 2004
IRL 80% P_{Imax}, 3 d·wk^{-1}, 10 wk	15	Y	75% $\dot{V}O_{2peak}$	34%	Cycling	Y	Gething et al., 2004a
>50% P_{Imax}, 30 b x 2 sets, 7 d·wk^{-1}, 6 wk	12	Y	Constant load to T_{lim}	14 ± 9%	Cycling	Y	Johnson & Sharpe, 2004
>50% P_{Imax}, 25' daily, 5 d·wk^{-1}, 6 wk	19	Y	6MAO effort	28%	Rowing	N	Vrabas & Riganas, 2005

Note: P_{Imax}, maximal inspiratory pressure; b, breaths; T_{lim}, limit of tolerance; VO_{2max}, maximal oxygen uptake; 6MAO, six minute all out effort; Y, yes; N, no.

Table 2.1. *cont.* Research investigating the benefits of inspiratory muscle training on sport performance.

Intervention	Sample Size (N)	Control or Sham Training Group/Trial	Exercise Test	Increase in P_{Imax}	Modality /Exercise	Improved Performance	References
50 cm H_2O, 30' (60 b every 30 sec), 5 d·wk^{-1}, 4 wk	30	Y	Underwater swim endurance	11%	Swimming	Y	Wyegala et al., 2006
>50% P_{Imax}, 25 min daily, 5 d·wk^{-1}, 6 wk	15	N	80% $\dot{V}O_{2max}$ to T_{lim}	M: 41 ± 20% F: 34 ± 18%	Cycling	Y	Guenette et al., 2006
>50% P_{Imax}, 30 b x 2 sets, 5 d·wk^{-1}, 6 wk	18	Y	25 km time trial & Constant power cycling	17 ± 12%	Cycling	Y	Johnson et al., 2007
>50% P_{Imax}, 40 b, 5 d·wk^{-1}, 4 wk	12	Y	85% $\dot{V}O_{2max}$ to T_{lim}	25 ± 3.1%	Running	N	Downey et al., 2007
50 cm H_2O, 30' (60 b every 30 sec), 5 d·wk^{-1}, 4 wk	30	Y	Underwater & surface swim endurance	12%	Swimming	Y	Wyegala et al., 2007
>50% P_{Imax}, 30 b, 7 d·wk^{-1}, 4 wk	16	Y	5 km time trial	15 ± 7% Control: 8 ± 7%	Running	Y	Edwards et al., 2008
>50% P_{Imax}, 30 b x 2 sets, 5 d·wk^{-1}, 6 wk	19	Y	2 km time trial	28%	Rowing	N	Riganas et al., 2008
>50% P_{Imax}, 30 b x 2 sets, 6 d·wk^{-1}, 6 wk	13	Y	Yo-Yo endurance shuttle	>30%	Shuttle Running	Y	Tong et al., 2008
>50% P_{Imax}, 30 b x 2 sets, 6 d·wk^{-1}, 5 wk	27	Y	Yo-Yo intermittent recovery test	20%	Running	Y	Nicks et al., 2009
IRL 80% P_{Imax}, 3 d·wk^{-1}, 6 wk	24	Y	Constant load to T_{lim}		Running	Y	Mickleborough et al., 2009

Note: P_{Imax}, maximal inspiratory pressure; b, breaths; T_{lim}, limit of tolerance; VO_{2max}, maximal oxygen uptake; 6MAO, six minute all out effort; Y, yes; N, no.

Table 2.1. *cont.* Research investigating the benefits of inspiratory muscle training on sport performance.

Abstracts (Rowing only)							
~75% P_{Imax}, 5 sets x 12 reps, 5 d·wk^{-1}, 8 wk	13	N	2 km time trial	46%	Rowing	Y	Feutz et al., 2006
>50% P_{Imax}, 30 min, 5 d·wk^{-1}, 6 wk	16	Y	5MAO effort	35%	Rowing	Y	Vrabas et al., 2007
>50% P_{Imax}, 30 min, 5 d·wk^{-1}, 6 wk	20	Y	2 km time trial 5MAO effort	39%	Rowing	Y	Riganas et al., 2007
IMT Warm-up							
Submaximal rowing warm up (RWU), RWU, RWU + IMT WU	14	Y	85% $\dot{V}O_{2max}$	Not measured	Rowing	Y	Volianitis et al., 2001b
IMT warm-up, 30 b at15% P_{Imax} or 30 b at 40% P_{Imax}	10	Y	Maximal repetitions of 20m shuttle run	Not measured	Running	Y	Tong & Fu, 2006
IMT warm-up, 30 b at 15% P_{Imax} or 30 b at 40% P_{Imax}	10	Y	Maximum incremental badminton footwork test	Not measured	Badmin-ton	Y	Lin et al., 2007

Note: P_{Imax}, maximal inspiratory pressure; d· wk^{-1}, days per week; wk, week; km, kilometre; b, breaths; VO_{2max}, maximal oxygen uptake; 5MAO, five minute all out effort; Y, yes; N, no.

Research using time trials as an outcome variable of performance following IMT has shown more consistently positive outcomes. For example, Sonnetti et al. (2001) investigated the effects of 5 wk of inspiratory resistance strength training and hyperpnoea endurance training (placebo group) on three tests of exercise performance: fixed work rate test, incremental maximal oxygen consumption test to exhaustion and an 8 km cycling time trial. Only the resistance training group demonstrated an improvement in P_{Imax} (8%, $P < 0.05$), however there was no significant differences in any of the exercise tests compared to the placebo group. The authors did report a significant 1.8 ± 1.2% ($P < 0.01$) increase in the 8 km time trial performance test in the resistance training group; however, no improvement was evident in the placebo group (-0.3 ± 2.7).

Subsequent IMT studies using time trial outcomes have observed significant improvements in exercise performance compared to the placebo/control group (Edwards, Wells & Butterly, 2008; Johnson, Sharpe & Brown, 2007; Romer et al., 2002a; Volianitis et al., 2001c). Romer et al. (2002a) studied the effects of IMT in which 16 male cyclists completed a 20 and 40 km time trial. Following 6 wk of IMT, the training group improved in both the 20 km (3.8 ± 1.7%) and 40 km (4.6 ± 1.9%) time trial performance compared to the control group. Similarly, Volianitis et al. (2001c) demonstrated a 1.9% increase (compared to the placebo group) in distance covered in the 6 min all out rowing effort (6MAO) as well as a 25 s decrease in 5 km rowing ergometer performance time post-IMT. Similarly, more recent investigations have demonstrated a positive impact on repeated intermittent sprint exercise performance following pressure threshold IMT (Nicks et al., 2009; Romer et al., 2002c; Tong et al., 2008). Unlike constant workload or endurance tests, improvements seen in time trial and intermittent sprint performances may be due to the sport specific nature of the testing in which the athletes are able to self-motivate and push themselves to a clear target. Collectively, the literature suggests

that rigorous and well-controlled, placebo designed RMT studies using appropriate and measurable outcomes of performance have demonstrated that RMT may improve exercise performance (see Table 2.1).

IMPACT OF IMT UPON ROWING

Elite performers may experience an increase in inspiratory muscle strength (Klusiewicz, Barkowski, Zdanowicz, Boros & Weselowski, 2008; Riganas et al., 2008) and may improve exercise performance after IMT (Feutz, Brilla, Mathers-Schmidt & Knutzen, 2006; Riganas et al., 2007; Volianitis, McConnell & Jones, 2001b, Volianitis et al., 2001c; Vrabas et al., 2007). The first study to investigate the ergogenic benefit of IMT in rowers studied competitive female rowers (Volianitis et al., 2001c). The authors conducted an 11 wk IMT study using 14 highly trained female rowers; subjects performed both a 6MAO (rowing ergometer test which is a simulation of competitive rowing) and a 5 km time trial performance before and after the intervention. Following 11 wk of IMT, the training group improved more than the placebo group in both the 6MAO (1.9%) and 5 km (25 s faster) rowing ergometer time trial. This increase in inspiratory muscle strength was associated with an ablation of IMF and decreased perception of dyspnoea, which may be possible mechanisms for increasing exercise tolerance. However, Riganas et al. (2008) observed no changes in 2 km rowing ergometer performance time following 6 wk IMT in a controlled trial of elite male and female rowers (n = 19). Inspiratory muscle strength (P_{Imax}) increased after IMT (28%) and IMF decreased following a $\dot{V}O_{2max}$ test, but no changes in 2 km rowing ergometer performance time or perceptions of dyspnoea were observed compared to the control group. More research is needed to, 1) explore the link between the increase in P_{Imax} and subsequent changes in exercise performance and 2) determine the specific ergogenic benefit of IMT, if any, in elite male rowers, as both of the above studies investigating elite rowers used either female or a combination of male and female

rowers. In general, females have smaller lung volumes and maximal flow rates as well as weaker respiratory muscles compared to men (ATS, 1991; ATS/ERS, 2002); however, in trained male and females both V_T and $\dot{V}_E$ have been shown to be constrained when performing at near maximal intensities (Johnson et al., 1992; McClaren, Harms, Pegelow & Dempsey, 1998). As discussed previously, increased baseline respiratory muscle strength may be sufficient enough to protect male rowers from IMF (Donnelly et al., 1991); hence there may be no ergogenic benefit for male rowers performing IMT.

INSPIRATORY MUSCLE TRAINING PRESCRIPTION

Similar to the underlying principles for resistance training of the limb muscles, the inspiratory muscles also require an appropriate stimulus to create a physiological change in the muscle structure and function (Caine & McConnell, 1998b; Morrissey, Harman & Johnson, 1995; Romer & McConnell, 2003). Pressure threshold IMT focuses on increasing the strength, power and endurance of the diaphragm and accessory inspiratory muscles (Caine & McConnell, 1998a; McConnell & Romer, 2004b). Appropriate training principles have been identified when using IMT to achieve a change in both physiological responses and athletic performance (Romer & McConnell, 2003). It seems it is the pressure-flow specificity of IMT that determines the nature of adaptation within the inspiratory muscles. Respiratory muscle strength (RMS) will increase by training at high pressure loads and low flow rates (preferably >50% P_{Imax}), whereas RME will improve by training at high flow rates with low pressure loads (Caine & McConnell, 1998a; Romer & McConnell, 2003; Tzelepsis et al., 1994).

In the context of skeletal muscle training, endurance training is typically performed at loads of 40-60% of the individual's one repetition maximum (1 RM). This is particularly true for untrained individuals, whilst they are learning the technique (Kraemer & Ratamess, 2004).

Skeletal muscle strength may be improved by performing 1-3 sets of lifts at 60-100% 1 RM (1-12 repetitions) for intermediate and advanced training. Novice to intermediate exercisers are recommended to start resistance training at loads of 60–70% RM (~8-12 repetitions) while more advanced lifters should train at higher intensities of 80-100% RM (~1-6 repetitions) (Kraemer, Adams & Cafarelli, 2002; Heyward, 2006). Kraemer & Ratamess (2004) suggest that an optimal repetition range for increasing both muscle strength and inducing hypertrophy is between 70-80% 1 RM (~6-12 repetitions). However, training intensity is exercise-dependent, meaning that different exercises may have a different load and volume relative to their 1RM. Whilst skeletal muscle training has received a lot of attention and the effects of training load and volume have been well defined, to date, there is no research that has specifically explored the inter-relationships of P_{Imax}, training load, and RM for IMT.

The current recommendation for performing threshold loading IMT is 30 RM at ~50% P_{Imax}, which has been shown to provide a sufficient overload to increase P_{Imax} (Caine & McConnell, 1998a). As shown in Table 2.1, most IMT studies reported using training loads of approximately 50-60% P_{Imax} as indicated by performing training sets of 30 RM. Pressure threshold training load is seldom expressed as a percentage of P_{Imax} but rather as a repetition maximum. However, one recent study using IMT reported that subjects were able to perform > 75 repetitions at measured loads of ~50% P_{Imax} (Riganas et al., 2008) a longer duration than the 30 RM suggested by Caine & McConnell (1998a). Indeed a study by Klusiewicz et al. (2008) reported no changes in inspiratory muscle strength after 4 wk of IMT using a measured load of 50% P_{Imax}. However, once the participants increased the training intensity to 60% $P_{Imax,}$ they observed an increase in P_{Imax} (34 ± 19% improvement; $P < 0.05$). Although, previous research has looked at the specificity of IMT and has outlined the specific parameters to achieve increases in inspiratory muscle strength in healthy subjects (Caine & McConnell, 1998a;

McConnell & Romer, 2004b), no study has looked at the effects of magnitude and volume on acute responses to IMT sessions in athletic populations.

Like other skeletal muscles the respiratory muscles are susceptible to detraining (Baker, Davenport & Sapienza, 2005; Romer & McConnell, 2003). The principle of reversibility states that when detraining occurs the body will readjust in relation to the decrease in physiological demand and any benefits may be lost (Mujika & Padilla, 2001). A study to investigate the effects of detraining following 6 wk IMT compared the impact of reducing IMT frequency (Romer & McConnell, 2003). One group performed no IMT whilst the other group performed an IMT maintenance programme in which mouth pressures were reassessed at 9 and 18 wk post-training. A decrease in P_{Imax} was evident after 9 wk of detraining. Increases in RMS were sustained with the maintenance programme. These results are similar to the detraining effects of skeletal muscle and the ability to maintain strength gains when performing a lower rate of exercise as part of a maintenance phase (Kraemer et al., 2002).

Most RMT studies have looked at performing both respiratory training and/or the testing manoeuvres in an upright seated or standing posture. This is the recommended posture used for both clinical and research testing for respiratory pressures and training (Green et al., 2002). Some medical studies have shown significant improvements in pulmonary function and respiratory pressures in postures specific to patient conditions, such as the 'seated slump' position (sitting down chest forward with elbows on knees) often chosen by COPD patients to relieve sensations of breathlessness (Ogiwara & Miyachi, 2002; Kera & Marumaya, 2001a, 2001b, 2005). Thus, it may be possible to enhance the potential effectiveness of IMT by modifying the traditional IMT posture (upright standing or seated) to a more sport specific training posture.

Further investigations are needed to determine if performing IMT in sport specific postures, particularly rowing, would enhance the ergogenic effectiveness. Two specific rowing postures seem appropriate for consideration. First, the catch phase of the rowing stroke when the body is compressed making it more difficult for the diaphragm to expand. Secondly, in the finish position when the body is extended the abdominal muscles are in a position of co-contraction. Since the respiratory muscles are responsible for both trunk stabilisation and maintaining high minute ventilation during exercise, they are particularly susceptible to fatigue. If there is a loss in the ability to maintain ventilation at these points of the rowing stroke there may be an argument to train in these sport specific postures.

In addition to its influence upon inspiratory muscle function, the effects of IMT upon cardiovascular strain and perceptual responses during exercise have been studied (McConnell& Romer, 2004a; Sheel, 2002). More research is needed to determine the expected ergogenic benefits associated with RMT, in particular the specific benefit, if any, to rowing performance. Moreover, research is needed to define what aspect of RMT, either IMT or EMT or combined IMT/EMT provides the most benefit to exercise and sports performance in both trained and untrained individuals. If IMT or EMT provides an ergogenic benefit to enhance rowing performance in oarsmen, then more research is needed to explore how to optimise these training protocols and to incorporate this training into whole-body exercise training programmes.

2.1.3: EXPIRATORY MUSCLE TRAINING

The exercise performance benefits associated with EMT remains controversial (Mota, Guell, Barreiro, Solanes, Ramirez-Sarmiento, Orcozco-Levi, Casan, Gea & Sanchis, 2007; Smeltzer, Lavietes & Cook, 1996; Weiner, Magadle, Beckerman, Weiner & Berar-Yanay, 2003b; see

table 2.2). Studies have shown a decrease in EMF following EMT along with a change in pulmonary function, exercise tolerance and/or reductions in respiratory distress in healthy individuals (Baker et al., 2005; Sasaki, Kurosawa & Kohzuki, 2005; Suzuki, Sato & Okubo, 1995) and clinical populations (Mota et al., 2007; Smeltzer et al., 1996; Weiner et al., 2003b). In contrast, other EMT studies have showed either no significant change in P_{Emax} (Gosselink, Kovacs, Ketelaer, Carton & Decramer, 2000), or a decrease in EMF with no change in any physiological parameter or functional benefit (Weiner et al., 2003a).

Recent studies have examined the influence of EMT on pulmonary function and/or levels of exercise tolerance in healthy adults (see table 2.2). Generally, these studies have shown that EMT does not alter pulmonary function. A recent study by Sasaki (2007) investigated the effect of EMT on pulmonary function; they compared two EMT groups, one using a natural expiratory flow rate and the second performing at a faster expiratory flow rate, compared to a control group. Both training groups performed EMT for 15 min daily at 30% of P_{Emax} for 4 wk. Although P_{Emax} increased significantly post-EMT (~19% increase in both training groups), there was no difference in any of the pulmonary function values measured (FVC, FEV_1, peak flow rate and peak cough flow rate).

Table 2.2 Summary of EMT research in healthy adults.

Intervention	Exercise Test	Increase in P_{Emax}	References
15' 2 d·wk^{-1}, 4 wk @ 30% P_{Emax}	None	25%	Suzuki et al., 1995
15' 2 d·wk^{-1}, 2 wk @ 30% P_{Emax}	None	33%	Akiyoshi et al., 2001
4 sets x 6 b, 5 d·wk$^{-1,}$ 4 wk @ 75% P_{Emax}	None	84%	Sapienza et al., 2001
4 sets x 6 b, 5 d·wk$^{-1,}$ 2 wk @ 75% P_{Emax}	None	47%	Sapienza et al., 2002
15' 7 d·wk^{-1}, 2 wk @ 30% P_{Emax}	progressive exercise treadmill test	10%	Sasaki et al., 2005
5 sets x 5 b, 4-8 wk @ 75% P_{Emax}	None	41% and 51%, respectively	Baker et al., 2005
5 sets x 5 b, 5 wk @ 75% P_{Emax}	2 km rowing time trial	No	Miller, 2005
15' 7 d·wk^{-1}, 4 wk @ 30% P_{Emax} at a natural flow rate or fast flow rate	None	19.9% and 9.6%, respectively	Sasaki, 2007

Similar to some IMT studies, EMT has been shown to increase P_{Emax}, decrease the sensation of breathlessness, and reduce breathing frequency and $\dot{V}_E$ during exercise in healthy adults (see table 2.2). Studies investigating the influence of EMT on P_{Emax} show variable improvements in P_{Emax} ranging from as little as 10% (Sasaki, 2007; Sasaki et al., 2005) to as much as 51% (Baker et al., 2005). These differences may be attributable to the different training protocols (intensity, frequency and duration of training) employed.

Interestingly, one study found that EMT had a positive impact on P_{Imax}. Akiyoshi, Takahashi, Sugawara, Satake & Shioya (2001) studied the effects of 2 wk of EMT using a dead-space expiratory pressure device (Souffle, Kayaku Co. Ltd, Tokyo, Japan) on respiratory muscle pressures, noting a ~32% increase in both P_{Emax} and P_{Imax} following EMT. Given the recent evidence that expiratory loading also fatigues the inspiratory muscles (Taylor & Romer, 2009),

this is not perhaps surprising. Another study comparing the physiological effects of IMT and EMT in healthy adult subjects (Sasaki et al., 2005) showed similar results. Participants in this study performed either pressure threshold IMT (Threshold-IMT, HealthScan, New Jersey, USA) or EMT using Souffle for 15 min twice daily for 2 wk at 30% of respective maximal pressure. The subjects in the IMT group increased both P_{Imax} (16.1%; $p < 0.01$) and P_{Emax} (7.3%; $p < 0.05$); there seemed to be a similar trend in the EMT group with an increase in P_{Imax} (8.0%; $p > 0.05$) and P_{Emax} (10.3%; $p < 0.05$). These increases in opposing mouth pressures may be due to increases in diaphragmatic and abdominal muscle work associated with increases in V_T, lung hyperinflation and muscle tension (see Taylor & Romer, 2009). However, there is limited EMT research to support these findings.

The effect of EMT on parameters such as exercise performance, f_c, $\dot{V}O_2$, $[La^-]_B$ and RRE during exercise, remain unresolved. There is one unpublished study by Miller (2005) investigating the effects of 5 wk of pressure threshold EMT (Respiratory Power Trainer; Sapienza et al., 2002) on 2 km rowing time trial performance in university male oarsmen. The EMT required 5 breaths of 5 sets at 75% P_{Emax}. Following 5 wk of EMT, there were no significant differences in P_{Emax} or 2 km rowing time trial performance above that of the sham-training group. Both the training and sham-training group had considerable increases in P_{Emax} (32% and 22%, respectively; $P < 0.05$) and 2 km rowing performance (+4.6% and 3.1%, respectively; $P < 0.05$). The authors speculated that the lack of significance between groups may have been due to the elevated load for the sham training group (15% of P_{Emax}); thus both groups may have been training at sufficient loads to induce a performance effect.

Similar to IMT, it seems that perceptions of respiratory effort may be decreased following EMT (Sasaki et al., 2005; Suzuki et al., 1995). Sasaki et al. (2005) observed exercise-induced

increases in relative $\dot{V}O_2$ and a decrease in respiratory effort, but no difference in f_c. To date, there is limited research investigating the physiological changes associated with EMT in healthy subjects, thus more research is needed to determine the physiological effect of this type of training, if any.

Only one study has examined the effects of reversibility with EMT (Baker et al., 2005); the response of two different training groups was examined following 4 wk (Group 1) and 8 wk (Group 2) of EMT. Group 1 demonstrated a 41% increase in P_{Emax} compared to a 51% increase in Group 2. All participants were shown to detrain at the same rate at both 4 wk and 8 wk of detraining, regardless of the length of EMT performed. Clearly, if EMT is found to generate functionally meaningful improvements, more research is needed to examine different training and maintenance programmes.

2.1.4: CONCURRENT SPECIFIC INSPIRATORY AND EXPIRATORY MUSCLE TRAINING

The effects of concurrent IMT and EMT on exercise performance also remain inconclusive. In order to distinguish between VIH and a combined specific IMT and EMT, within this dissertation the terms concurrent or combined IMT/EMT is defined as the deliberate and isolated training of these specific muscle groups performed within the same training session. Recent studies examining the use of a combined IMT and EMT on competitive swimmers and trained cyclists have shown an improvement in dynamic pulmonary function (Butts, Swensen & Pfaff, 2005; Wells, Plyley, Thomas, Goodman & Duffin, 2005), but no change in exercise outcome variables compared to placebo groups. Similar to IMT, research has shown improvements to both P_{Imax} and P_{Emax} with no difference in $\dot{V}O_{2max}$ following concurrent IMT/EMT (Amonette & Dupler, 2002). The results of concurrent IMT/EMT studies still leave

the question of whether functional improvements are due to changes in the function of the inspiratory muscles, expiratory muscles or both.

Such a comparison has been made in patients with COPD. Weiner and colleagues (Weiner et al., 2003a) demonstrated that 12 wk of EMT significantly improved expiratory muscle strength and walking endurance, but did not decrease the sensation of dyspnoea compared to a control group. However, in a subsequent study comparing IMT, EMT, and a combined program of IMT/EMT, the same authors reported no additional benefit of EMT, or a combined program of IMT/EMT compared to the benefits of IMT alone (Weiner et al., 2003b).

2.2 EFFECTS OF RESPIRATORY MUSCLE TRAINING UPON PHYSIOLOGICAL MARKERS

It has been suggested that the mechanisms by which IMT improves exercise performance and functional capacity are twofold: (1) reduced effort sensation (Suzuki et al., 1995; Romer et al., 2002a, 2002b; Volianitis et al., 2001c; Williams et al., 2002); and (2) delayed onset of the respiratory muscle metaboreflex (McConnell & Lomax, 2006; Witt et al., 2007). Inspiratory muscle training (IMT) seems to decrease the perceptions of dyspnoea and limb discomfort, allowing participants to exercise for longer durations at higher exercise intensities. Moreover, a delay in the metaboreflex activation would maintain limb blood flow for longer and at higher exercise intensities.

Besides the attenuation or the delayed onset of RMF, there are a number of physiological variables that have been shown to change following RMT. Variables such as perceptions of dyspnoea, $\dot{V}O_2$, f_c, and $[La^-]_B$ during both submaximal and maximal exercise have been

examined, and shown to change following RMT. However, the extent and the mechanistic significance of these changes remain unresolved.

PERCEPTIONS OF RESPIRATORY EFFORT

Research investigating clinical populations with respiratory disease has observed significant improvements in the relief of dyspnoea (feeling out of breath) during both daily and laboratory physical activities following IMT (Beckerman, Magadle, Weiner, & Weiner, 2005; Weiner, Magadle, Berar-Yanay, Davidovitch & Weiner, 2000; Weiner et al., 2003a, 2003b; Weiner & McConnell, 2005; Weiner, Waizman, Magadie, Berar-Yanay & Pellad, 1999). Some, but not all, research in healthy adults has shown attenuation of respiratory effort or reduced sensation of breathlessness during exercise following IMT (Gething, Passfield & Davies, 2004b; Huang, Martin & Davenport, 2009; Redline, Gottfried & Altose, 1991; Romer et al., 2002a, 2002b, 2002c; Sasaki et al., 2005; Spengler, Roos, Laube & Boutellier, 1999; Suzuki et al., 1995).

During high intensity exercise, the sensation of breathlessness is one of the primary factors affecting the ability to maintain exercise intensity. A decrease in this sensation may allow individuals to sustain exercise for longer and/or at higher exercise intensities (Harms et al., 2000). The research relating to the impact of RMT upon decreases in the perception of respiratory effort during exercise, in particular time trial performance, remains equivocal. Decreases in perception of respiratory and peripheral effort during cycling have been reported following RMT (Boutellier et al., 1992; Chatham et al., 1999; Romer et al., 2002a; Suzuki et al., 1995; Volianitis et al., 2001b, 2001c); whereas other studies showed no significant change (Riganas et al., 2008; Suzuki, Yoskiike, Suzuki, Akahori, Haegawa & Okubo, 1993; Volianitis et al., 2001c; Williams et al., 2002). For example, Volianitis et al. (2001c) showed a significant

decrease in sensations of breathless in the training group following the 5 km time trial but not the 6MAO race.

Although RMT has been shown to increase RME and RMS as well as attenuate IMF, the influence these factors have on respiratory sensation is still poorly understood. A study performed by Suzuki et al. (1993) found that although IMT increased both P_{Imax} and diaphragmatic strength (by 30%) it failed to affect respiratory effort sensation as measured by the Borg CR-10 scale during treadmill exercise. Similar results were found by Williams et al. (2002) when they investigated the effect of IMT on endurance capacity in seven distance runners. They found an increase in both RMS and RME, but no significant difference in rating of perceived dyspnoea at steady state or at the end of the test following IMT. The effect of IMT upon respiratory sensation does not seem to be directly related to an increase in RMS or RME. Rather it has been suggested that with the attenuation of RMF, there may be a decrease or a delay in the recruitment of accessory respiratory muscles at higher exercise intensities (Johnson et al., 1993; Mador et al., 1993) potentially reducing the overall sensation of respiratory effort.

As fatigue of the respiratory muscles develops there is a progressive increase in the sense of effort required to maintain inspiratory pressure (Gandevia, Killian & Campbell, 1981). This increased respiratory muscle work may heighten the awareness of an increased perceptual or respiratory effort, which may lead to a decrease in exercise tolerance (Harms et al., 2000). A recent study by Huang et al. (2009) investigated the influence of 4 wk IMT on the detection of load magnitude on inspiratory airflow through a pressure threshold loading device. Following IMT, they observed a significant increase in inspiratory muscle strength and the load magnitude required to detect inspiratory resistive loads when breathing through a pressure threshold device. In general terms, this means that by increasing inspiratory muscle strength subjects

were able to compensate for higher respiratory loads; hence adjusting the perception of respiratory effort at that load.

OXYGEN UPTAKE AND VENTILATION

Many studies have examined the response of $\dot{V}O_{2max}$ and the oxygen cost of exercise post-RMT (Belman & Gaesser, 1988; Boutellier, 1998; Boutellier et al., 1992, 1998; Edwards & Cooke, 2004; Hanel & Secher, 1991; Inbar, Weiner, Azgad, Rotstein & Weinstein, 2000; Markov, Spengler, Knopfli-Lenzin, Stuessi & Boutellier, 2001; Romer et al., 2002a; Williams et al., 2002). It is apparent from these studies that O_2 uptake kinetics and $\dot{V}O_{2max}$ are not affected by RMT. For instance, Markov et al. (2001) demonstrated an increase in cycling endurance using both RMT (VIH) and aerobic endurance training, however only their aerobic endurance training group showed an increase in SV (17%) and reduced f_c (12%) (> 60% maximal aerobic power) following RMT. Thus, suggesting that the change in cycling endurance after RMT was not due to cardiovascular adaptations.

The effect of RMT on VO_{2max} and exercise economy has also been studied after RMT, with the rationale that RMT may decrease the $\dot{V}O_2$ required for a given $\dot{V}_E$, or reduce $\dot{V}_E$ and its associated O_2 cost. However, only a small group of studies have shown a statistically significant decrease in submaximal $\dot{V}O_2$ following RMT (Guenette et al., 2006; Haas & Haas, 1981; Sasaki et al., 2005). One Japanese study investigating the effects of IMT and EMT in normal subjects measured $\dot{V}O_2$ at submaximal intensities (Sasaki et al., 2005). Following training, both groups showed a significant increase in P_{Imax} and P_{Emax} and a significant decrease in $\dot{V}O_2$ at submaximal exercise intensities. Similarly, Hass & Haas (1981) showed a decrease in submaximal $\dot{V}O_2$ and f_c after 16d of VIH. These results suggest there may be a reduction in cardiovascular strain and/or metabolic demand at submaximal exercise intensities. More

research is required to assess cardiovascular responses to IMT at submaximal intensities to clarify these issues.

Generally there does not appear to be a systematic influence of RMT upon $\dot{V}_E$ or $\dot{V}O_2$ during exercise. However, the research is equivocal as to the influence of RMT upon exercise $\dot{V}_E$; with some studies showing no change in $\dot{V}_E$ (Fairbarn, Coutts, Pardy & McKenzie, 1991; Hanel & Secher, 1991; Inbar et al., 2000; Markov et al., 2001; Romer et al., 2002a; Sonnetti, Wetter¸ Pegelow & Dempsey, 2001; Stuessi, Spengler, Knopfli-Lenzin, Markov & Boutellier, 2001; Williams et al., 2002) some showing a decrease (Boutellier & Piwko, 1992; Boutellier et al., 1992) and some showing an increase (Boutellier, 1998; Holm et al., 2004; Spengler et al., 1999). The significance of changes in exercise $\dot{V}_E$ is controversial. Holm et al. (2004) found an increase in cycle performance during constant work rate exercise following RMT. The authors suggested that the associated increase in exercising $\dot{V}_E$ allowed participants to work at higher workloads without an increase in sensations of breathlessness. However, a reduction in exercise $\dot{V}_E$ has shown to reduce respiratory muscle blood flow increasing blood availability to the limb locomotor muscles (Harms et al., 1998, 2000).

HEART RATE

Some studies have shown no change in f_c following IMT (Guenette et al., 2006; Romer et al., 2002a; Williams et al., 2002), whereas some have shown a decrease in f_c at maximal and submaximal exercise intensities (Gething, Williams & Davies, 2004a; Haas & Haas, 1981; Swanson et al., 1998). Gething et al. (2004a) investigated the effects of IMT on f_c and RPE following 6 wk of flow resistive IMT at maximal or submaximal intensity. Both groups improved P_{Imax} compared to the control group, but the maximal group showed a -6 (± 9) beats min^{-1} (P = 0.02) decrease in f_c at submaximal exercise intensities following IMT. Similarly,

Swanson et al. (1998) showed a 6.6% decrease in f_c during T_{lim} exercise in cyclists after a 6 wk VIH intervention with an increase in T_{lim} cycling performance of 34.9%.

A plausible explanation for a reduction in f_c during exercise following IMT is that it reflects an absent or delayed activation of the inspiratory muscle metaboreflex. As stated previously, during fatiguing work there is a generalised increase in sympathetic outflow that stimulates both an increase in arterial blood pressure and f_c (Witt et al., 2007). It seems plausible that post-IMT, metaboreflex activation is delayed, thus reducing the level of sympathetic activation and f_c during exercise.

BLOOD LACTATE CONCENTRATION

Although RMT seems to have no significant effect on $\dot{V}O_{2max}$, many studies have shown a decrease in $[La^-]_B$ concentrations during and post-RMT (Boutellier & Piwko, 1992; Boutellier et al., 1992; Brown, Sharpe & Johnson, 2008; Johnson, Sharpe & McConnell, 2006; McConnell & Sharpe, 2005; Mickleborough et al., 2009; Romer et al., 2002b; Spengler et al., 1999). The precise mechanisms for this decrease in $[La^-]_B$ have yet to be determined, although it has been suggested that the decrease may be due to an increase in $[La^-]_B$ uptake by the respiratory muscles during exercise (Boutellier, 1998; Brown et al., 2008; Chiappa, Roseguini, Vieira, Alves, Tavares, Winkelmann, Ferlin, Stein & Ribiero, 2008), or possibly through decreased production of $[La^-]_B$ in better perfused limb muscles following RMT (Wetter & Dempsey, 2000). It has been noted that the increased $[La^-]_B$ uptake occurs in parallel with a decrease in $\dot{V}_E$ following RMT (Boutellier, 1998). It has therefore been argued that RMT enhances respiratory muscle efficiency, thus delaying RMF; this in turn depresses the rise in $\dot{V}_E$ at higher exercise intensities and muscles consume more $[La^-]_B$ (Boutellier, 1998). To date, there is no evidence to suggest that the mechanical efficiency of breathing improves after RMT.

However, this latter mechanism does not explain the decrease in $[La^-]_B$ production observed following 10 min hyperpnoea at rest following 6 wk of pressure threshold IMT (Brown et al., 2008). These findings demonstrated that the repiratory muscles increased $[La^-]_B$ independent of exercise and that this increase was attenuated following IMT. The authors highlighted that the decrease in $[La^-]_B$ following RMT may have been due to: an increase in monocarboxylate transport protein content which may have facilitated lactate shuttling (Brooks, Brown, Butz, Sicurello & Dubouchaud, 1999); an improved oxidative capacity of the respiratory muscles either by increasing type 1 muscle fibres (Ramirez-Sarmiento, Orozco-Levi, Guell, Barriero, Hernandez, Mota, Sangenis, Broquetas, Casan & Gea, 2002) or by an increase in the oxidative enzyme activity of the muscle (Costill, Coyle, Fink, Lesmes & Witzmann, 1979; Sale, MacDougall & Gardner, 1990), all of which may have attenuated, at least in part, the $[La^-]_B$ response following IMT (Brown et al., 2008).

No study has yet to confirm that there is a direct link between decreasing $[La^-]_B$ concentrations and improved exercise performance following RMT, although it seems likely that something related to the change in $[La^-]_B$ may be an underlying mechanism. Several authors have investigated different possibilities to identify a causal link between changes in post-RMT $[La^-]_B$ and exercise performance. McConnell & Sharpe (2005) showed a decrease in $[La^-]_B$ without a substantial change in maximal lactate steady state (MLSS) suggesting that RMT induced increases in exercise tolerance. Since there was no change in MLSS they concluded that the mechanism for a decrease in $[La^-]_B$ was not related to an improvement in the lactate threshold.

2.3: CONCLUSION

The conflicting results of studies investigating RMT have caused much confusion in respect of its effectiveness as an ergogenic aid to sport performance. These contradictory results may be

due to the variety of study designs, some with inappropriate outcome measures. The message to the sporting community regarding RMT is therefore unclear, and worse, unhelpful. However, as new research is published, and more information about the mechanistic underpinnings of RMT becomes known, we are better equipped to understand why controversy exists. This being the case, we are also better equipped to design and undertake studies that clarify the message regarding RMT and sports performance.

Over the past decade, research has been able to identify the specific physiological outcomes affected by RMT, these include: P_{Imax}, IMF, MVV, T_{lim} at MVV, MSVC, and a decrease in sensation of breathlessness. Similarly, other indices (e.g.,$\dot{V}O_{2max,}$ exercise economy) are not affected by RMT. However, there are still many questions that remain unresolved, such as the impact of EMT as an ergogenic aid, as well as the physiological effects of IMT on f_c and $[La^-]_{B,}$ during submaximal exercise. Finally, if RMT is to be recommended as an ergogenic aid, there are unresolved issues relating to practical advice such as protocol prescription that require further study.

2.3.1: PURPOSE

Inspiratory muscle training (IMT) has the potential to benefit competitive oarsmen. This dissertation has identified two specific subgroups of well-trained oarsmen (elite heavyweight men and club-level) in which IMT may have an impact on rowing performance. The results of this research may also prove beneficial to other athletic populations that are seeking information about the likely functional benefits and alternative training protocols for IMT and EMT. Hence, the aim of this research will be to outline the functional benefits provided by IMT and EMT for improving time trial performance in competitive oarsmen.

This dissertation has been divided into two main sections: three laboratory based studies and one applied training intervention study. The laboratory based studies (Section one) addressed the following:

1) whether IMT, EMT or combined IMT/EMT programme provided differing ergogenic benefits to club-level oarsmen.

2) whether rowing induced any postural impairment to respiratory muscle pressure and/or flow generating capacities that merited further investigation into posture-specific IMT protocols.

3) characterisation of the acute physiological response to various pressure threshold loads.

The applied training intervention study (Section two) was conducted at the invitation of the British International Rowing Organisation to determine:

1) the ergogenic effectiveness of IMT upon rowing performance in the elite ('World-Class' athletes) Great Britain heavyweight men's squad.

CHAPTER THREE

GENERAL METHODS

The following chapter provides detailed information regarding the general equipment and procedures used throughout this dissertation. Additional methodological information relevant to each particular study is contained within those chapters.

3.1: PRE-TEST DATA

3.1.1 PARTICIPANT INFORMATION

Prior to the start of each study, all participants were required to complete a written informed consent (Appendix A-1) and a general health questionnaire (Appendix A-2). Participants were removed from the study if they reported illness or respiratory infections prior to the start or throughout the course of the studies.

For each study, participants were provided with a detailed description of the testing procedures, the risks involved, the benefits of taking part in the testing and assured confidentiality of their data. All participants were familiarised with the testing procedures and provided proper instruction and detailed objectives for each testing session. Participants were requested to restrain from performing strenuous or maximal exercise 1-2 days prior to the testing session. On testing days, participants were requested to maintain a normal diet, avoid alcohol and caffeine intake and not take any drug that may affect the outcome of their performance or change the results of the study. We specifically asked participants not to ingest caffeine at least 24 hours prior to the testing session, as it has been shown to significantly reduce effort perception and muscular fatigue, reduce the catabolism of glycogen, increase fat utilisation (Astrand, Rodahl, Dahl & Stromme, 2003; Spriet, MacLean, Dyck, Hultman, Cederblad & Graham, 1992), improve respiratory muscle function (Supinski, Leven & Kelsen, 1986) and improve exercise performance (Graham, 2001).

3.1.2 LABORATORY ENVIRONMENT

Testing sessions performed in the laboratory were not maintained to standardised conditions; however, where appropriate, environmental conditions (temperature, humidity and barometric measurements) were measured and recorded for equipment calibration (tests using Oxycon Pro). Field testing sessions, including inspiratory and expiratory muscle training (IMT and EMT, respectively) sessions, were performed outside the laboratory in uncontrolled conditions. All testing sessions were performed at sea-level.

3.2: EQUIPMENT AND PROCEDURES

3.2.1: ANTHROPOMETRIC MEASUREMENTS

Anthropometric measurements of stature and body mass were assessed for individual comparisons and to determine predictive values of lung function and oxygen uptake ($\dot{V}O_2$). Freestanding stature was measured to the nearest cm with a fixed (Harpenden Stadiometer, Birmingham, UK; Seca Telescopic Height Rod, Seca Ltd., Birmingham, UK) or portable (Seca Stadiometer, Seca Ltd., Birmingham, UK) stadiometer. Measurements were made with participants standing barefoot, heels together with arms hanging naturally by their sides while looking straight ahead (Eston & Reilly, 2001).

Body mass in lightweight clothing and barefoot was measured to the nearest 0.1 kg using either a calibrated electronic scale (Tanita Body Composition Analyser/Scale, Tanita UK Ltd., Yiewsley, UK) or balance beam scale (Seca Classic Mechanical Column Scale 710, Seca Ltd., Birmingham, UK) (Eston & Reilly, 2001). During field tests, when stadiometers and weight scales were not accessible, information provided by the athlete or coach was applied.

3.2.2 PULMONARY FUNCTION TESTS

RESTING SPIROMETRY

Resting pulmonary measurements, such as volumes and flows, provide useful screening information about overall lung health and respiratory muscle performance (ATS/ERS, 2005). Spirometric measurements were assessed at rest by performing maximal flow volume loops (MFVLs). This manoeuvre is used to detect small airway disease as the shape of the loops is indicative of obstructive or restrictive airways (ATS/ERS, 2002). The loops also provide a visual display of whether the flows are appropriate for the particular lung volume as they include both a maximal inspiratory and expiratory flow from one breath graphed against volume changes. The following respiratory measures were evaluated: peak inspiratory flow (PIF), peak expiratory flow (PEF), forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1). As defined by the ATS/ERS (2005), PIF is the maximum flow of inspired air achieved from a maximum inspiration starting at residual volume, whereas PEF is 'the highest flow achieved from a maximum forced expiratory manoeuvre started without hesitation from a position of maximal lung inflation.' FVC is 'the maximal volume of air exhaled with maximally forced effort from a maximal expiration'; whereas, FEV_1 is 'the maximal amount of air exhaled in the first second of a forced expiration starting from a full inspiration'. Spirometric measurements were assessed using a portable hand held spirometer (MicroLoop Spirometer, Micro Medical Ltd., Kent, UK; see fig 3.1). Measurements were made according to the recommendations of the ATS/ERS for pulmonary function tests (ATS/ERS, 2002; Quanjer et al., 1993).

Figure 3.1 Microloop Spirometer (Micro Medical Ltd., Kent, UK).

The MFVL manoeuvre has four phases: 1) maximal inspiration prior to the start, 2) a forceful expiration, 3) continued exhalation until the lungs are empty and 4) forceful inspiration until the lungs are full. Participants were instructed to inspire fully to total lung capacity (TLC) and to hold their breath for < 1 second (s). Then place the flanged mouthpiece in their mouth and close their lips fully to create a tight seal. The participant was instructed to expire maximally and forcefully to residual volume (RV) (~6 s for a healthy lung); followed by a sharp forceful inspiration to TLC. Nose clips were worn during the manoeuvre to occlude the nares. Participants were given careful instruction to perform the expiratory and inspiratory loop as a single manoeuvre and were verbally coached throughout the manoeuvre.

A minimum of 3 technically acceptable MFVL attempts (maximum of 8 attempts) were performed. The criteria for acceptable repeatability for spirometry measurements were: a minimum of 6 s on exhalation, free from artefact such as cough, leak, obstructed mouthpiece, hesitation or extra breath during the manoeuvre, and a good start. The attempt was accepted

when measurements with the largest values of FVC and FEV_1 were within 0.150 L. The attempt with the largest sum of FVC and FEV_1 was recorded and utilised to determine other pulmonary indices. Peak inspiratory and expiratory flow (PIF and PEF, respectively) were expressed in $L \cdot sec^{-1}$, whilst FVC and FEV_1 are expressed in litres (L) at body temperature and ambient pressure saturated (BTPS) with water vapour (ATS/ERS, 2005). A sample graph of a MFVL is presented in Figure 3.2 representing flow rate ($L \cdot sec^{-1}$) against lung volume (L).

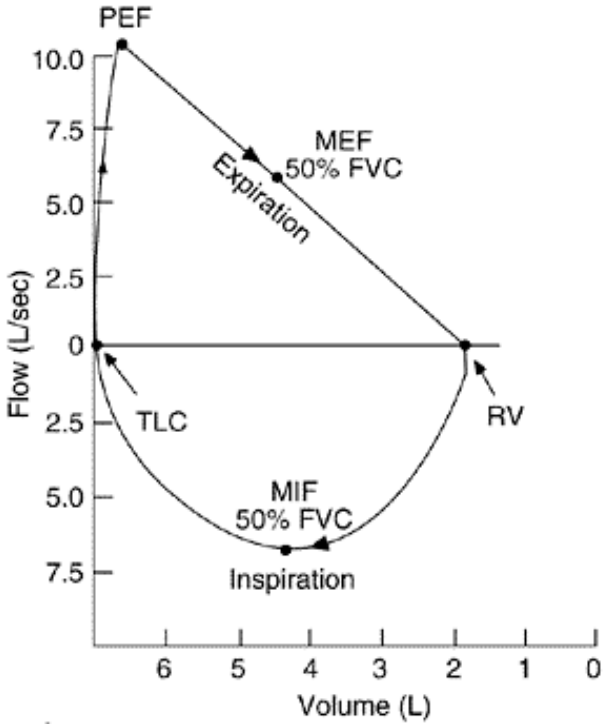


Figure 3.2 Maximum flow volume loop. TLC, total lung capacity; PEF, peak expiratory flow; MEF, mean expiratory flow; RV, residual volume; MIF, mean inspiratory flow; FVC, forced vital capacity.

RESPIRATORY MUSCLE STRENGTH

Maximal static inspiratory and expiratory mouth pressures (P_{Imax} and P_{Emax}, respectively) were measured as a surrogate of RMS (Green et al., 2002). Both measurements were made using a portable hand held mouth pressure meter (Micro MPM, Micro Medical Ltd., Kent, United Kingdom; Precision Medical MPM, UK; see fig. 3.3). Mouth pressure meters have been shown to be an accurate and reliable method of measuring respiratory mouth pressures in healthy, motivated subjects (Black & Hyatt, 1969; Hamnegard et al., 1994).

Maximal inspiratory pressure (P_{Imax}) was initiated at RV; the participants were required to inhale fully with a sharp, forceful effort maintained for a minimum of ~2 s. For $P_{Emax,}$ participants were asked to inhale fully (to TLC) then exhale forcibly and maximally. The mouth pressure meter incorporated a 1 millimetre (mm) leak to prevent glottic closure during the P_{Imax} manoeuvre and to reduce buccal muscle contribution during the P_{Emax} manoeuvre (Black & Hyatt, 1969). All measurements were performed using a flanged mouthpiece and were performed in an upright standing position, unless otherwise stated. Then place the flanged mouthpiece in their mouth and close their lips fully to create a tight seal. Nose clips were worn to occlude the nares while performing the measurement. Participants were given careful instruction prior to the test and were verbally coached throughout the manoeuvre. A minimum of five satisfactory inspiratory and/or expiratory efforts were conducted and the highest of three measurements with less than 5% variability or within 5 cm H_2O difference was defined as maximal (Green et al., 2002). A minimum of a 1 min rest interval was observed between each successive measurement to ensure that the respiratory muscles were not fatigued.

A *B*

Figure 3.3 Mouth pressure metres. *A*, Micro Medical MPM; *B*, Precision Medical MPM.

RESPIRATORY MUSCLE WARM-UP

Research has shown that there may be significant variability when performing repeated measurements to assess respiratory mouth pressures (Astrand et al., 2003; Fiz, Montserrat, Picado, Plaza & Agusti-Vidal, 1989; Wen, Woon & Keens, 1997). This variability may be due to day-to-day fluctuations (Astrand et al., 2003) or a 'learning effect' during repeated measures (Fiz et al., 1989; Wen et al., 1997) which may affect the reproducibility of the testing measures. A study conducted by Volianitis et al. (2001c) assessed whether performing a specific respiratory warm-up prior to performing maximal inspiratory mouth pressure testing would enhance the repeatability of the measurements. They found that performing a specific respiratory muscle warm-up using a pressure threshold training device set at an intensity of ~40% P_{Imax} minimises the 'learning effect' and variability when performing repeated inspiratory mouth pressure measurements. Therefore, prior to performing baseline respiratory

mouth pressure measurements, participants were instructed to perform 2 sets of 40 inspiratory breaths, against an intensity ~40% $P_{Imax,}$ using a pressure threshold inspiratory muscle trainer (POWERbreathe® Inspiratory Muscle Trainer, Gaiam Ltd., Southam, UK).

RESPIRATORY MUSCLE FATIGUE

Respiratory muscle fatigue (RMF) was assessed using volitional maximal respiratory mouth pressures. Measures of inspiratory and expiratory muscle fatigue (IMF and EMF, respectively) using P_{Imax} or P_{Emax} have been shown to be reliable in highly motivated participants (Supinski et al., 2002). Inspiratory muscle fatigue (IMF) was determined by calculating the difference between the pre-exercise P_{Imax} (PreEx–P_{Imax}) and post-exercise P_{Imax} (PostEx–P_{Imax}) measurements. Post-exercise measurements were scheduled at 2 min of termination following the exercise test session. Due to individual variations in recovery from maximal exercise performance the exact timing for testing was within 2-3 min post exercise (see methodology within studies for exact timing of post-exercise measurements). All post-exercise measurements, including the exact time of measurement post-exercise termination, were recorded to ensure the timing of post-exercise measurements was kept consistent at subsequent testing sessions. All measures of RMF were presented as the percentage change from baseline.

3.2.3: RESPIRATORY MUSCLE TRAINING

INSPIRATORY MUSCLE TRAINING

Inspiratory muscle training (IMT) was performed using a commercially available pressure threshold spring loaded inspiratory muscle trainer (POWERbreathe®, Gaiam Ltd., Southam, U.K.). Unlike other respiratory muscle training (RMT) devices, the POWERbreathe® is a lightweight, portable handheld device, which allows the user to specifically train the inspiratory

muscles by increasing resistance as needed. A detailed illustration of the trainer is shown in Figure 3.4.

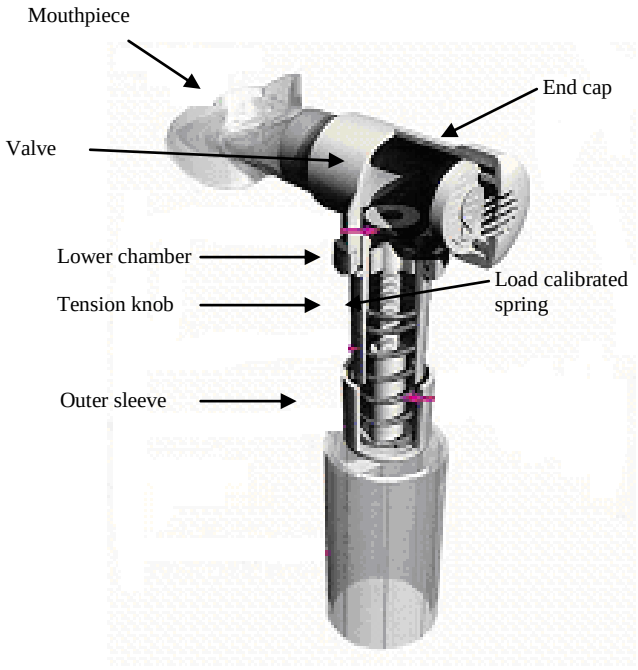


Figure 3.4 Schematic of the POWERbreathe® Inspiratory Muscle Trainer. Picture taken from www.powerbreathe.com.

During the IMT studies, all participants were familiarised with the POWERbreathe® device at the baseline testing session. As illustrated above, the tension knob was used to adjust the load calibrated spring to increase and/or decrease training resistance. Training was performed by placing the mouthpiece into the mouth with teeth on the inner grip and lips closed tightly around the outer shield. Participants were instructed to inhale maximally and fully against the resistance and then exhale slowly to empty.

Unless otherwise stated, the IMT sessions required participants to perform one set of 30 maximal inspiratory efforts twice daily for a period of 4-11 wk depending on the study. Each

effort required the participant to inspire against a threshold load equivalent to 30 repetitions maximum (RM). This protocol has been shown to be effective for IMT in previous studies (Romer & McConnell, 2003; Romer et al., 2002a, 2002c; Volianitis et al., 2001c).

RESPIRATORY MUSCLE TRAINING DIARY

During the training studies, all participants were requested to provide a detailed physical activity-training programme and complete a RMT diary in order to monitor training adherence. The diary was used to monitor changes in RMT volume and intensity. Respiratory muscle training (RMT) adherence was presented as a percentage of their overall training programme. A copy of the RMT diary supplied to all participants is shown in Appendix A-3.

3.2.4: EXERCISE TESTS

MAXIMAL ROWING EXERCISE TEST

The influence of IMT or EMT on exercise performance was assessed by comparing 2 km rowing ergometer time trial performance or six min all out rowing (6MAO) effort pre and post-IMT. The test distance of 2 km, or the distance covered within six min, is commonly used to monitor rowing training and performance as it is the standard distance for on-water elite racing. Although, racing on-water requires different skills than the rowing ergometer, it has been shown to reflect similar biochemical and metabolic demands (Maestu, Jurimae & Jurimae, 2005). This distance on a wind braked rowing ergometer (Concept II, Nottingham, UK) has also been shown to have a high reliability in well–trained rowers (Schabort, Hawley, Hopkins & Blum, 1999).

Rowing ergometer performance time was recorded and presented as the total time to perform the designated distance in minutes, seconds, deciseconds (m:s.ds). The electronic monitoring

device standard on the rowing ergometer was set to the designated 2 km distance prior to the start of the test. During the 6MAO effort, the monitor was set to six min and the total distance achieved, mean power output, and split time was recorded. The drag factor was set to 138 (damper setting 4) to ensure that all participants were rowing at the same resistance settings when performing at different locations (Ingham, Whyte, Jones & Nevill, 2002).

3.2.5: PHYSIOLOGICAL PARAMETERS

OXYGEN UPTAKE

An online gas analyser system (Oxycon, Jaeger-Toennies, Hoechberg, Germany) was used to measure peak oxygen uptake ($\dot{V}O_2$) during each stage of the rowing 'step-test'. All measurements were made according to the BASES Physiological Testing guidelines (Winter, Jones, Davison, Bromley & Mercer, 2006). The system was calibrated before each exercise testing session. Although ambient conditions are automatically calculated in the system, manual measurements of laboratory conditions were monitored simultaneously to ensure system validity.

CALIBRATION OF THE ONLINE GAS ANALYSIS SYSTEM

Standard calibration of the Oxycon Pro® on-line gas analysis system (Jaeger-Toennies, Hoechberg, Germany; fig. 3.5*A*) was divided into three parts: ambient conditions, volume calibration, and gas analyser calibration. Ambient conditions were checked manually using a mercury barometer, and hygrometer; values were manually entered into the system. For manual volume calibration, a 3 litre (L) syringe (Jaeger Calibration Pump, Hoechberg, Germany) was attached to the Triple V mouthpiece (fig. 3.5*B*) via a plastic tube. Multiple pumps of the piston within the nearest 1% of the 3 L were sufficient for calibration while simultaneously calibrating flow rates.

Next, a gas analyser calibration was conducted by connecting the system to a mixed standard gas cylinder (15.0% O_2 and 5.0% CO_2). The automatic calibration performed a systematic check on the analysis of ambient air, zero adjustment, gains settings, zero checkpoints, and then calculated the calibration factors. A warning signal provided notification if zero or gain values were outside the normal limits and the calibration procedure was repeated until acceptable values were obtained. Prior to the start of participant testing, personal information (stature, body mass, age and gender) was entered into the computer to determine predictive values.

A

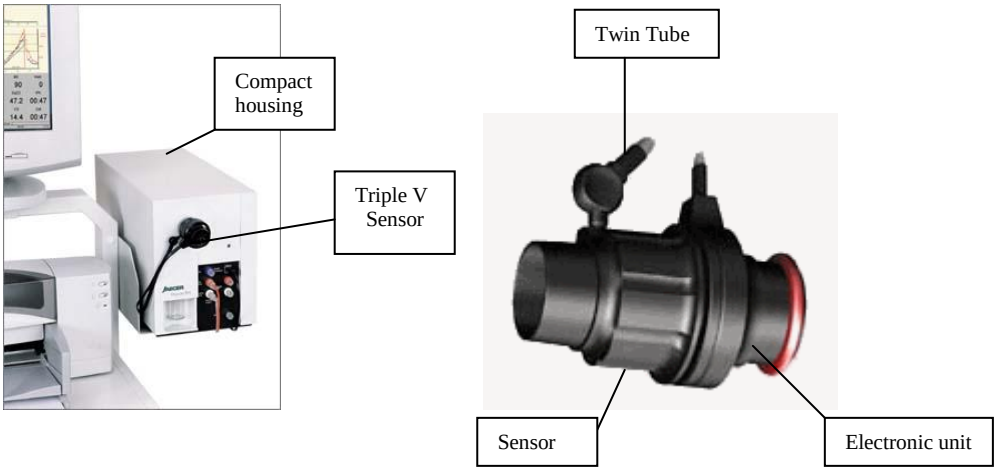


Figure 3.5 *A*, Oxycon Pro® Online Gas Analysis System. *B*, Triple V-sensor used to analyse 'breath-by-breath' gas analysis and spirometry.

HEART RATE

Heart rate (f_c) was used to continuously monitor exercise intensity during the incremental rowing 'step test' and to determine the cardiovascular fitness effects of RMT. The heart rate monitor used a short-range telemetry system (Polar Sport Tester, Polar Electro Oy, Finland); the validity, reliability and functionality of this system have been well documented (Laukkanen & Virtanen, 1998; Thivierge & Leger, 1988, 1989; Wajciechowski, Gayle, Andrews &

Dintiman, 1991). An elastic electrode belt was slightly moistened with water and strapped around the chest, directly under the pectoralis muscles. A receiver attached to the rowing ergometer provided a visual display of f_c. Peak f_c during the last 30 s of each exercise stage was recorded through visual inspection of the receiver and presented in beats per minute (bpm).

BLOOD LACTATE CONCENTRATION

Blood lactate concentration ($[La^-]_B$) measurements were taken using a portable lactate analyser, this system uses electrochemical-enzymatic analysis to measure $[La^-]_B$ (Lactate Pro Blood Lactate Test Meter, Arkray, Inc, Japan; see fig. 3.6*A*). Previous research has been demonstrated this device to be valid method of measuring $[La^-]_B$ (Medbo, Mamen, Holt Olsen & Evertsen, 2000; Pyne, Martin & Logan, 2000). Table 4.5 provides absolute and ratio limits of agreement for between-day reliability testing for $[La^-]_B$ measurements. All $[La^-]_B$ sampling followed the recommendations according to the BASES Physiological Testing guidelines for safe blood handling and disposal (Winter et al., 2006).

The lactate analyser was calibrated using a Lactate Pro Check Strip, followed by a Calibration Strip for each particular box of test strips. Prior to taking a blood sample, the puncture site was cleaned with an alcohol wipe and allowed to dry. A small prick was initiated using an automatic lancing device (Accu-chek Softclix Pro, Roche Diagnostics, UK) on the participant's earlobe and then wiped with a tissue to remove blood with perspiration or alcohol residue. Pressure was applied around the surrounding site to obtain a drop of blood for sampling. A Test Strip was prepared in the lactate analyser, in which a 5µl blood sample was taken (fig. 3.6*B*). A beep sounds when a complete sample was taken and the analysis was completed in 60 s. All measurements were expressed in millimoles per litre ($mmol \cdot L^{-1}$).

A *B*

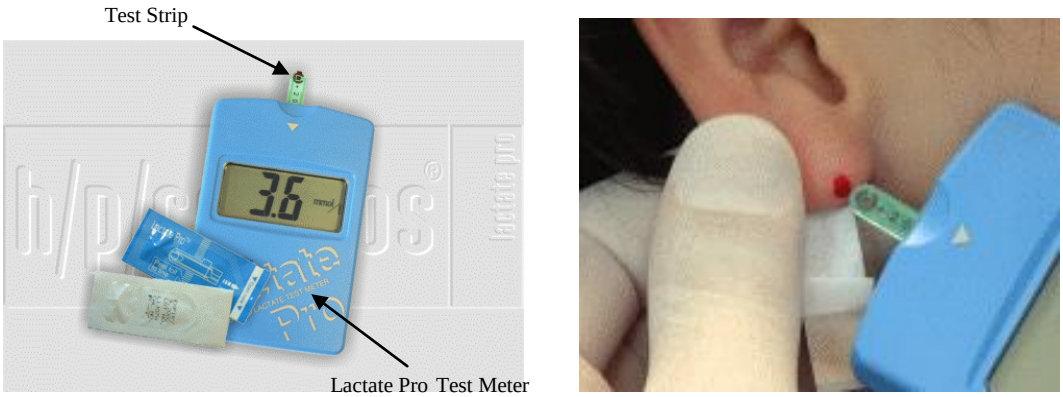


Figure 3.6 *A*, Lactate Pro Blood Lactate Test Meter. *B*, blood-sampling technique using the Lactate Pro.

3.2.6: STATISTICAL ANALYSIS

Two methods of analyses were used to determine reproducibility of outcome variables. Ratio limits of agreement were calculated to determine the within-subject variation according to the procedures of Bland and Altman (1986). These were used to estimate the effect of the measurement upon statistical power. The ratio limits of agreements allows for a differentiation between systematic bias (e.g., general learning effects) and random error (inherent testing errors), thus providing a reference range to determine whether a change has taken place between pairs of measurements. This measure of agreement is based on calculating the standard deviation of the mean difference; if the mean difference is anything other than 0 this suggests that a systematic bias exists between the two methods.

Confidence intervals were used to estimate the reliability of a measurement and /or changes due to an intervention. Any change within the 95% confidence intervals was considered a normal variation; any change above or below this range suggested that a real change had occurred. The confidence limits provide a lower and upper value (or boundary), defining the range of the

confidence interval. Thus, the 95% ratio limits of agreements were used to estimate sample sizes for a range of treatment effects for a repeated-measures design, given a statistical power of 0.9 and an alpha level of 0.05. Logarithmic transformation of differences was performed to create dimensionless quantities to allow for a comparison across parameters with different units of measure. These estimates were performed using a bespoke Excel spreadsheet (Romer, personal communication), based upon the calculations of Zar (1998).

Where appropriate, a repeated measures analysis of variance (ANOVA) was used to determine physiological and/or performance changes over time. Violations of the assumption of sphericity were measured using Mauchly's sphericity test and corrected using the Greenhouse-Geisser adjustment. Planned pairwise comparisons were made to analyse significant interaction effects using the Bonferroni adjustment. Probability values $\leq$ 0.05 were considered significant. Statistical and mean data were calculated using the statistical software SPSS V16.0 for Windows (SPSS Inc, Chicago, IL, USA). All results are expressed in mean ± standard deviation (SD) unless otherwise stated. A detailed description of the statistical analyses used can be found in the methods section for each study.

SECTION 1

LABORATORY BASED STUDIES

CHAPTER FOUR

DIFFERENTIATING THE INFLUENCE OF INSPIRATORY AND EXPIRATORY MUSCLE TRAINING UPON ROWING PERFORMANCE IN CLUB-LEVEL OARSMEN

This chapter was presented at the 2006 ACSM Annual Conference and published in collaboration with my dissertation supervisor (see Appendix A-5).

4.1: INTRODUCTION

Respiratory muscle training (RMT) yields improvements in exercise performance in both healthy young adults (see table 2.1) and in patients with chronic obstructive pulmonary disease (COPD) (McConnell & Romer, 2004a; Weiner & McConnell, 2005). In studies of healthy young people, two different forms of respiratory training have been employed: 1) voluntary isocapnic hyperpnoea (VIH) and, 2) inspiratory resistive loading (McConnell & Romer, 2004b; Sheel, 2002). The former is an endurance training approach that involves both the inspiratory and expiratory muscles, whilst the latter employs resistance training principles and is confined primarily to the inspiratory muscles. Both techniques apparently result in a similar pattern of physiological changes post-RMT (see McConnell & Romer, 2004b), which suggests that the underlying mechanism(s) for improved performance following RMT is independent of the training stimulus employed. However, it is unclear whether the addition of the expiratory muscle training (EMT), as occurs during VIH, provides any additional benefit to inspiratory muscle training (IMT) alone.

A potential mechanism for the improved exercise performance that follows RMT centres around the notion that fatiguing respiratory muscles elicit a sympathetically mediated reflex vasoconstriction in locomotor muscles (Harms et al., 2000), thereby limiting limb blood flow, with obvious repercussions for performance. Since IMT has been shown to diminish exercise-induced inspiratory muscle fatigue (IMF) (Romer et al., 2002b; Volianitis et al., 2001c), it has been suggested that IMT delays or attenuates this respiratory muscle 'metaboreflex' (McConnell & Lomax, 2006; Witt et al., 2007). Thus, IMT may act to preserve limb blood flow, and improve performance by minimising IMF. The existence of this metaboreflex has been demonstrated in both inspiratory and expiratory muscles (Derchak et al., 2002; Sheel et al., 2001). If a mechanism based on the ablation of the respiratory muscle metaboreflex makes

an important contribution to the improvements in exercise performance that follow RMT, we would predict that both IMT and EMT would improve performance. Further, we would predict that a combination of specific IMT and EMT would provide superior benefits compared with IMT or EMT alone.

To date, there have been no published studies examining the influence of isolated EMT upon exercise performance in healthy adults, or an athletic population. Suzuki et al. (1993, 1995) conducted two separate experiments investigating the effects of EMT and IMT on respiratory sensation during exercise. They observed a decrease in respiratory sensation during exercise after 4 wk EMT, showing an increase of 25% in $P_{Emax.}$ Although they concluded that IMT increased diaphragmatic strength by 30%, there was no significant effect on respiratory effort sensation. These latter observations contradict with those of Volianitis et al. (2001c) and Romer et al. (2002a, 2002c) who noted reductions in respiratory and/or whole body effort sensations after IMT. Similarly, other researchers have noted a reduction in respiratory effort sensation during loaded breathing after IMT (Gandevia et al., 1981; Supinski, Clary, Bark & Kelsen, 1987; Williams et al., 2002).

Sasaki et al. (2005) conducted an investigation comparing the effects of IMT and EMT in healthy subjects. The study consisted of 3 groups, the IMT, EMT and control group; the respective group performed either 15 min of IMT or EMT at 30% maximum pressure for 2 wk. The IMT group showed an increase in both P_{Imax} and P_{Emax} (16.2% and 7.3%, respectively), the EMT group only showed a significant increase in P_{Emax} (10.3%); no change in the control group. Exercise-induced increases in heart rate (f_c), $\dot{V}O_2$/kg and ratings of perceived exertion (RPE) decreased following training in the IMT group, only $\dot{V}O_2$/kg and RPE decreased in the EMT group. Unfortunately, the study did not measure exercise performance; instead the test

protocol was a progressive incremental treadmill test that measured peak values at different stages until the subject reached 85% of maximal $f_{c.}$ One EMT study, did measure the effects of specific EMT on rowing performance in university oarsmen (Miller, 2005). However, the findings remain inconclusive as both the EMT group (training intensity at 75% P_{Emax}) and the sham-training group (training intensity at 15% P_{Emax}) significantly increased P_{Emax} and rowing performance following training; no significant differences were evident between groups. Thus, the impact of EMT on exercise performance remains unclear.

Recently, a few papers have investigated the benefits of combined IMT and EMT programmes on sport performance. Wells et al. (2005) studied the effects of a 12 wk combined IMT/EMT programme in adolescent competitive swimmers in which they showed a significant improvement in FEV_1 in the training group, however both the sham and the experimental group showed similar improvements in dynamic pulmonary functional variables and swimming critical speed. Interestingly, these results are supported by a recent abstract investigating the effects of a combined programme on maximal, submaximal and 20 km time trial cycling performance (Butts et al., 2005) in healthy, trained adult cyclists. After 6 wk of RMT, the experimental group showed a 131% improvement in respiratory muscle endurance (RME) and a lowered rating of perceived exertion (RPE) during the 20 km time trial (9.7%). Although the combined programme enhanced respiratory muscle performance and decreased dyspnoea they found no significant improvement in exercise performance compared to the control group. These findings are controversial in comparison to the benefits shown from IMT on exercise performance.

Hence, the purpose of the present study was to compare the effect of 4 wk of IMT or EMT upon club-level oarsmen and to investigate changes in rowing performance, as well as the

effect of a subsequent 6 wk period of combined IMT/EMT. The hypotheses for this study included the following outcomes: 1) an improvement in the maximal strength of the inspiratory muscles in the IMT group, and expiratory muscles in the EMT group, with further increases in respiratory muscle strength following combined IMT/EMT, 2) an improvement in 6 minute all-out (6MAO) effort performance in both the IMT and EMT groups, 3) and an attenuation of exercise induced respiratory muscle fatigue (RMF) in both groups.

4.2: METHODOLOGY

4.2.1: PARTICIPANTS

Seventeen competitive male rowers gave written informed consent to participate in the study. All participants were recruited from a local rowing club, and whilst they were at different stages of their rowing careers (competing > 6 months) they trained under the same coach, and participated in an identical cardiovascular and resistance-training program during the period of the study. All participants were naive to the study aims/outcomes and volunteered to participate in agreement with the researcher and the team rowing coach. Although participants were aware they were using different respiratory muscle training devices; they were not told about differences in the devices, the specific training protocols or any physiological benefits that respiratory muscle training could potentially offer. The coach instructed the participants that the physiological testing was part of a developmental rowing training programme. Table 4.1 contains the descriptive characteristics of the participants.

Prior to the start of the exercise testing sessions all participants were required to complete a written informed consent form approved by the School Ethics Committee. A copy of the informed consent and health questionnaire can be found in Appendix A-1 and A-2. All participants were non-smokers and free from any upper respiratory tract infections, though two

participants (1 IMT and 1 EMT subject) were previously diagnosed with asthma. Only the IMT subject's diagnosis of asthma was supported by evidence of a mild obstruction at rest (FEV_1%FVC value of 77.5% {< 80% predicted}). Neither participant regularly suffered from episodes of bronchoconstriction and stated that symptoms were only brought on by upper respiratory tract infections and specific allergies. These participants were required to have their inhaler available during testing; however no participants needed to use their inhaler at any time over the course of the testing.

Participants reported to the Department of Sport Sciences, Physiology Laboratory, at Brunel University, Uxbridge for all testing. All participants were requested to maintain a normal diet for a few days prior to testing and to refrain from vigorous exercise and alcohol two days preceding the testing and to avoid caffeinated beverages the day of the test.

4.2.2: GENERAL DESIGN

Participants made at least three visits to the laboratory, but eight made four visits. The additional visit by these participants was at Baseline 1, and was used to assess the within-subject variation (reliability) of the testing procedures. There was a 4 wk interval between testing at Baseline 1 and Baseline 2. Following the baseline visit(s), participants were ranked according to their 2 km rowing ergometer performance time and then divided into two groups. One group undertook 4 wk of IMT (n = 10), whilst the other undertook 4 wk of EMT (n = 7). Initially, both groups had 10 subjects, however due to practicality reasons (i.e. travel across London, evening testing) a few of the participants removed themselves from the study. Immediately at the end of this phase, the first post-intervention visit took place (Post-intervention 1). After the post-intervention 1, two of the IMT group stopped training due to illness and difficulty with maintaining their rowing training program due to personal reasons. The remaining participants (n = 15) undertook a 6 wk

period of combined IMT (n = 8) and EMT (n = 7). At the end of this combined phase of training a second post-intervention test took place (Post- intervention 2). This phase was extended to 6 wk due to events in their rowing program (fig. 4.1). All participants were kept naive to the study aims.

Ideally, the study design would have consisted of four groups all using the same training device: an IMT group, an EMT group, a combined IMT/EMT group and a control group. However, due to practicality reasons (i.e. the number of available respiratory training devices, the number of eligible athletes on the same rowing squad undertaking the same whole body and rowing training programme), this was not possible. Therefore, two groups were used to perform the IMT and EMT separately, and then using a cross-over design both groups performed combined IMT/EMT, essentially acting as their own controls. Even so, both groups could have used the same training device (i.e. Powerlung); however at the start of the study we did not have sufficient number of these devices to allocate to all the athletes. Therefore the decision was made to provide the IMT-group with the POWERbreathe® and the EMT-group with the Powerlung until the start of the combined training programme when we were able to supply all athletes with the same training device.

Baseline 1 (n=8)	Baseline 2 (n=17)	RMT Phase 1	Post-intervention 1 (n=17)	RMT Phase 2	Post-intervention 2 (n=15)
Resting pulmonary & respiratory muscle function testing	Resting pulmonary & respiratory muscle function testing	7 d·wk^{-1} 4 wk	Resting pulmonary & respiratory muscle function testing	7 d·wk^{-1} 6 wk	Resting pulmonary & respiratory muscle function testing
Rowing ergometer 'step test' & 6MAO	Rowing ergometer 'step test' & 6MAO	IMT: 2 sets of 30 breaths at repetition maximum (n=10)	Rowing ergometer 'step test' & 6MAO	IMT group (n=8)* EMT group (n=7)	Rowing ergometer 'step test' & 6MAO
PostEx-P_{Imax} & P_{Emax}	PostEx-P_{Imax} & P_{Emax}	EMT: 2 sets of 30 breaths at repetition maximum (n=7)	PostEx-P_{Imax} & P_{Emax}	Combined IMT/EMT: 2 sets of 30 breaths at repetition maximum	PostEx-P_{Imax} & P_{Emax}

Figure 4.1 Schematic diagram of testing sessions. Note: *, 2 IMT participants dropped out.

During their baseline visit to the laboratory, participants were familiarised with all of the testing procedures and each participant performed the entire testing protocol. Pre- and post-exercise respiratory mouth pressures and spirometry, including maximal flow volume loops (MFVLs) were recorded. A progressive incremental rowing exercise test was used to evaluate physiological variables and rowing ergometer performance. Rowing ergometer performance was measured as mean power (W), distance travelled (m) and split time. The final stage of the 'step test' consisted of a 6MAO, which was used as a time trial to compare rowing performance between conditions. Physiological variables measured included the following: peak f_c in the last 30 s of each stage; peak oxygen uptake ($\dot{V}O_2$); $[La^-]_B$ concentration and RRE at the end of each stage.

4.2.3: PROCEDURES

A detailed description of the instrumentation and testing procedures are provided in section 3.2.2.

ANTHROPOMETRY

Anthropometric measurements, including stature and body mass, were assessed prior to each testing session. Details of anthropometric measurements are described in section 3.2.1. Measurements recorded at baseline are presented in Table 4.1.

Table 4.1 Descriptive characteristics of the participants at Baseline 2 (mean ± SD).

	IMT-group (n = 10)	EMT-group (n = 7)
Anthropometry		
Age (y)	24.9 ± 5.6	28.7 ± 9.1
Stature (m)	1.87 ± 0.1	1.86 ± 0.1
Body mass (kg)	83.7 ± 4.8	82.6 ± 13.5

RESPIRATORY MUSCLE STRENGTH

Maximal inspiratory and expiratory pressure manoeuvres (P_{Imax} and P_{Emax}, respectively), were measured as surrogates of inspiratory and expiratory muscle strength using a hand-held mouth pressure meter (Micro Medical MPM, Micro Medical Ltd., Kent, UK). Measurements were performed in the standing position before and 2 min after the 6MAO test to determine improvements in maximal pressures due to training and to assess the extent of exercise-induced RMF following maximal rowing. As no research into the effectiveness of an expiratory warm-up exists, no respiratory warm-up was used in this study for either group. A detailed

description of the equipment and the procedures used for testing respiratory pressures are presented in section 3.2.2.

INSPIRATORY AND EXPIRATORY MUSCLE FATIGUE

The following dependent variables were measured: pre-exercise maximal inspiratory pressure (PreEx-P_{Imax}), pre-exercise maximal expiratory pressure (PreEx-P_{Emax}), post-exercise maximal inspiratory pressure (PostEx-P_{Imax}), and post-exercise maximal expiratory pressure (PostEx-P_{Emax}). Muscle fatigue was expressed as a percentage of the baseline value.

INCREMENTAL ROWING ERGOMETER EXERCISE TEST

A discontinuous incremental rowing ergometer test, also known as a 'step test', was used to evaluate physiological variables and rowing ergometer performance (Godfrey & Williams, 2007). The 'step-test' consisted of five stages: four stages of 4 min duration, followed by one 6MAO effort. All participants were familiar with the exercise test as they perform both incremental and maximal exercise tests as part of their routine training and monitoring. All exercise tests were performed on the same rowing ergometer; drag factor set to 138, damper setting 4. Participants completed a standardised 4 min warm-up at their typical UT2 power output. Following the warm-up, the participants rested on their ergometer for ~ 3 min awaiting the start of the step-test. During this time, there participants were given a brief reminder of the testing procedures and the Borg CR-10 scale used to assess RRE.

Starting power output was determined by calculating the power of each athlete's typical UT2 split time (light intensity aerobic training band), then subtracting 50 W. Subsequent stages were incremented by 25 W to create an incremental 'step test' (e.g., 150 W, 175 W, 200 W). Power output was identical at all testing phases of the intervention. There was a 1 min interval

between each stage to allow for $[La^-]_B$ and perceptual measurements (see fig 4.2). The athlete self-selected the power and pace during the final 6MAO effort; all participants were instructed to perform maximally and to attempt to achieve the greatest distance possible (using the ergometer distance output) in the 6-min time period allotted. The ergometer also calculated and displayed mean power output, mean 250 m split time and distance covered which were recorded at the end of the 6MAO stage.

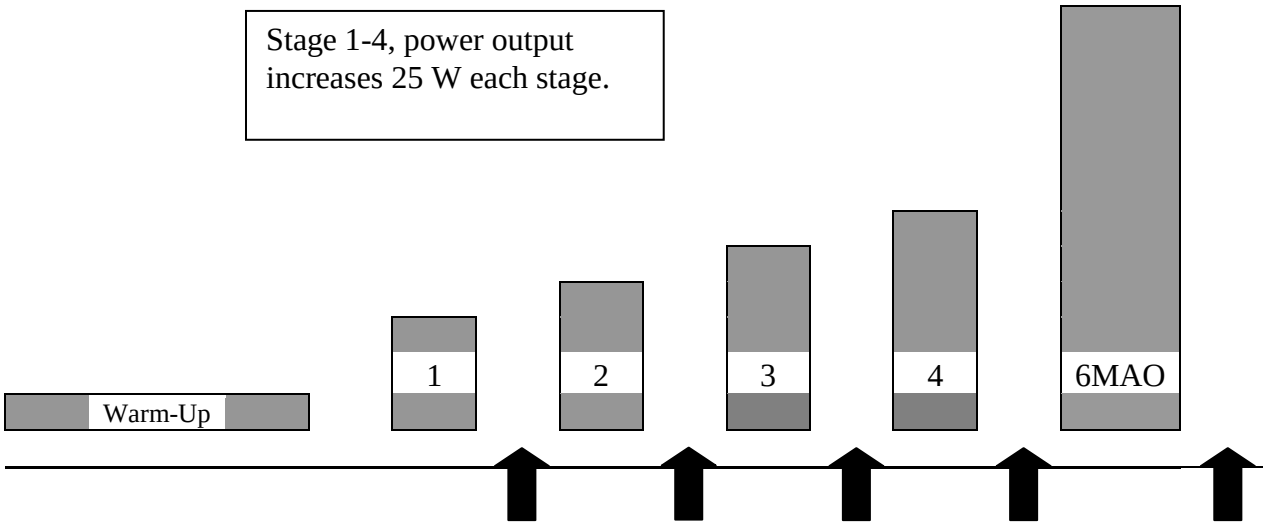


Figure 4.2 Rowing ergometer incremental 'step-test' protocol.

Stages 1-4 are work periods of 4 minutes. Stages 1-4 have a 1 min rest interval in which RRE and earlobe $[La^-]_B$ were taken. 6MAO, six minute all-out maximal effort. Figure recreated from Godfrey & Williams, 2007 p.173.

BREATHING PATTERN AND OXYGEN UPTAKE

Minute ventilation ($\dot{V}_E$), breathing pattern and pulmonary gas exchange indices were assessed continuously during exercise using an on-line turbine ergospirometry system (Oxycon, Jaeger-Toennies, Hoechberg, Germany). Resting MFVLs were assessed using the online system

according to European Respiratory Society guidelines (Quanjer et al., 1993). The MFVLs were performed and the following measures were recorded: peak inspiratory flow (PIF), peak expiratory flow (PEF), forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and forced expiratory flow at 50% of FVC ($FEF_{50\%}$). Mean $\dot{V}_E$, V_T and inspiratory flow rate (MIFR) was calculated for each stage of the step test and 6MAO effort. Measures of peak $\dot{V}O_2$ were recorded for each stage and are presented in millilitres per kilogram of body weight per minute ($ml \cdot kg^{-1} \cdot min^{-1}$). A detailed description of the MFVL manoeuvre, measures of gas exchange and the calibration procedures for the online gas analyser are described in section 3.2.2.

OTHER PHYSIOLOGICAL PARAMETERS

HEART RATE

Heart rate (f_c) was assessed throughout exercise using a short-range telemetry system (Polar Sport Tester, Polar Electro Oy, Finland). The 'peak end-stage f_c' value was obtained visually on the rowing ergometer monitor and recorded during the final 30 s of each stage of the exercise 'step test'. Full details of heart rate assessment are provided in section 3.2.5.

BLOOD LACTATE CONCENTRATION

Earlobe $[La^-]_B$ concentration was measured to monitor the intensity of exercise during the 'step test' and at termination of the 6MAO effort to compare changes in exercise intensity pre and post RMT. Blood measurements were taken at the end of each stage and immediately following the 6MAO effort; in which blood samples were measured within 15 s of the termination of exercise. A detailed description of the equipment and procedures for monitoring $[La^-]_B$ is presented in section 3.2.5.

RATING OF RESPIRATORY EFFORT SENSATION

A modified version of the RPE scale known as the Category Ratio 10 (CR10) scale (Borg, 1998) was used to produce a symptom profile of perceived breathlessness during exercise (Appendix A-4); hence, this value was used to represent RRE. Both scales are designed to produce estimates of exertion as to the degree of heaviness and strain experienced during physical work. The CR10 scale differs from the traditional RPE scale as it is specifically designed to analyse specific responses to exercise, such as sensations of breathlessness, quadriceps fatigue, etc; whereas, the RPE scale is intended to provide an index of overall impending fatigue. The CR10 scale rises exponentially from 0-maximal and has additional points at the higher end compared to the traditional linear RPE scale (6-20).

Prior to exercise testing, participants were provided with verbal instructions on how to read the CR10 scale by using the 'verbal anchors' (i.e., weak, moderate, very strong) to identify their RRE. Participants were requested to assess their RRE during the final 30 s of each submaximal exercise stage and 6MAO.

INSPIRATORY AND EXPIRATORY MUSCLE TRAINING

Participants were instructed on correct usage of the inspiratory (POWERbreathe®, Gaiam Ltd., Southam, UK) or expiratory (Powerlung®, Powerlung Inc., USA) pressure threshold-loading device for RMT. The Powerlung® provides a threshold load during both inspiration and expiration. To isolate the expiratory load for the EMT group, the inspiratory load was disabled by removing the valve tensioning spring. These changes had no effect on expiratory loading, and successfully removed the IMT challenge. A picture of this device and the coil removed is shown in Figure 4.3.

A

B

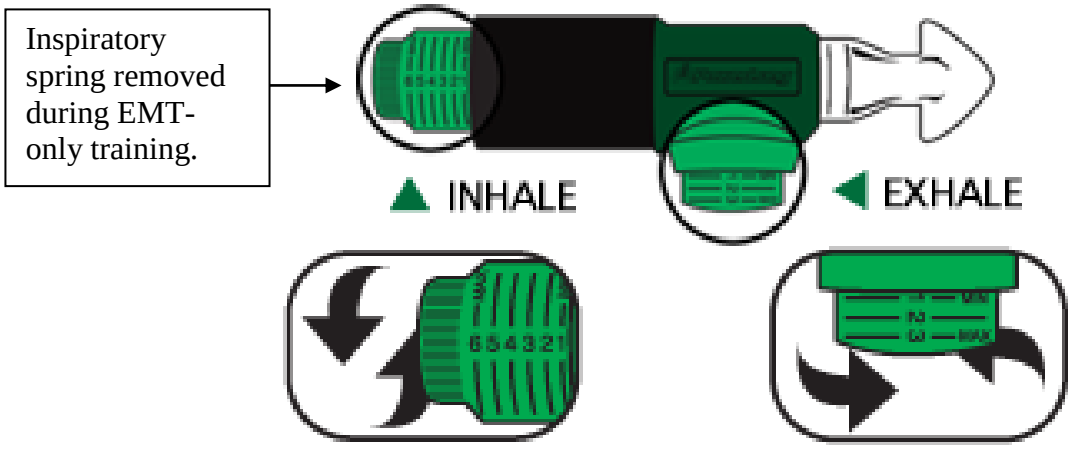


Figure 4.3 *A*, PowerLung® Sports Trainer; *B*, schematic of the breathing effort index (1-6) and the inspiratory spring removed during EMT-only exercise.

Both groups were instructed to perform 30 inspiratory or expiratory efforts twice daily for 4 wk against a pressure load equivalent to their individual 30 repetition maximum (30 RM, equivalent to ~50% of P_{Imax} or P_{Emax}), a protocol that has been shown to be effective for IMT in previous studies (Romer et al., 2002a, 2002c, 2003; Volianitis et al., 2001c). Participants in both groups were instructed to breathe rapidly and with maximal effort against the training load. The IMT group was instructed to initiate each breath from RV and to sustain the effort

until their lungs felt full. The EMT group was instructed to initiate each breath from TLC and to sustain the effort until their lungs were empty. Participants were instructed to increase the training load in order to maintain it at the 30 RM (Romer et al., 2002a), which increased throughout the training period.

After Post-intervention test 1, participants performed a combined program of IMT/ EMT using a respiratory muscle trainer (PowerLung®, PowerLung® Inc., USA) for a further 6 wk. For this phase of the study, the device was intact and applied both an inspiratory and expiratory load. Participants were directed to perform both maximal inspirations and expirations for 30 RM, twice daily and to increase resistance when necessary to maintain the intensity at the 30 RM loads. In order to prevent hyperventilation, participants were encouraged to momentarily hold their breath at RV and TLC.

During their training, participants completed respiratory training diaries (Appendix A-3), which were used to monitor training adherence and to track increases in training loads throughout the interventions. Participants continued with their scheduled whole body exercise training throughout the intervention. Participants were instructed on correct usage and cleaning of the inspiratory (POWERbreathe®, Gaiam Ltd., Southam, UK) or expiratory (PowerLung®, PowerLung® Inc., USA) pressure threshold-loading devices for respiratory muscle training. Detailed pictures of the devices and instructions for use are outlined in section 3.2.2.

4.2.4: STATISTICAL ANALYSES

Two methods of analyses were used to determine reproducibility of outcome variables. Ratio limits of agreement were calculated to determine the within-subject variation according to the procedures of Bland and Altman (1986). These were used to estimate the effect of the measurement upon statistical power. Thus, the 95% ratio limits of agreements were used to

estimate sample sizes for a range of treatment effects (including those measured post-intervention) for a repeated-measures design, given a statistical power of 0.9 and an alpha level of 0.05. These estimates were performed using a bespoke Excel spreadsheet, based upon the calculations of Zar (1996).

A repeated measures analysis of variance (ANOVA) was used to evaluate 'treatment' (IMT and EMT) and 'time' on breathing pattern, physiological parameters and performance changes throughout the intervention. A mixed between-within analysis of variance was conducted to compare the three performance indices of mean power, distance and split time achieved during the 6MAO effort; Baseline 2, Post-intervention test 1, and Post-intervention test 2. Planned pairwise comparisons were made with repeated measures *t* tests to compare main effects; the Bonferroni adjustment was used to modify for per family type I error rate per comparison and probability values of ≤ 0.05 were considered significant. Pearson's bivariate correlations were used to determine significant relationships between variables. Data were analysed using the statistical software package SPSS V10.0 for Windows (Chicago, IL, USA) and results are reported as the mean ± standard deviation (SD).

4.3: RESULTS

Two of the IMT group participants were unable to attend the Post-intervention test 2; Thus, n = 8 for the IMT group at the Post-intervention 2 time point.

4.3.1: INTER-TEST PRECISION

The estimated sample sizes for a range of effect magnitudes are provided in Table 4.2. Parameters derived for the MFVL and mouth pressures showed the lowest reliability compared

with the other variables measured. The data suggested that the study had sufficient power to detect changes in most parameters with a magnitude of effect > 20% (assuming n = 7 per group). The exceptions to this were FVC and FEV_1, which required effect magnitudes of > 5% and 10%, respectively. In contrast, the exercise performance outcome measures of mean power and distance, and the physiological variables of $\dot{V}O_{2\ 6MAO}$ and f_c required effect magnitude of > 5%. The highest reliability was observed in the RRE, which required an effect magnitude of < 5%.

The limits of agreement for within-subject variation for all outcome measures are summarised in Tables 4.3 to 4.7. Significant differences in baseline measurements highlighted by the independent sample *t*-tests are also noted. These tables provide for visual inspection of the agreement between the mean and the difference of the means on the two separate occasions. The correlation of the mean difference was very low suggesting that the data were not heteroscedastic. Additionally, all data were log transformed to create dimensionless quantities allowing for a comparison across parameters with different units of measure.

Table 4.2 Estimated sample size for effect.

Variable	Effect magnitudes (percentage of measured value)			
	5%	10%	20%	30%
PIF	125	31	8	3
PEF	130	32	8	4
FVC	5	1	1	1
FEV_1	12	3	1	1
FEF_{25}	37	9	2	1
FEF_{50}	50	12	3	1
FEF_{75}	673	168	42	19
PreEx-P_{Imax}	104	26	7	3
PreEx-P_{Emax}	52	13	3	1
PostEx-P_{Imax}	303	76	19	8
PostEx-P_{Emax}	125	31	8	3
$\dot{V}O_2$ step 1	20	5	1	1
$\dot{V}O_2$ step 2	30	7	2	1
$\dot{V}O_2$ step 3	18	4	1	1
$\dot{V}O_2$ step 4	1	1	1	1
$\dot{V}O_2$ 6MAO	5	1	1	1
f_c	2	1	1	1
$[La^-]_B$	397	99	25	11
RRE	1	1	1	1
Mean power	5	1	1	1
Distance	3	1	1	1

Note**:** PIF, peak inspiratory flow; PEF, peak expiratory flow; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 sec; FEF_{25}, forced expiratory flow at that point that is 25% from FVC; FEF_{50}, forced expiratory flow at the point that is 50% from FVC; FEF_{75}, forced expiratory flow at the point that is 75% from FVC. Pre-P_{Imax}, maximal inspiratory pressure before the exercise testing session; Pre-P_{Emax}, maximal expiratory pressure before the exercise testing session; Post-P_{Imax}, maximal inspiratory pressure ~2' after the 6MAO effort; Post-P_{Emax}, maximal expiratory pressure ~2' after the 6MAO effort. $\dot{V}O_2$, oxygen consumption; f_c, heart rate in beats per minute (bpm); $[La^-]_B$, earlobe blood lactate concentration; RRE, rating of respiratory effort.

Table 4.3 Absolute and ratio limits of agreement for pulmonary function.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
PIF ($L \cdot sec^{-1}$)	8	9.5 (2.0)	10.1 (1.6)	-0.7 (1.4)	-0.65	0.48	-1.68 to 0.38	2.67	0.83	-5.10 to -1.53	0.23 to 3.80
PEF ($L \cdot sec^{-1}$)	8	10.2 (2.0)	10.7 (1.3)	-0.6 (1.4)	-0.64	0.51	-1.73 to 0.46	2.83	0.88	-5.36 to -1.57	0.30 to 4.08
FVC (L)	8	5.9 (0.5)	6.1 (0.5)	-0.2 (0.2)*	-0.21	0.07	-0.35 to -0.07	0.36	0.11	-0.82 to -0.33	-0.09 to 0.40
FEV_1 (L)	8	5.0 (0.5)	5.1 (0.5)	-0.1 (0.3)	-0.11	0.09	-0.30 to 0.09	0.51	0.16	-0.96 to -0.28	0.06 to 0.74
FEF_{25} ($L \cdot sec^{-1}$)	8	9.0 (2.3)	9.1 (2.2)	-0.1 (0.8)	-0.13	0.29	-0.74 to 0.48	1.58	0.49	-2.77 to -0.65	0.39 to 2.51
FEF_{50} ($L \cdot sec^{-1}$)	8	6.1 (1.9)	6.1 (1.8)	-0.0 (0.5)	-0.02	0.18	-0.41 to 0.37	1.01	0.32	-1.72 to -0.36	0.31 to 1.67
FEF_{75} ($L \cdot sec^{-1}$)	8	3.0 (1.5)	2.9 (1.5)	0.2 (0.8)	0.16	0.28	-0.44 to 0.76	1.55	0.49	-2.43 to -0.35	0.67 to 2.76

Log transformed measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Ratio	SE	95% CI	Ratio	SE	95% CI for Lower L of A	95% CI for Upper L of A
PIF ($L \cdot sec^{-1}$)	8	2.2 (0.2)	2.3 (0.2)	-0.1 (0.2)	0.929	0.052	-0.187 to 0.038	1.338	0.091	0.499 to 0.889	1.047 to 1.437
PEF ($L \cdot sec^{-1}$)	8	2.3 (0.2)	2.4 (0.1)	-0.1 (0.2)	0.929	0.053	-0.188 to 0.041	1.345	0.093	0.492 to 0.889	1.051 to 1.448
FVC (L)	8	1.8 (0.1)	1.8 (0.1)	-0.0 (0.0)	0.965	0.011	-0.060 to -0.011	1.066	0.020	0.863 to 0.948	0.986 to 1.071
FEV_1 (L)	8	1.6 (0.1)	1.6 (0.1)	-0.0 (0.1)	0.978	0.018	-0.060 to 0.016	1.104	0.031	0.820 to 0.952	1.013 to 1.146
FEF_{25} ($L \cdot sec^{-1}$)	8	2.2 (0.3)	2.2 (0.2)	-0.0 (0.1)	0.983	0.030	-0.082 to 0.048	1.183	0.053	0.718 to 0.943	1.050 to 1.276
FEF_{50} ($L \cdot sec^{-1}$)	8	1.8 (0.3)	1.8 (0.3)	-0.0 (0.1)	0.992	0.035	-0.083 to 0.067	1.213	0.060	0.689 to 0.947	1.074 to 1.333
FEF_{75} ($L \cdot sec^{-1}$)	8	1.0 (0.5)	1.0 (0.5)	0.1 (0.3)	1.056	0.104	-0.170 to 0.278	1.785	0.181	0.203 to 0.980	1.496 to 2.272

Note: PIF, peak inspiratory flow; PEF, peak expiratory flow; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 sec; FEF_{25}, forced expiratory flow at that point that is 25% from FVC; FEF_{50}, forced expiratory flow at the point that is 50% from FVC; FEF_{75}, forced expiratory flow at the point that is 75% from FVC;
*, significant difference ($p \leq 0.05$).

Table 4.4 Absolute and ratio limits of agreement for maximal inspiratory and expiratory pressures.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
PreEx-P_{Imax} (cm H_2O)	8	113.6 (18.9)	133.5 (14.9)	-19.9 (16.6)*	-19.88	5.85	-32.43 to -7.32	32.45	10.14	-74.07 to -30.58	-9.17 to 34.32
PreEx-P_{Emax} (cm H_2O)	8	119.0 (11.6)	134.6 (21.3)	-15.4 (14.7)*	-15.63	5.19	-26.75 to -4.50	28.75	8.98	-63.64 to -25.11	-6.14 to 32.39
PostEx-P_{Imax} (cm H_2O)	8	111.6 (17.8)	117.0 (20.8)	-5.4 (23.9)	-5.38	8.45	-23.49 to 12.74	46.83	14.63	-83.59 to -20.82	10.07 to 72.84
PostEx-P_{Emax} (cm H_2O)	8	113.5 (20.7)	119.4 (13.7)	-5.9 (16.1)	-5.88	5.70	-18.11 to 6.36	31.61	9.88	-58.67 to -16.30	4.55 to 46.92

Log transformed measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Ratio	SE	95% CI	Ratio	SE	95% CI for Lower L of A	95% CI for Upper L of A
PreEx-P_{Imax} (cm H_2O)	8	4.7 (0.0)	4.9 (0.1)	-0.2 (0.1)	0.846	0.049	-0.272 to -0.063	1.309	0.084	0.466 to 0.827	0.927 to 1.288
PreEx-P_{Emax} (cm H_2O)	8	4.8 (0.1)	4.9 (0.2)	-0.1 (0.1)	0.890	0.036	-0.193 to -0.041	1.218	0.061	0.599 to 0.863	0.951 to 1.215
PostEx-P_{Imax} (cm H_2O)	8	4.7 (0.2)	4.7 (0.2)	-0.1 (0.2)	0.956	0.076	-0.209 to 0.118	1.527	0.132	0.342 to 0.909	1.175 to 1.742
PostEx-P_{Emax} (cm H_2O)	8	4.7 (0.2)	4.8 (0.1)	-0.1 (0.2)	0.941	0.053	-0.173 to 0.052	1.338	0.091	0.508 to 0.899	1.065 to 1.455

Note: PreEx-$P_{Imax,}$ maximal inspiratory pressure before the exercise testing session; PreEx-P_{Emax}, maximal expiratory pressure before the exercise testing session; PostEx-P_{Imax}, maximal inspiratory pressure ~2' after the 6MAO effort; PostEx-P_{Emax}, maximal expiratory pressure ~2' after the 6MAO effort. *, significant difference ($p \leq 0.05$).

Table 4.5 Absolute and ratio limits of agreement for blood lactate concentrations during the incremental exercise test.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
Stage 1 $[La^-]_B$ ($mmol \cdot L^{-1}$)	8	1.8 (0.6)	1.8 (0.6)	0.0 (0.6)	0.03	0.23	-0.37 to 0.42	1.25	0.39	-1.91 to -0.54	0.59 to 1.96
Stage 2 $[La^-]_B$ ($mmol \cdot L^{-1}$)	8	1.8 (1.1)	2.1 (0.9)	-0.3 (0.5)	-0.25	0.19	-0.57 to 0.07	1.03	0.32	-1.84 to -0.71	-0.21 to 1.34
Stage 3 $[La^-]_B$ ($mmol \cdot L^{-1}$)	8	2.6 (1.8)	2.8 (1.4)	-0.2 (0.8)	-0.19	0.26	-0.65 to 0.27	1.46	0.46	-2.45 to 0.85	0.47 to 2.07
Stage 4 $[La^-]_B$ ($mmol \cdot L^{-1}$)	8	3.9 (1.9)	5.0 (4.0)	-1.1 (2.3)	-1.10	0.88	-2.66 to 0.46	4.57	1.53	-8.38 to -2.97	0.77 to 6.18

Log transformed measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Ratio	SE	95% CI	Ratio	SE	95% CI for Lower L of A	95% CI for Upper L of A
Stage 1 $[La^-]_B$ ($mmol \cdot L^{-1}$)	8	0.6 (0.4)	0.5 (0.4)	0.0 (0.3)	1.017	0.120	-0.194 to 0.228	1.948	0.223	0.131 to 0.912	1.591 to 2.372
Stage 2 $[La^-]_B$ ($mmol \cdot L^{-1}$)	8	0.5 (0.5)	0.6 (0.5)	-0.2 (0.4)	0.834	0.123	-0.397 to 0.035	1.979	0.228	0.022 to 0.821	1.252 to 2.052
Stage 3 $[La^-]_B$ ($mmol \cdot L^{-1}$)	8	0.8 (0.6)	0.9 (0.5)	-0.1 (0.3)	0.878	0.097	-0.300 to 0.039	1.709	0.179	0.200 to 0.827	1.186 to 1.814
Stage 4 $[La^-]_B$ ($mmol \cdot L^{-1}$)	8	1.3 (0.4)	1.4 (0.7)	-0.1 (0.3)	0.882	0.118	-0.335 to 0.083	1.844	0.204	0.116 to 0.840	1.264 to 1.988

Note : $[La^-]_B$, earlobe blood lactate concentration.

Table 4.6 Absolute and ratio limits of agreement for physiological variables during the six minute all out (6MAO) effort.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$\dot{V}O_2$peak ($ml \cdot kg^{-1} \cdot min^{-1}$)	8	58.0 (6.6)	56.0 (6.1)	2.0 (2.0)*	1.99	0.07	0.46 to 3.51	3.94	1.23	-4.59 to 0.69	3.29 to 8.57
f_c (bpm)	8	184.9 (10.3)	185.9 (10.3)	-1.0 (4.4)	-1.00	1.56	-4.34 to 2.34	8.64	2.70	-15.43 to -3.85	1.85 to 13.43
$[La^-]_B$ ($mmol \cdot L^{-1}$)	8	15.2 (1.2)	16.1 (3.3)	-0.9 (3.1)	-0.91	1.18	-3.00 to 1.18	6.12	2.04	-10.66 to -3.41	1.59 to 8.83
RRE	8	8.8 (1.3)	8.8 (1.3)	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Log transformed measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Ratio	SE	95% CI	Ratio	SE	95% CI for Lower L of A	95% CI for Upper L of A
$\dot{V}O_2$peak ($ml \cdot kg^{-1} \cdot min^{-1}$)	8	4.1 (0.1)	4.0 (0.1)	0.0 (0.0)	1.035	0.012	0.008 to 0.061	1.070	0.021	0.922 to 1.013	1.062 to 1.153
f_c (bpm)	8	5.2 (0.1)	5.2 (0.1)	-0.0 (0.0)	0.995	0.008	-0.023 to 0.012	1.046	0.014	0.920 to 0.981	1.010 to 1.071
$[La^-]_B$ ($mmol \cdot L^{-1}$)	8	2.7 (0.1)	2.8 (0.3)	-0.0 (0.3)	0.963	0.091	-0.199 to 0.123	1.603	0.158	0.321 to 0.880	1.264 to 1.822
RRE	8	2.2 (0.2)	2.2 (0.2)	0.00	1.000	0.000	0.000	1.000	0.000	1.000 to 1.000	1.000 to 1.000

Note: $\dot{V}O_2$peak, peak oxygen consumption; f_c, heart rate; $[La^-]_B$, earlobe blood lactate concentration; RRE, rating of respiratory effort. *, significant difference ($p \leq 0.05$).

Table 4.7 Absolute and ratio limits of agreement for performance indices during the six minute all out (6MAO) effort.

Measurements

Variable	N	Mean (SD)1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
Mean power (W)	8	346.1 (29.0)	344.5 (33.8)	1.7 (11.7)	1.65	4.15	-7.26 to 10.56	23.02	7.19	-36.80 to -5.94	9.24 to 40.10
Distance (m)	8	1772.6 (79.5)	1788.5 (58.3)	16.0 (43.8)	-15.93	15.5	-48.80 to 16.94	85.95	26.85	-158.81 to -44.95	13.09 to 126.95

Log transformed measurements

Variable	N	Mean (SD)1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Ratio	SE	95% CI	Ratio	SE	95% CI for Lower L of A	95% CI for Upper L of A
Mean power (W)	8	5.8 (0.1)	5.8 (0.1)	0.0 (0.0)	1.006	0.012	-0.021 to 0.033	1.072	0.022	0.892 to 0.985	1.032 to 1.124
Distance (m)	8	7.5 (0.1)	7.5 (0.0)	-0.0 (0.0)	0.991	0.009	-0.028 to 0.009	1.050	0.015	0.911 to 0.976	1.008 to 1.073

4.3.2: ADHERENCE TO RESPIRATORY TRAINING

Training adherence to the IMT-only and EMT-only phases during the first 4 wk was similar between groups. The IMT group completed a total of 43 ± 10% sessions (76 ± 17% of prescribed) and the EMT group a total of 43 ± 5.5 (78 ± 9.8%). During the combined program phase of the study, the IMT group adherence remained unchanged at 75 ± 33% whilst the adherence of the EMT group fell to 60 ± 37% ($P < 0.05$) of the prescribed training sessions.

4.3.3: RESPIRATORY MUSCLE AND PULMONARY FUNCTION

INSPIRATORY MUSCLE STRENGTH

There was a significant interaction over time within groups ($P = 0.000$), as well as between the two groups over time ($P = 0.008$) in P_{Imax} (see table 4.7). PreEx-P_{Imax} in the IMT group was 26% higher compared to baseline after 4 wk of IMT ($P = 0.000$) and improved by a further 3.3% after the combined IMT/EMT phase to 30% ($P = 0.002$ relative to baseline). The EMT group also showed a small improvement in P_{Imax} after the combined IMT/EMT program to 13% ($P = 0.029$) (fig. 4.4*A*).

EXPIRATORY MUSCLE STRENGTH

As shown in Figure 4.4*B*, the IMT group showed no change in P_{Emax} during the IMT-only phase of training or combined IMT/EMT phase (23%; $P = 0.056$). During the EMT-only phase, the EMT group showed no improvement in P_{Emax} (18%; $P > 0.05$); however, during the combined IMT/EMT phase this group showed a further improvement (relative to baseline) in P_{Emax} (to 31%) ($P = 0.033$).

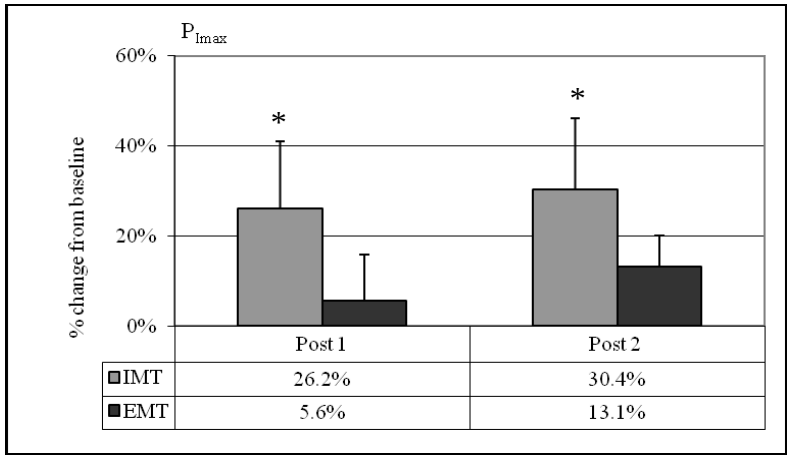
EXERCISE-INDUCED INSPIRATORY AND EXPIRATORY MUSCLE FATIGUE

Only the IMT group exhibited a change over time in the exercise-induced fall in P_{Imax} following IMT and the combined IMT/EMT ($P = 0.029$). As illustrated in Figure 4.4*C* & *D*, IMF

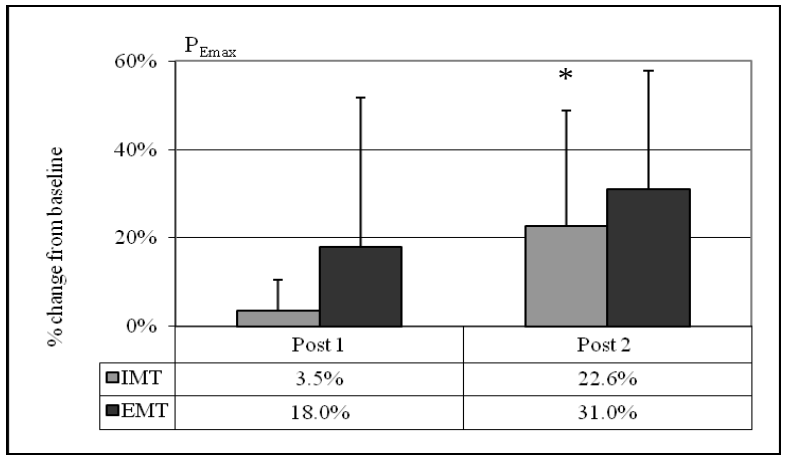
persisted following the IMT phase, but was abolished following combined IMT/EMT (fig. 4.4*C*). In contrast, the EMT group showed no change in the severity of IMF or EMF at any stage (fig. 4.4*C*& *D*). The IMT group showed no change in EMF during the IMT phase, but EMF was reduced from 15% to 5% following the combined training programme (fig. 4.4*D*), but not significantly.

In contrast to the percentage changes in P_{Imax} and P_{Emax} post-exercise, which largely persisted at the same magnitude following the interventions (fig. 4.4*C* & *D*), the absolute values for PostEx-P_{Imax} and PostEx-P_{Emax} showed consistent improvements in both groups (see table 4.7). There were no correlations between the changes in RMF and the changes in exercise performance in either group at any stage of the intervention.

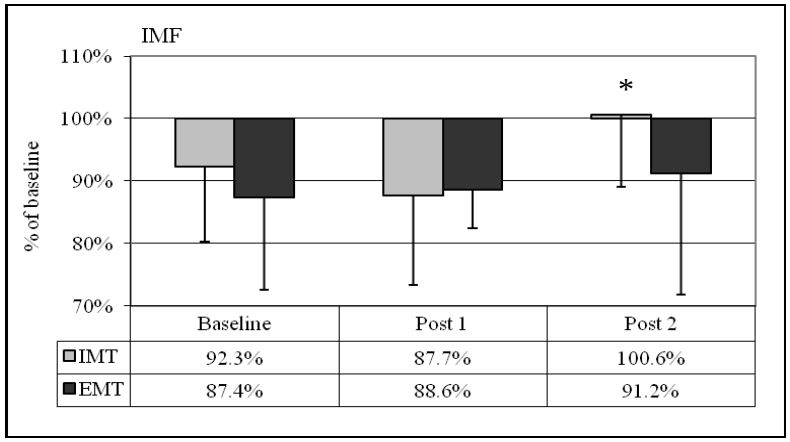
A



B



C



D

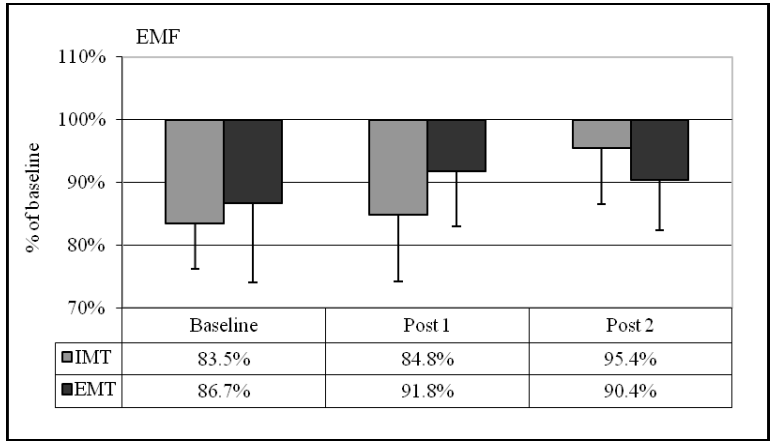


Figure 4.4 *A & B*, Percent change in maximal inspiratory and expiratory pressure post-intervention. *C & D*, Percent of inspiratory (IMF) and expiratory muscle fatigue (EMF) post-6MAO effort. Values measured in percent (%). *, significantly different to baseline 2 ($p \leq 0.05$).

MAXIMAL FLOW VOLUME LOOP

Post-intervention pulmonary function data were similar to baseline (see table 4.8). The only changes in pulmonary function were an increase in PIFR in the IMT group following the IMT phase (P = 0.043), and a decrease in FVC following the combined IMT/EMT phase (P = 0.033).

BREATHING PATTERN

No change was found in the following parameters after IMT, EMT, or combined IMT/EMT in either group: mean V_T, IFR, $\dot{V}_E$ and f_R (see table 4.9). However, for the IMT group, there was a strong positive correlation between the individual changes in P_{Imax} and $\dot{V}_E$ (the value at stage 4 was selected for comparison) between baseline and post-intervention test 2 (r = 0.846, P = 0.016), as well as between post-intervention test 1 and post-intervention test 2 (r = 0.896, P = 0.016).

Table 4.8 Summary of respiratory muscle and pulmonary function data for the IMT and EMT groups.

	Baseline 1 (n=8)	Baseline 2 (n=17)	Post-intervention 1 (n=17)	Post-intervention 2 (n=15)
PreEx- P_{Imax} (cm H_2O)				
IMT	113.6 ± 18.9	129.1 ± 16.5*	162.9 ± 24.1*	168.3 ± 31.2*
EMT	133.5 ± 14.8	138.6 ± 27.4	146.3 ± 28.7	156.7 ± 29.7*†
PreEx- P_{Emax} (cm H_2O)				
IMT	119.0 ± 11.6	144.8 ± 22.0*	149.9 ± 28.1	177.5 ± 49.3
EMT	134.6 ± 21.3	133.7 ± 15.4	157.7 ± 27.7	175.1 ± 36.3*
PostEx- P_{Imax} (cm H_2O)				
IMT	111.6 ± 17.8	119.1 ± 21.4	142.8 ± 43.0*	169.4 ± 43.3*†
EMT	117.0 ± 20.8	121.1 ± 30.3	129.6 ± 25.4	142.9 ± 31.5
PostEx- P_{Emax} (cm H_2O)				
IMT	113.5 ± 20.7	120.9 ± 22.8	127.1 ± 21.3	169.3 ± 41.3*†
EMT	119.4 ± 13.7	115.9 ± 16.4	144.7 ± 24.1*	158.3 ± 25.6*
FVC (L)				
IMT	6.21 ± 0.27	6.19 ± 0.78	6.27 ± 0.65	6.02 ± 0.56*
EMT	5.03 ± 0.58	6.05 ± 0.67	6.03 ± 0.74	5.94 ± 0.56
FEV_1 (L)				
IMT	5.33 ± 0.13	5.37 ± 0.71	5.37 ± 0.69	5.22 ± .071
EMT	4.38 ± .027	4.86 ± 0.51	4.74 ± 0.52	4.74 ± 0.38
PIF ($L \cdot sec^{-1}$)				
IMT	10.11 ± 1.38	10.28 ± 1.66	10.92 ± 1.29*	10.55 ± 1.38
EMT	9.35 ± 1.99	10.47 ± 1.64	10.55 ± 0.61	10.55 ± 0.38
PEF ($L \cdot sec^{-1}$)				
IMT	11.09 ± 1.13	10.7 ± 1.66	10.52 ± 1.29	10.85 ± 1.76
EMT	7.85 ± 0.41	10.31 ± 1.02	10.44 ± 1.24	10.59 ± 0.76
$FEF_{50\%}$ ($L \cdot sec^{-1}$)				
IMT	7.33 ± 1.57*	6.74 ± 1.45	6.56 ± 1.56	6.51 ±1.84
EMT	4.46 ± 1.18	5.27 ± 1.14	5.05 ± 0.85	4.73 ± 0.51

Note: Post-intervention 1 and Post-intervention 2 contain different number of participants (Post 1: IMT, n = 10; EMT, n = 7. Post 2: IMT, n = 8; EMT, n = 7). PreEx-P_{Imax}, maximal inspiratory pressure pre-exercise testing session; PreEx-P_{Emax}, maximal expiratory pressure pre-exercise testing session; PostEx-P_{Imax}, maximal inspiratory pressure ~2 min post-6MAO effort; PostEx-$P_{Emax,}$ maximal expiratory pressure ~2 min post-6MAO effort. FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 second; PIF, peak inspiratory flow; PEF, peak expiratory flow; $FEF_{50\%,}$ forced expiratory flow at 50% of FVC. *, significantly different from Baseline 2 ($p \leq 0.05$); †, significantly different from the preceding time point ($p \leq 0.05$).

Table 4.9 Comparison of V_T, MIFR, V_E, and f_R.

	Step 1	Step 2	Step 3	Step 4	6MAO
IMT Group					
Baseline 2 (n=10)					
V_T (L)	2.3 ± 0.6	2.4 ± 0.5	2.5 ± 0.6	2.7 ± 0.7	3.1 ± 0.6
MIFR ($L \cdot sec^{-1}$)	2.1 ± 0.3	2.6 ± 0.3	3.1 ± 0.3	3.4 ± 0.5	5.4 ± 0.4
V_E ($L \cdot min^{-1}$)	62.3 ± 6.3	74.1 ± 10.1	89.5 ± 9.0	102.8 ± 12.5	157.0 ± 12.0
f_R ($breaths \cdot min^{-1}$)	29.3 ± 6.8	32.2 ± 6.7	38.5 ± 9.6	30.3 ± 7.5	52.4 ± 10.4
Post-intervention 1 (n=10)					
V_T (L)	2.3 ± 0.6	2.4 ± 0.7	2.5 ± 0.7	2.5 ± 0.5	3.1 ± 0.7
MIFR ($L \cdot sec^{-1}$)	2.3 ± 0.3	2.6 ± 0.3	3.0 ± 0.4	3.4 ± 0.4	5.2 ± 0.6
V_E ($L \cdot min^{-1}$)	65.8 ± 6.7	77.1 ± 10.0	89.4 ± 12.1	101.7 ± 10.2	159.2 ± 9.5
f_R ($breaths \cdot min^{-1}$)	30.4 ± 8.6	35.5 ± 11.9	36.6 ± 9.9	41.7 ± 11.2	55.3 ± 5.8
Post-intervention 2 (n=8)					
V_T (L)	2.1 ± 0.5	2.2 ± 0.5	2.4 ± 0.6	2.5 ± 0.5	3.1 ± 0.6
MIFR ($L \cdot sec^{-1}$)	2.1 ± 0.3	2.6 ± 0.4	2.9 ± 0.6	3.4 ± 0.4	5.6 ± 0.4
V_E ($L \cdot min^{-1}$)	64.6 ± 10.0	75.0 ± 8.4	84.4 ± 15.5	101.1 ± 13.3	164.3 ± 14.1
f_R ($breaths \cdot min^{-1}$)	31.9 ± 8.8	35.4 ± 8.5	39.0 ± 8.7	42.4 ± 6.8	53.9 ± 10.5
EMT Group					
Baseline 2 (n=7)					
V_T (L)	2.0 ± 0.8	1.9 ± 0.7	2.1 ± 0.9	2.4 ± 1.0	2.4 ± 1.1
MIFR ($L \cdot sec^{-1}$)	2.1 ± 0.7	2.7 ± 0.7	3.1 ± 0.6	3.4 ± 0.7	5.1 ± 0.7
V_E ($L \cdot min^{-1}$)	63.4 ± 10.7	77.3 ± 12.8	85.7 ± 11.6	98.0 ± 12.9	157.0 ± 12.0
f_R ($breaths \cdot min^{-1}$)	30.6 ± 8.2	35.4 ± 7.2	36.3 ± 7.7	37.9 ± 9.3	52.9 ± 7.9
Post-intervention 1 (n=7)					
V_T (L)	1.7 ± 0.6	1.9 ± 0.7	2.0 ± 0.8	2.1 ± 0.8	2.4 ± 1.0
MIFR ($L \cdot sec^{-1}$)	2.0 ± 1.0	2.5 ± 1.0	3.1 ± 0.6	3.6 ± 0.8	5.2 ± 0.7
V_E ($L \cdot min^{-1}$)	66.6 ± 11.6	76.1 ± 10.6	88.0 ± 11.8	103.0 ± 13.3	152.5 ± 7.1
f_R ($breaths \cdot min^{-1}$)	34.4 ± 6.3	35.4 ± 6.3	38.7 ± 6.6	42.8 ± 6.0	55.3 ± 5.4
Post-intervention 2 (n=7)					
V_T (L)	2.1 ± 0.7	2.2 ± 0.8	2.2 ± 0.7	2.2 ± 0.7	2.7 ± 0.2
MIFR ($L \cdot sec^{-1}$)	2.4 ± 0.8	2.8 ± 0.9	3.1 ± 0.9	3.4 ± 1.0	5.4 ± 0.9
V_E ($L \cdot min^{-1}$)	68.0 ± 14.6	79.6 ± 14.7	88.9 ± 16.2	101.7 ± 14.6	153.3 ± 15.6
f_R ($breaths \cdot min^{-1}$)	33.3 ± 9.4	35.4 ± 8.5	39.0 ± 8.7	42.4 ± 6.8	57.7 ± 5.3

Note: IMT, inspiratory muscle training. V_T, tidal volume; MIFR, mean inspiratory flow rate; mean V_E, minute ventilation; f_R, breathing frequency; EMT, expiratory muscle training. *, significantly different from Baseline 2 ($p \leq 0.05$).

4.3.4: PHYSIOLOGICAL VARIABLES DURING ROWING

As stroke rate was not controlled between trials or at different stages of the exercise step test, this may have directly affected the amount of effort performed during each stage. An increase or decrease in stroke rate, even at the same power output, would have led to a change in the amount of effort performed during the 'step-test', thus potentially influencing the physiological variables measured. This is not as relevant to the results achieved during the 6MAO effort as athletes typically do not regulate stroke rate during racing conditions, rather variations in stroke rate are used to control effort throughout the race to ensure optimal performance/pace.

OXYGEN UPTAKE AND HEART RATE

No change in $\dot{V}O_2$ was evident at any stage of the 10 wk intervention (fig 4.5) in either training group ($P > 0.05$). However, there seemed to be a trend in which $\dot{V}O_2$ peak increased during the 6MAO in the IMT-group following both phases of training (8.6%; $P > 0.05$). Previous pressure threshold loading IMT studies have observed a reduction in both metabolic and f_c during constant power exercise (Downey et al., 2007; Gething et al., 2004; Romer et al., 2002c). Although RMT has not typically been shown to improve VO_{2max} or cardiac output (Markov et al., 2001), it may have been that following IMT, these athletes were able to work at higher exercise intensities thus reaching a higher $\dot{V}O_2$ peak.

Although there was no change in peak end-stage f_c for the 6MAO ($P = 0.283$), there was a decrease of 2% to 5% for the IMT group across all steps following the IMT phase of the intervention ($P = 0.001$) and during the sub-maximal steps following the combined IMT/EMT phase ($P = 0.000$) (fig. 4.6). Paired t-tests were performed (corrected using the Bonferroni adjustment; P set at <0.013), to interpret the significance of the change in peak end-stage f_c values in the IMT groups across all submaximal steps. No significant

differences were observed in the IMT group at any particular time point. There was no change in the EMT group in f_c at any stage after either phase of training.

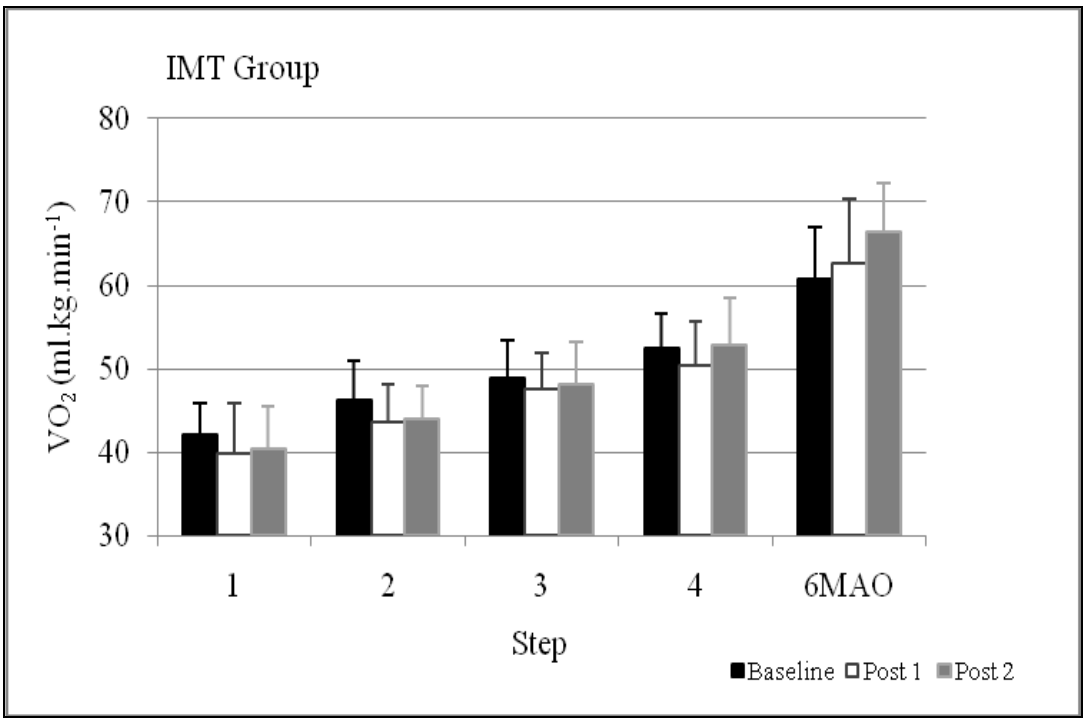
BLOOD LACTATE CONCENTRATION

There was no interaction within groups (P = 0.084) or between the two groups over time (P = 0.383); however, there was a tendency for $[La^-]_B$ to be lower in the IMT group after training. As seen in Figure 4.7, alterations to the $[La^-]_B$ relationship in this group were greatest following the combined IMT/EMT phase (average decrease in $[La^-]_B$ across all steps = 30%) and $[La^-]_B$ was lower immediately after the 6MAO effort (14%, P = 0.047). We calculated that a sample size of > 10 (per group) would be required in order to detect a significant effect of 30% during the sub-maximal stages. It is therefore possible that the lack of significance for the sub-maximal steps is a type 2 error. There was a weak correlation between the individual changes in P_{Imax} and $[La^-]_B$ between baseline and post-intervention test 1 (r = 0.614; P = 0.059). There was no change in mean $[La^-]_B$ in the EMT group after either phase of training.

RATING OF RESPIRATORY EFFORT SENSATION

No differences in the stage and group interaction for the RRE sensation was found between the IMT and EMT groups (P = 0.065) over the three testing sessions (fig. 4.8). However, there was a reduction in RRE in the IMT group following the 6MAO (P = 0.05). No correlation was found between the individual changes in P_{Imax} and RRE (the rating at stage 4 was selected for comparison) between baseline (r = -0.539, P = 0.107) and post-intervention test 1 or 2 (r = -0.002, P = 0.995; r = -0.342, P = 0.407, respectively). No change was evident in RRE for the EMT group throughout the study.

A



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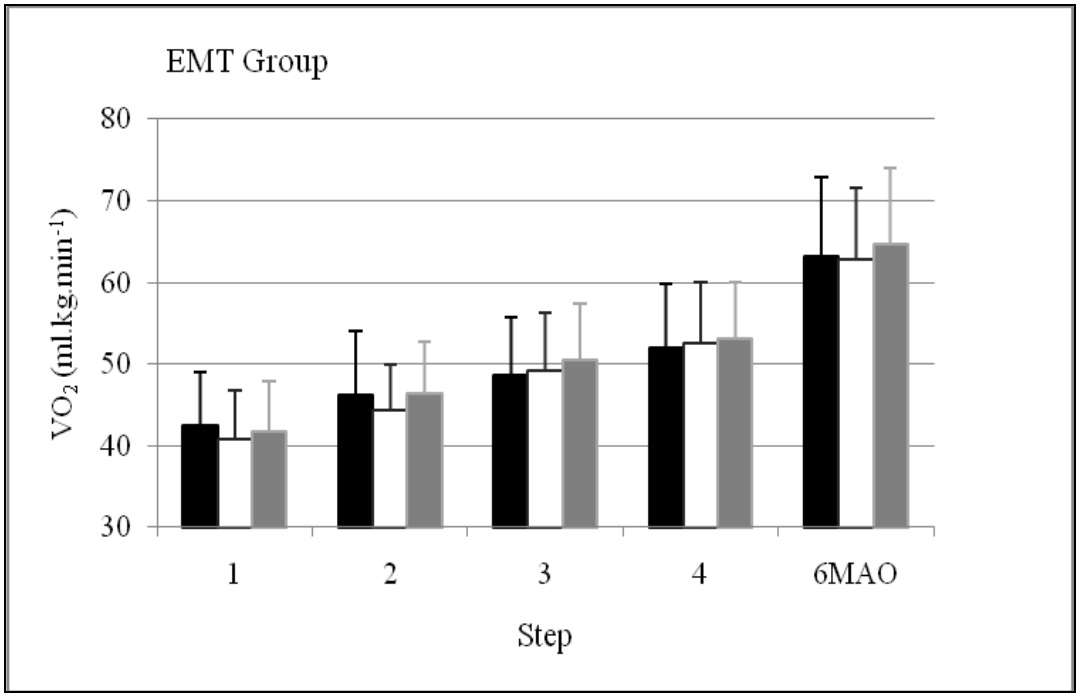
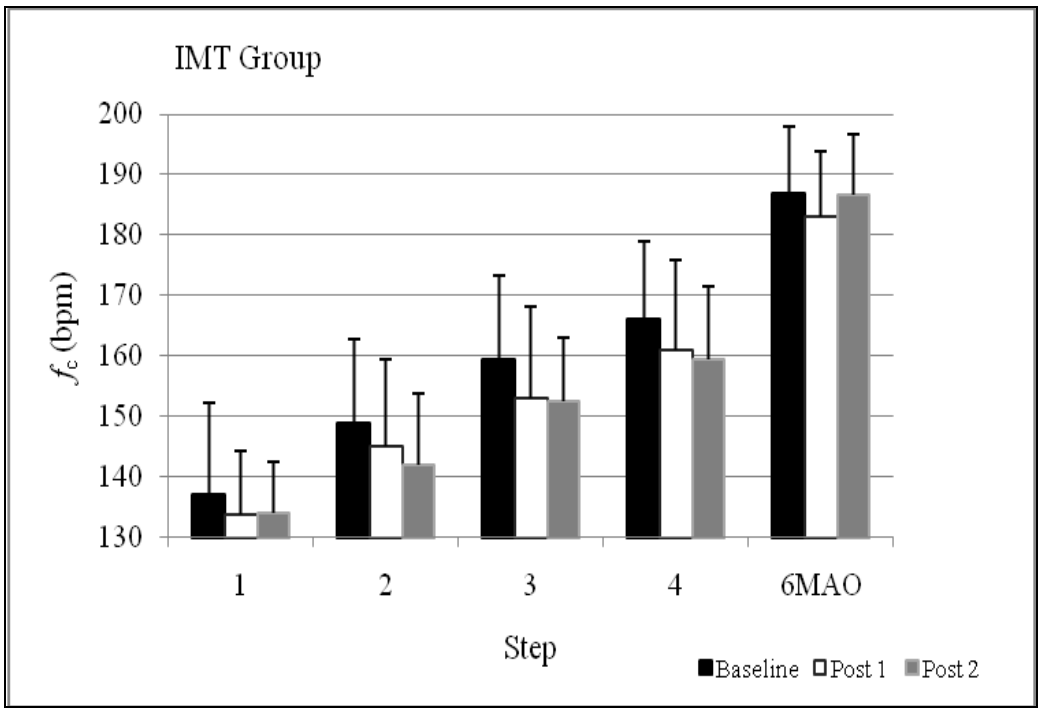


Figure 4.5 Comparison of peak oxygen uptake ($\dot{V}O_{2peak}$) during the step test and 6MAO effort..

A



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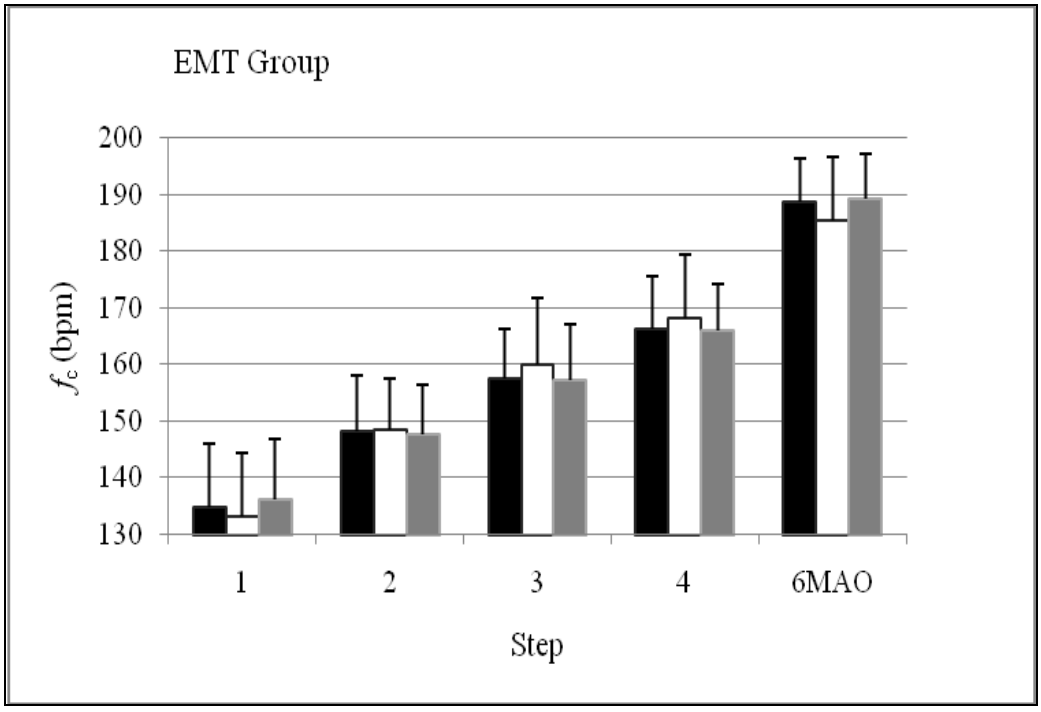
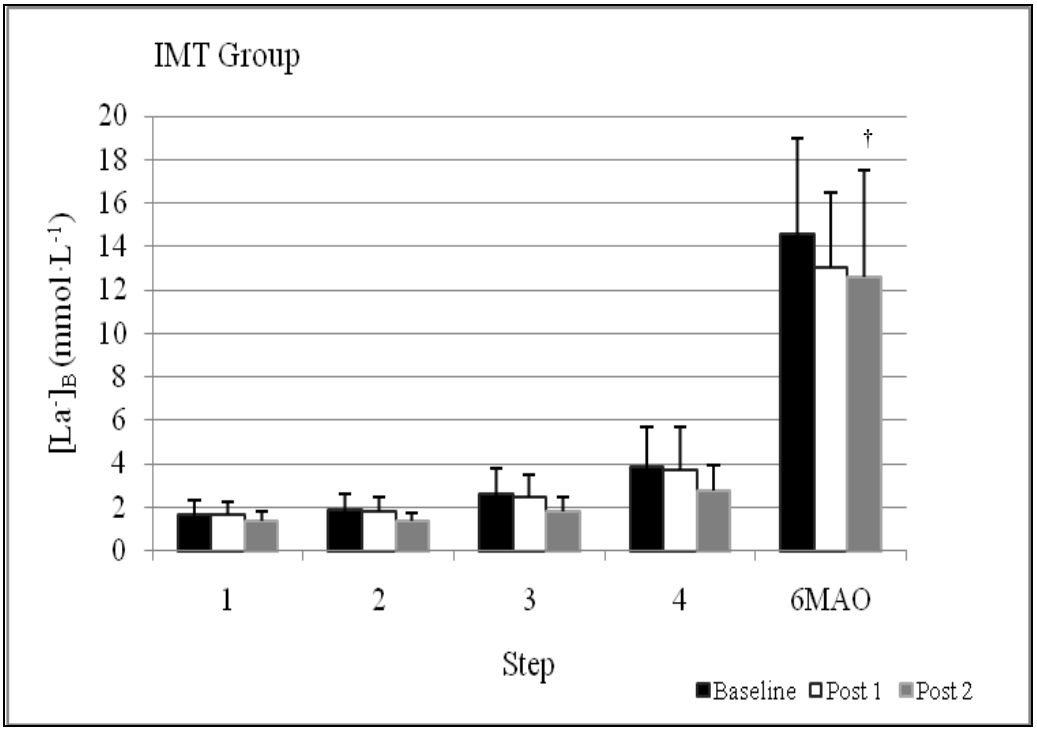


Figure 4.6 Comparison of heart rate (f_c) during the step test and 6MAO effort.

A



B

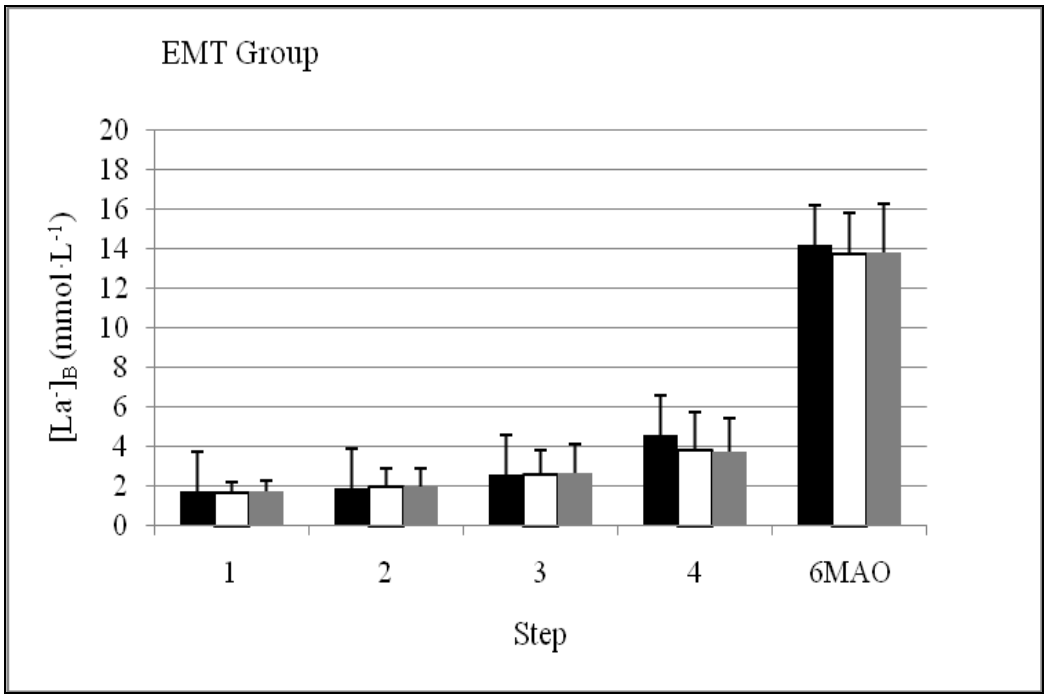
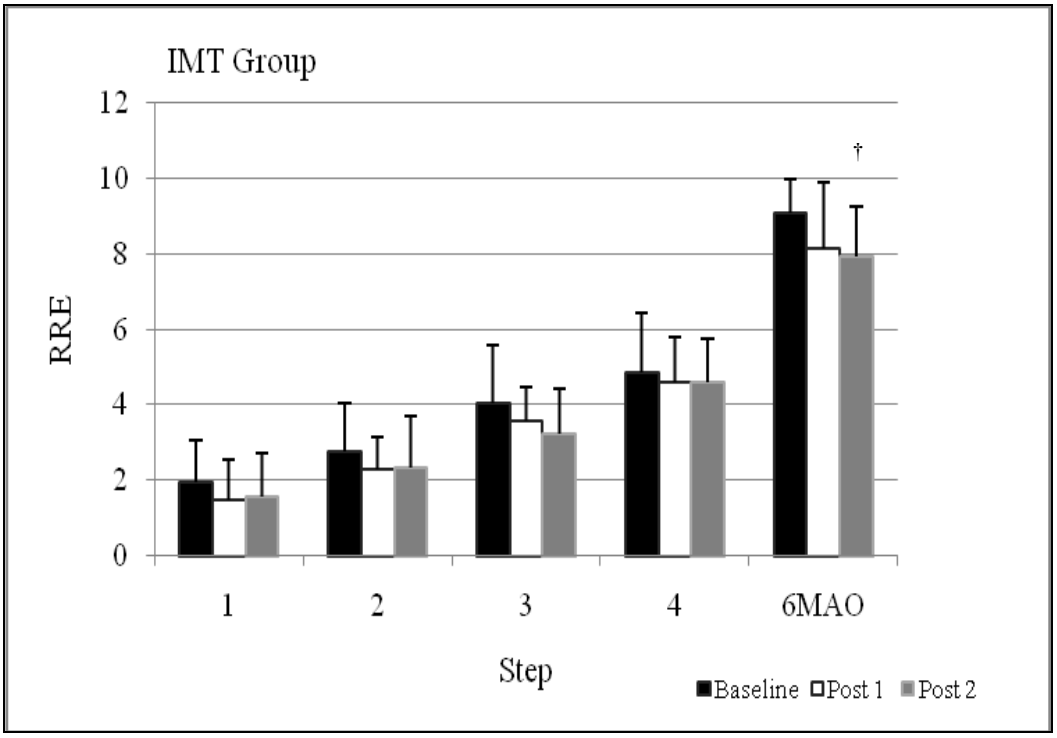


Figure 4.7 Comparison of earlobe blood lactate concentration ($[La^-]_B$) during the step test and 6MAO effort. Note:.†, significantly different from the preceding test ($p \leq 0.05$).

A



B

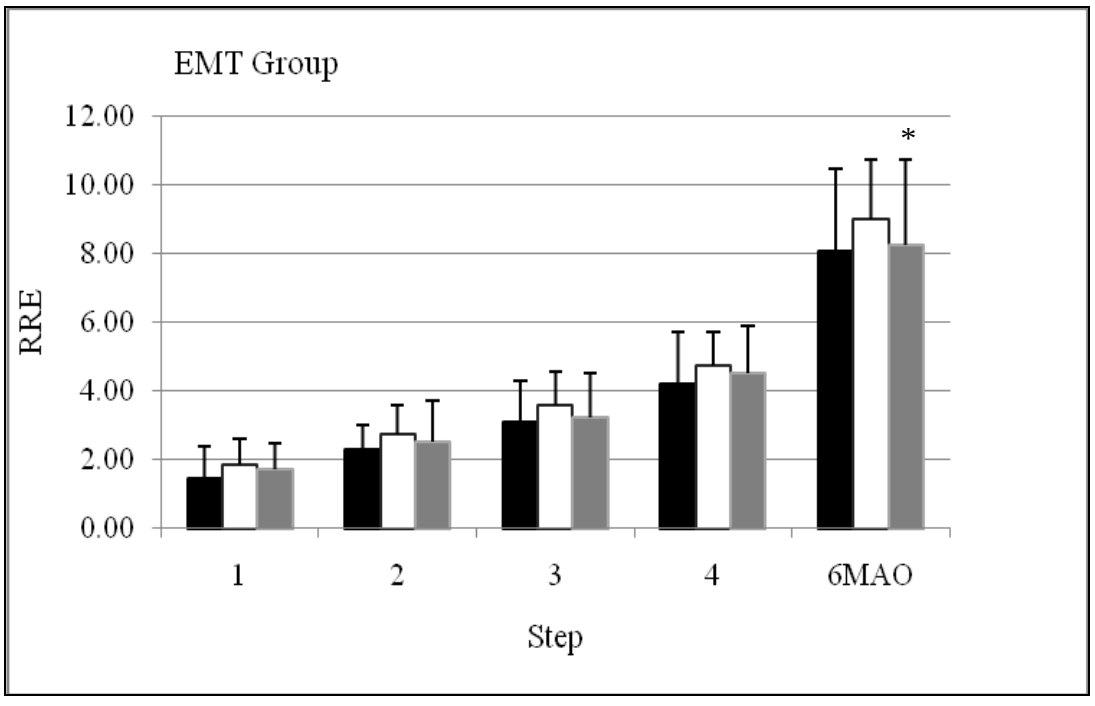


Figure 4.8 Comparison of rating of respiratory effort (RRE) during the step test and 6MAO effort. Note: *, significantly different from baseline ($p \leq 0.05$); †, significantly different from the preceding test ($p \leq 0.05$).

4.3.5: ROWING ERGOMETER PERFORMANCE

There were no differences between groups after the 4 wk of training and the combined training period for mean power output ($P = 0.053$), distance ($P = 0.081$) and split time ($P = 0.058$) in the 6MAO effort.

The IMT group showed a 2.7% improvement in mean power output during the 6MAO effort after the IMT phase ($P = 0.015$), with no further improvement with the addition of EMT (1.6% increase; $P = 0.076$) (fig. 4.9*A*). No change was evident in the EMT group after either training phase.

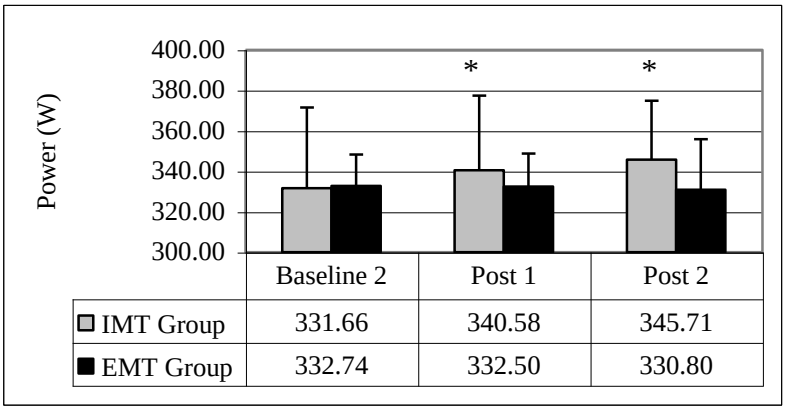
The IMT group improved their overall distance travelled in the 6MAO efforts by 0.92% following 4 wk of IMT ($P = 0.019$). The total increase in overall distance travelled during the 6MAO effort was 26.1 m (1.1 ± 1.4%) in the IMT group following the combined IMT/EMT (fig. 4.9*B*); whereas, no change was evident in the EMT group following training (0.2 ± 1.7%).

Following the IMT phase, the change in IMT group split time (0.9 s, 0.88%; $P = 0.023$) improved compared to the EMT group (no change) during the 6MAO effort (fig. 4.9*C*). The IMT group continued to improve their split times following the combined IMT/EMT phases of training (1.7%; $P = 0.004$). No change in split time was evident in the EMT group at any testing stage.

Figures 4.10 and 4.11 provide a visual comparison of the changes in RMS to distance (representing the change in rowing performance) from Baseline test 2 to Post-intervention 1 and from Post-intervention 1 to Post-intervention 2, respectively.

Arrows pointing up and toward the right demonstrate an association between improvement in P_{Imax} and improvement in rowing performance (distance). This pattern was apparent for the majority of the IMT group. In contrast, only one participant in the EMT group displayed this pattern; improvements in P_{Emax} were not accompanied by improvements in distance.

A



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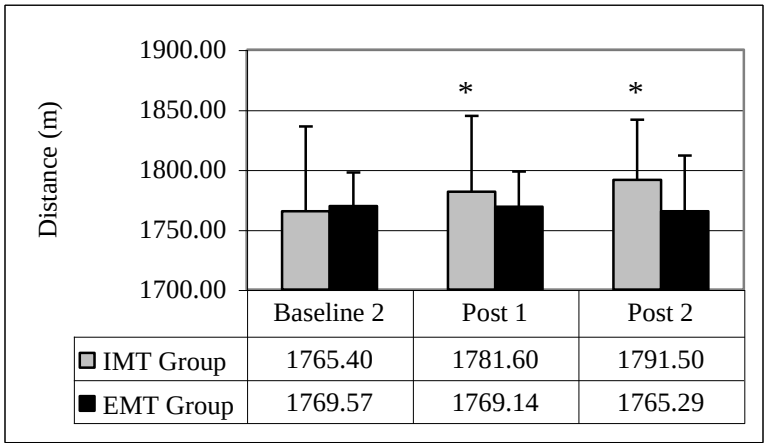


C

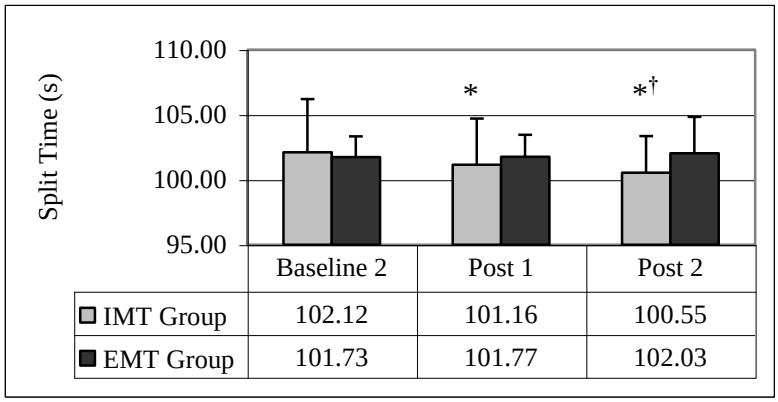
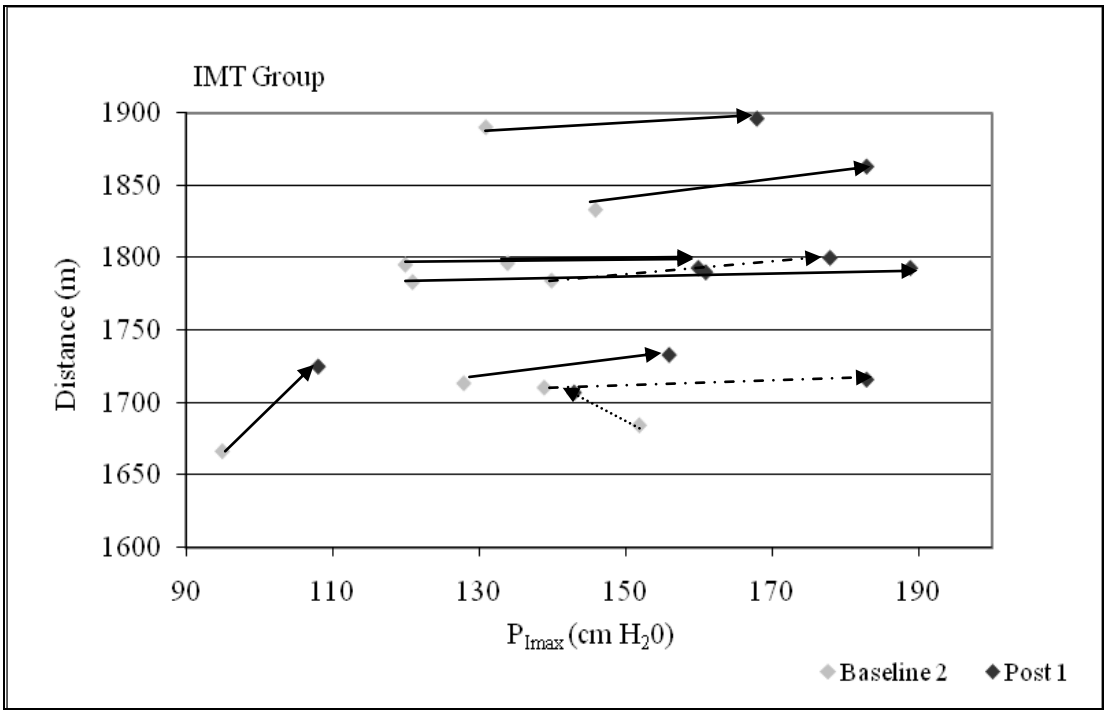


Figure 4.9 Comparison of changes between groups during the 6MAO effort. *A*, power output; *B*, distance; *C*, split times Note: Baseline 2 and Post 1: IMT, n = 10; EMT, n =7; Post 2: IMT, n = 8; EMT, n = 7; *, significantly different compared to baseline ($p \leq 0.05$); †, significantly different from the preceding test ($p \leq 0.05$).

A



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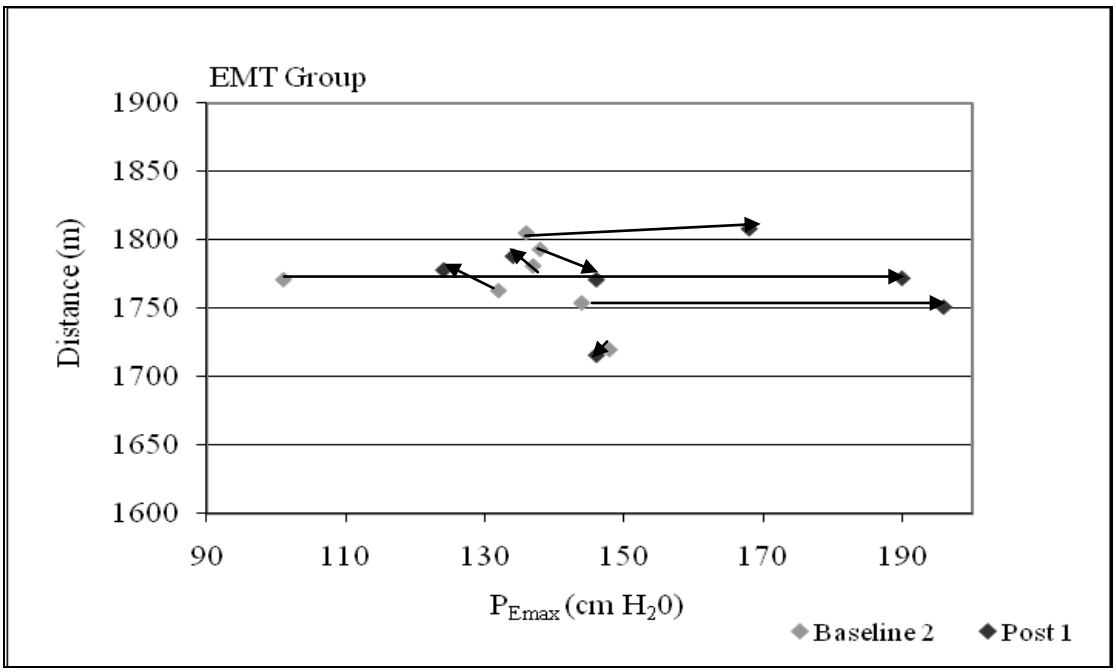
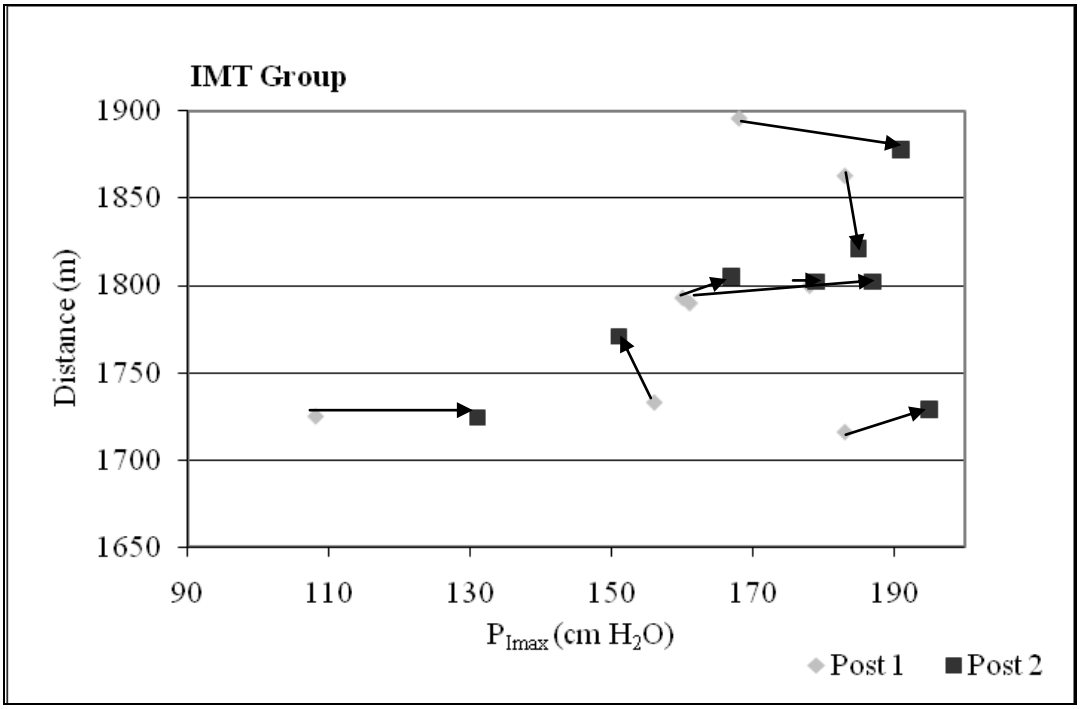


Figure 4.10 Comparison of respiratory pressures from Baseline 2 to Post-intervention 1. *A*, comparison of P_{Imax} vs. Distance (n=10); *B*, comparison of P_{Emax} vs. Distance (n=7). Note: dashed line in figure *A* represents participants that did not complete Post-intervention 2.

A



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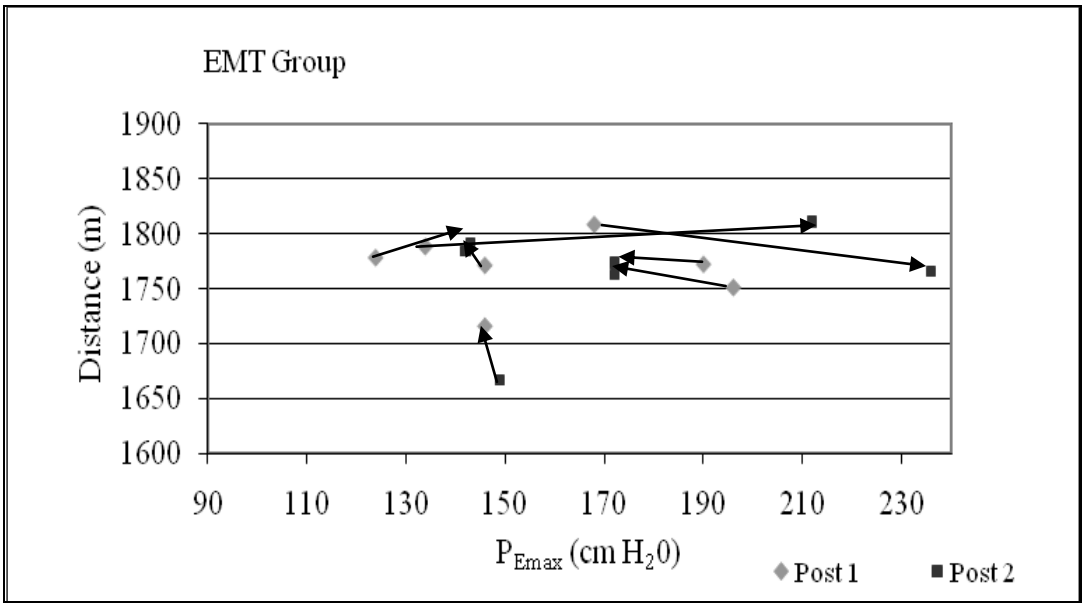


Figure 4.11 Comparison of respiratory pressures from Post-intervention 1 to Post-intervention 2**.** *A*, comparison of P_{Imax} vs. distance (n = 8); *B*, comparison of P_{Emax} vs. distance (n = 7).

There was no correlation between the individual changes in P_{Imax} and any index of rowing performance between baseline and post-intervention test 1 or 2. Similarly, there was no correlation between the absolute or percentage change in rowing performance and the changes in VO_2, f_c, $[La^-]_B$, or RRE between baseline and post-intervention test 1 or 2 (see table 4.10 & 4.11).

Table 4.10 Correlations among percentage changes in physiological variables and rowing performance indices at Post-test 1.

		1	2	3	4	5	6	7
IMT-group								
1. f_c	Pearson Correlation		-0.082	-0.179	-0.346	-0.489	-0.513	0.536
	Sig. (2-tailed)		0.822	0.620	0.327	0.151	0.129	0.110
2.RRE	Pearson Correlation			-0.164	-0.156	-0.307	-0.293	0.279
	Sig. (2-tailed)			0.650	0.667	0.389	0.411	0.435
3.$[La^-]_B$	Pearson Correlation				-0.210	0.248	0.246	-0.246
	Sig. (2-tailed)				0.560	0.491	0.494	0.493
4. $\dot{V}O_2peak$	Pearson Correlation					-0.010	-0.001	-0.005
	Sig. (2-tailed)					0.979	0.997	0.989
5.Avg watts	Pearson Correlation						0.999**	-0.998**
	Sig. (2-tailed)						0.000	0.000
6.Distance	Pearson Correlation							-1.000**
	Sig. (2-tailed)							0.000
7.Split time	Pearson Correlation							
EMT-group								
1. f_c	Pearson Correlation		0.364	0.333	0.691	-0.397	-0.389	0.364
	Sig. (2-tailed)		0.422	0.465	0.086	0.378	0.389	0.422
2.RRE	Pearson Correlation			0.484	0.048	0.063	0.066	-0.080
	Sig. (2-tailed)			0.271	0.919	0.893	0.888	0.865
3.$[La^-]_B$	Pearson Correlation				0.623	-0.454	-0.451	0.439
	Sig. (2-tailed)				0.135	0.306	0.309	0.325
4. $\dot{V}O_2peak$	Pearson Correlation					-0.577	-0.564	0.542
	Sig. (2-tailed)					0.175	0.187	0.208
5. Power output	Pearson Correlation						1.000**	-0.999**
	Sig. (2-tailed)						0.000	0.000
6.Distance	Pearson Correlation							-0.999**
	Sig. (2-tailed)							0.000
7.Split time	Pearson Correlation							
	Sig. (2-tailed)							

Note: f_c, heart rate; RRE, rating of perceived respiratory effort; $[La^-]_B$, blood lactate; $\dot{V}O_2max$, maximal oxygen consumption. Sig., significance.**, significant at $p < 0.000$.

Table 4.11 Correlations among percentage changes in physiological variables and rowing performance indices at Post-test 2.

		1	2	3	4	5	6	7
IMT-group								
1. f_c	Pearson Correlation		0.211	-0.267	-0.145	-0.633	-0.600	0.672
	Sig. (2-tailed)		0.616	0.522	0.732	0.092	0.116	0.068
2.RRE	Pearson Correlation			-0.498	0.126	-0.452	-0.422	0.432
	Sig. (2-tailed)			0.209	0.767	0.261	0.298	0.285
3. $[La^-]_B$	Pearson Correlation				0.580	0.250	0.179	-0.221
	Sig. (2-tailed)				0.131	0.550	0.671	0.599
4. $\dot{V}O_2peak$	Pearson Correlation					-0.257	-0.360	0.252
	Sig. (2-tailed)					0.540	0.380	0.547
5.Avg watts	Pearson Correlation						0.959**	-0.998**
	Sig. (2-tailed)						0.000	0.000
6.Distance	Pearson Correlation							-0.945**
	Sig. (2-tailed)							0.000
7.Split time	Pearson Correlation							
EMT-group								
1. f_c	Pearson Correlation		-0.016	0.621	0.699	-0.092	-0.104	0.102
	Sig. (2-tailed)		0.973	0.137	0.081	0.844	0.825	0.828
2.RRE	Pearson Correlation			0.080	-0.219	-0.403	-0.383	0.363
	Sig. (2-tailed)			0.864	0.637	0.370	0.397	0.423
3. $[La^-]_B$	Pearson Correlation				0.246	-0.641	-0.652	0.649
	Sig. (2-tailed)				0.595	0.121	0.113	0.115
4. $\dot{V}O_2peak$	Pearson Correlation					0.273	0.265	-0.273
	Sig. (2-tailed)					0.554	0.565	0.553
5.Avg watts	Pearson Correlation						1.000**	-0.999**
	Sig. (2-tailed)						0.000	0.000
6.Distance	Pearson Correlation							-1.000**
	Sig. (2-tailed)							0.000
7.Split time	Pearson Correlation							

Note: f_c, heart rate; RRE, rating of perceived respiratory effort; $[La^-]_B$, blood lactate; $\dot{V}O_2max$, maximal oxygen consumption. Sig., significance.**, significant at $p < 0.000$.

4.4: DISCUSSION

4.4.1: MAIN FINDINGS

The aim of this study was to differentiate the influence of IMT, EMT and concurrent IMT/EMT programme upon rowing ergometer performance and the physiological response to maximal and submaximal rowing. After the initial phase of IMT or EMT, the IMT group showed a 26% improvement in P_{Imax}, which was accompanied by a significant improvement in mean power output (2.7%) and distance completed (0.92%) in the 6MAO rowing test. During combined IMT/EMT, the IMT group showed a further small improvement in P_{Imax} (to 30%), and a significant improvement in P_{Emax} (to 23%).

The EMT group showed an 18% improvement in P_{Emax} following EMT, although it is evident from Figure 4.10*B* that the response showed large inter-subject variability. Despite changes in P_{Emax} in some participants, no change was found in either their rowing performance, or in any of the physiological variables assessed. After combined IMT/EMT (post-intervention test 2), the EMT group showed a further increase in P_{Emax} (to 31%) and a non-significant improvement in P_{Imax} (to 13%), but no associated change in rowing performance. The relative temporal patterns of the changes in RMS and rowing performance leads to the conclusion that IMT induced the greatest improvements in rowing performance following combined IMT/EMT.

4.4.2: INTER-TEST PRECISION

The 95% ratio limits of agreement (Bland & Altman, 1986) were used to determine within-subject variation for pulmonary function, RMS, physiological variables and performance data. The range defined by the limits of agreement provides a reference

range for differences between measurements; any change beyond the stated limits indicates that a real change has occurred due to the training or intervention (Hopkins, 2000).

When comparing the pulmonary function values in Table 4.2, the FEF_{25} and FEF_{50} had the best agreement with an insignificant bias of 0.99 and 1.05 and very good agreement ratios (x/÷1.18 and 1.21, respectively). This is not surprising since these values are effort independent. However, the effort dependent variables, PIF and PEF, had the worst agreement with the same ratio bias of 0.93 (agreement ratio x/÷ 1.34). Romer & McConnell (2004) also showed a poor agreement ratio (x/÷ 1.15) in PEF in their study on the inter-reliability of respiratory muscle function. The agreement ratio means that if the participants retested PIF or PEF they are likely to achieve results 1.34 times larger (or smaller) than their measurements compared to their baseline tests.

The values for P_{Imax} and P_{Emax} in Table 4.3 had the worst agreement with a large ratio bias of 0.85 and 0.89 with an agreement ratio of (x/÷) 1.31 and 1.22, respectively. The coefficient of variation (CV) for P_{Imax} was 12.4%, which is higher in comparison to the 8.7% reported by Aldrich & Spiro (1995) in 10 healthy participants. The wide range of values for P_{Imax} is high when compared to similar studies, which have shown a much greater agreement between tests (Maillard et al., 1998; Romer & McConnell, 2003; Volianitis et al., 2001b). Romer & McConnell (2003) demonstrated an almost perfect inter-test reliability with P_{Imax} and P_{Emax} agreement ratios ranging from (0.99 – 1.00), stating that both variables can be measured with equal reliability.

The inconsistency in the reliability values for P_{Imax} and P_{Emax} remains unclear, but procedural and population differences may be part of the reason (Romer & McConnell, 2003). However, in our study, the large systematic bias ratio most likely indicates that a general learning effect occurred between the two baseline tests. Larson, Covey, Vitalo, Alex, Patel & Kim (1993) has shown similar results in which they reported P_{Imax} improved with practice in 91 naïve COPD patients; P_{Imax} improved by 9 cm H_2O from the first to the fourth trial, but performance appeared to plateau between the third trial and fourth trial.

Table 4.4 summarised the limits of agreement for the physiological variables tested during the 6MAO effort. The RRE had a perfect agreement with a ratio and agreement bias of x/÷1.00. Both mean power and distance (Table 4.5) had a negligible ratio bias of 1.00 and 0.99 and an excellent agreement ratio (x/÷1.07 to 1.05) showing good reliability for rowing performance measurements. However, $[La^-]_B$ had the least agreement with a ratio bias of 0.96 and a poor agreement ratio of x/÷1.6 (CV = 8.75%). The lack of within-day measurements and the low reliability observed for the between-day measurements (see Table 4-5), severely limits the application of the $[La^-]_B$ findings.

With only 17 participants (IMT-group = 10; EMT-group = 7), it is possible to assume that a type 2 error may have occurred in assessing significance in changes with small effect magnitudes. The reliability data provided estimated sample sizes used to identify if the current sample size was too small to detect meaningful changes in the measured variables (see table 4.2). Although variables including $\dot{V}O_2$, peak end-stage f_c and mean power did not achieve statistical significance, they all had small

magnitudes of effect (< 4.7%). Therefore, it would be reasonable to assume that a larger sample size may have provided statistical significance in these variables.

4.4.3: CHANGES IN RESPIRATORY MUSCLE STRENGTH

INSPIRATORY MUSCLE STRENGTH

The IMT group showed progressive improvement in P_{Imax} at both post-intervention testing sessions (totalling 30%); these results are similar in magnitude to those of previous studies using pressure-threshold training, which range from 15-45% (Edwards et al., 2008; Volianitis et al., 2001c). After the combined training programme, the IMT group displayed a small non-significant improvement in P_{Imax} of 3%. This small change in improvement was most likely due to the 'plateau' effect experienced in the skeletal and respiratory muscles after 4-6 wk of the same training; this is consistent with previous studies (Romer & McConnell, 2003; Volianitis et al., 2001c).

Following the EMT-only phase, the EMT group showed a 5.6% improvement in $P_{Imax.}$ Although they were not directly training the inspiratory muscles this slight increase is consistent with previous observations that it is impossible to load the expiratory muscles without also loading the inspiratory muscles (Taylor & Romer, 2006, 2009). Following the combined IMT/EMT phase, the EMT group showed only a small, but significant, increase in P_{Imax} (to 13%). This suggests that an improvement in P_{Imax} > 13% may be necessary before an ergogenic effect is observed. This is consistent with data from Hart, Sylvester, Ward, Cramer, Moxham & Polkey (2001) who showed no change in incremental treadmill performance after a 6 wk IMT that increased P_{Imax} by 10%. From the results reported by authors that have seen a change in performance, it

seems that an increase > 15% in P_{Imax} is necessary for a change in physical performance (see table 2.1).

EXPIRATORY MUSCLE STRENGTH

Maximal expiratory pressures (P_{Emax}) increased in both groups, with the greatest overall improvement in the EMT group (31% over both training phases), along with a noticeable decrease in EMF. During the combined IMT/EMT phase, the IMT-group showed an increase in P_{Emax} of 23% ($P < 0.05$) compared to Post-intervention 1. We hypothesised this improvement in the EMT group during both phases of training and the IMT group during the 2nd phase. Unlike the plateau evident in the IMT-group in P_{Imax} after IMT-only training, the EMT-group continued to increase expiratory muscle strength at both phases of the training intervention. One possible explanation for the lack of plateau in expiratory muscle strength may be that the training load prescribed for the EMT group was based on previous IMT literature. To date, no published literature has provided evidence to support a specific training protocol for pressure threshold EMT; therefore the training load prescribed may not have been sufficient enough to fully overload the muscles. The inclusion of IMT during the combined training phase may have increased the overall training intensity thereby eliciting further improvements in P_{Emax}.

Although there was an improvement in P_{Emax} and EMF in the EMT group there was no evidence that EMT provided a significant improvement in any of the physiological outcomes tested. Similarly, there was no significant improvement in rowing performance observed in the EMT group after either method of training.

4.4.4: ADHERENCE TO RESPIRATORY TRAINING

During IMT or EMT only, adherence was comparable between groups; however, a noticeable decrease in training adherence was apparent in the EMT group during the combined IMT/EMT (from 77% of prescribed sessions to 60%). The decrease in mean adherence was partly due to two of the EMT participants contracting upper respiratory infections during the combined IMT/EMT. However, it is also possible that a decrease in motivation may have played a part, as the EMT group realised that EMT yielded no change in their rowing performance. In contrast, the IMT group perceived the benefits of IMT and this might have encouraged them to train more diligently during the combined IMT/EMT.

At Post-intervention 2, participants from both groups reported anecdotally that they had found the combined IMT/EMT exhausting and they often found it difficult to complete the entire training session. This most likely explains the smaller improvement observed in P_{Imax} during this phase (to 13%) compared to 1st phase for the IMT group (to 26%). Oftentimes, participants reported stopping during the 30 repetitions to recover by taking full satisfying breaths. They also reported that RMT was often more challenging after resistance or water training sessions.

4.4.5: RESPIRATORY MUSCLE FATIGUE

The lack of improvement in IMF following IMT is in contrast to previous studies, where IMF has been virtually abolished (Romer et al., 2002b; Volianitis et al., 2001c). Even with an improvement in 6MAO performance, the PostEx-P_{Imax} was virtually unchanged following the IMT phase, but was attenuated after the IMT/EMT phase. There are two potential explanations for this: 1) the attenuation of IMF does not play a

role in the improved performance following IMT and 2) that the pre-exercise measurements of P_{Imax} were not truly maximal. As the participants did not perform a specific respiratory warm-up (RWU) prior to the start of testing for P_{Imax}, the muscles may have not been fully prepared to perform a maximal manoeuvre. The poor inter-test reliability of the P_{Imax} data would tend to support this. Volianitis, McConnell, Koutedakis & Jones (2001a) showed that inspiratory muscles show evidence of a 'warm-up' effect in response to repeated measurements, but that this can be minimised by performing a specific RWU to negate the 'learning effect', which contributes to test variability. To overcome this, Volianitis et al. (2001b) recommend a specific RWU using a pressure threshold loading device for 2 sets of 30 breaths at 40% P_{Imax} prior to maximal inspiratory testing. Indeed, a recent study has demonstrated that this inspiratory warm up protocol facilitates the between day reliability of P_{Imax} measurements (Lomax & McConnell, 2009).

In contrast, EMF showed attenuation after EMT. It is possible that the P_{Emax} measurements were more representative of maximal values because they were always preceded by P_{Imax} measurements, which required 'squeezing' down to RV. Thus, the P_{Emax} measurements may not have been affected in the same manner as the P_{Imax} values.

4.4.6: PERFORMANCE IMPLICATIONS

After the initial phase of IMT or EMT, only the IMT group demonstrated an improvement in indices of rowing performance during the 6MAO effort, with only a slight further decrease in split time after combined IMT/EMT. These results are

consistent with the 'plateau' in P_{Imax} observed by Romer et al. (2002a) and performance by Volianitis et al. (2001c) after 4-6 wk of IMT.

The EMT group showed no improvement in rowing performance at any time during the study. This is in contrast to Miller (2005), who observed an improvement in both the experimental ($p < 0.05$) and sham-training group in P_{Emax} (32% and 22%, respectively) and 2 km rowing performance (+4.6% and +3.1%, respectively; $P > 0.05$) following 5 wk EMT. However following training, both groups had similar magnitudes of improvement in expiratory muscle strength and rowing performance, in which there was no significant difference between groups in either variable. The authors speculated that this may have been due to the sham-training load being set to high (15% of P_{Emax}) in which both groups underwent a sufficient training load to induce strength gains. However, it renders the data inconclusive as to whether the improvements in 2 km rowing performance were due to EMT or the athletes' whole body and rowing training programme.

Collectively, the data suggests that improvement in P_{Imax} was associated with a significant improvement in functional performance; whereas the significant increase in P_{Emax} was not associated with changes in any parameter of rowing performance. Thus, the data suggest that improvements in performance following RMT are most likely ascribable to training-induced changes in inspiratory muscle function.

4.4.7: CHANGES IN PHYSIOLOGICAL VARIABLES

No significant change was found in $\dot{V}O_2$ in either training group; this may have been due to small sample sizes as addressed in section 4.4.1. However, there was a change

in cardiovascular reflex response during submaximal power outputs in the IMT group, in the form of a (non-significant) decrease in peak end-stage f_c (~3-6 bpm) during the 'step test'. Simply stated, although there was no significant change in metabolic response ($\dot{V}O_2$) there was a decrease in the cardiovascular strain (decrease in f_c) at the same work intensity.

These observations in peak f_c are consistent with those of previous investigators. Gething et al. (2004b) observed a decrease in exercising f_c of ~6 ± 9 bpm at the end of a 5 min bout of constant power cycling after their participants performed 6 wk of IMT that increased P_{Imax} by 29%. Haas & Haas (1981) also observed a lower f_c and $\dot{V}O_2$ during submaximal exercise after 16 d combined programme of voluntary isocapnic hyperpnoea (VIH) and inspiratory resistance loading (IRL) in healthy adults. Similarly, Swanson et al. (1998) reported a decrease in f_c after 6 wk of VIH intervention. The mechanism by which f_c might decrease following IMT is unknown, but one possible explanation for changes in cardiovascular response to exercise is an improvement in inspiratory muscle efficiency following IMT, thereby preserving blood flow to the working muscles (i.e. leg muscles), and/or a delay/attenuation of the metaboreflex induced increase in sympathetic vasomotor outflow that follows activation of the inspiratory muscle metaboreflex (McConnell & Lomax, 2006; Sheel et al., 2001; Witt et al., 2007).

The shift in the $[La^-]_B$ curve of the IMT group after IMT (downwards and to the right), is reminiscent of the training response of $[La^-]_B$ in response to whole body endurance exercise training. Spengler et al. (1999) made similar observations following 4 wk of VIH in healthy participants. Our findings of a combined improvement in P_{Imax} (31%)

with a decreased in $[La^-]_B$ following the 6MAO (13.7%) is very similar to the findings of Romer et al. (2002a), in which they also observed an improvement of PreEx-P_{Imax} of 31% in the training group with a 15.7% change in $[La^-]_{B.}$ These data would suggest that IMT reduces $[La^-]_B$ concentration during exercise, however we did not observe a significant correlation between P_{Imax} and $[La^-]_B$ after IMT-only (r = 0.614, *P* = 0.059).

Spengler et al. (1999) suggested the reason for RMT-induced changes in the lactate curve was that trained respiratory muscles use more lactate as fuel to maintain work output. Since there was no further decrease after EMT, or combined IMT/EMT, it appears likely that decreases in $[La^-]_B$ are attributable only to IMT. Although there was a reduction in $[La^-]_B$ and an improvement in rowing performance the data do not support a causal relationship between changes in $[La^-]_B$ and changes in performance.

Since RPE corresponds to peak f_c and $[La^-]_B$ (Borg, Hassmen & Lagerstrom, 1987) the changes in the RRE for the IMT group may be explained by the reduction of both of these variables after IMT alone. However, we did not observe a significant correlation between RRE and performance after training (P > 0.05). Other possible explanations for the decrease in RRE may be an altered perception of their breathing effort or an improvement in the physiological conditioning of the inspiratory muscles after training, which has been suggested by other studies finding a decrease in respiratory effort following IRL and IMT (Huang et al., 2009; Kellerman, Martin & Davenport, 2000; Sheel et al., 2001; Volianitis et al., 2001c).

4.4.8: ANECDOTAL SUBJECTIVE FEEDBACK

EMT participants stated they felt little benefit from EMT alone and stated there was no subjective change in their rowing performance. However, the IMT group often stated they felt a decrease in breathlessness during exercise. All participants stated that the combined IMT/EMT was challenging and oftentimes difficult to complete a full set of 30 repetitions without stopping.

4.5: CONCLUSION

The present study compared the effects of IMT and EMT in healthy young adult subjects. We observed no apparent benefits of EMT, either alone, or in combination with IMT. However, the use of two different training devices may have introduced an unnecessary variable to the study; it would have been preferable to have performed IMT-only and EMT-only with the same training device. Even so, the data suggests that the positive changes in rowing performance and physiological variables observed were due to functional improvements within the inspiratory muscles over the testing period, accompanied by improvement in factors such as f_c, $[La^-]_B$ and RRE. This is an interesting finding, as Derchak et al. (2002) showed the existence of a metaboreflex originating in expiratory muscles, similar to that found in the inspiratory muscles (Sheel et al., 2001; St. Croix et al., 2000). If preventing this reflex were part of the mechanistic basis of the benefits of RMT, then we would expect similar physiological changes with both IMT and EMT. It is therefore surprising that EMT did not improve rowing performance or any of the physiological variables tested in this study. A possible explanation for this may reside in the training status of the expiratory muscles of most well trained individuals, especially rowers. The expiratory muscles of the abdominal wall already form part of the conditioning programmes of athletes, and it may be that this training is sufficient to raise the expiratory muscle

metaboreflex threshold such that it is not stimulated during activities that initiate the inspiratory muscle metaboreflex.

Our findings suggest that the significant increases in inspiratory muscle strength were associated with an improvement in rowing performance following IMT-only training. Furthermore, the study demonstrated that IMT alone is more effective than EMT for improving rowing performance. More research is required to determine if the effects of EMT or combined IMT/EMT could benefit other sports, however, these results suggest that EMT does not provide a functional benefit for rowing performance.

CHAPTER FIVE

INFLUENCE OF POSTURE ON RESPIRATORY MUSCLE PRESSURES AND LUNG FUNCTION

5.1: INTRODUCTION

During the rowing stroke, the respiratory muscles are responsible for postural control, trunk stabilisation, generation/transmission of propulsive forces and ventilation (Bierstaker et al., 1986; Mahler et al., 1991b). The challenge of these sometimes competing requirements is exacerbated in certain parts of the rowing stroke due to flexed and extended postures. For instance, Szal and Schoene (1989) suggested that the high exercise ventilation during rowing was most likely due to the variable seated position and that this may alter respiratory timing and mechanics (i.e. entrainment of breathing) in order to meet the increasing oxygen demands. Siegmund et al. (1999) investigated the stroke-by-stroke and breath-by breath inter-relationships of male varsity rowers in an effort to understand the entrained breathing pattern adopted by these athletes. The authors observed two dominant breathing strategies. In the first strategy, the subjects maintained peak expiratory flow (PEF) rates, but used short duration breaths during the recovery phase of the stroke and long duration breaths during the drive. The second strategy relied upon maintaining inspiratory reserve volume (IRV), whilst generating high PEF rates during the drive and low PEF rates during recovery. They showed that changes in peak flow rates and V_T depended upon when the breath was initiated during the stroke cycle; trained rowers tended to adopt an entrained breathing pattern at what the authors considered to be the most advantageous times of the stroke for large inspired and expired volumes (Siegmund et al., 1999). Further, they postulated that the observed decrease in peak inspiratory flow (PIF) rates at the end of the drive indicated there was a decrease in the vital capacity (VC) at stroke 'finish', and not at the 'catch' as was thought previously (Cunningham et al., 1975). It is unclear what underlying mechanism(s) contribute to the hypothesised decrease in VC in the 'finish' position, but it may be attributable to

changes in inspiratory and/or expiratory muscle mechanics, which may in turn be due to the competing postural and respiratory roles of the respiratory muscles at the 'finish'.

Hence, this study is specifically interested in the co-contraction of the postural role of the respiratory muscles during rowing. Particularly, if there is any impairment due to the simultaneous engagement of the respiratory muscles, which may cause a potential conflict between their postural and respiratory role. If there is a significant reduction in the respiratory muscle pressure and/or flow generating capacity in specific rowing postures, this would warrant further investigations into the effects of posture-specific respiratory muscle training (RMT), with the aim of maximising ergogenic benefits. Thus, the purpose of this study was to determine whether the respiratory muscle pressures and/or flow generating capacity are influenced in various seated postures relevant to rowing. In addition, the independent influences of postural muscle activation and body position will be examined by comparing responses to postures in the supported and unsupported states. A significant difference in respiratory muscle function at different postures performed during the rowing stroke may help to explain the data of Siegmund et al. (1999), as well as providing a rationale to investigate the possible benefits of RMT in those postures where function is impaired.

5.2: METHODOLOGY

5.2.1: PARTICIPANTS

Sixteen healthy adult participants, eleven males and five females, who regularly performed moderate to vigorous exercise, volunteered to participate in the study. All participants reported to the laboratory on two separate occasions. Nine of the

participants made four visits; these additional visits were used to collect reliability data to determine inter-test precision of within-subject variation of the testing procedures.

Testing was conducted at the physiology laboratory at Brunel University and Buckinghamshire New University. Written informed consent from all participants and School Ethics Committee approval were obtained prior to the start of testing sessions. A copy of the informed consent and health questionnaire can be found in Appendix A-1 and A-2. Participants were asked to refrain from vigorous exercise 24 hours prior to testing.

5.2.2: GENERAL DESIGN

Participants were asked to visit the laboratory to complete two different testing protocols (see figure 5.1). Prior to testing, all participants performed an inspiratory warm-up using an inspiratory muscle trainer (Volianitis et al., 2001b; see section 3.2.2). Testing Protocol 1 (T1) required the participants to perform either maximal inspiratory pressure (P_{Imax}) or maximal expiratory pressure (P_{Emax}) manoeuvres whilst maintaining a variety of specified static rowing-related postures. Testing Protocol 2 (T2) consisted of maximal flow volume loops (MFVLs) and P_{Emax} manoeuvres in the same postures used in T1.

Due to the nature of maximal mouth pressure testing, it would be impossible to perform these tests during dynamic activity (such as during the rowing stroke); therefore we were limited to performing the pulmonary testing in static postures. We recognise this would limit the external validity of the study, but would allow an

uncontaminated assessment of the postural role of the respiratory muscles in postures relevant to rowing.

Two static positions were chosen for comparison, the flexed 'catch' position and the extended 'finish' position, both of which have been cited as potentially impairing pulmonary function during the rowing stroke (Cunningham et al, 1975; Siegmund et al., 1999). The 'catch' position was defined as a 75º angle of flexion at the hip (but with legs straight), whilst the 'finish' postures were defined as extended hip angles of 110º, 130º and 150º. These positions were designed to simulate relevant postures performed during a normal rowing stroke. Three different 'finish' postures were utilised to incorporate individual variations of the 'finish' position. The postures were assigned randomly and were either 'supported' (S) by a bench or 'unsupported' (U). 'Unsupported' postures required the participants to sustain the specified posture against the force of gravity during the manoeuvres.

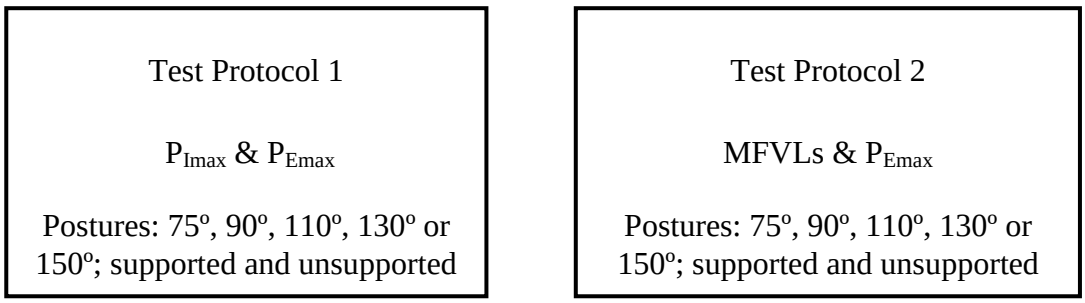


Figure 5.1 Schematic diagram of testing sessions.

5.2.3: PROCEDURES

A detailed description of the instrumentation and testing procedures are provided in Chapter 3.

ANTHROPOMETRY

Anthropometric measurements, including body mass and stature, were performed at the start of the study. Measurements recorded at baseline are presented in Table 5.1. A detailed description of the procedures for collecting anthropometric data is described in Chapter 3.2.2.

Table 5.1 Descriptive characteristics of the participants (mean ± SD).

	N	Age (yr)	Body mass (kg)	Stature (cm)
Males	11	25.6 ± 6.5	86.8 ± 18.7	181.7 ± 9.1
Females	5	23.6 ± 2.5	71.9 ± 15.7	175.4 ± 12.0

PULMONARY AND RESPIRATORY MUSCLE FUNCTION MEASUREMENTS

Prior to all testing sessions, participants performed an inspiratory muscle warm-up using an inspiratory muscle trainer. Pulmonary measurements included: P_{Imax}, P_{Emax} and MFVLs.

INSPIRATORY WARM-UP

Participants were instructed on the proper usage of the pressure threshold-loading device for the inspiratory warm-up (POWERbreathe®, Gaiam Ltd., Southam, UK). A detailed description of the warm-up procedures is outlined in section 3.2.2. No benefits of an expiratory muscle warm-up are currently available, so this was not implemented.

RESPIRATORY MUSCLE STRENGTH

Maximal inspiratory and expiratory mouth pressure manoeuvres (P_{Imax} and P_{Emax}, respectively) were measured as surrogates of inspiratory and expiratory muscle strength. Measurements were performed using a portable handheld mouth pressure metre (Micro Medical MPM, Micro Medical Ltd., Kent, UK). Regardless of the manoeuvre, all unsupported measurements were started in the 90º upright position. Participants were required to either inhale fully or exhale completely while in the upright position and were then manually positioned by the researcher into the specified unsupported posture. Once the participant was in the correct posture, the participant performed the manoeuvre. Participants were required to maintain head and neck alignment (head upright looking forward) for all respiratory manoeuvres. During all manoeuvres, the participants held the measuring device handle with one hand while the other hand was relaxed by their side. The procedure was repeated until two P_{Imax} or P_{Emax} values were reproduced within 3-5 cm H_2O. The highest reproducible value was recorded and presented in cm H_2O. A detailed description of the equipment and the procedures used for testing respiratory pressures are presented in section 3.2.2.

MAXIMAL FLOW VOLUME LOOP

Maximal flow-volume loop (MFVL) measurements were made using a handheld spirometer (MicroLoop, Micro Medical Ltd., Kent, UK). The following measures were recorded: PIF, PEF, forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1). Peak flows are presented in litres per minute ($L \cdot min^{-1}$). Forced expiratory volumes are presented in litres (L). A detailed description of the manoeuvre is described in section 3.2.2.

ASSESSMENT OF ROWING-RELATED POSTURES

The postures were determined by simulating a complete rowing stroke. The stroke was divided into three distinct phases: the 'catch', sitting upright and the 'finish'. Since oarsmen choose various points to complete the stroke, three different finish positions were chosen for analysis (Figure 5.2).

The rowing-related postures included hip flexion to 75º with legs straight (simulated 'catch' position), sitting upright at 90º and lumbar extension to 110º, 130º or 150º (the 'finish'). A goniometer was positioned on the supporting bench adjacent to the lumbar region of the spine to determine the joint angle of the back when performing the breathing manoeuvres.

Flexion of the hip to 75º was performed to simulate the upper body's action during the catch position of the rowing stroke. The straight legged position was used for two reasons: 1) lack of within-test reliability in standardising the compression of the abdomen and 2) to isolate the influence of postural control factors. Postures >90º are consistent with the normal range of back extension during the driving phase of the rowing stroke (Mahler et al., 1984). An extended range of motion to 150° was utilised to examine the fully extended position of each individual.

Participants were positioned on a bench, sitting upright with legs straight. Breathing manoeuvres were performed in either 'supported' (S) or 'unsupported' (U) postures. In total, there were eight different postures for each breathing manoeuvre: three 'supported' positions 110º, 130º and 150º (S-110º, S-130º and S-150º, respectively);

and five ‘unsupported’: 75º, 90º, 110º, 130º and 150º (U-110º, U-130º and U-150º, respectively).

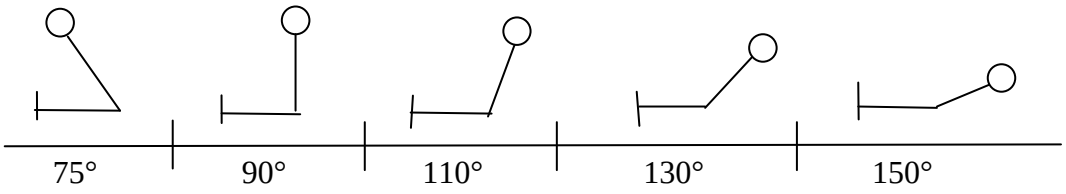


Figure 5.2 Schematic diagram of the rowing-relevant postures.

All ‘unsupported’ manoeuvres were initiated at 90º, and participants were required to prepare for their manoeuvre by inhaling to TLC or exhaling to RV, whereupon they moved into the required position as quickly as possible. Participants were required to start the manoeuvre with head facing forward and chin parallel to the floor. Using a goniometer, manual assistance was provided to get the participant into the appropriate posture. Once in the correct posture, participants performed the designated breathing manoeuvre and were required to maintain the specified posture and head and neck alignment (e.g., chin did not collapse to the chest) throughout the entire manoeuvre.

TESTING PROTOCOL

As shown in table 5.2 and 5.3, the postures were randomised and the manoeuvres were alternated allowing a timed one minute rest between each breathing manoeuvre, including a short rest break between each block of measurements. The participants were required to perform all measurements three times in one session. The order of the testing protocols (T1 and T2) was randomly assigned. The acceptable criteria for repeatability of volume and pressure measurements, as described in section 3.2.2, were applied.

Originally, we intended to assess an additional dynamic flow measurement in the T2 protocol, however due to technical failure with the device we were unable to continue with this measurement. As a small number of the participants already completed a portion of the testing with this measurement, it was decided to substitute this measurement with P_{Emax} to mimic the muscular effort required. Therefore, P_{Emax} was assessed in both protocols however only those values measured in the T1 protocol were used for analysis.

Table 5.2 Testing protocol 1 (T1) for assessment of respiratory pressures.

90º	**75º**	**110º**	**150º**	**130º**
1. U-P_{Imax}	2. U-P_{Emax}	3. U-P_{Imax}	4. S-P_{Emax}	5. S-P_{Imax}
6. U-P_{Emax}	7. U-P_{Imax}	8. S-P_{Emax}	9. S-P_{Imax}	10. U-P_{Emax}
		11. S-P_{Imax}	12. U-P_{Emax}	13. U-P_{Imax}
		14. U-P_{Emax}	15. U-P_{Imax}	16. S-P_{Emax}

Note: U-P_{Imax}, unsupported maximal inspiratory pressure; U-P_{Emax}, unsupported maximal expiratory pressure; S-P_{Emax}, supported maximal expiratory pressure; S-P_{Imax}, supported maximal inspiratory pressure.

Table 5.3 Testing protocol 2 (T2) for MFVLs.

90º	**75º**	**110º**	**150º**	**130º**
1. U-MFVL	2. U-P_{Emax}	3. S-MFVL	4. U-P_{Emax}	5. U-MFVL
6. U- P_{Emax}	7. U-MFVL	8. U- P_{Emax}	9. U-MFVL	10. S- P_{Emax}
		11.U- MFVL	12. S- P_{Emax}	13. S- MFVL
		14. S- P_{Emax}	15. S-MFVL	16. U- P_{Emax}

Note: U-MFVL, unsupported maximal flow volume loop; U-P_{Emax}, unsupported maximal expiratory pressure; S-MFVL, supported maximal flow volume loop; S-P_{Emax}, supported maximal expiratory pressure.

5.2.4: STATISTICAL ANALYSIS

Limits of agreement were used to ascertain the reliability of the respiratory pressures and pulmonary function measurements performed on two separate days (as described previously in section 3.2.6). A repeated measures analysis of variance (ANOVA) was used to determine intra-subject differences in outcome variables between postures. Planned pairwise comparisons were made to analyse significant interaction effects using the Bonferroni adjustment. Pearson's correlation coefficient was used to determine relationships between absolute variables and the percent change from 90º in the various postures. Probability values ≤ 0.05 were considered significant. All results are expressed in mean ± standard deviation (SD) unless otherwise stated.

5.3: RESULTS

5.3.1: INTER-TEST PRECISION

The estimated sample sizes for a range of effect magnitudes are provided in Table 5.4. Parameters derived for P_{Emax} and the U-150º posture showed the lowest reliability compared with the other variables measured. The data suggests that the study had sufficient power to detect changes in most parameters with an effect magnitude >20% (assuming n=9 per group). However, there were a few exceptions, five postures required >16 subjects to detect a >20% change. Required sample sizes were very high for effects of 5%, especially for respiratory pressures. MFVLs required smaller samples than respiratory pressures for effect magnitude <5%. The highest reliability was shown for FVC, which required an effect magnitude of <5%.

The limits of agreement for within-subject variation for all outcome measures are summarised in Tables 5.5 to 5.9. A significant difference in baseline measurements

was found for P_{Emax} at U-110º (P = 0.032), all other parameters were within normal limits (P > 0.05) compared to baseline measurements. The tables provide data on the agreement between the mean and the difference of the means on two separate days. The correlation of the mean difference was very low suggesting that the data were not heteroscedastic. Additionally, all data were log transformed to create dimensionless quantities allowing for a comparison across parameters with different units of measure.

Table 5.4 Estimated sample size for effect.

Variable	Effect magnitude (percentage of measured value)			
	5%	10%	20%	30%
$P_{Imax75°}$	98	24	6	3
$P_{Imax90°}$	83	21	5	2
S-$P_{Imax110°}$	50	12	3	1
U-$P_{Imax110°}$	138	35	9	4
S-$P_{Imax130°}$	267	67	17	7
U-$P_{Imax130°}$	71	18	4	2
S-$P_{Imax150°}$	115	29	7	3
U-$P_{Imax150°}$	290	73	18	8
$P_{Emax75°}$	152	38	9	4
$P_{Emax90°}$	51	13	3	1
S-$P_{Emax110°}$	287	72	18	8
U-$P_{Emax110°}$	90	22	6	2
S-$P_{Emax130°}$	69	17	4	2
U-$P_{Emax130°}$	67	17	4	2
S-$P_{Emax150°}$	257	64	16	7
U-$P_{Emax150°}$	611	153	38	17
$PIF_{75°}$	48	12	3	1
$PIF_{90°}$	49	12	3	1
S-$PIF_{110°}$	106	26	7	3
U-$PIF_{110°}$	67	17	4	2
S-$PIF_{130°}$	59	15	4	2
U-$PIF_{130°}$	51	13	3	1
S-$PIF_{150°}$	119	30	7	3
U-$PIF_{150°}$	99	25	6	3
$PEF_{75°}$	34	9	2	1
$PEF_{90°}$	8	2	1	0
S-$PEF_{110°}$	16	4	1	0
U-$PEF_{110°}$	7	2	0	0
S-$PEF_{130°}$	16	4	1	0
U-$PEF_{130°}$	16	4	1	0
S-$PEF_{150°}$	5	1	0	0
U-$PEF_{150°}$	83	21	5	2
$FVC_{75°}$	20	5	1	1
$FVC_{90°}$	5	1	0	0
S-$FVC_{110°}$	3	1	0	0
U-$FVC_{110°}$	3	1	0	0
S-$FVC_{130°}$	5	1	0	0
U-$FVC_{130°}$	2	0	0	0
S-$FVC_{150°}$	5	1	0	0
U-$FVC_{150°}$	4	1	0	0

Note: S, supported; U, unsupported P_{Imax}, maximal inspiratory pressure; P_{Emax}, maximal expiratory pressure; PIF, peak inspiratory flow; PEF, peak expiratory flow; FVC, forced vital capacity.

Table 5.5 Absolute and ratio limits of agreement for performance indices for maximal inspiratory pressure.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$P_{Imax75°}$	9	116 (24)	116 (34)	0.3 (17)	0.33	5.5	-11.35 to 12.01	32.4	9.54	-52.30 to -11.83	12.50 to 52.97
$P_{Imax90°}$	9	122 (30)	121 (34)	0.1 (13)	0.11	4.4	-9.28 to 9.50	26.05	7.67	-42.20 to -9.67	9.89 to 42.42
S-$P_{Imax110°}$	9	117 (30)	116 (35)	1.2 (11)	1.22	3.7	-6.68 to 9.13	21.93	6.46	-34.40 to -7.01	9.46 to 36.85
U-$P_{Imax110°}$	9	118 (4)	118 (35)	-0.8 (14)	0.78	4.7	-10.75 to 9.19	27.65	8.14	-45.69 to -11.16	9.60 to 44.13
S-$P_{Imax130°}$	9	122 (36)	116 (35)	5.7 (18)	5.67	6.0	-6.95 to 18.29	35.01	10.30	-51.20 to -7.48	18.81 to 62.53
U-$P_{Imax130°}$	9	115 (32)	118 (26)	-2.1 (12)	-2.11	4.0	-10.48 to 6.25	23.20	6.83	-39.80 to -10.82	5.54 to 36.64
S-$P_{Imax150°}$	9	110 (33)	111 (35)	-1.6 (15)	-1.56	5.1	-12.27 to 9.16	29.73	8.76	-49.85 to -12.72	9.61 to 46.73
U-$P_{Imax150°}$	7	108 (31)	103 (30)	5.1 (24)	5.14	9.1	-14.62 to 24.91	47.03	15.70	-76.12 to -7.66	17.94 to 86.41

Log transformed measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Ratio	SE	95% CI	Ratio	SE	95% CI for Lower L of A	95% CI for Upper L of A
$P_{Imax75°}$	9	4.7 (0.2)	4.7 (0.2)	0.02 (0.1)	1.024	0.04	-0.070 to 0.118	1.299	0.05	0.689 to 0.889	1.230 to 1.430
$P_{Imax90°}$	9	4.8 (0.2)	4.8 (0.3)	0.01 (0.1)	1.010	0.04	-0.077 to 0.098	1.276	0.04	0.699 to 0.885	1.196 to 1.382
S-$P_{Imax110°}$	9	4.7 (0.3)	4.7 (0.3)	0.02 (0.1)	1.023	0.03	-0.047 to 0.092	1.213	0.04	0.769 to 0.917	1.167 to 1.314
U-$P_{Imax110°}$	9	4.7 (0.3)	4.7 (0.3)	0.00 (0.2)	1.005	0.05	-0.105 to 0.052	1.356	0.06	0.625 to 0.858	1.246 to 1.479
S-$P_{Imax130°}$	9	4.8 (0.3)	4.7 (0.3)	0.05 (0.2)	1.052	0.07	-0.094 to 0.196	1.494	0.07	0.551 to 0.858	1.418 to 1.726
U-$P_{Imax130°}$	9	4.7 (0.3)	4.7 (0.2)	-0.03 (0.1)	0.970	0.04	-0.112 to 0.052	1.256	0.04	0.686 to 0.860	1.132 to 1.306
S-$P_{Imax150°}$	9	4.7 (0.3)	4.7 (0.3)	-0.02 (0.1)	0.984	0.05	-0.117 to 0.086	1.325	0.05	0.635 to 0.850	1.197 to 1.412
U-$P_{Imax150°}$	7	4.6 (0.3)	4.6 (0.3)	0.04 (0.2)	1.047	0.08	-0.129 to 0.221	1.515	0.08	0.528 to 0.854	1.423 to 1.750

Note: Maximal inspiratory pressure (P_{Imax}) presented in cm H_2O; S, supported posture; U, unsupported posture.

Table 5.6 Absolute and ratio limits of agreement for performance indices for maximal expiratory pressure.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$P_{Emax75°}$	9	110 (33)	115 (25)	-5.7 (18)	-5.78	6.10	-18.70 to 7.15	35.84	10.56	-64.01 to -19.24	7.68 to 52.45
$P_{Emax90°}$	9	113 (33)	117 (32)	-3.2 (9)	-3.22	3.09	-9.77 to 3.33	18.17	5.35	-32.74 to -10.05	3.60 to 26.30
S-$P_{Emax110°}$	9	117 (32)	116 (35)	1.6 (18)	1.56	6.05	-11.28 to 14.39	35.59	10.48	-56.26 to -11.81	14.92 to 59.38
U-$P_{Emax110°}$	9	108 (31)	120 (27)	-12.0 (13)*	-12.00	4.65	-21.85 to -2.15	27.32	8.05	-56.38 to -22.26	-1.74 to 32.38
S-$P_{Emax130°}$	9	111 (28)	118 (35)	-7.9 (13)	-7.89	4.39	-17.19 to 1.41	25.79	7.60	-49.78 to -17.57	1.80 to 34.00
U-$P_{Emax130°}$	9	111 (32)	117 (34)	-5.3 (10)	-5.33	3.41	-12.57 to 1.90	20.06	5.91	-37.92 to -12.87	2.20 to 27.25
S-$P_{Emax150°}$	9	112 (36)	114 (31)	-1.2 (21)	-1.22	7.31	-16.73 to 14.28	43.01	12.67	-71.08 to -17.37	14.93 to 68.64
U-$P_{Emax150°}$	7	99 (37)	119 (27)	-20.6 (27)	-20.57	10.46	-43.37 to 2.23	54.26	18.12	-114.32 to 35.34	-5.80 to 73.17

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$P_{Emax75°}$	9	4.7 (0.3)	4.7 (0.5)	-0.07 (0.2)	0.933	0.054	-0.183 to 0.045	1.372	0.057	0.559 to 0.801	1.160 to 1.402
$P_{Emax90°}$	9	4.7 (0.3)	4.7 (0.3)	0.03 (0.1)	0.973	0.033	-0.098 to 0.043	1.216	0.035	0.725 to 0.875	1.108 to 1.258
S-$P_{Emax110°}$	9	4.7 (0.3)	4.7 (0.4)	0.03 (0.2)	1.028	0.070	-0.121 to 0.177	1.512	0.075	0.522 to 0.838	1.397 to 1.713
U-$P_{Emax110°}$	9	4.6 (0.3)	4.8 (0.3)	-0.12 (0.1)	0.889	0.043	-0.208 to -0.027	1.286	0.045	0.595 to 0.787	1.047 to 1.240
S-$P_{Emax130°}$	9	4.7 (0.3)	4.7 (0.3)	-0.05 (0.1)	0.946	0.038	-0.136 to 0.025	1.251	0.040	0.671 to 0.842	1.097 to 1.270
U-$P_{Emax130°}$	9	4.7 (0.3)	4.7 (0.3)	-0.04 (0.1)	0.959	0.038	-0.122 to 0.037	1.247	0.040	0.684 to 0.853	1.111 to 1.280
S-$P_{Emax150°}$	9	4.7 (0.3)	4.7 (0.3)	-0.02 (0.2)	0.982	0.067	-0.161 to 0.124	1.484	0.071	0.510 to 0.812	1.306 to 1.608
U-$P_{Emax150°}$	7	4.5 (0.4)	4.8 (0.3)	-0.23 (0.3)	0.797	0.108	-0.462 to 0.007	1.747	0.101	0.236 to 0.675	1.173 to 1.611

Note: Maximal expiratory pressure (P_{Emax}) presented in cm H_2O; S, supported posture; U, unsupported posture.*, significant difference ($p \leq 0.05$).

Table 5.7 Absolute and ratio limits of agreement for performance indices for peak inspiratory flow.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$PIF_{75°}$	9	479 (129)	492 (138)	-12.7 (49.9)	-12.67	16.65	-47.96 to 22.63	97.90	28.84	-171.70 to -49.43	24.10 to 146.37
$PIF_{90°}$	9	509 (137)	519 (149)	-10.3 (48.5)	-10.27	16.16	-44.52 to 23.99	95.01	27.99	-164.64 to -45.94	25.41 to 144.07
$S\text{-}PIF_{110°}$	9	493 (145)	505 (144)	-11.6 (59.4)	-11.60	19.81	-53.59 to 30.39	116.47	34.31	-200.81 to -55.34	32.14 to 177.61
$U\text{-}PIF_{110°}$	9	488 (140)	495 (141)	-7.0 (46.9)	-6.96	15.63	-40.10 to 26.19	91.92	27.08	-156.29 to -41.48	27.56 to 142.37
$S\text{-}PIF_{130°}$	9	482 (137)	484 (148)	-1.4 (47.1)	-1.40	15.69	-34.66 to 31.86	92.25	27.17	-151.27 to -36.04	33.24 to 148.47
$U\text{-}PIF_{130°}$	9	470 (126)	480 (140)	-9.9 (48.6)	-9.87	16.20	-44.21 to 24.48	95.26	28.06	-164.61 to -45.64	25.90 to 144.88
$S\text{-}PIF_{150°}$	9	456 (115)	471 (146)	-15.1 (62.8)	-15.13	20.92	-59.48 to 29.21	123.00	36.23	-214.94 to -61.32	31.05 to 184.67
$U\text{-}PIF_{150°}$	7	440 (112)	462 (144)	-22.1 (61.1)	-22.06	23.05	-72.29 to 28.18	119.55	39.93	-228.61 to -54.60	10.49 to 184.50

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$PIF_{75°}$	9	6.1 (0.3)	6.2 (0.3)	-0.02 (0.1)	0.980	0.032	-0.088 to 0.049	1.208	0.034	0.739 to 0.884	1.112 to 1.257
$PIF_{90°}$	9	6.2 (0.3)	6.2 (0.3)	-0.02 (0.1)	0.987	0.033	-0.082 to 0.056	1.212	0.035	0.741 to 0.888	1.123 to 1.270
$S\text{-}PIF_{110°}$	9	6.2 (0.3)	6.2 (0.3)	-0.02 (0.1)	0.980	0.046	-0.118 to 0.077	1.311	0.049	0.644 to 0.851	1.181 to 1.388
$U\text{-}PIF_{110°}$	9	6.2 (0.3)	3.2 (0.3)	-0.01 (0.1)	0.989	0.038	-0.090 to 0.069	1.248	0.040	0.708 to 0.878	1.150 to 1.319
$S\text{-}PIF_{130°}$	9	6.1 (0.3)	6.1 (0.3)	0.00 (0.1)	1.006	0.035	-0.069 to 0.081	1.232	0.038	0.737 to 0.897	1.160 to 1.319
$U\text{-}PIF_{130°}$	9	6.1 (0.3)	6.1 (0.3)	-0.01 (0.1)	0.988	0.033	-0.083 to 0.058	1.217	0.035	0.737 to 0.887	1.127 to 1.277
$S\text{-}PIF_{150°}$	9	6.1 (0.3)	6.1 (0.4)	-0.01 (0.2)	0.989	0.048	-0.114 to 0.091	1.329	0.051	0.635 to 0.853	1.205 to 1.423
$U\text{-}PIF_{150°}$	7	6.1 (0.3)	6.1 (0.3)	-0.03 (0.1)	0.970	0.051	-0.141 to 0.080	1.301	0.048	0.642 to 0.849	1.158 to 1.365

Note: Peak inspiratory flow (PIF) presented in $L \cdot min^{-1}$; S, supported posture; U, unsupported posture.

Table 5.8 Absolute and ratio limits of agreement for performance indices for peak expiratory flow.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$PEF_{75°}$	9	536 (128)	531 (145)	4.8 (40.7)	4.80	13.58	-23.99 to 33.59	79.84	23.52	-124.90 to -25.18	34.78 to 134.50
$PEF_{90°}$	9	547 (141)	551 (151)	-4.3 (21.0)	-4.33	7.01	-19.20 to 10.54	41.24	12.15	-71.32 to -19.82	11.15 to 62.66
$S\text{-}PEF_{110°}$	9	545 (135)	545 (154)	-0.3 (28.7)	-0.27	9.55	-20.51 to 19.98	56.16	16.54	-91.49 to -21.35	20.82 to 90.96
$U\text{-}PEF_{110°}$	9	530 (137)	531 (138)	-0.5 (20.3)	-0.53	6.77	-14.90 to 13.83	39.83	11.73	-65.24 to -15.49	14.43 to 64.18
$S\text{-}PEF_{130°}$	9	534 (139)	533 (148)	0.9 (34.8)	0.87	11.59	-23.70 to 25.43	68.13	20.07	-109.81 to -24.72	26.45 to 111.54
$U\text{-}PEF_{130°}$	9	530 (132)	528 (151)	1.7 (32.2)	1.67	10.74	-21.10 to 24.44	63.15	18.60	-100.92 to -22.05	25.38 to 104.25
$S\text{-}PEF_{150°}$	9	532 (138)	532 (145)	-0.3 (15.8)	-0.33	5.26	-11.49 to 10.83	30.95	9.12	-50.62 to -11.96	11.29 to 49.95
$U\text{-}PEF_{150°}$	7	504 (133)	521 (144)	-17.2 (63.2)	-17.20	23.88	-69.23 to 34.83	123.82	41.36	-231.14 to -50.90	16.50 to 196.74

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$PEF_{75°}$	9	6.3 (0.3)	6.5 (0.3)	0.0 (0.1)	1.019	0.028	-0.040 to 0.077	1.178	0.029	0.803 to 0.928	1.137 to 1.262
$PEF_{90°}$	9	6.3 (0.3)	6.3 (0.3)	-0.0 (0.0)	0.996	0.014	-0.034 to 0.026	1.087	0.015	0.885 to 0.948	1.051 to 1.114
$S\text{-}PEF_{110°}$	9	6.3 (0.3)	6.3 (0.3)	0.0 (0.1)	1.008	0.019	-0.032 to 0.049	1.120	0.020	0.857 to 0.944	1.086 to 1.172
$U\text{-}PEF_{110°}$	9	6.2 (0.3)	6.2 (0.3)	-0.0 (0.0)	1.000	0.013	-0.028 to 0.028	1.081	0.014	0.896 to 0.955	1.051 to 1.110
$S\text{-}PEF_{130°}$	9	6.3 (0.3)	3.2 (0.3)	0.0 (0.1)	1.006	0.02	-0.036 to 0.048	1.122	0.021	0.852 to 0.941	1.085 to 1.173
$U\text{-}PEF_{130°}$	9	6.2 (0.3)	6.2 (0.3)	0.0 (0.1)	1.013	0.019	-0.029 to 0.054	1.121	0.021	0.859 to 0.947	1.092 to 1.179
$S\text{-}PEF_{150°}$	9	6.2 (0.3)	6.2 (0.3)	0.0 (0.0)	1.002	0.011	-0.021 to 0.026	1.067	0.012	0.914 to 0.964	1.045 to 1.095
$U\text{-}PEF_{150°}$	7	6.2 (0.3)	6.2 (0.3)	-0.0 (0.1)	0.971	0.047	-0.132 to 0.073	1.276	0.044	0.665 to 0.857	1.144 to 1.336

Note: Peak expiratory flow (PEF) presented in $L \cdot min^{-1}$; S, supported posture; U, unsupported posture.

Table 5.9 Absolute and ratio limits of agreement for performance indices for forced vital capacity.

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$FVC_{75°}$	9	5.2 (1.2)	5.1 (1.2)	0.2 (1.2)	0.15	0.11	-0.10 to 0.39	0.67	0.20	-0.95 to -0.11	0.40 to 1.24
$FVC_{90°}$	9	5.4 (1.2)	5.3 (1.2)	0.1 (0.2)	0.06	0.08	-0.10 to 0.22	0.44	0.13	-0.66 to -0.10	0.23 to 0.78
$S\text{-}FVC_{110°}$	9	5.3 (1.2)	5.3 (1.2)	0.0 (0.2)	0.01	0.05	-0.10 to 0.12	0.32	0.09	-0.51 to -0.11	0.13 to 0.53
$U\text{-}FVC_{110°}$	9	5.2 (1.2)	5.2 (1.2)	-0.0 (0.1)	-0.03	0.05	-0.13 to 0.07	0.27	0.08	-0.47 to -0.13	0.08 to 0.42
$S\text{-}FVC_{130°}$	9	5.3 (1.2)	5.4 (1.2)	-0.0 (0.2)	-0.03	0.06	-0.15 to 0.08	0.33	0.10	-0.56 to -0.16	0.09 to 0.49
$U\text{-}FVC_{130°}$	9	5.1 (1.2)	5.1 (1.2)	-0.0 (0.1)	-0.04	0.04	-0.12 to 0.04	0.21	0.06	-0.38 to -0.12	0.04 to 0.30
$S\text{-}FVC_{150°}$	9	5.3 (1.2)	5.3 (1.2)	-0.0 (0.2)	-0.03	0.06	-0.15 to 0.09	0.34	0.10	-0.59 to -0.16	0.10 to 0.52
$U\text{-}FVC_{150°}$	7	5.1 (1.3)	5.1 (1.3)	0.01 (0.2)	0.01	0.06	-0.12 to 0.14	0.31	0.10	-0.52 to -0.07	0.10 to 0.55

Measurements

Variable	N	Mean (SD) 1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
$FVC_{75°}$	9	1.6 (0.2)	1.6 (0.2)	0.0 (0.1)	1.030	0.022	-0.016 to 0.076	1.136	0.023	0.858 to 0.956	1.121 to 1.219
$FVC_{90°}$	9	1.7 (0.2)	1.6 (0.2)	0.0 (0.0)	1.010	0.012	-0.014 to 0.034	1.070	0.012	0.918 to 0.970	1.055 to 1.107
$S\text{-}FVC_{110°}$	9	1.7 (0.2)	1.7 (0.2)	0.0 (0.0)	1.000	0.009	-0.019 to -0.019	1.053	0.009	0.930 to 0.969	1.033 to 1.073
$U\text{-}FVC_{110°}$	9	1.6 (0.2)	1.6 (0.2)	-0.0 (0.0)	0.995	0.008	-0.023 to 0.012	1.05	0.009	0.929 to 0.966	1.026 to 1.063
$S\text{-}FVC_{130°}$	9	1.7 (0.2)	1.7 (0.2)	-0.0 (0.0)	0.994	0.011	-0.028 to 0.017	1.064	0.011	0.910 to 0.958	1.034 to 1.082
$U\text{-}FVC_{130°}$	9	1.6 (0.2)	1.6 (0.2)	-0.0 (0.0)	0.991	0.007	-0.023 to 0.006	1.041	0.007	0.937 to 0.968	1.017 to 1.048
$S\text{-}FVC_{150°}$	9	1.6 (0.2)	1.6 (0.2)	-0.0 (0.0)	0.993	0.011	-0.030 to 0.017	1.067	0.012	0.906 to 0.956	1.035 to 1.085
$U\text{-}FVC_{150°}$	7	1.6 (0.3)	1.6 (0.3)	0.0 (0.0)	1.001	0.012	-0.024 to 0.027	1.063	0.011	0.918 to 0.966	1.040 to 1.088

Note: Forced vital capacity (FVC) presented in L. S, supported posture; U, unsupported posture.

5.3.2 RESPIRATORY STRENGTH MEASUREMENTS

Respiratory mouth pressures did not differ with any of the supported or unsupported postures (P = 0.181). Mean data for all measurements is presented in Table 5.10. As shown in figure 5.3*A*, there was a small, non-significant decrease from 90º (upright-seated) when compared to 75º (catch position) for both P_{Imax} and P_{Emax} (~4.8%, P = 1.00). No difference was detected at the typical finish position of 110º in comparison to sitting upright; however, P_{Imax} tended to be lower when reclining at U-130º (6.0%, P = 0.742), S-150º (8.6%, P = 1.00) and U-150º (9.8%, P = 1.00). We calculated that a sample size of >64 subjects would be required to detect a significance effect of 10% for postures exceeding 130º.

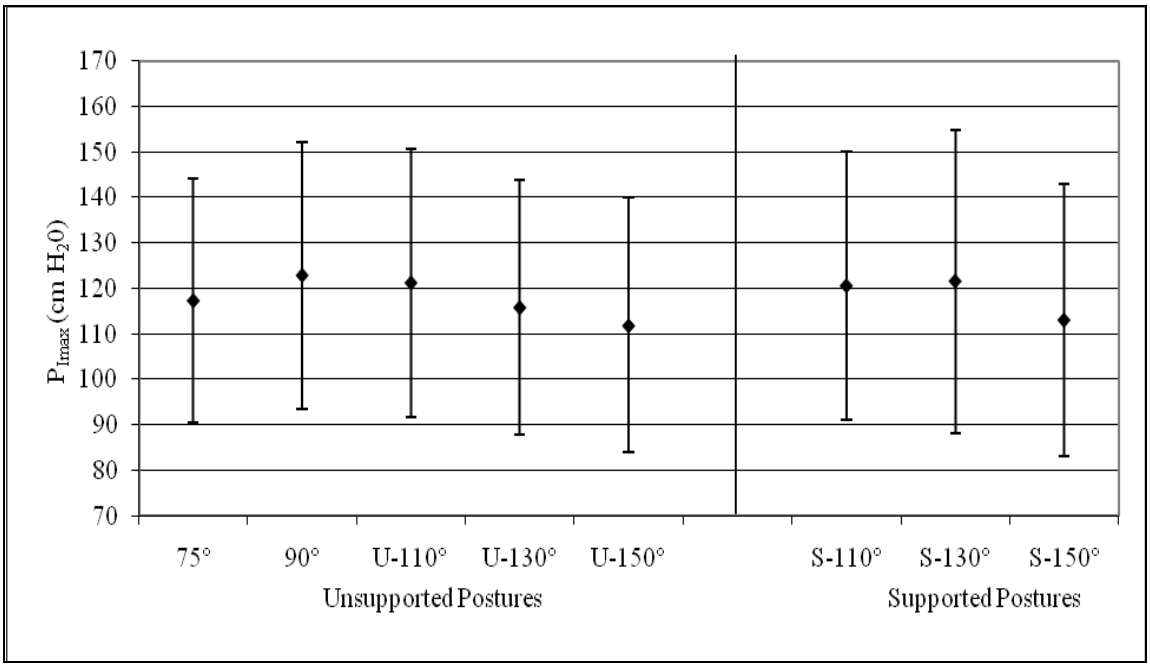
Similar to P_{Imax}, P_{Emax} showed no differences between any of the rowing specific postures (P = 0.696). As shown in figure 5.3*B*, U-150º showed the greatest decrease (8.1%; P = 1.00) compared to sitting upright (90º). P_{Emax} was lower overall compared to $P_{Imax.}$ There was a correlation between P_{Imax} and P_{Emax} observed at all postures (P < 0.05); however there were no relationships evident between these variables when expressed as a percent change from the 90º posture.

Table 5.10 Mean values for respiratory pressures and pulmonary measurements.

	75º	90º	S-110º	U-110º	S-130º	U-130º	S-150º	U-150º
P_{Imax} (cm H_2O)	117.3 ± 26.9	122.8 ± 29.4	120.6 ± 29.4	121.2 ± 29.4	121.6 ± 33.3	115.8 ± 28.0	113.1 ± 29.9	111.9 ± 28.0
P_{Emax} (cm H_2O)	110.2 ± 33.0	115.6 ± 30.3	115.9 ± 31.3	111.8 ± 29.5	113.6 ± 28.9	112.9 ± 29.0	112.7 ± 35.1	106.9 ± 32.4
PIF ($L \cdot min^{-1}$)	450.3 ± 124.9	472.7 ± 139.1	470.8 ± 130.7	448.1 ± 129.9	452.9 ± 128.2	441.9 ± 135.6	434.9 ± 117.5	423.2 ± 118.4
PEF($L \cdot min^{-1}$)	536.4 ± 117.9	542.9 ± 128.7	540.6 ± 126.5	525.7 ± 116.3	525.8 ± 120.7	522.0 ± 123.7	522.1 ± 125.1	501.6 ± 127.9†
FVC (L)	5.15 ± 1.14	5.27 ± 1.14	5.25 ± 1.13	5.07 ± 1.12†	5.20 ± 1.13	5.03 ± 1.14†	5.14 ± 1.13	4.88 ± 1.17*†
FEV_1 (L)	4.08 ± 0.86	4.19 ± 0.91	4.21 ± 0.89	4.07 ± 0.87	4.13 ± 0.89†	4.03 ± 0.87	4.04 ± 0.87†	3.95 ± 0.91

Note: P_{Imax}, maximal inspiratory pressure; P_{Emax}, maximal inspiratory pressure; PIF, peak inspiratory flow; PEF, peak inspiratory flow; FVC, forced vital capacity; FEV_1, forced expired volume in 1 second; *, significantly different to 90º ($p \leq 0.05$); †, significantly different to S-110º ($p \leq 0.05$).

A



B

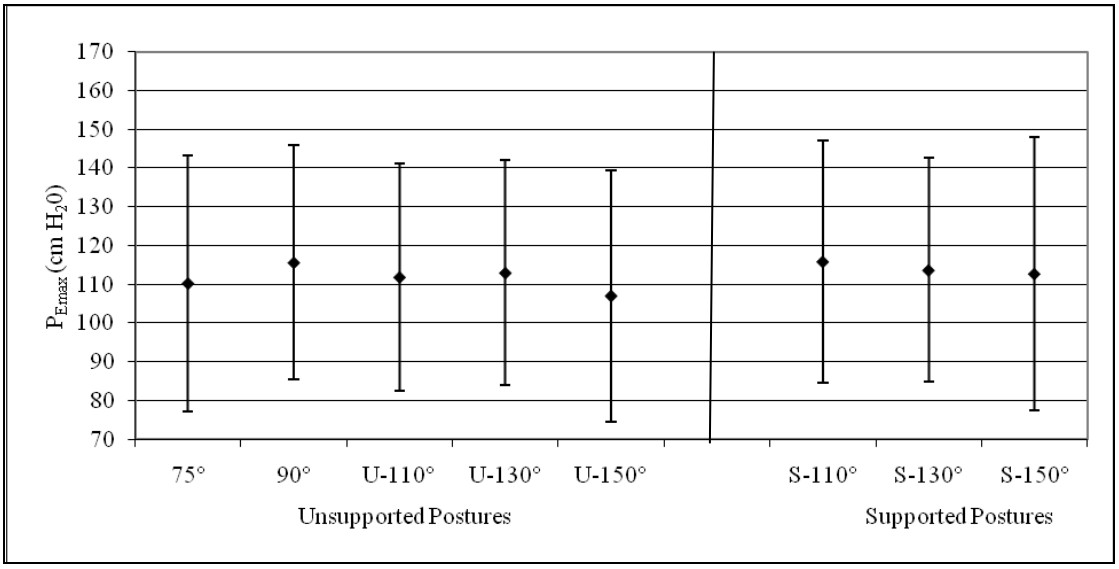


Figure 5.3 *A*, Comparison of maximal inspiratory pressure (P_{Imax}) in unsupported and supported postures; *B*, Comparison of maximal expiratory pressure (P_{Emax}) in unsupported and supported postures.

SPIROMETRIC MEASUREMENTS

Pairwise comparisons showed a statistically significant interaction effect with posture for both PEF (P = 0.020) and FVC (P = 0.033).

The interaction effect for PIF and posture was not significant (P = 0.057). As shown in Figure 5.4, there was no change in PIF at 90º compared to the catch position (5.0% decrease; P > 0.05). As the participants reclined to unsupported postures > 110º, PIF tended to decrease by a further 5.4% (P = 0.177). The S-150º and U-150º postures showed a decrease of 8.7% (P = 0.186) and 11.7% (P = 0.84), respectively, when compared to upright-seated (90º). We calculated that a sample size of >25 participants would be required to detect a significant effect of 10% for postures at 150º. No correlation was observed, neither absolute nor as a percentage change from 90º, between PIF and P_{Imax} at any posture (P > 0.05).

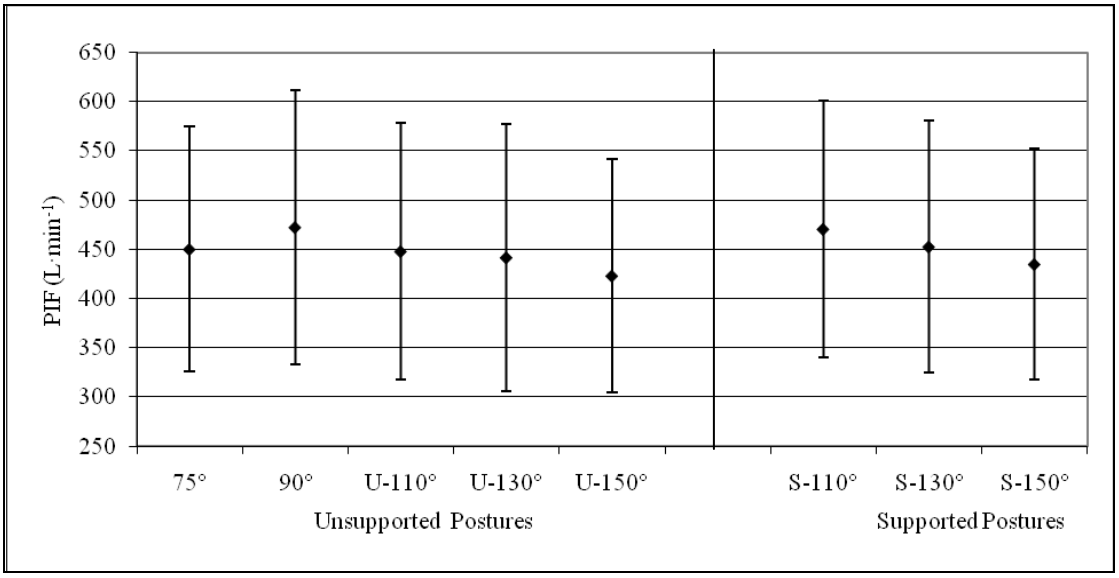


Figure 5.4 Comparison of peak inspiratory flow (PIF) to unsupported and supported postures.

There was an interaction effect between PEF and posture (P = 0.020; see fig. 5.5), but PEF decreased only in the S-110º posture when compared to U-150º (8.3%, P = 0.044). There was no correlation between absolute PEF and P_{Emax} (P > 0.05); but there was a moderate inverse correlation as a percentage change from 90º at the S-130º posture (r = -0.509; P = 0.044).

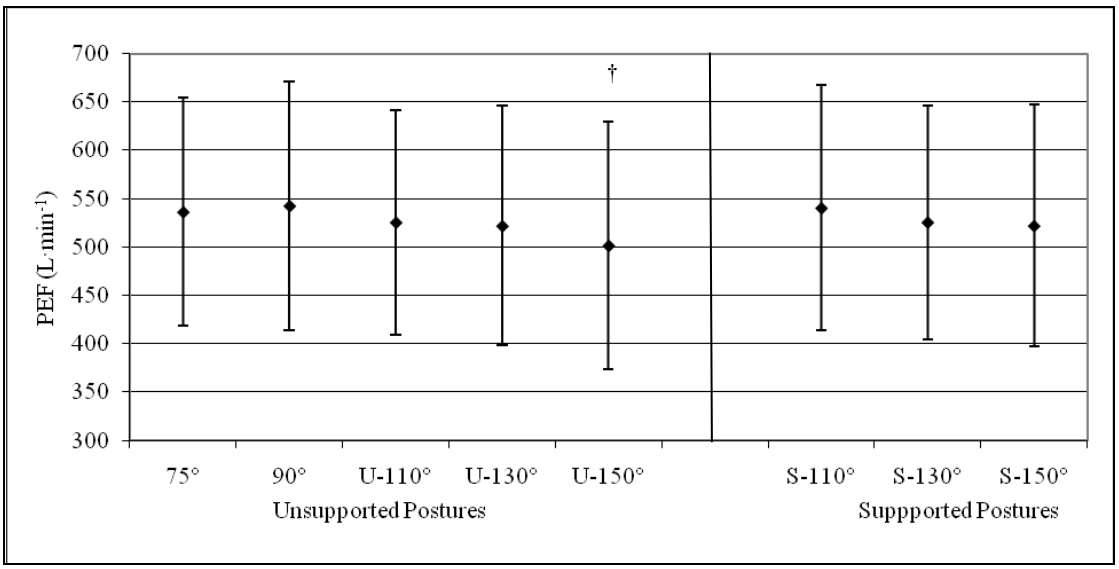


Figure 5.5 Comparison of peak expiratory flow (PEF) in unsupported and supported postures. †, significantly different to S-110º ($p \leq 0.05$).

Forced vital capacity (FVC) also showed an interaction with posture (P = 0.033). Both 90º and S-110º postures were different compared to U-150º (7.8% and 7.0% respectively, P = 0.019; see fig. 5.6). There was also a difference between S-110º compared to U-110º (3.4%, P = 0.014). Bivariate correlations revealed relationships between absolute FVC and PIF (P < 0.05) and PEF (P < 0.001) at all postures. As shown in Figure 5.8*A*, there was also a strong positive correlation between absolute FVC and P_{Emax} at the U-150° posture (r = 0.712; P = 0.003); no correlation was

evident between absolute or percent change from 90º for FVC and P_{Imax} ($P > 0.05$) at any posture.

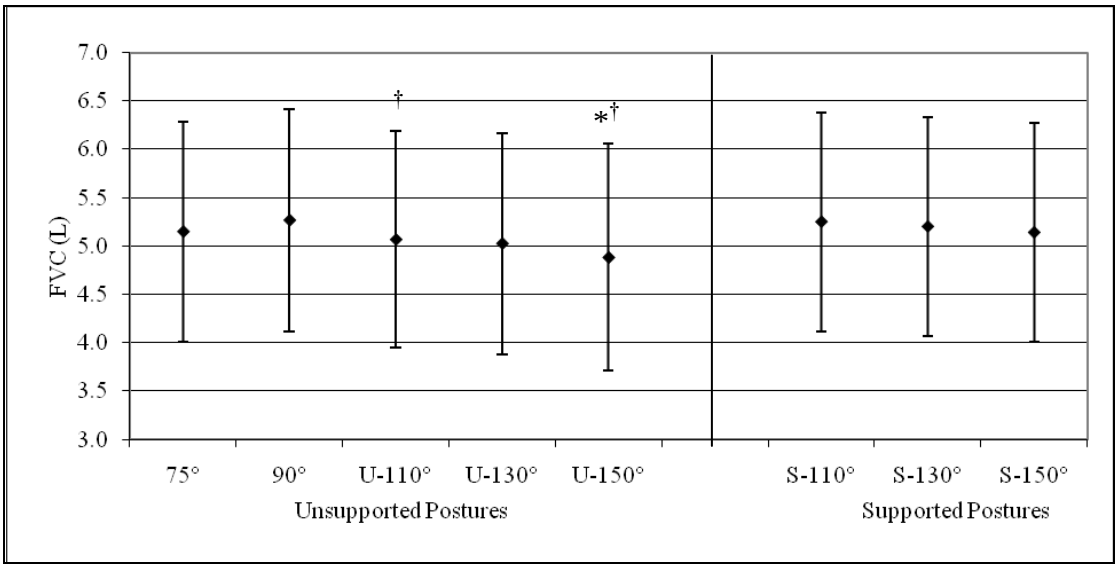


Figure 5.6 Comparison of forced vital capacity (FVC) in unsupported and supported postures. *, significantly different to 90º ($p \geq 0.05$); †, significantly different to S-110º ($p \geq 0.05$).

Forced expiratory volume in 1 second (FEV_1) exhibited an interaction effect with posture ($P = 0.021$). As shown in Figure 5.7, FEV_1 was higher in the S-110º posture compared to S-130º (1.8%; $P = 0.044$), and both the S-110º and S-130º were higher compared to S-150º (3.9% and 2.1%; $P = 0.001$ and 0.049, respectively). Positive correlations were evident for FEV_1 and FVC ($P < 0.001$), PIF ($P < 0.05$) and PEF ($P < 0.001$). There was a moderate positive relationship between absolute FEV_1 and P_{Emax} at the U-150° posture ($r = 0.537$; $P = 0.039$; see fig 5.8*B*); as well as a positive relationship as a percent change from 90º at S-110º (r = 0.778; $P = 0.000$) and U-110° (r = 0.590; $P = 0.016$). No relationship was observed between FVC and P_{Imax} at any posture.

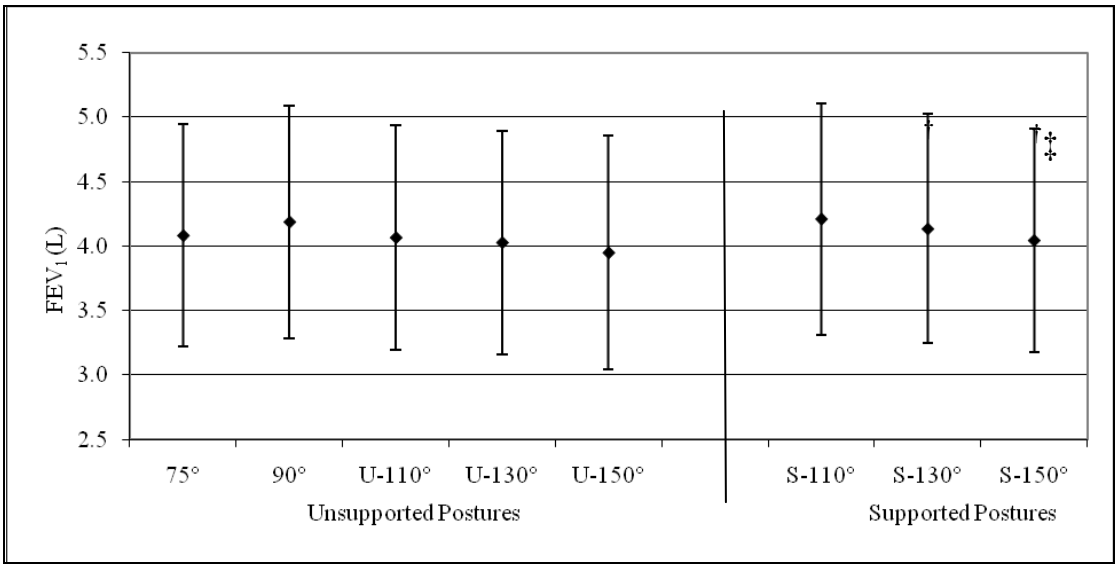
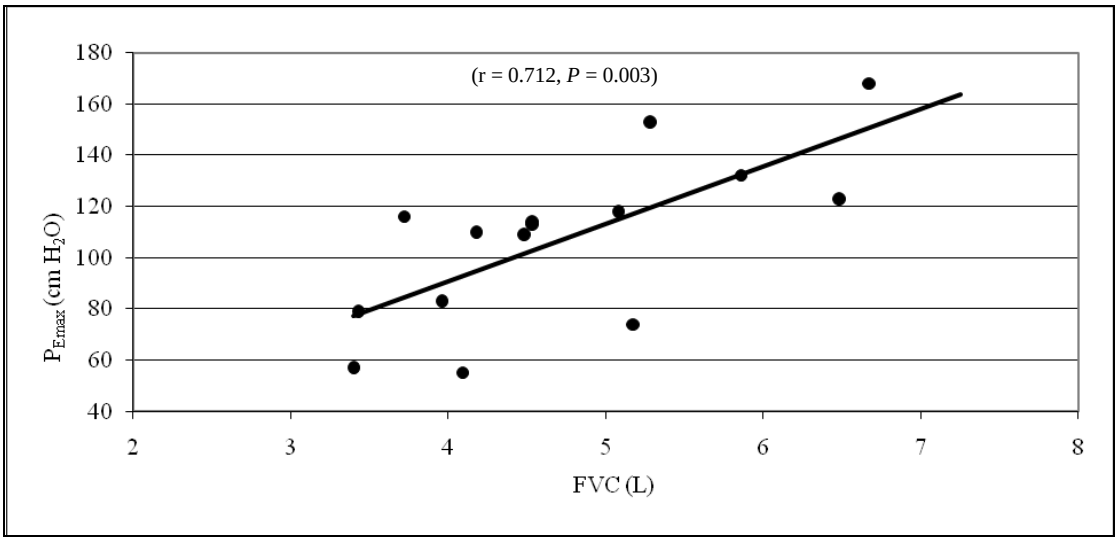


Figure 5.7 Comparison of forced expiratory volume in one second (FEV_1) in unsupported and supported postures. †, significantly different to S-110º ($p \geq 0.05$); ‡, significantly different to S-130º ($p \geq 0.05$).

A



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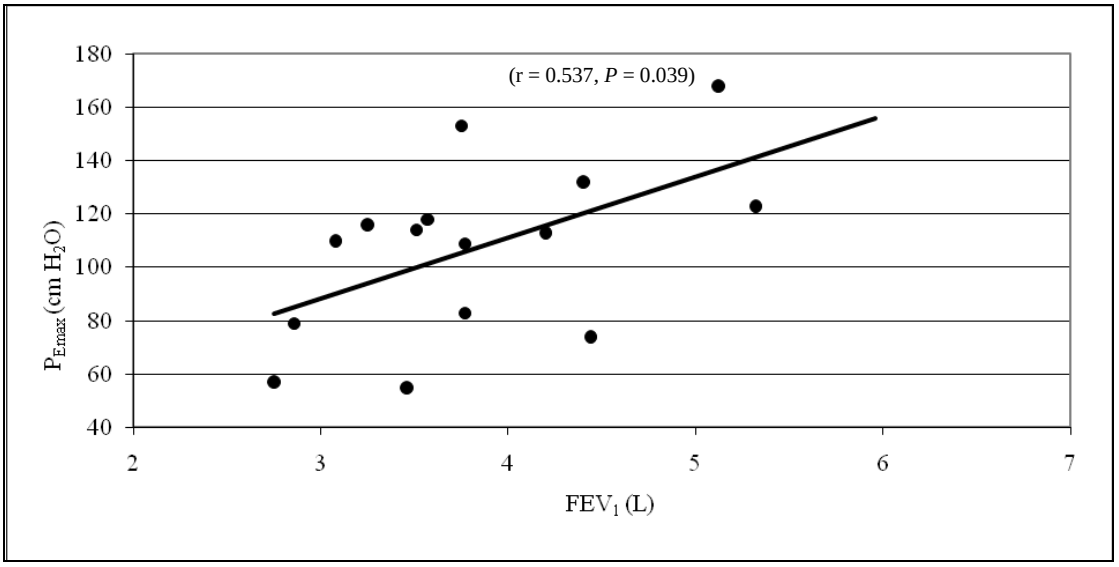


Figure 5.8 *A*, Relationship between forced vital capacity (FVC) and maximal expiratory pressure (P_{Emax}) at U-150° posture. *B*, Relationship between forced expiratory volume (FEV_1) and maximal expiratory pressure (P_{Emax}) at U-150° posture.

5.4: DISCUSSION

5.4.1: MAIN FINDINGS

The aim of this study was to determine whether respiratory pressure and flow generating capacity differed due to postural adjustments in various seated postures relevant to rowing, and to assess the effect of supported and unsupported positions. To this end we used a series of static rowing-related postures, in which we controlled for starting lung volume to isolate the co-contraction of trunk stabilising muscles, but found no significant change in respiratory mouth pressures in any of the postures tested. Thus, respiratory muscles appear to work effectively in all rowing-related postures; notwithstanding this observation, there was a clear tendency for function to be optimised in the seated and more upright postures. However, both PEF and FVC showed decreases in the 'finish' positions (> 110°) compared to sitting upright or supported at 110º. All outcome variables showed a tendency to be reduced as the postures became more reclined and they were noticeably lower in the unsupported postures and at both S-150º and U-150º. The lack of any significant changes in respiratory pressures generated in the rowing-related postures suggests that there would likely be no measurable benefit to using posture specific IMT compared to performing IMT in the upright seated position.

5.4.2: INTER-TEST PRECISION

Most measurements lacked the reliability required to detect changes with physiologically relevant effect magnitudes, and required relatively large sample sizes (n > 16). These findings also indicate that a study examining the effects of posture-specific RMT would require impractical sample sizes, unless the between day reliability could be improved from that achieved in the current study.

5.4.3: EFFECT OF POSTURE ON RESPIRATORY MUSCLE STRENGTH

No differences were evident in respiratory mouth pressures between supported and unsupported postures. Albeit, this may be due to the comparatively low reliability observed between baseline tests for P_{Emax}. There were some clear trends in the data and some clear interrelationships between physiologically related variables were also evident, i.e., significant differences in pulmonary function seemed to be related to changes in respiratory muscle function. For example, respiratory mouth pressures and the spirometric measures tested were highest in the upright-seated (90º) position and S-110º compared to all other postures. The decision to standardise the starting lung volume by initiating each manoeuvre from the upright posture (inhaling or exhaling before adopting the test posture) would have minimised the influence of posture upon the measured pressures. However, this was performed in order to isolate the effect of co-contraction of trunk muscles during the effort, and to minimise the effect of starting lung volumes upon the measured pressures.

Although non-significant, there was a small decrease in both P_{Imax} and P_{Emax} of ~5 cm H_2O (4.8%) at 75° compared to sitting upright and a noticeable decline in both pressures as the postures became more recumbent. As stated previously, the control of starting lung volume means that these alterations were the result of co-contraction of trunk stabilising muscles in recumbent postures. These findings are consistent with previous studies that have investigated changes in mouth pressures in various seated, supine and recumbent postures (Badr, Elkins & Ellis, 2002; Druz & Sharp, 1981; Kera & Maruyama, 2001a, 2001b; Koulouris et al., 1989; Meysman & Vincken, 1998; Ogiwara & Miyachi, 2002; Tsubaki et al., 2009). For example, Meysman & Vincken (1998) found a non-significant 6% decrease in P_{Imax} and a 2-5% decrease in P_{Emax} in

the right and left lateral recumbent positions. They also observed a decrease in P_{Imax} (8%; P = 0.04) from upright seated compared to the supine position. In contrast, Ogiwara & Miyachi (2002) investigated the effects of posture on mouth pressures in various seated and supine postures but found no differences in either P_{Imax} or P_{Emax} at any of the postures tested. Similar to the present study, they observed non-significant differences in both P_{Imax} and P_{Emax} from sitting compared to half lying (5.1% and 7.3%, respectively), 'slumped' half lying (9.1% and 8.7%) and supine half lying (9.1% and 10.9%). The improved respiratory muscle function in the upright positions is perhaps due to an increased capacity for rib expansion (i.e. activation of the accessory respiratory muscles to increase the diameter of the thoracic cage), thereby increasing and optimising the length of the expiratory muscles (Druz & Sharp, 1981). Any increase in respiratory muscle lengths would increase the potential elastic recoil pressure thereby augmenting the compression of the thoracic cage, hence gaining higher P_{Imax} and P_{Emax} values (Ogiwara & Miyachi, 2002).

The tendency for respiratory muscle pressures to decline in recumbent postures in these previous studies is most likely due to alterations in starting lung volumes in these positions (Ogiwara & Miyachi, 2002; Talwar et al., 2002). As discussed in section 1.1.2, the force generating capacity of the respiratory muscles is dependent upon the starting lung volume, which influences both the length-tension relationship and the elastic contribution from the chest wall. Although we did not measure TLC or RV, our data showed a decline in FVC (5-7%), PIF (5-9%) and PEF (3-7%) with reclining postures compared to upright seated, which supports the notion that posture influenced the ability of the respiratory muscles to generate maximal volume and flow excursions. Furthermore, we observed correlations between P_{Emax} and FVC, and

between P_{Emax} and FEV_1, as well as a correlation between the change in P_{Emax} and PEF, and the FEV_1 from the upright position to recumbent postures. In other words, impairment of expiratory muscle function (due to co-contraction of trunk postural stabilising muscles) was inter-related with the ability to maximise lung volume change and expiratory flow rate.

Recumbency has been shown previously to induce changes in lung volumes and flow rates (Badr et al., 2002; Castile, Mead, Jackson, Wohl & Stokes, 1982; D'Angelo & Agostini, 1995; Kera & Maruyama, 2005; Talwar et al., 2002). Studies investigating the effects of posture on flow volume loops have noted decreases in expiratory flows and lung volumes in the supine posture compared to seated (Castile et al., 1982; Talwar et al., 2002). Kera & Maruyama (2005) observed a decrease in the TLC and vital capacity (VC) of the lungs in the supine position compared to upright sitting and standing. The authors suggested that this was due to a shift in blood flow from the lower extremities to the thoracic cavity. In addition, they also observed a decrease in functional residual capacity (FRC) in the supine position, and attributed this change to an increase in intra-abdominal pressure due to the contents of the abdominal cavity pushing upwards onto the diaphragm in the supine position. Consequently, a decrease in FRC or TLC in the supine position may result in lower P_{Imax} or P_{Emax} as a result of a change in starting lung volume and muscle length (i.e. force-length tension relationship). However, since the effects of gravity upon fluid and organ shifts were absent in the present study, these mechanisms cannot have played a role in the changes that we observed.

There was not a substantial difference (not > 5%) between the corresponding supported vs. unsupported recumbent postures (i.e. S-130° compared to U-130°) for respiratory pressures. This was surprising, as we had speculated that the competing demands for postural and respiratory functions in unsupported recumbent body positions would have a substantial negative impact upon respiratory muscle pressure generating capacity. The contraction of the diaphragm along with the expiratory muscles assists in maintaining spine stabilisation by increasing intra-abdominal pressure (Hodges & Gandevia, 2000b; Siegmund et al., 1999). Hence, the co-contraction of the diaphragm and abdominal muscles during simultaneous postural and respiratory manoeuvres in the recumbent positions could conceivably impair, or compromise the motion of the rib cage and abdomen (Siegmund et al., 1999); particularly as the expiratory muscles would also be in a less advantageous position on the length-tension relationship during forced expiratory manoeuvres (Badr et al., 2002; Ogiwara & Miyachi, 2002). A potential explanation for the relatively small magnitude of this effect in the data may reside in the nature of maximal mouth pressure measurements. Under conditions of bracing and static co-contraction it is conceivable that P_{Imax} and P_{Emax} are relatively unaffected. However, under conditions where respiratory muscle shortening must take place in the presence of static, stabilising contraction of muscles stabilising the trunk, i.e., during production of MFVLs in the extended unsupported positions, the competing demands upon the trunk muscles for breathing and postural functions may be greater. The data suggest that this is the case, since the unsupported postures tended to have a greater effect upon dynamic flow and volume generation than on static pressure generation.

Typically, P_{Emax} is higher compared to P_{Imax} when measured in both normal seated and standing positions; however the participants in this study had a lower P_{Emax} in all postures tested (see table 5.11). Badr et al. (2002) observed a similar non-significant decrease in P_{Emax} in 'long sitting' (90º supported with legs straight) and '¾ sitting' (135º supported with legs straight) compared to standing or chair seated. The authors suggested the reduction was most likely due to muscle mechanics or a different starting lung volume. In our study, all postures required the participants to stiffen their upper body to maintain the specified position with legs straight, hence the abdominal muscles would have been activated. Thus, the postural role of the abdominal muscles to maintain these postures may have limited their ability to generate maximal pressure. An alternative explanation may be the use of a full inspiratory warm-up prior to P_{Imax} efforts in which previous studies suggest this adds up to 10-12% to the resulting maximal value for P_{Imax} (Lomax & McConnell, 2009; Volianitis et al., 2001a).

5.4.4: EFFECT OF POSTURE ON SPIROMETRIC MEASUREMENTS

Although not statistically different at all postures, all spirometric values tended to decrease as the postures became more recumbent. Our data are consistent with previous research that has demonstrated a significant reduction in spirometric indices (FVC, FEV_1, PEF) when posture changes from the upright seated to supine position (Allen et al., 1985; Badr et al., 2002; Crosby & Myles, 1985; Domingos-Benecio, Gastaldi, Perecin, Avena Kde, Guimaraes, Sologuren & Lopes-Filho, 2004; Meysman & Vincken, 1998; Tsubaki et al., 2009; Vilke et al., 2000). As discussed above, the influence of being supine appears to be due, at least in part, to the fluid and organ shifts due to gravity. All of the spirometric indices were similar at 90º and S-110º,

suggesting that the small influence of recumbency was due to the competing postural role of the respiratory muscles. For instance, there was a decrease in PIF of 5% at the catch and a 5-9% reduction in the recumbent postures compared to sitting upright. Peak expiratory flow (PEF) remained relatively unchanged at the catch position compared to 90º (1.3%), but declined in unsupported postures exceeding U-110º (3-7%). Peak flow rates and FVC began to decrease in the recumbent postures compared to sitting upright. These results are similar to Siegmund et al.'s (1999) findings in which they also observed no differences in peak flow rates in seated compared to catch position (knees bent). However, they did find a decrease in PIF at stroke finish and FVC in the catch position compared to upright seated. The authors suggested that the decline in PIF was likely due to the co-contraction of the diaphragm and abdominal muscles to maintain trunk extension resulting in impaired diaphragm function, whereas the ~5% decrease in expired volume in the catch position (compared to upright) may have been due to the compressed posture limiting lung volume.

Our finding of a 5-7% decrease in FVC from 90º to all unsupported postures is consistent with previous research demonstrating a 4-12% decrease in FVC when transferring between the upright seated and supine positions (Allen et al., 1985; Crosby & Myles, 1985; Meysman & Vincken, 1998; Vilke et al., 2000), as well as recumbent postures (Meysman & Vincken, 1998) in normal healthy subjects. Although FVC is influenced by respiratory pressure generating capacity (Leith & Brown, 1999), particularly the activation of the rectus abdominus muscle during forced expiratory manoeuvres, it is interesting that we found no correlation between absolute FVC and P_{Emax} at any posture except in the U-150°. Similarly, we observed a

non-significant decrease in FEV_1 at postures >U-110º (3-6%), which may be due to the initiation of the expiration from a lower starting volume (reducing the parenchymal pull on the airways, reducing their starting diameter). It is possible that the decrease in forced expiratory volumes, particularly in the U-150° posture, may be attributed to the mechanically disadvantageous position of the rectus abdominus during hip extension (Tsubaki et al., 2009). These results are consistent with other research showing a decrease in the ability to generate fast forced expiration in the supine or reclined postures (Crosby & Myles, 1985; Meysman & Vincken, 1998; Tsubaki et al., 2009; Vilke et al., 2000), albeit with slightly different underlying mechanisms in operation in the case of supine postures.

5:4.5: METHODOLOGICAL CONSIDERATIONS

To maintain consistency between trials, the 75° position was performed with straight legs (see fig. 5.2). This decision was made because of the difficulty in standardising the catch position (with knees bent) with sufficient reliability between trials. It is recognised that during the rowing stroke the 'catch' position is characterised by knees fully bent pressed against the chest and abdomen. Thus, the two positions are not directly comparable as there is less abdominal compression in our participants compared to the real rowing stroke. However, this modified position allowed for an uncontaminated assessment of the postural role of respiratory muscles in this position. In the tilt forward position (75° position) performed by our subjects, there was greater potential for excursion of the abdominal wall. During the actual rowing stroke, the thighs may limit or prohibit abdominal excursion, which may impair the ability to generate maximal pressures and flows. Hence, the results of this study are not directly

applicable to the catch position, and probably represent a best case scenario in terms of the detrimental influence of this posture upon respiratory function in this position.

Previous research has shown that the position of the neck could affect expiratory flow rates by altering tracheal stiffness (Mellisions & Mead, 1977). In order to minimise this affect, participants performed the manoeuvres with the same head and neck flexion-extension and rotation (i.e., head upright, chin maintaining same distance from the chest). Whilst performing recumbent respiratory manoeuvres, participants were required to start the manoeuvre by either inhaling to TLC or exhaling to RV in the upright seated position. Participants were then assisted to the correct recumbent position before initiating the respiratory manoeuvre thus maintaining a consistent head and neck posture and assuring subjects reached the appropriate lung volume for each manoeuvre. Although we cannot be certain that all participants were able to sustain the achieved lung volume while being repositioned, each manoeuvre was performed a minimum of three times with at least two values within the specified parameters (see section 3.2.2) to ensure reliable measurements. However, it is important to acknowledge there may have been a degree of error in the positioning of the participants to the required postures at specified hip angles which contributed to the relatively poor reliability that we observed.

5.5: CONCLUSION

Significant interaction effects between posture and PEF ($P = 0.020$), FVC ($P = 0.033$) and FEV_1 ($P = 0.021$) suggest that respiratory function was influenced by posture, but poor reliability rendered some paired comparisons non-significant. Notwithstanding this limitation, it seems clear that respiratory function tended to be optimised in the seated or more upright postures, and minimised in unsupported recumbent postures; thus suggesting that respiratory function was influenced by postural co-contraction of the trunk muscles. The lack of between day reliability of the outcome measures in this study would be insufficient to distinguish any influence of RMT; therefore no further investigation is warranted for posture-specific IMT in simulated rowing postures.

CHAPTER SIX

THE INFLUENCE OF LOAD MAGNITUDE UPON REPETITION MAXIMUM AND CARDIOVASCULAR RESPONSES TO ACUTE INSPIRATORY LOADING

6.1 INTRODUCTION

In 1976, Leith and Bradley demonstrated that ventilatory muscle strength and endurance could be increased by the application of an appropriate respiratory muscle training regimen (RMT). Inspiratory muscle endurance and strength training have been shown to evoke different, training-specific, muscle adaptations, but despite the differing adaptations that they elicit, both forms of training improve whole body exercise performance (Markov et al., 2001; Romer et al., 2002a). This is most likely because both forms of training elicit adaptations that affect a common primary mechanism in the ergogenic effect of RMT; both delay the activation of the inspiratory muscle metaboreflex (McConnell & Lomax, 2006).

During a flow resistive loading breathing task, activation of the respiratory muscle metaboreflex can be identified by a time-dependent increase in mean arterial blood pressure (MAP) (McConnell & Lomax, 2006; Witt et al., 2007) and a rise in heart rate (f_c) (St Croix et al., 2000; Sheel et al., 2001; McConnell & Lomax, 2006; Witt et al., 2007). However, following a 4-5 wk period of pressure threshold IMT these cardiovascular changes were attenuated, as demonstrated by a blunted increase in both MAP and f_c (Witt et al., 2007), as well as a failure to elicit a reduction in limb blood flow post-IMT (McConnell & Lomax, 2006). However, it is unclear whether the metaboreflex is activated during acute inspiratory pressure threshold loading (of the type employed during IMT), or indeed, whether this is an obligatory stimulus to adaptations that result in changes to activation of this reflex following IMT.

Traditionally, studies of pressure threshold IMT in healthy young adults have used loads of 50-60% maximal inspiratory pressure (P_{Imax}) to increase inspiratory muscle

strength and endurance and to assess the effects of IMT upon exercise tolerance (see table 2.1). Studies employing alternative loading methods have trained the inspiratory muscles at higher intensities (> 60% P_{Imax} or sustained maximal inspiratory pressure), but elicited similar improvements in inspiratory muscle strength (Enright, Unnithan, Heward, Withnall & Davies, 2006; Feutz et al., 2006; Huang, Martin & Davenport, 2003, 2009; Gething et al., 2004b).

It is important to optimise the potential benefits and to minimise the time investment for athletes in training interventions such as IMT. Characterising the acute responses to a range of inspiratory loading intensities is the first step to achieving this. However, to date, there is no published data reporting the relationship between inspiratory pressure threshold load and repetition maximum (RM) as a percentage of P_{Imax}. Previous studies have measured T_{lim} and cardiovascular responses using inspiratory flow resistive loading (Bellemare & Grassino, 1982a, 1982b; Mador & Acevedo, 1991; McKenzie, Allen, Butler & Gandevia, 1997a; Roussos & Macklem, 1977; Sheel et al., 2001). However unlike inspiratory flow resistive loading, the principal difference using pressure threshold loading is the interaction of the fixed load with the inspiratory muscle length-tension (pressure volume) relationship. This interaction is such that, the greater the magnitude of the inspiratory pressure threshold load, the smaller the tidal volume excursion that can be achieved. Thus, not only do higher loads result in a smaller number of repetitions to task failure, they may also be associated with a reduction in the amount of external work undertaken by the inspiratory muscles. To date, no study has examined any aspects of breathing pattern, its response to a range of loads, or the influence of load magnitude upon external work of breathing using pressure threshold loading. Similarly, the cardiovascular

responses to pressure threshold loading remain uncharacterised, so it is unknown whether traditional pressure threshold IMT at 50-60% P_{Imax} activates the inspiratory muscle metaboreflex.

The aim of this study was to characterise the RM for a range of inspiratory pressure threshold loads, and to determine whether activation of the respiratory muscle metaboreflex accompanies acute pressure threshold loading. It is hypothesised that one or more of the loading protocols will activate the inspiratory muscle metaboreflex.

6.2: METHODOLOGY

6.2.1: PARTICIPANTS

Eight healthy competitive male rowers volunteered to participate in this study, which was approved by the School Ethics Committee (Appendix A-5). All participants were recruited from Buckinghamshire New University. Prior to testing, all participants completed a health questionnaire and informed consent form.

Participants were recommended to maintain their normal diet in the few days preceding the exercise tests. Participants were also requested to avoid alcohol and vigorous exercise two days before the testing sessions and to avoid caffeinated beverages on test day. To minimise the effects of inspiratory muscle fatigue (IMF), participants were limited to one test session per day; thus requiring participants to be tested on seven separate occasions.

6.2.2: GENERAL DESIGN

Each participant was required to attend seven testing sessions. During the first session, P_{Imax}, resting tidal volume (V_T) and forced vital capacity (FVC) were assessed. On the following six test sessions, participants (n = 8) were required to perform a series of pressure threshold breathing tasks at various loads using a pressure threshold inspiratory muscle trainer. Inspiratory loads corresponding to 50%, 60%, 70%, 80% and 90% of P_{Imax} were assessed, and participants breathed against each load to the limit of tolerance (T_{lim}) at a breathing frequency of 15 breaths per minute. During each test session, pulmonary and cardiovascular responses, including V_T, beat by beat blood pressure and $f_{c,}$ were measured. Using these data, the relative training load and breath volume at each load were determined.

6.2.3: PROCEDURES

A detailed description of the instrumentation and testing procedures are provided in Chapter 3.

PARTICIPANT CHARACTERISTICS

Stature, body mass and respiratory function were assessed at the initial testing session. Details of anthropometric measurements are described in section 3.2.1. Measurements recorded at baseline are presented in Table 6.1.

Table 6.1 Descriptive characteristics of the participants (mean ± SD).

	Participants (n = 8)
Anthropometry	
Age (y)	22.0 ± 2.1
Stature (cm)	183.7 ± 11.9
Body mass (kg)	86.0 ± 9.9
Respiratory Function	
P_{Imax} (cm H_2O)	193.4 ± 26.7
FVC (L)	5.2 ± 1.02
Resting V_T (L)	1.3 ± 0.26

Note: P_{Imax}, maximal inspiratory mouth pressure; FVC, forced vital capacity; $V_{T,}$ tidal volume.

INSPIRATORY WARM-UP

Prior to inspiratory muscle strength testing, participants were instructed on proper usage of the pressure threshold-loading device for the inspiratory warm-up (POWERbreathe®, Gaiam Ltd., Southam, UK). Participants were instructed to perform 2 sets of inspiratory breaths at a resistance set to 40 RM (~40% P_{Imax}). This loading intensity has been shown to effectively warm-up the inspiratory muscles and attenuates the effect of repeated inspiratory measurements (Volianitis et al., 2001a).

A detailed description of the warm-up procedures is outlined in section 3.2.2.

INSPIRATORY MUSCLE STRENGTH

Maximal inspiratory pressure (P_{Imax}), used to determine the strength of the inspiratory muscles, was assessed using a portable hand held mouth pressure meter (Morgan

Medical, UK). The assessment of P_{Imax} required a sharp, forceful effort maintained for a minimum of ~2 s. The pressure meter incorporated a 1 mm leak to prevent glottic closure during the P_{Imax} manoeuvre (Black & Hyatt, 1969). Measurements were repeated until three technically acceptable manoeuvres were achieved within 3-5 centimetres of water (cm H_2O); the best of these three were recorded and presented in cm H_2O. A detailed description of the equipment and the procedures used for testing P_{Imax} is presented in section 3.2.2.

PULMONARY FUNCTION

Resting and loaded breath volumes were assessed using an online computer software package. Participants breathed through a differential pressure transducer (BIOPAC MP30, © BIOPAC Systems Inc., Goleta, USA), which measured changes in airflow and volume. Spirometry was performed to assess resting V_T and FVC; both are presented in litres (L).

MEASUREMENT OF TIDAL VOLUME DURING PRESSURE THRESHOLD LOADING

Participants performed a series of loaded inspiratory breathing tasks using a pressure threshold training device (POWERbreathe®, Gaiam Ltd., Southam, UK). The breathing tasks consisted of loads of 50%, 60%, 70%, 80% and 90% of P_{Imax} performed in randomised order. The participants were asked to complete each load to the limit of tolerance (T_{lim}), but no encouragement was provided during the task, and no indication was provided as to how many breaths they should perform. A timer was used to regulate breathing frequency to 15 breaths per minute (4 s per breath; duty cycle 0.3 - 0.5). After 15 minutes, any participants able to maintain the pressure

threshold load were stopped. Participants were not informed of the cut-off time of 15 min until they reached that point. All participants were encouraged to perform to task failure and not towards a target time, or number of breaths. The duration from the onset of the task to the point the subject removed the mouthpiece was termed T_{lim} and was presented in minutes: seconds (m:s).

Tidal volume (V_T) was measured during each loading task, and was predicted to decline with increasing load and with increasing repetitions (due to the effects of the length-tension relationship and fatigue). Since the time course of the within-test change in V_T was unknown, an objective V_T threshold was determined retrospectively to define the maximum number of repetitions. Following data analysis, a V_T value of 10% of FVC was defined as a threshold for determining the RM at each load; any breaths occurring after V_T had fallen below 10% FVC were not counted (for RM purposes).

ASSESSMENT OF CARDIOVASCULAR RESPONSES

Measures of f_c and arterial blood pressure were made non-invasively during the loaded breathing task using an automated combined continuous blood pressure monitor (Colin CBM-7000, COLIN, Scanmed, UK). Blood pressure was measured using arterial tonometry in which a solid-state blood pressure transducer sensor was attached to the participants left wrist over the radial artery. An oscillometric brachial cuff provided calibration for the pressure transducer sensor. A change in beat by beat MAP was used to determine the threshold for activation of the 'inspiratory metaboreflex' (Witt et al., 2007). Measures of MAP, systolic and diastolic blood pressure (SBP and DBP, respectively) were automatically calculated by the Colin

software and are presented in millimetres of mercury (mmHg). Continuous f_c was recorded and presented as beats per minute (bpm).

6.2.4: DATA ANALYSIS

Data were analysed using two distinct time intervals. Firstly, to account for differences in the number of repetitions achieved and changes in V_T, each breathing task was divided into isotime quartiles. Secondly, pulmonary and cardiovascular data were analysed every 30 s for the first 3 min at loads of 50% and 60% P_{Imax} and every 15 s for the first minute at loads of 70%, 80% and 90% P_{Imax} to determine the onset, if present, of the inspiratory muscle metaboreflex. Mean values were calculated for each outcome variable and subjected to statistical analysis. Participants not achieving 4 breaths for a given task were excluded from the analysis at that particular load. In addition, an approximate estimation of inspiratory work of breathing was made to determine if the combination of load and volume resulted in more or less work at any given inspiratory load. Average external work of breathing was calculated for each resistive load using the following equation:

External work of breathing = force [pressure] x distance [volume], therefore

External work of breathing = inspiratory load (cm H_2O) x V_T (L)

External work of breathing=cm H_2O.L

A repeated measures analysis of variance (ANOVA) was used to determine physiological changes over time. Planned pairwise comparisons were made to analyse significant interaction effects using the Bonferroni adjustment. Pearson's correlation

coefficients were performed to determine relationships between physiological and performance variables. Probability values $\leq$ 0.05 were considered significant. Statistical and mean data were calculated using the statistical software SPSS V16.0 for Windows (SPSS Inc, Chicago, IL, USA). All results are expressed in mean ± SD unless otherwise stated.

6.3: RESULTS

6.3.1: PULMONARY DATA

REPETITIONS AT EACH RESISTIVE LOAD

The total number of breaths performed at each load was assessed. A repeated measures analysis showed a within-subject effect for the total number of breaths (P = 0.001; Greenhouse-Geisser) demonstrating differences in the number of repetitions performed at different loads. As shown in Figure 6.1, there was a decrease in the total repetitions performed at 50% P_{Imax} compared to 70% (P = 0.011), 80% (P = 0.009) and 90% P_{Imax} loads (P = 0.010).

Similarly, there was a decrease in the number of repetitions in V_T >10% FVC (T) within- subjects (P = 0.001; Greenhouse-Geisser) at 50%T compared to 70%T (P = 0.013), 80%T (P = 0.010) and 90%T (P = 0.011). Average total number of repetitions, repetitions performed at a V_T > 10% FVC threshold load (T), and average T_{lim} at each load is presented for comparison in Table 6.2.

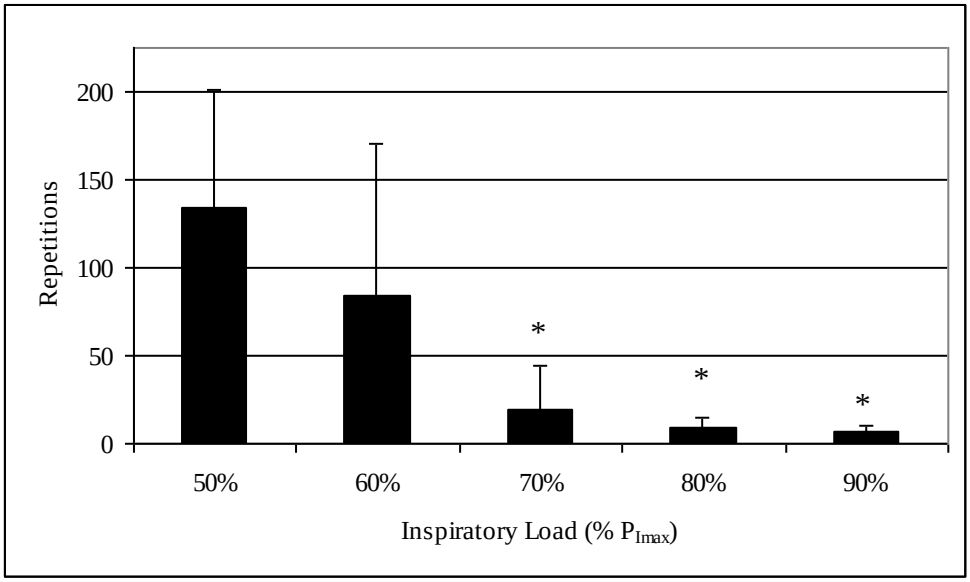


Figure 6.1 Total repetitions performed per inspiratory load. *, significantly different compared to 50% P_{Imax} load ($p \leq 0.05$).

Table 6.2 Average total repetitions, number of repetitions with V_T >10% FVC (T) and average time at each load.

	Mean (breaths)	**Minimum (breaths)**	**Maximum (breaths)**	**T_{lim} (min:sec)**
Total Repetitions				
50%	134.6 ± 66.9	57	217	8:57 ± 4:28
60%	84.6 ± 85.4	14	217	5:39 ± 5:42
70%	19.5 ± 24.4*	6	76	1:18 ± 1:36
80%	8.9 ± 6.0*	4	21	0:32 ± 0:24
90%	7.1 ± 3.3*	2	12	0:28 ± 0:13
Repetitions >10%FVC				
50% T	133.6 ± 68.2	54	217	8:54 ± 4:30
60% T	84.5 ± 85.5	14	217	5:34 ± 5:35
70% T	17.3 ± 25.5†	0	76	1:12 ± 1:35
80% T	7.1 ± 7.0†	2	21	0:28 ± 0:28
90% T	**4.6 ± 3.7**†	2	11	0:18 ± 0:15

Note: n=8. V_T >10% FVC, tidal volume greater than 10% of forced vital capacity; T_{lim}, limit of tolerance. *, significantly different compared to 50% P_{Imax} load ($p \leq 0.05$); †, significantly different compared to 50%T ($p \leq 0.05$).

Each participant completed the various breathing tasks at different time points. As shown in Figure 6.2, there was an abrupt drop in T_{lim} at loads $\geq$ 70% P_{Imax}. During the 50% and 60% loads, there were a few participants (n = 3 and n = 2, respectively) who maintained the task to the 15 min threshold; at which point their session was ended. However, on average, for this group of subjects, the 30 RM corresponded to about 65% P_{Imax}.

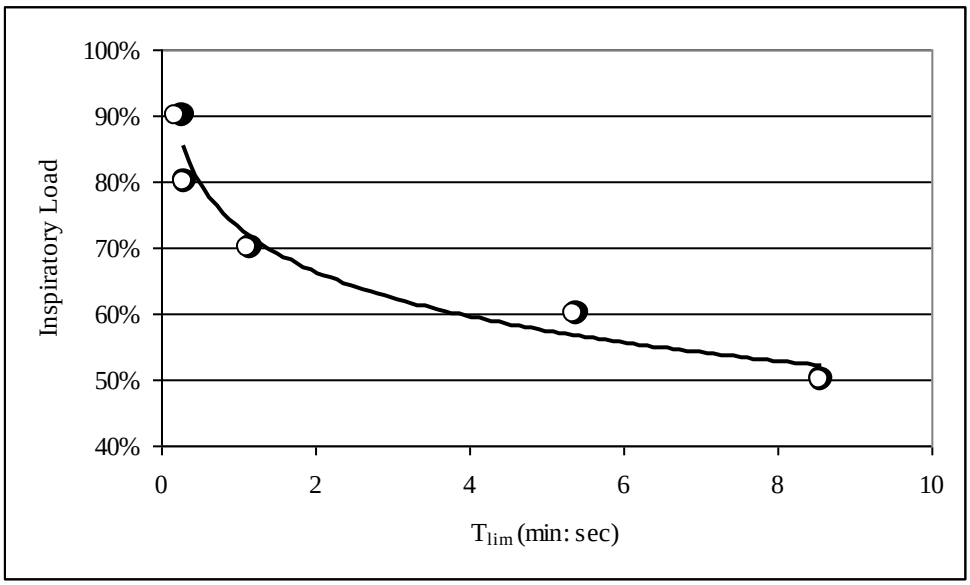


Figure 6.2 Endurance time (T_{lim}) for total repetitions and repetitions at V_T >10%FVC for each load. Note: ●, total repetitions; ○, repetitions at >10%FVC.

Bivariate correlations between anthropometric and pulmonary data were compared to the number of repetitions performed to determine variations in task performance. Strong positive correlations were found between FVC and the number of repetitions performed at 70% P_{Imax} (r = 0.783; *P* = 0.022), 70%T (r = 0.806; *P* = 0.016), 80% P_{Imax} (r = 0.717; *P* = 0.045) and 90%T (r = 0.841; *P* = 0.009). Although these relationships were significant, it seems that these were due primarily to one or two

participants who were outliers. There was no correlation between the number of repetitions performed, stature, body mass, V_T, or P_{Imax} at any load ($P > 0.05$).

WITHIN TASK CHANGES IN TIDAL VOLUME

At isotime 1 (first quartile of the test), there was a difference between ($P = 0.009$) and within-subjects ($P = 0.007$) as shown in a reduced V_T at loads of 70% and 80% P_{Imax} compared to 60% P_{Imax} ($P < 0.05$). Albeit close, there were no differences at 50% compared to 80% P_{Imax} load ($P = 0.054$). Significant differences were detected in V_T between subjects at 60%T ($P = 0.039$) and within-subjects over time at 50%T ($P = 0.023$), 60%T ($P = 0.006$), 70%T ($P = 0.041$) and 80%T ($P = 0.000$; see table 6.3). As shown in Figure 6.3, V_T decreased over time at 50%T from isotime 2 to isotime 4 ($P = 0.027$) and at 60%T from isotime 1 to isotime 4 ($P = 0.038$), and from isotime 2 to isotime 3 ($P = 0.040$) and isotime 4 ($P = 0.002$). A decline in V_T was also detected at 70%T (n = 6) from isotime 1 to isotime 4 ($P = 0.023$), at 80%T (n = 6) from isotime 1 to isotime 2 ($P = 0.016$), isotime 3 ($P = 0.008$) and isotime 4 ($P = 0.011$). Only 2 participants (n = 2) achieved a sufficient number of breaths at the 90% P_{Imax} load, consequently no analysis was performed at this load.

Table 6.3 Comparison of V_T and V_T%FVC across isotime quartiles (Q) at each load.

	50% P_{Imax} (n = 8)	**60% P_{Imax}** (n = 8)	**70% P_{Imax}** (n = 6)	**80% P_{Imax}** (n = 6)
V_T (L)				
Q1	2.4 ± 1.0	2.3 ± 0.8	1.6 ± 1.1§	1.4 ± 0.6§
Q2	2.3 ± 0.8	2.2 ± 0.8	1.4 ± 1.2	1.1 ± 0.5*
Q3	2.1 ± 0.7	2.0 ± 0.8†	1.4 ± 1.2	1.0 ± 0.4*
Q4	2.0 ± 0.9†	1.9 ± 0.8*†	1.0 ± 0.8*	0.8 ± 0.4*
V_T%FVC (%)				
Q1	44.8 ± 16.1	43.7 ± 12.5	30.7 ± 4.2‡§	29.6 ± 8.1‡
Q2	44.0 ± 14.8	41.5 ± 12.1	26.4 ± 7.8	20.5 ± 5.6
Q3	42.6 ± 15.7	38.3 ± 15.2	24.1 ± 9.9	18.5 ± 4.3*
Q4	41.7 ± 18.9	36.2 ± 14.7†	21.6 ± 8.3	14.4 ± 4.9*

Note: P_{Imax}, maximal inspiratory mouth pressure; V_T, tidal volume; V_T%FVC, tidal volume as a percent of forced vital capacity. *, significantly different to isotime 1. †, significantly different to isotime 2. ‡, significantly different compared to 50% load ($p \leq 0.05$). §, significantly different compared to 60% load ($p \leq 0.05$).

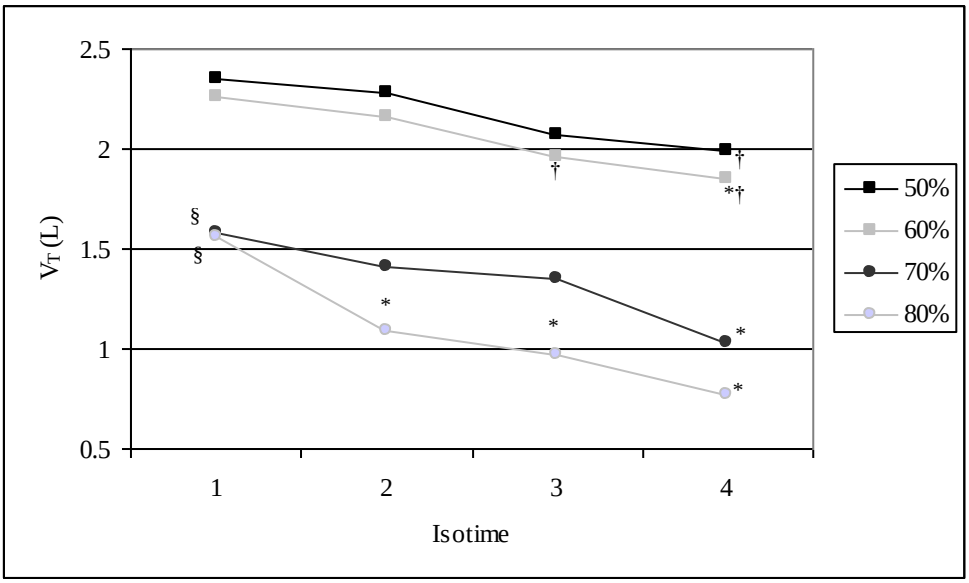


Figure 6.3 Comparison of V_T over isotime quartiles at different training intensities.

Note: V_T, tidal volume. *, significantly different compared to isotime 1 ($p \leq 0.05$). †, significantly different compared to isotime 2 ($p \leq 0.05$). ‡, significantly different compared to 50% load ($p \leq 0.05$). §, significantly different compared to 60% load ($p \leq 0.05$).

WITHIN TASK CHANGES IN TIDAL VOLUME AS A PERCENT OF FORCED VITAL CAPACITY

As shown in Figure 6.4, the V_T as a percent of FVC (V_T%FVC) was calculated for loads of 50-80%T. Significant differences were detected within-subjects at isotime 1 ($P = 0.024$) in which paired t-tests showed a lower V_T at loads > 70% P_{Imax} compared to 50% and 60% P_{Imax} ($P < 0.05$). Over time there were reductions in V_T%FVC within-subjects at 60%T, 70%T and 80%T ($P < 0.05$); no changes were evident at 50%T. Post hoc tests revealed a decrease in V_T%FVC at 60%T from isotime 2 to isotime 4 ($P = 0.047$) and at 80%T from isotime 1 and isotime 3 ($P = 0.031$) and 4 ($P = 0.050$).

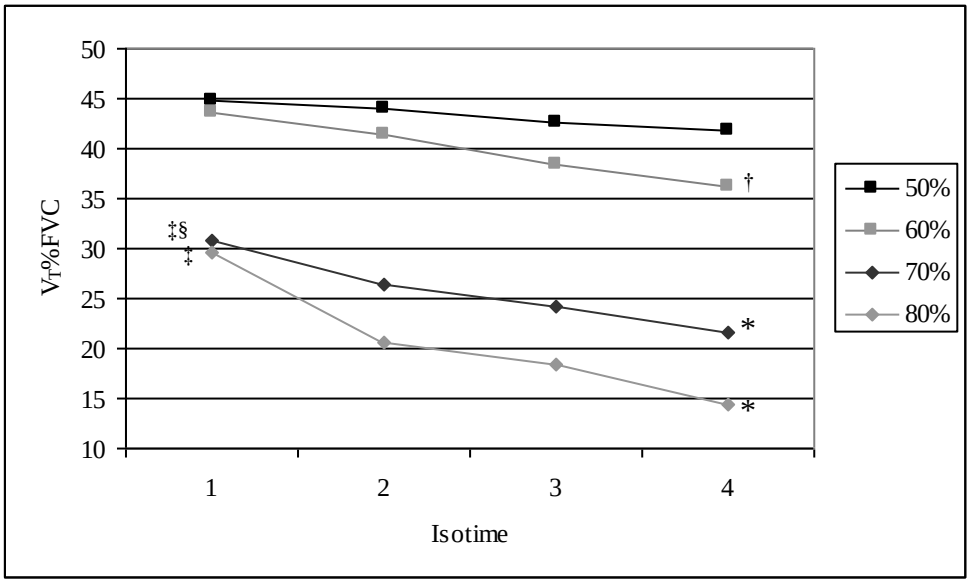


Figure 6.4 Comparison of V_T%FVC across isotime quartile for all resistive loads. Note: V_T%FVC, tidal volume as a percent of forced vital capacity. *, significantly different over time ($p \leq 0.05$). †, significantly different to isotime 2 ($p \leq 0.05$). ‡, significantly different compared to 50% load ($p \leq 0.05$). §, significantly different compared to 60% P_{Imax} load ($p \leq 0.05$).

ESTIMATION OF AVERAGE WORK PERFORMED

There was a within-subject effect over time (P = 0.006; Greenhouse Geisser) when comparing the average amount of work performed for each load (see table 6.4). The estimated work performed was highest in the 60% P_{Imax} load in which there was a increase in the average work performed compared to 50% (13.6%; P = 0.012), 70% (22.5%; P = 0.023) and 80% (40.6%; P = 0.043) P_{Imax} loads (see fig. 6.4). Inspiratory work performed at all loads was highest within the first quartile followed by a decrease in the amount of work performed over time at all loads ($P < 0.05$). Bivariate correlations were performed to compare the relationship between average work performed to average f_c at each load and to the number of repetitions at each load; no correlation was found at any load.

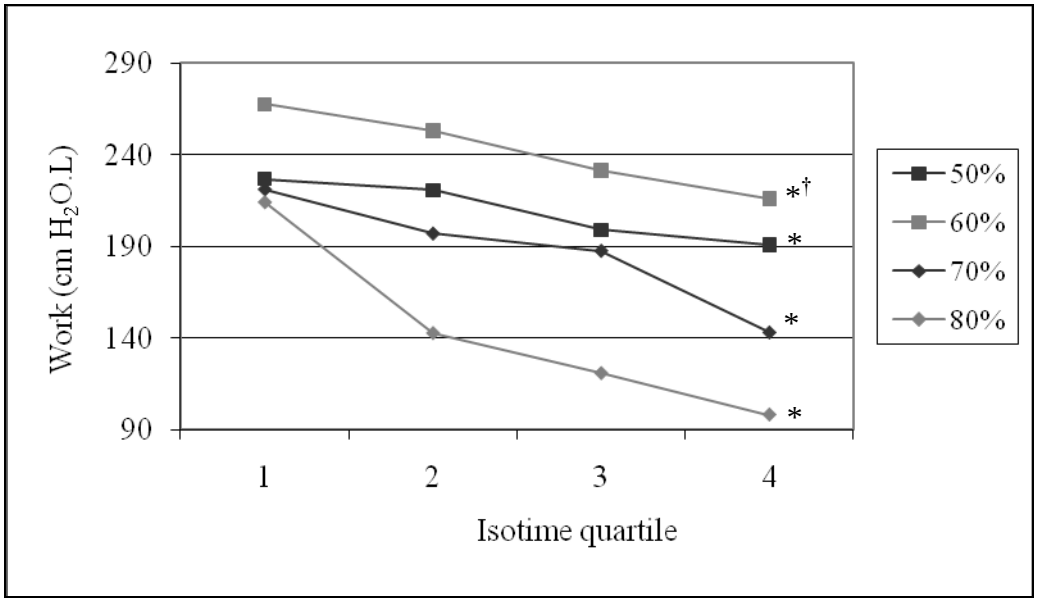


Figure 6.5 Comparison of work performed for each resistive load. *, significantly different over time ($p \leq 0.05$). †, significantly different compared to other loads ($p \leq 0.05$).

Table 6.4 Comparison of inspiratory work performed across isotime quartiles for each resistive load.

	50%	60%	70%	80%
Work (cm H_2O.L)				
Q1	226.8 ± 112.9	267.6 ± 114.7	221.3 ± 170.5	214.0 ± 100.6
Q2	220.8 ± 89.3	253.3 ± 107.5	197.1 ± 177.7	142.5 ± 86.6
Q3	199.1 ± 79.2	231.4 ± 110.0	187.7 ± 187.0	120.7 ± 74.2
Q4	190.9 ± 92.8*	216.1 ± 105.1*	143.0 ± 124.7*	98.0 ± 63.0*
Average Work Performed	209.4 ± 17.2	242.1 ± 22.8†	187.3 ± 32.7	143.8 50.2
Total Work Performed	1064.2 ± 17.1	1233.2 ± 22.8	969.1 ± 32.7	769.2 ± 50.2

Note: *, significantly different over time ($p \leq 0.05$). †, significantly different compared to other loads ($p \leq 0.05$).

6.3.2: CARDIOVASCULAR RESPONSE

There was a significant reduction in the number of repetitions performed at loads ≥ 70% P_{Imax} (< 19 breaths; see table 6.2) with a lot of variation in individual tolerance. Hence, two separate time analyses were performed. Analysis of loads at 50% and 60% P_{Imax} were analysed in 30 s intervals for the first three minutes. Whereas loads of ≥ 70% P_{Imax} were analysed in 15 s intervals for the first minute as some participants were unable to maintain breathing for > 30 s.

CARDIOVASCULAR RESPONSES TO PRESSURE THRESHOLD LOADING AT ≤ 60% P_{Imax}

It has been shown that MAP and f_c increases within the first 2-3 min of resistive breathing at 60% P_{Imax} (Witt et al., 2007); therefore in order to determine the time

interval for the rise in MAP and $f_{c,}$ an analysis of cardiovascular responses for each load was performed in 30 s intervals up to the first three min (see table 6.5). There were no differences between loads for MAP (P = 0.343), SBP (P =0.314) or DBP (P = 0.313); however there was a sharp and sustained rise in blood pressure response in the 60% compared to the 50% P_{Imax} load (see fig. 6.6). Therefore, planned pairwise comparisons were performed, corrected using a Bonferroni adjustment based on the number of comparisons, to determine if there were any significant changes within-loads (P set at $\leq$ 0.016) and between-loads (P set at $\leq$ 0.025) at the 30 s, 60 s and 90 s time intervals compared to baseline. The lack of significance in parameters beyond the 90 s time interval is most likely due to the decreasing number of participants being able to continue with the task at the 60% P_{Imax} load. Using the critical P values above, there was an increase from baseline to 60 s time interval in MAP (P = 0.016) and DBP (P = 0.015) at the 60% P_{Imax} load. The 60% P_{Imax} load elicited a sharp and sustained rise in SBP from baseline to the 60 s (P = 0.002) and 90 s (P = 0.002) time interval; there was also a rise in SBP at the 30 s time interval compared to the 50% P_{Imax} load (P = 0.020). No change in blood pressure was evident over time in the 50% P_{Imax} load.

A repeated measures ANOVA revealed a between subjects (P = 0.002) and within-subject effect (P = 0.001) over time in f_c when comparing the 50% and 60% P_{Imax} load. Heart rate (f_c) exhibited a sustained increase from baseline to 30 s in the 50% (P = 0.000) and 60% (P = 0.002) P_{Imax} load. Pairwise comparisons performed using a Bonferroni correction (P set at $\leq$ 0.016) revealed an increase in f_c at 60 s (P = 0.015) and 90 s (P = 0.002) time intervals compared to baseline.

Figure 6.6 shows the percentage change in cardiovascular responses compared to baseline. A repeated measures ANOVA showed an increase over time in f_c in the 60% P_{Imax} load (P = 0.048; Greenhouse Geisser); but no change in the 50% P_{Imax} load (P = 0.963).

Table 6.5 Comparison of physiological responses at 30 s intervals for the first 3 min at loads of 50% and 60% P_{Imax}.

	V_T (L)	**MAP** (mmHg)	**SBP** (mmHg)	**DBP** (mmHg)	**f_c** (bpm)	**Work** (cm H_2O.L)
50% (n=8)						
Pre-test	1.3 ± 0.5	87.9 ± 11.3	136.0 ± 19.8	69.8 ± 10.5	73.3 ± 11.4	
30 s	2.3 ± 1.0	90.2 ± 13.4	133.0 ± 20.5	71.4 ± 14.1	86.7 ± 12.6*	225.6 ± 107.6
60 s	2.4 ± 1.1	87.1 ± 13.9	132.9 ± 21.8	67.6 ± 14.4	88.6 ± 17.7*	234.5 ± 123.8
90 s	2.5 ± 1.1	90.8 ± 13.1	135.9 ± 20.8	71.0 ± 13.4	88.2 ± 17.0*	241.2 ± 119.8
120 s	2.3 ± 1.0	90.0 ± 13.2	135.0 ± 20.1	70.2 ± 13.6	89.4 ± 18.8	226.5 ± 113.8
150 s	2.4 ± 1.0	87.5 ± 12.2	131.9 ± 18.8	68.0 ± 12.9	87.4 ± 15.0	228.2 ± 116.6
180 s	2.2 ± 0.9	87.3 ± 12.8	131.8 ± 20.7	67.9 ± 12.5	88.9 ± 16.0	214.3 ± 103.7
60%						
Pre-test (n=8)	1.3 ± 0.5	92.3 ± 7.9	139.0 ± 16.8	70.6 ± 7.4	70.1 ± 13.2	
30 s (n=8)	2.3 ± 0.8	94.6 ± 9.7	142.8 ± 15.2†	73.5 ± 10.7	88.9 ± 18.5	269.1 ± 118.8
60 s (n=8)	2.1 ± 0.8	99.7 ± 10.1*	145.3 ± 20.0*	77.7 ± 8.1*	92.0 ± 20.8*	249.3 ± 112.3
90 s (n=7)	2.1 ± 0.8	103.3 ± 12.2	151.7 ± 22.4*	80.4 ± 11.1	98.0 ± 22.8*	250.1 ± 131.8
120 s (n=6)	2.1 ± 0.8	104.3 ± 16.7	149.1 ± 26.5	82.6 ± 13.5	95.8 ± 24.9	255.6 ± 134.6
150 s (n=4)	2.2 ± 1.0	109.2 ± 18.4	151.8 ± 29.4	87.7 ± 14.3	95.6 ± 29.2	271.3 ± 141.1
180 s (n=4)	2.3 ± 1.0	109.1 ± 20.1	153.8 ± 31.5	87.7 ± 15.0	100.7 ± 23.8	237.2 ± 145.0

Note: *, significantly different compared to baseline ($p \leq 0.05$); †, significantly different compared to 50% P_{Imax} load ($p \leq 0.05$). NB. Pairwise comparisons were only made at 30, 60 and 90 s, since there were insufficient subjects at later times for the 60% P_{Imax} load.

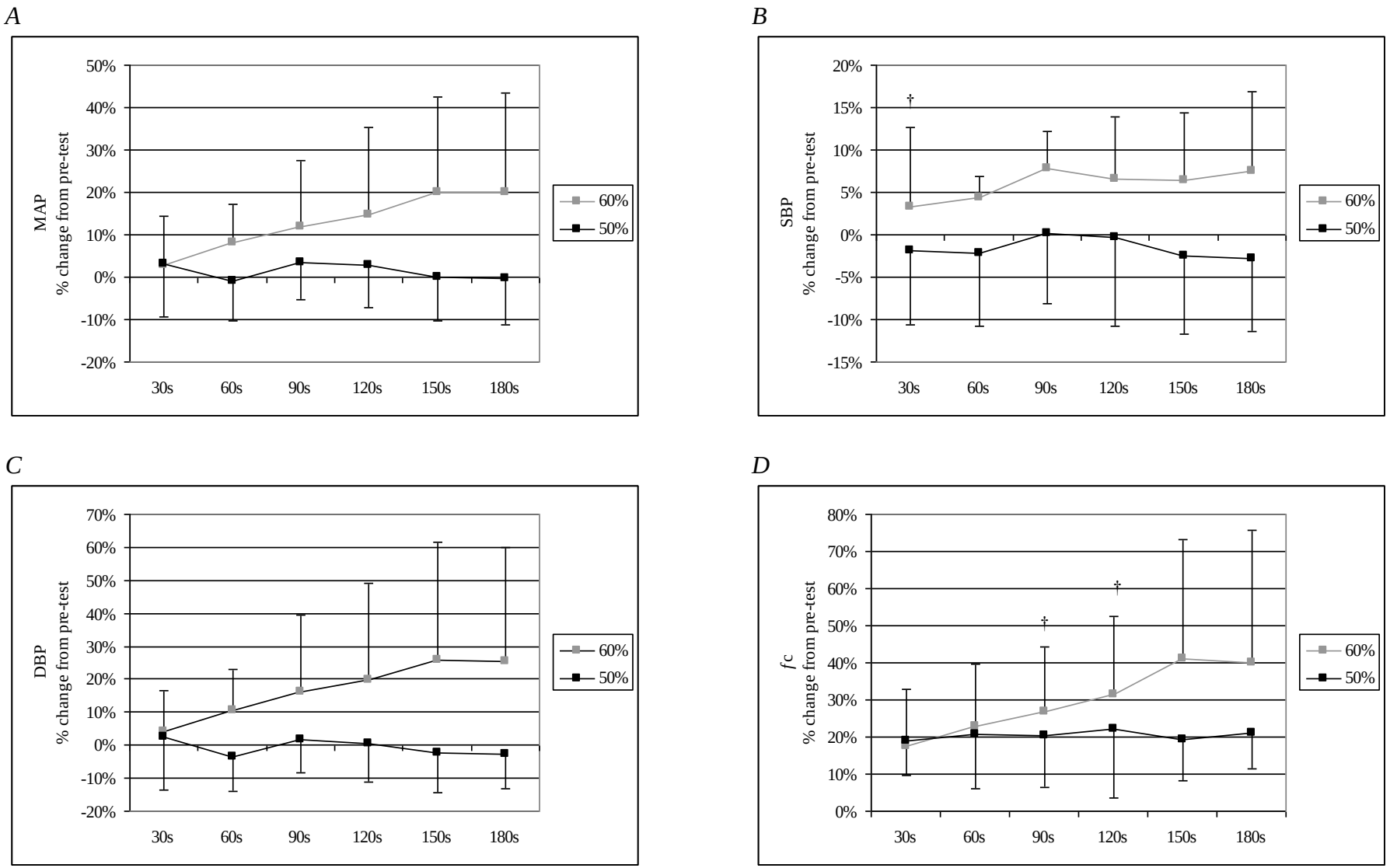


Figure 6.6 Comparison of percentage change from pre-test values for MAP (*A*), SBP (*B*), DBP (*C*) and f_c (*D*) at loads of 50% and 60% P_{Imax} loads. Note: MAP, SBP and DBP measured in mmHg. †, significantly different compared to 50% P_{Imax} load ($p \leq 0.05$).

ESTIMATION OF INSPIRATORY WORK PERFORMED

A comparison of average work at 50% and 60% P_{Imax} loads was performed to determine if there was a relationship between the increase in inspiratory work and a change in cardiovascular responses. There were no differences in average work performed across the different loads (P = 0.262). There was a 10.7% higher average work performed at 60% (255.4 ± 12.9 cm H_2O.L) compared to the 50% P_{Imax} load (228.4 ± 9.1 cm H_2O.L) during the first 2 min (see fig. 6.7). No statistical analysis was performed from 120 s to 180 s due to an insufficient number of participants (n=4) able to continue the task for 3 min at the 60% P_{Imax} load.

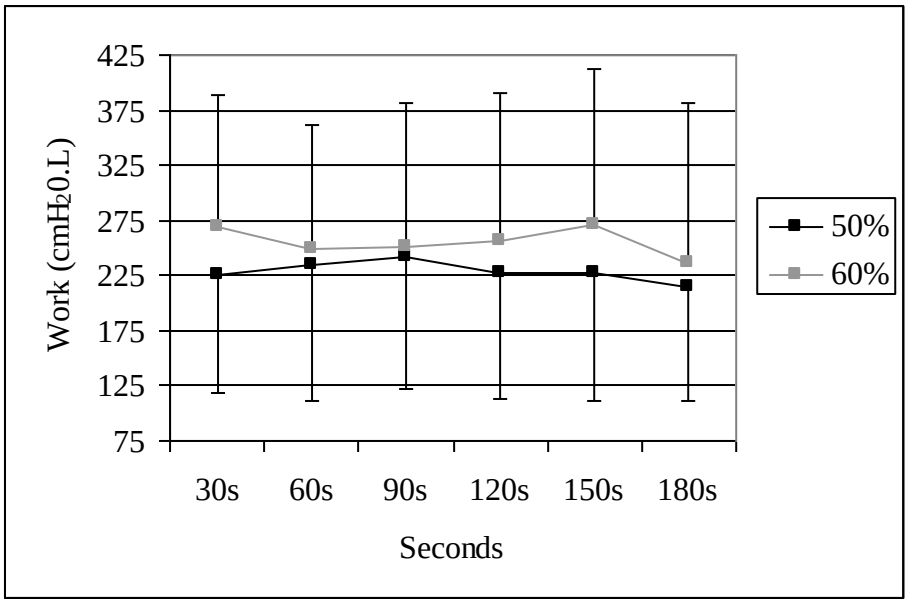


Figure 6.7 Comparison of inspiratory work performed at 30 s intervals for 50% and 60% P_{Imax} loads.

CARDIOVASCULAR RESPONSES TO PRESSURE THRESHOLD LOADING AT LOADS ≥70% P_{Imax}

Only those participants that performed a sufficient number of repetitions (at least 30 s of breathing) were considered in the temporal analysis. Table 6.6 provides a comparison of the cardiovascular responses at 15 s intervals for loads of 70%, 80% and 90% P_{Imax}. Although there was a within subject effect in all variables ($P < 0.05$), there was no differences between loads. Albeit not significant, there was a similar rise in MAP (7-9 mmHg), SBP (5-7 mmHg) and DBP (3-8 mmHg) at loads of 70-90% P_{Imax} from baseline to the first 30 s (see fig. 6.8). Each of these loads elicited an increase in f_c over time compared to baseline ($P < 0.05$). The data at loads ≥70% P_{Imax} respond similarly to the 50% and 60% P_{Imax} loads at the equivalent time points. Comparatively across all loads, the resistance at the 60% P_{Imax} load was the only load to demonstrate a sustained rise in MAP, SBP and f_c over time.

A repeated measures ANOVA revealed a change in cardiovascular responses over time at the 90% P_{Imax} load ($P < 0.05$) when comparing the percentage change at each time interval. There was a rise in MAP ($P = 0.005$), SBP ($P = 0.016$), DBP ($P = 0.004$) and f_c ($P = 0.049$) between baseline compared to the 30 s time interval.

Table 6.6 Comparison of cardiovascular responses at 15 s intervals for the first 45 s at loads of 70%, 80% and 90% P_{Imax}.

	V_T (L)	MAP (mmHg)	SBP (mmHg)	DBP (mmHg)	f_c (bpm)
70% P_{Imax}					
15 s Intervals					
Pre-test (n=8)	1.3 ± 0.5	91.0 ± 14.3	131.4 ± 20.9	74.2 ± 13.0	76.2 ± 10.3
15 s (n=8)	1.0 ± 0.6	94.8 ± 25.5	133.7 ± 33.5	75.8 ± 19.7	89.3 ± 14.9*
30 s (n=8)	0.8 ± 0.6	98.7 ± 26.0	136.4 ± 39.0	77.2 ± 19.5	91.8 ± 16.5*
45 s (n=3)	1.0 ± 0.8	95.2 ± 16.0	122.7 ± 15.9	75.0 ± 10.8	98.7 ± 17.5*
80% P_{Imax}					
15 s Intervals					
Pre-test (n=8)	1.5 ± 0.5	90.5 ± 9.6	133.1 ± 15.8	72.1 ± 11.4	73.4 ± 11.6
15 s (n=8)	1.3 ± 0.9	89.4 ± 6.9	126.0 ± 18.1	67.7 ± 9.8	92.2 ± 11.0*
30 s (n=6)	0.9 ± 0.8	97.3 ± 15.2	140.5 ± 24.8	78.3 ± 13.8	87.9 ± 11.7*
45 s (n=2)	1.5 ± 0.6	96.2 ± 0.21	142.2 ± 9.8	68.6 ± 4.0	109.4 ± 7.9
90% P_{Imax}					
15 s Intervals					
Pre-test (n=8)	1.3 ± 0.5	92.1 ± 7.9	133.1 ± 7.9	74.4 ± 7.9	70.6 ± 6.4
15 s (n=8)	0.8 ± 0.3	94.1 ± 11.5	129.5 ± 13.2	75.1 ± 10.0	86.3 ± 13.9*
30 s (n=5)	0.5 ± 0.3	101.0 ± 15.9	139.8 ± 19.6	82.3 ± 13.9	96.0 ± 19.2*
45 s (n=1)	0.5 ± 0.3	100.1	139.3	76.7	87.5

Note: MAP, mean arterial pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; f_c, heart rate. *, significantly different compared to baseline.

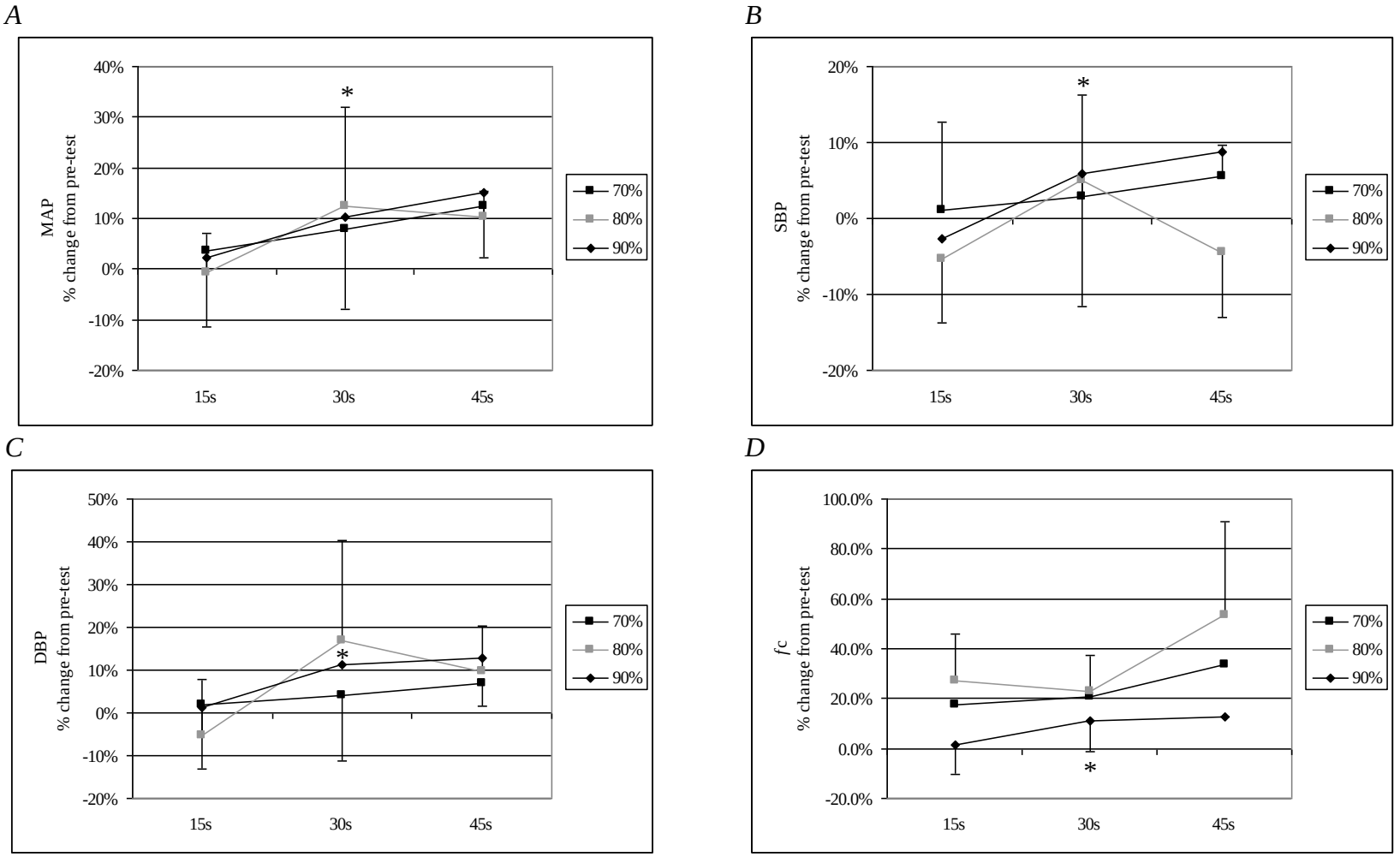


Figure 6.8 Comparison of percentage change from pre-test values for MAP (*A*), SBP (*B*), DBP (*C*) and f_c (*D*) at loads of 70%, 80% and 90%. *, significantly different compared to baseline.

6.4: DISCUSSION

6.4.1: MAIN FINDINGS

The purpose of this study was to characterise the acute cardiorespiratory responses to a range of pressure threshold loading protocols. To this end, all participants performed loaded breathing tasks at loads ranging from 50-90% P_{Imax} during which pulmonary and cardiovascular responses were assessed. Time to the T_{lim} was shown to decrease progressively as loading intensity increased (% P_{Imax}), and reduced abruptly at loads equal to and above 70% P_{Imax}. Estimated work was greatest at the 60% P_{Imax} load and lowest at the 80% P_{Imax} load. Although all loads elicited a sustained increase in f_c, only the 60% P_{Imax} load elicited a sustained rise in SBP and MAP after 2 minutes, providing some evidence for a metaboreflex response at this load.

6.4.2: TRAINING LOAD AND REPETITIONS

There was little difference between the total number of repetitions performed and repetitions defined objectively using the V_T%FVC threshold (see Table 6.2). At the lower intensity loads, participants were able to perform an average of 134 ± 68 repetitions (8.5 ± 4.0 min) at 50% P_{Imax} load and 85 ± 85 repetitions (5.39 ± 5.4 min) at the 60% P_{Imax} load. The T_{lim} at 60% P_{Imax} is consistent with Sheel et al. (2002) in which their subjects performed an average of 3-8 min against an inspiratory resistive load of 60 ± 10% P_{Imax} to task failure. In comparison, the participants in Witt et al.'s (2007) study breathed against a load of 60% P_{Imax} and were able to perform for an average of 8.55 min ± 52 s (range of 4.25 min – 16.36 min). These subjects sustained the task an average of ~3 min longer during their flow resistive breathing task compared to the present study. The difference between endurance times (T_{lim}) between the studies at the 60% P_{Imax} load may be due to the differences in

methodologies; for example, Witt et al. (2007) employed prolonged duty cycles following a pre-set target tracing of mouth pressure on a computer monitor and provided supplemental CO_2. The higher inspiratory flow rates associated with the pressure threshold loading used in the present study increases the relative load upon the inspiratory muscles, since they experienced functional weakening at higher velocities of shortening (Leblanc, Summers, Inman, Jones, Campbell & Killian, 1988). This may hasten the T_{lim} during pressure threshold loading.

Similar to skeletal muscles, there was an inverse relationship between the number of repetitions performed during inspiratory resistive breathing and the load (Roussos & Macklem, 1977). All participants appeared to experience a critical threshold for tolerance at loads >70% P_{Imax} above which T_{lim} decreased abruptly (> 1:18 min compared to 5:39 min at 60% P_{Imax} load). Our results showed a broadly similar relationship between inspiratory muscle load and number of repetitions to that of limb muscles, in that participants performed an average of 1-7 repetitions at training loads > 80% P_{Imax}, 7-17 repetitions between 70-80% P_{Imax} and >18 repetitions at loads $\leq$ 60% P_{Imax}. In traditional resistance training, loads >80-85% of the one RM (1 RM) are equivalent to 1-6 repetitions, intermediate loads at 70-80% 1 RM range from ~6-12 repetitions, with an increase in number of repetitions (>12-15 repetitions) at loads of $\leq$ 60% 1 RM (Kraemer et al., 2002). According to these findings, the magnitudes of inspiratory resistive loads are similar to the recommended workloads adopted for resistance training of the limb skeletal muscles.

Previous research has shown that inspiratory resistive loads can be tolerated indefinitely until they reach a critical level that result in task failure (McKenzie et al.,

1997a; Roussos & Macklem, 1977). Task failure, in this study, refers to when the subject was no longer able to continue with the breathing task (McKenzie et al., 1997a). This usually occurs when the muscle(s) are unable to maintain the target level of contraction for a period of time (Laghi, Topeli & Tobin, 1998). Research investigating task failure during voluntary contractions in limb skeletal muscle research has led to the principle of 'task dependency of muscle fatigue, which states that there is no single cause of muscle fatigue and that the dominant mechanism depends on the details of the task being performed' (Enoka, 2008). Potential mechanisms identified as influencing time to task failure following inspiratory resistive loading include: lack of subject motivation, decreased force generating capacity of the muscles due to diaphragmatic fatigue, hypercapnia, hypoxemia, sensory limitations, the load exceeding maximum strength or possibly a reduction in neural drive or voluntary activation of the respiratory muscles (Aubier et al., 1981; Bellemare & Grassino, 1982a, 1982b; Eastwood, Hillman & Finucane, 1994; Gorman, McKenzie & Gandevia, 1999; Luo, Hart, Mustfa, Lyall, Polkey & Moxham, 2001; McKenzie et al., 1997a; McKenzie, Allen & Gandevia, 1997b). As the variables listed previously were not assessed during this study, we cannot be sure exactly what led to task failure; whatever the cause of task failure in our subjects, the increase in load magnitude > 60% P_{Imax} resulted in a significant and sharp decrease in T_{lim}.

During inspiratory resistive loading, diaphragmatic fatigue has been shown to contribute to ventilatory task failure (Aubier et al., 1981; Mador & Acevedo, 1991: Rohrbach, Perret, Kayser, Boutellier & Spengler, 2003; Roussos & Macklem, 1977). Although numerous studies have investigated the causal link between ventilatory task

failure and diaphragm fatigue, the research remains contradictory. Rohrbach et al. (2003) found both diaphragm and rib cage muscle fatigue as assessed by internal pressures and cervical magnetic stimulation during inspiratory resistive loading (67% P_{Imax} load); however breathing endurance times were not related to IMF. In contrast, McKenzie et al. (1997a) demonstrated that the onset of severe dyspnoea and hypercapnoea (demonstrated by an increase in P_{ETCO2}) was associated with task failure during inspiratory resistive loading (65% P_{Imax} load); but their results showed no evidence of diaphragmatic fatigue as assessed by bilateral phrenic nerve stimulation (BPNS) and maximal mouth pressures. These data suggest that peripheral diaphragm fatigue does not necessarily coincide with ventilatory task failure during inspiratory resistive loading (Eastwood et al., 1994; Gorman et al., 1999; McKenzie et al., 1997a, 1997b; Yan, Sliwinski, Gauthier, Lichros, Zakynthinos & Macklem, 1993). Eastwood et al. (1994) also studied the effect of progressive threshold loading of the inspiratory muscles. The test protocol was designed to increase inspiratory resistance every 2 min until subjects reached task failure. They did not see diaphragm fatigue until very late in the breathing sessions, and even then it was not enough to elicit task failure. Their results showed that with increasing loads, breathing pattern and respiratory muscle recruitment were coordinated to generate more inspiratory force and increase endurance. At higher training intensities, these studies observed conditions of hypoxia, hypercapnoea (CO_2 retention) and sensory limitations (e.g., breathing discomfort) which may have contributed to task failure. Indeed, Gorman et al. (1999) in a similar study investigating task failure during inspiratory resistive loading concluded that the amount of breathing discomfort (e.g., due to a rise in CO_2) was related to the magnitude of the inspiratory resistive load. As we did not assess

IMF or CO_2 following each load, there is no way of knowing whether global IMF was present, and thus contributed to task failure in our subjects.

It has been suggested that stronger inspiratory muscles per se, lead to an increased ability to perform more repetitions to task failure, irrespective of the magnitude of the inspiratory load (Eastwood et al., 1994). However our findings do not support this, as there was only a weak correlation between P_{Imax} and number of repetitions performed at all loads. Other possible explanations for the wide range in individual ability include FVC, the individual's ability to work at a smaller V_T, or the metabolic profile of the inspiratory muscles (a more aerobic phenotype). It is possible that the correlation between FVC and number of breaths (70%T; r = 0.806; *P* = 0.016) may explain why some individuals were able to perform more repetitions at the same intensity. However, this relationship may be misleading as not all participants with high FVCs performed the highest number of repetitions. Alternatively, it may be that some individuals were able to breath at a smaller V_T during loaded breathing thus changing the magnitude of force output; by using a smaller V_T, the respiratory muscles are exerting less force and doing less work, which may lower effort perception (Oliven, Kelsen, Deal & Cherniack, 1993) during resistive breathing. Thus, a decreased effort perception or respiratory sensation would allow them to maintain breathing for longer. Conversely, these individual differences in performance, particularly at loads $\geq$ 60% P_{Imax}, may be due to cardiovascular or metabolic changes (i.e. respiratory muscle metaboreflex), which may occur at differing time points.

RELATIONSHIP BETWEEN IMT AND THE 30RM

Our subjects showed significant differences in their individual ability during inspiratory pressure threshold loading, particularly at loads $\geq$ 60% P_{Imax}. For example, at the lowest load of 50% P_{Imax} some participants (n = 3) were able to continue the breathing for the maximum 15 minute time period, whereas others reached task failure in less than 4 min (n = 2). These individual differences warranted further investigation as to whether a predetermined RM load (i.e. 30 RM) is more appropriate than a % of P_{Imax} as an appropriate training intensity. To explore this possibility, we grouped participant data according to the total repetitions performed nearest to a 30 RM load. The data sets used for the 30 RM load were between the 60% (n = 6) and 70% P_{Imax} load (n = 2). Comparatively, the 30 RM load was similar to the 60% P_{Imax} load and did not elicit any changes in average work performed or cardiovascular responses.

In previous IMT research (see table 1.1), training loads equivalent to the 30 RM have been shown to elicit increases in inspiratory muscle strength ($P < 0.05$). This load is also the recommended training intensity for IMT using pressure threshold loading for the POWERbreathe® training device (www.powerbreathe.com). It has been suggested by some studies that 30 RM is equivalent to ~50% P_{Imax} (see table 1.1); however this may be an underestimation for the athlete population. These results are supported by a recent abstract (Buckley, McConnell, Gorman & Mills, 2007), which specifically assessed breathing repetitions at all the set intensities on the POWERbreathe® Inspiratory Muscle Training device (Gaiam Ltd., Southam, UK). They evaluated the rating of perceived breathing effort during a 30 RM IMT session, concluding that 30

RM was equivalent to ~62 ± 21% of $P_{Imax.}$ Thus support our findings that loads ranging from 60-70% P_{Imax} are equivalent to the 30 RM.

A study investigating the use of IMT in elite male rowers (Klusiewicz et al., 2008) showed no change in P_{Imax} using a load of 50% P_{Imax}, whereas they found a 21% increase in P_{Imax} ($P > 0.05$) following 6 wk of IMT using a load of ~62 ± 3% P_{Imax}. Similarly, the untrained subjects in Witt et al.'s (2007) study trained using a pressure threshold loading device, performing three sets of 75 breaths at 50% P_{Imax}, thus demonstrating that loads > 50% P_{Imax} may be required to elicit a 30 RM.

6.4.3: RESPIRATORY CHANGES DURING PRESSURE THRESHOLD LOADING

The initial differences in V_T at isotime 1 for loads $\leq$ 60% P_{Imax} compared to loads $\geq$70% P_{Imax} (as shown in fig. 6.2) is presumably related to the non-linearity of the length-tension relationship being steeper above 60% P_{Imax} such that the same change in volume results in a larger change in P_{Imax}. It was evident from the estimate of inspiratory muscle work (calculated as V_T x Pressure load) that the greatest amount of work was performed at the 60% P_{Imax} load.

Albeit no change was evident over time in either V_T or V_T%FVC at the 50% P_{Imax} load, there was a decrease in V_T and V_T%FVC at loads $\geq$ 70% P_{Imax} compared to loads $\leq$ 60% P_{Imax} (see fig. 6.3 & 6.4; $P < 0.05$). At loads $\geq$ 60% P_{Imax}, both values decreased over time and proportionally with each increasing load. This temporal-related decline in V_T during resistive breathing has been shown in similar studies (Sheel et al., 2001; St Croix et al., 2001; Witt et al., 2007) and has been attributed to increasing inspiratory muscle fatigue. When inspiratory muscles become overloaded,

breathing pattern is altered such that f_R and $\dot{V}_E$ increases, while V_T declines or remains unchanged (Babcock et al., 1998; Eastwood et al., 1994; Mador & Acevedo, 1991; Sheel et al., 2002; Sliwinski et al., 1996; Witt et al., 2007); this hyperventilatory response results in hypercapnia one of the mechanisms contributing to ventilatory task failure (Gorman et al., 1999). The higher resistive loads were sufficient to overload the muscles as all participants showed a significant decrease in their ability to perform repetitions at workloads $\geq$ 70% P_{Imax} (an average of < 17 repetitions). Thus indicating a possible threshold for the influence of load upon V_T, which arises at loads > 60% P_{Imax}. As the 50% P_{Imax} load showed no change in V_T over time it may indicate that loads $\leq$ 50% P_{Imax} are insufficient for 'taxing' the inspiratory muscles.

6.4.4: CARDIOVASCULAR RESPONSE TO PRESSURE THRESHOLD LOADING

All participants demonstrated a rapid decrease in arterial blood pressure response during inspiration and an increase with each expiration at all loads. These characteristic 'respiratory swings' are generated by fluctuations in intrathoracic pressure, such that when airway resistance is high the forced expiration (i.e. partial Valsalva) causes compression of the thoracic organs by contracting the rib cage (Lee, Matthews & Sharpey-Schaffer, 1954).

Heart rate (f_c) increased following 2 min of resistive breathing at the 60% P_{Imax} load with a concomitant rise in both SBP and MAP when compared to the 50% P_{Imax} load. Earlier studies using resistive breathing tasks at workloads of 60% and 95% P_{Imax} (Sheel et al., 2001, 2002; St. Croix et al., 2000) showed a similar cardiovascular response. Both of these studies, designed to elicit diaphragm failure to mimic the demands during sustained high intensity exercise, demonstrated an increase in f_c

within the first minute with a non-significant rise in MAP. The initial increases in f_c are due to a rapid withdrawal of parasympathetic activity most likely attributed to centrally mediated factors or local mechanoreceptors (Tipton et al., 2006). The increase in inspiratory effort due to the larger pressures generated during loaded breathing may have lead to a significant vagal withdrawal (Hollander & Bouman, 1975); whereas the greater contractile force would increase mechanical deformation of the diaphragm thereby stimulating local mechanoreceptors (Jammes & Speck, 1995). However, changes due to mechanoreceptor usually appear earlier and are of a decreased magnitude compared to that of a chemoreflex response. Both Sheel et al. (2002) and Witt et al. (2007) showed a time dependent increase in both f_c and MAP within 2-3 min of the start of resistive breathing at 60% P_{Imax}. During heavy or maximal intensity exercise when diaphragmatic fatigue occurs, sympathetic activation is elevated thus eliciting an increase in f_c and arterial blood pressure. These time-delayed changes in the cardiovascular response have been linked to activation of the inspiratory muscle metaboreflex.

It is unlikely that any change in MAP at higher intensities ($\geq$ 70% P_{Imax}) is due to an inspiratory metaboreflex as the short time that the respiratory muscles were working (less than 1.12 ± 1.35 minutes) were unlikely to cause an accumulation in local metabolites. Previous studies have demonstrated no change in MAP in near maximal trials of 95% P_{Imax} (St Croix et al., 2000; Sheel et al., 2001); the authors suggested that although this load elicited near maximal recruitment of the diaphragm it did not cause diaphragmatic fatigue; hence, very heavy loading is also unlikely to induce activation of a metaboreflex response. Both the 60% and 70% P_{Imax} load elicited the highest magnitude of response in MAP and SBP compared to the other loads.

Similarly, limb muscle loading at submaximal loads performed to volitional fatigue (a % of maximum) generate an elevated haemodynamic response as compared to 1 RM loads (Fleck & Dean, 1987; MacDougall, Tuxen, Sale, Moroz & Sutton, 1985). In this study, the largest amount of work performed was during the 60% P_{Imax} load which also demonstrated the highest increase in MAP and SBP. It seems that training at a moderate intensity load (e.g., 60% P_{Imax}) for a longer duration generates more work, hence an increase in effort perception (Yan & Bates, 1999), compared to the higher loads ($\geq$70% P_{Imax}). Witt et al. (2007) report that a minimum of 2-3 min of resistive breathing before a change in MAP was identified. Therefore, it may be possible that there is a minimal threshold of training intensity and/or a time dependent response that must occur during acute inspiratory loading before local metaboreflexes respond (Augustyniak, Collins, Ansorge, Rossi & O'Leary, 2001; O'Leary, 1993; Rowell & O'Leary, 1990; Sheel et al., 2002).

Therefore, the training intensity may need to be taxing enough to elicit the inspiratory muscle metaboreflex to actually incur a training stimulus. This may be important in respect of the activation of the inspiratory muscle metaboreflex, since the data suggest that only the 60% P_{Imax} loading protocol created the muscle milieu required to induce activation. This is consistent with the findings from Sheel et al. (2001, 2002) and Witt et al. (2007) in which they demonstrated that a minimum inspiratory flow resistive load of ~60% P_{Imax} was required to sufficiently fatigue the diaphragm to initiate a metaboreflex response. In Sheel et al.'s (2002) study, the subjects were able to sustain the lower inspiratory flow resistive loads of 30%, 40% and 50% P_{Imax} for a longer duration, the intensity was insufficient to adequately fatigue the diaphragm; thus suggesting that fatiguing loads of 60% P_{Imax} or greater were necessary to activate the

metaboreflex response. As shown in Figure 6.5, there was a higher amount of inspiratory muscle work generated at the 60% P_{Imax} load compared to the other loads. It may be possible that a sufficient level of inspiratory muscle work (e.g., a threshold) must be achieved before the onset of the metaboreflex is activated. As estimated inspiratory muscle work was calculated as pressure x volume, it is plausible that the lower load (50% P_{Imax}) had insufficient 'pressure' whereas the higher loads ($\geq$ 70% P_{Imax}) were lacking sufficient volume (V_T) to generate sufficient levels of inspiratory muscle work to activate the inspiratory muscle metaboreflex.

Collectively, it appears that inspiratory loads must be of sufficient intensity and duration to induce fatiguing contractions which elicit a metaboreflex response. Further studies are required in order to identify whether this activation during training is an obligatory feature of the IMT-induced increase in metaboreflex threshold (McConnell & Lomax, 2006; Witt et al., 2007).

6.4.5: METHODOLOGICAL CONSIDERATIONS

There are a number of methodological considerations that may have limited interpretation of the results of this study. Firstly, the use of a volitional measurement to determine inspiratory muscle strength; ideally, P_{Imax} would be supplemented by electrically evoked measurement of diaphragm function. Although we did not train the inspiratory muscles or look for an improvement in inspiratory muscle strength, the work measurements were based on a percentage of each individual P_{Imax}. However, the utilisation of voluntary measurements performed according to the recommendations of the ATS/ERS (Green et al., 2002) has been shown to be a

reliable and effective method to assess inspiratory muscle strength in healthy, highly motivated subjects.

The P_{Imax} values of the participants in this study are comparably higher than typical values of healthy adult men (Green et al., 2002); albeit comparable to that of other research (Klusiewicz et al., 2008; Mickleborough et al., 2009). Previous research has already demonstrated that rowers have higher respiratory mouth pressures than their aged match counterparts (Shephard, 1998). In combination with the specific inspiratory warm up performed prior to the start of the inspiratory loading may explain why these values are considerably higher as specific inspiratory muscle warm-up has already been shown to increase inspiratory mouth pressure measurements by ~11-17% (Lomax & McConnell, 2009; Volianitis et al., 2001c). Even so, all inspiratory loads were calculated as a percentage of their maximum P_{Imax} values allowing for a systematic comparison across participants.

Also, no measurements of inspiratory muscle function were taken following the loading tasks to determine if IMF occurred. So there was no way of knowing if subjects became fatigued following loading. However, previous studies investigating acute inspiratory resistive loading at these same intensities showed no diaphragmatic fatigue even when using maximum voluntary manoeuvres (i.e. PreEx-P_{Imax} compared to PostEx-P_{Imax}) and electrically evoked bilateral nerve stimulation (Eastwood et al., 1994; Laghi et al., 1998).

Finally, the relatively high pretest values of blood pressure are not representative of the normal population of this age. By comparison, the SBP of participants in this

study (139 ± 16) was much higher than that reported in Witt et al. (2007) (117.5 ± 1.8 mmHg). There may be a few potential reasons for the inflated elevated blood pressure values: 1) a lack of sufficient rest between the inspiratory muscle warm-up and the inspiratory loading session; 2) inaccurate measurements due technical issues with the blood pressure monitor; and 3) elevated resting blood pressure in some of the athletes. Typically the time between inspiratory warm-up and the start of the inspiratory loading session was ~ 5 min, as this time was spent setting up the blood pressure monitor (brachial cuff and radial sensor), metronome and pulmonary function equipment. Following set-up, typically there was a one minute rest following calibration to allow the blood pressure monitor to settle before inspiratory pressure threshold loading testing would begin.

Alternatively, the blood pressure monitor may not have calculated blood pressure correctly. The device used in this study (COLIM CBM-7000) was not originally designed to measure blood pressure in active participants; it was originally intended for resting measurements of clinical patients at the bedside (Scanmed representative, personal communication). Therefore, an erect seated posture and any movements by the participants (even sitting at rest) may have altered the calibration of the device enough to elevate blood pressure values. However, this same instrument was used by McConnell & Lomax (2006) in which they successfully assessed blood pressure during all active protocols, including during contractions of the lower limb.

It may also be possible that some of the subjects had slightly elevated resting blood pressure. Two of the subjects, both South-Asian students studying in the UK, had higher resting blood pressures values (> 140 mmHg) compared to the other athletes.

Both of these athletes were very fit (trained regularly > 4 days per week), had normal resting heart rate, and stated they had no medical concerns and were not taking any prescribed or recreational substances. A systematic review investigating blood pressures in South-Asian males compared to UK males, highlighted that seven studies have demonstrated that South Asian males, particularly those living in the London area, have higher blood pressures compared to their UK counterparts (Agyemang & Bhopal, 2002). Although the overall data is complex and does not show a clear picture of blood pressure and hypertension in the South-Asian community living in the UK, it does highlight a difference in population groups. The reviewers cite these differences may be due to environmental risk factors (i.e. living in immigrant communities). Nonetheless, the two athletes in this study demonstrated higher than normal resting blood pressure values which may have elevated the pre-test values.

6.5 CONCLUSION

The data suggests that there is an inverse relationship between load magnitude and endurance time when performing inspiratory resistive loads. Increases in blood pressure are evident within 60 s when using a pressure threshold load at 60% P_{Imax}. In addition, IMT loads of 60-70% P_{Imax} are required to meet a critical threshold to elicit metaboreflex activation in the inspiratory muscles. The respiratory muscles responded similarly in load magnitude upon RM to that of skeletal muscles at resistive loads >70% P_{Imax}. It is evident that there is much individual variation in the ability to perform IMT at differing training intensities. Future research is needed to determine whether the activation of the metaboreflex during IMT is obligatory to generate a training adaptation and for increasing the threshold for activation after IMT.

SECTION 2

FIELD BASED STUDY

CHAPTER SEVEN

THE INFLUENCE OF INSPIRATORY MUSCLE TRAINING UPON ROWING ERGOMETER PERFORMANCE IN ELITE HEAVYWEIGHT OARSMEN

This study was conducted by request of the British International Rowing Organisation (BIRO) as a grant funded research project. A consultancy report was presented to the coaches of the Amateur Rowing Association upon completion in 2003.

7.1: INTRODUCTION

The study described in Ch.4, demonstrated that inspiratory muscle training (IMT) increased inspiratory muscle strength, attenuated IMF and improved six minute all-out (6MAO) rowing performance in competitive club-level oarsmen. These data confirmed those obtained in a previous study in which well-trained female rowers showed improvements in both the 6MAO effort and a 5 km rowing ergometer time trial (1.9% above control) after 11 wk of IMT (Volianitis et al., 2001c). This research, and additional studies examining the impact of IMT in well-trained athletes, has shown a beneficial impact on sport performance (Caine & McConnell, 1998a, 1998b; Guenette et al., 2006; Lin, Tong, Huang, Nie, Lu & Quach, 2007; Romer et al., 2002a, 2002c). For example, Romer et al. (2002a) have demonstrated improvements in 20 and 40 km cycling time trial performance (3.8% and 4.6% above control, respectively) in well-trained male cyclists after 6 wk of IMT. However, a recently published controlled-trial conducted by Riganas et al. (2008) investigated the effects of threshold loading IMT on 2 km rowing performance in elite male and female rowers. Although they observed an increase in inspiratory muscle strength and a decrease in end-stage IMF, they found no significant improvement in $\dot{V}O_{2max}$ or 2 km rowing time trial performance. Hence, the specific performance benefits, if any, associated with IMT for elite athletes remain questionable.

The present study was conducted at a time (2002/2003) when the evidence supporting the efficacy of IMT, and the level of understanding of underlying mechanisms were at a preliminary stage. The rationale for the study was based upon evidence that sub-elite female rowers showed improvement in their rowing performance after IMT (Volianitis et al., 2001c), but the influence of IMT upon rowing performance in elite

male rowers remained untested. Hence the primary purpose of the study was to assess the potential role of IMT in this unique group of athletes. Because of the nature of the study population, considerable restrictions were imposed upon the experimental design, which was conducted in the field and under the complete control of the British International Rowing Organisation (BIRO).

We hypothesised the following outcomes; 1) an improvement in the maximal strength of the inspiratory muscles with IMT, 2) an improvement in the maximum rate of inspiratory muscle shortening, 3) an attenuation of exercise-induced inspiratory muscle fatigue, and 4) an improvement in rowing ergometer time trial performance.

7.2: METHODOLOGY

The present study was undertaken at the invitation of the BIRO to investigate the ergogenic efficacy of IMT in 'World-Class' heavyweight male rowers. The study design was limited by the availability of the participants to undertake maximal testing, and their ability to produce truly maximal performances under laboratory conditions. Accordingly, it was agreed that all testing would be undertaken in the context of scheduled squad/crew selection time trials, and that an attempt would be made to account for normal training improvements by comparing two groups of participants (see section 7.2.1); only one of which received IMT. These compromises limit the scientific rigor and internal validity of the study design, but have the benefit of making it highly externally valid in the context of the performance tests. The main outcome variable in the study was ergometer time trial performance. Additionally, we gathered supplementary data, such as inspiratory muscle and lung function, to support any performance changes in the time trial data.

7.2.1: PARTICIPANTS

School Ethics Committee approval of the research design and written informed consent was obtained prior to participation in the study. Fourteen heavyweight male competitive rowers were invited to participate in this study; unfortunately two of the athletes (one in each group) were removed from the study after baseline testing due to illness/injury unrelated to this study. The data presented throughout is limited to the 12 participants who completed all performance tests. As 'World-Class' athletes, all rowers had previous experience in maximal rowing ergometer exercise tests as part of their regular training routine.

Ideally, the intervention and control groups in a study of this type would be drawn from a single squad, training under the direction of one coach. However, this was not possible due to the elite nature of the athletes, the limited number of participants accessible at this level as well as the rigorous training and competition restrictions. A compromise was agreed in which two squads training under different coaches were compared. Thus, participants were allocated into a training group (T-group, n = 6) or a control group (C-group, n = 6) based on their current squad assignment. Although the two groups were in separate coaching groups, both groups were part of a squad system overseen by BIRO and under the direction of its head coach. The T-group consisted of 'World-Class' athletes, in which five of the six athletes went on to win a team bronze medal at the World Champions and a total of 3 individual gold medals at the next Olympics. The athletes in the C-group were on the BIRO reserve squad; as such we expected the rowing ergometer performance of the T-group to be faster compared to the C-group.

Since both groups were tested at similar phases of the training year (November to April), it is reasonable to assume that their training would have been formulated to achieve similar physiological adaptations and performance changes. We anticipated that there would be small differences in individualised programmes between the T & C groups; however, both groups performed their regular pre-season training programmes with periodic rowing-specific training camps. Accordingly, we expected cardiorespiratory and resistance-training routines would be similar between the two groups.

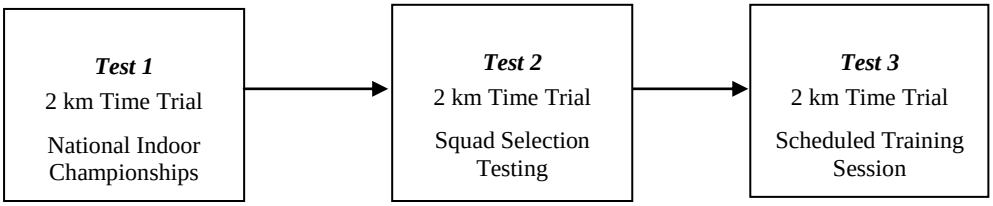
7.2.2: GENERAL DESIGN

Participants were assessed on rowing performance, pulmonary and respiratory muscle function, including maximal inspiratory pressures (P_{Imax}) pre- and post-2 km rowing ergometer time trial (PreEx-P_{Imax} and PostEx-P_{Imax}, respectively). All participants were familiarised with the testing procedures prior to the start of the first testing session. Participants' inspiratory muscles were trained 7 days per week ($d \cdot wk^{-1}$) for 11 wk, with time trial performance and pulmonary function retested at Test 2 and Test 3. At the beginning of the study, baseline rowing performance in the T-group was assessed at a National Indoor Rowing Championships. The C-group performed their baseline 2 km rowing ergometer test at their boathouse as part of a selection trial.

After Test 2, the T-group was instructed to continue their daily IMT, but with an increase in training intensity. Figure 7.1 shows a schematic diagram of the testing sessions and their respective dates and locations; whereas Table 7.1 illustrates the timetable for the testing schedule, including significant events that occurred throughout the study. The overall length of the IMT intervention was 11 wk. Test

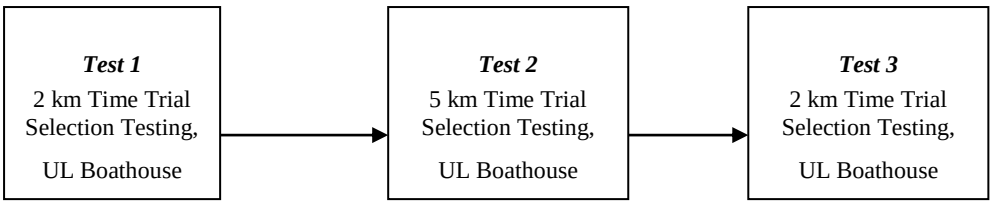
dates were selected by the coaches of the national rowing organisation, in conjunction with the athletes' current selection test process.

T-group Testing Sessions



Test 1-3: P_{Imax} & MFVLs were measured at rest prior to a 2 km rowing ergometer performance. Within 2-3 minpost-exercise participants performed a P_{Imax} manoeuvre.

C-group Testing Sessions



Test 1-3: P_{Imax} & MFVLs were measured at rest prior to a 2 km or 5 km rowing ergometer performance. Within 2-3 minpost-exercise participants performed a P_{Imax} manoeuvre.

Figure 7.1 Schematic diagram of testing sessions.

Note: P_{Imax}, maximal inspiratory pressure; MFVL, maximum flow volume loop; UL, University of London

Table 7.1 Chronological sequence of exercise testing and other significant events.

Date	Event	Group
November 02	Test 1: 2 km time trial	T
	Test 1: 2 km time trial	C
December 02	Test 2: 2 km time trial	T
	Christmas holidays	T & C
January 03	Test 2: 5 km time trial	C
	Altitude Training Camp	T
	San Moritz, Switzerland Illness/Colds –Self reported	T
February 03	Test 3: 2 km time trial	T
	Seville, Training Camp	T
March 03	Head of the River Race	T & C
April 03	Test 3: 2 km time trial	C

Note: T, training group; C, control group.

7.2.3: PROCEDURES

PULMONARY AND INSPIRATORY MUSCLE FUNCTION

Prior to all exercise testing, a series of three maximal flow volume loops (MFVLs) was obtained using a hand-held portable spirometer (MicroLoop, Micro Medical Ltd., Kent, United Kingdom). The following measures were derived: forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), peak inspiratory flow rate (PIF) and peak expiratory flow rate (PEF). A detailed explanation of the MFVL manoeuvres can be found in section 3.2.2.

Maximal inspiratory mouth pressures (P_{Imax}), measured for the evaluation of inspiratory muscle strength, were performed pre- and post-exercise (PreEx-P_{Imax} and

PostEx-P_{Imax}, respectively) using a mouth pressure meter (Micro MPM, Micro Medical Ltd., Kent, United Kingdom). All P_{Imax} measurements were initiated at residual volume (RV). The procedure was repeated until the highest of three P_{Imax} values were reproduced with less than 5% variability or within 5 cm H_2O. The highest reproducible value was recorded and presented in cm H_2O. All inspiratory pressure measurements were made according to the procedures described in section 3.2.2.

INSPIRATORY MUSCLE FATIGUE

Inspiratory muscle fatigue (IMF) was defined as a 'loss in capacity to develop force and/or shorten resulting from muscle fibre activity under load which was reversible by rest' (NHLBI, 1990). Therefore, exercise-induced IMF was calculated as the percentage difference between PreEx-P_{Imax} and PostEx-P_{Imax} measurements for each rowing ergometer time trial. PostEx-P_{Imax} was assessed within 2 to 3 min following the completion of the 2 km rowing ergometer time trial. The exact time of the measurement post-exercise was recorded and repeated on subsequent test trials. Inspiratory muscle fatigue (IMF) was expressed as the percent change (%) from PreEx-P_{Imax}. A detailed description of the measurement of IMF is described in section 3.2.2.

ERGOMETER TIME TRIAL PERFORMANCE

The influence of IMT was assessed by comparison of pre-IMT performance time (Test 1) for the 2 km rowing ergometer time trial, with those obtained following the training intervention period (Test 2 and Test 3). A wind-braked rowing ergometer (Concept II, Nottingham, UK) was used to assess time trial performance. The electronic monitoring device standard on the rowing ergometer was set to the

designated distance prior to the test. Each participant's performance time was recorded as the total time to perform the designated distance. Time was recorded in minutes, seconds, deciseconds (m:s.ds). To ensure that all participants were rowing at the same resistance settings when performing at different locations, the drag factor was set to 138 (damper setting 4) for all participants.

INSPIRATORY MUSCLE TRAINING

The T-group commenced IMT immediately following the National Indoor Rowing Championships (Test 1; see fig. 7.2). Prior to Test 1, an introductory group demonstration on proper usage of the IMT device (POWERbreathe®, Gaiam Ltd., Southam, UK) was provided. The T-group was instructed to perform IMT using maximal inspiratory efforts from residual volume (RV) and was encouraged to perform the breathing effort rapidly and to total lung capacity (TLC). A detailed description of the IMT device and training methods is described and illustrated in Chapter 3.2.3.

The T-group was instructed to perform one set of IMT, twice daily, at an intensity corresponding to 30 repetitions maximum (30 RM). Previous studies have shown this load to be effective in eliciting an adaptive response in trained athletes (Riganas et al., 2008; Romer et al., 2002a, 2002c; Volianitis et al., 2001c). Participants were requested to maintain a load of 30 RM during Phase 1 and 2 by independently increasing the intensity by manually adjusting the training device. Figure 7.2 provides an overview of the testing sessions and phases of IMT throughout the study.

Test 1	IMT Phase 1	Test 2	IMT Phase 2	Test 3
MFVL ↓ PreEx-P_{Imax} ↓ 2 km time trial ↓ PostEx-P_{Imax}	7 d·wk^{-1} 4 wk 1 set of 30 breaths at 30 RM, 2x day Training diary	MFVL ↓ PreEx- P_{Imax} ↓ 2 km/5 km time trial ↓ PostEx- P_{Imax}	7 d·wk^{-1} 7 wk 1 set of 30 breaths at 30 RM, 2x day Training diary	MFVL ↓ PreEx- P_{Imax} ↓ 2 km time trial ↓ PostEx- P_{Imax}

Figure 7.2 Schematic diagram of testing and inspiratory muscle training phases.

Note: MFVL, maximum flow volume loop; PreEx-P_{Imax}, pre-exercise maximal inspiratory mouth pressure; PostEx-P_{Imax}, post-exercise maximal inspiratory mouth pressure.

A daily training log, specifically designed for IMT, was used to monitor adherence to the prescribed training regimen (Appendix A-3). At Test 2, the T-group were informed of a motivational reward in which the top three participants able to improve their time trial performances over the total testing period would keep their inspiratory muscle trainer. The C-group performed no IMT during the entirety of the study.

7.2.4: STATISTICAL ANALYSIS

A detailed explanation of data analysis is presented in the Ch.3.2.3.

7.3: RESULTS

Subject 2 in the T-group was unable to perform the PostEx-P_{Imax} manoeuvre at Test 1 due to his inability to physically recover from the 2 km rowing ergometer time trial. Thus, the mean IMF has been calculated with n = 5 in the T-group for Test 1.

7.3.1: DESCRIPTIVE CHARACTERISTICS

Descriptive characteristics of the participants are presented in Table 7.2. An independent samples t-test revealed no significant differences at Test 1 in resting pulmonary function. However, the T-group had a significantly higher body mass compared to the C-group (6.8 kg; $P = 0.004$).

Table 7.2 Descriptive characteristics of the participants (mean ± SD).

	T-group (n = 6)	**C-group** (n = 6)
Anthropometry		
Age (y)	24 ± 2.1	22 ± 2.8
Stature (cm)	194.8 ± 2.0	191.9 ± 2.7
Body mass (kg)	96.0 ± 2.3*	89.2 ± 3.9

Note: *, significantly different compared to the C-group ($P = 0.004$).

7.3.2: TRAINING ADHERENCE

Detailed diaries were used to monitor IMT volume and intensity. The T-group demonstrated a good adherence to training during the first 4 wk (85 ± 14%). After Test 2 in December, the T-group reported having colds and chest infections and found it difficult to perform IMT during a high altitude training camp, resulting in deterioration in training compliance. Only two training diaries were returned at Test 2,

negating the usefulness of these instruments for data analysis purposes from Test 1 to Test 2.

7.3.3: PULMONARY FUNCTION

RESTING PULMONARY FUNCTION

As shown in Table 7.3, no significant differences were detected at Test 1 or following IMT Phase 1 or 2 for FEV_1, FVC, FEV_1/FVC, PIF or PEF values ($P > 0.05$) in either group.

MAXIMAL INSPIRATORY PRESSURES

A repeated measures ANOVA revealed a difference in P_{Imax} within groups over time ($P = 0.001$) and between groups ($P = 0.021$). A paired sample one-tail t-test, corrected using a Bonferroni adjustment (P set at ≤ 0.025) was performed to identify changes over time. After IMT Phase 1, the T-group improved their PreEx-P_{Imax} on Test 2 by 31.1 ± 23.9% ($P = 0.011$). Even though a majority of the T-group stated that they did not regularly perform IMT during Phase 2, the T-group maintained a PreEx-P_{Imax} at Test 3 that was 30.2 ± 23.7% ($P = 0.022$) higher compared to Test 1. Whereas the C-group's PreEx-P_{Imax} slightly increased over time, it did not change at Test 2 (3.3 ± 13.0%; $P = 0.295$) or Test 3 (9.2 ± 13.8%; $P = 0.085$) as compared to Test 1. A post-hoc one-way ANOVA revealed differences between groups at Test 2 ($P = 0.020$) and at Test 3 ($P = 0.046$). Figure 7.3 highlights the significant changes in PreEx-P_{Imax} over the three testing sessions.

Table 7.3 Summary of pulmonary function data for both groups.

	Test 1	Test 2	Test 3
PreEx-P_{Imax} (cm H_2O)			
T-group (n=6)	138.7 ± 18.2	180.2 ± 30.4*†	178.8 ± 29.7*†
C-group (n=6)	136.0 ± 16.2	140.0 ± 18.1	147.8 ± 17.3
PostEx-P_{Imax} (cm H_2O)			
T-group	95.0 ± 22.3a‡	168.7 ± 30.5*	176.0 ± 24.0*
C-group	109.3 ± 25.6‡	108.2 ± 28.4‡	120.0 ± 30.2
IMF (%)			
T-group	28.5 ± 15.8%a	6.1 ± 10.6%†	1.1 ± 6.8%*
C-group	20.3 ± 13.7%	23.6 ± 13.5%	18.5 ± 19.2%
FEV_1 (L)			
T-group	5.21 ± 0.59	5.04 ± 0.52	5.01 ± 0.81
C-group	5.07 ± 0.45	5.18 ± 0.49	4.99 ± 0.61
FVC (L)			
T-group	6.26 ± 0.33	6.13 ± 0.57	5.98 ± 0.55
C-group	6.33 ± 0.31	6.65 ± 0.40	6.27 ± 0.73
FEV_1 /FVC (%)			
T-group	83.2 ± 8.0	82.2 ± 5.9	83.9 ± 12.1
C-group	88.0 ± 5.5	77.8 ± 4.0	79.6 ± 4.4
PIF ($L \cdot min^{-1}$)			
T-group	635.4 ± 109.4	644.4 ± 103.6	647.3 ± 85.2
C-group	518.7 ± 103.9	539.2 ± 124.1	534.9 ± 146.9
PEF ($L \cdot min^{-1}$)			
T-group	728.9 ± 87.8	659.4 ± 104.8	689.1 ± 58.2
C-group	617.8 ± 103.6	608.0 ± 56.3	655.3 ± 80.0

Note: PreEx-P_{Imax}, maximal inspiratory pressure before ergometer time trial. PostEx-P_{Imax}, maximal inspiratory pressure after ergometer time trial. IMF, inspiratory muscle fatigue; FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; PIF, peak inspiratory flow; and PEF, peak expiratory flow. a, n = 5. *, significantly different to Test 1 ($p \leq 0.05$). †, significantly different compared to C-group. ‡, significantly different compared to PreEx-P_{Imax} ($p \leq 0.05$).

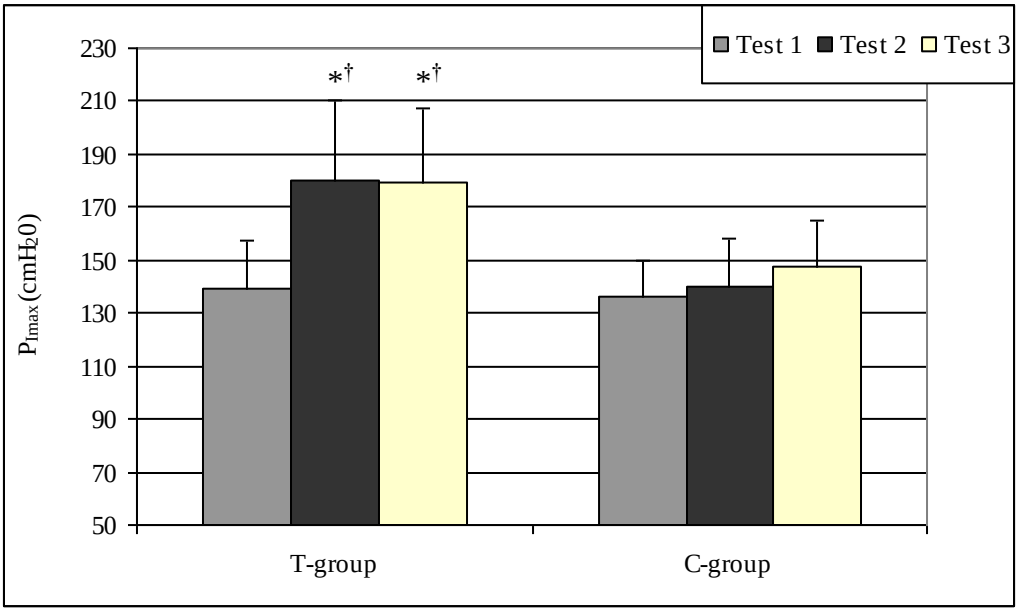


Figure 7.3 Comparison of P_{Imax} between groups over the three testing sessions.

Note: *, significantly different to Test 1 ($p \leq 0.05$). †, significantly different compared to the C-group ($p \leq 0.05$).

INSPIRATORY MUSCLE FATIGUE

Repeated measures ANOVA revealed a difference in the severity of IMF over time, both within subjects ($P = 0.010$) and between groups over time ($P = 0.010$). As shown in Table 7.3, both the T-group (28.5 ± 15.8%; $P = 0.009$) and the C-group (20.3 ± 13.7%; $P = 0.005$) experienced a decrease in P_{Imax} following their 2 km rowing performance at Test 1. However, the post-hoc paired sample t-test (P set at < 0.025) showed that following IMT Phase 1 and 2, the PreEx-P_{Imax} compared to PostEx-P_{Imax} values in the T-group decreased at both Test 2 (6.1 ± 10.6%; $P = 0.165$) and Test 3 (1.1 ± 6.8%; $P = 0.313$). The athletes in the C-group continued to experience IMF following their time trials at Test 2 (23.6 ± 13.5%; $P = 0.007$) and Test 3 (18.5 ± 19.2%; $P = 0.034$). As highlighted in Figure 7.4, IMF was reduced over time in the T-group at both Test 2 (~22%; $P = 0.029$) and Test 3 (~27%; $P = 0.010$) compared to

Test 1. A one-way ANOVA revealed differences in IMF between groups at Test 2 (P = 0.031).

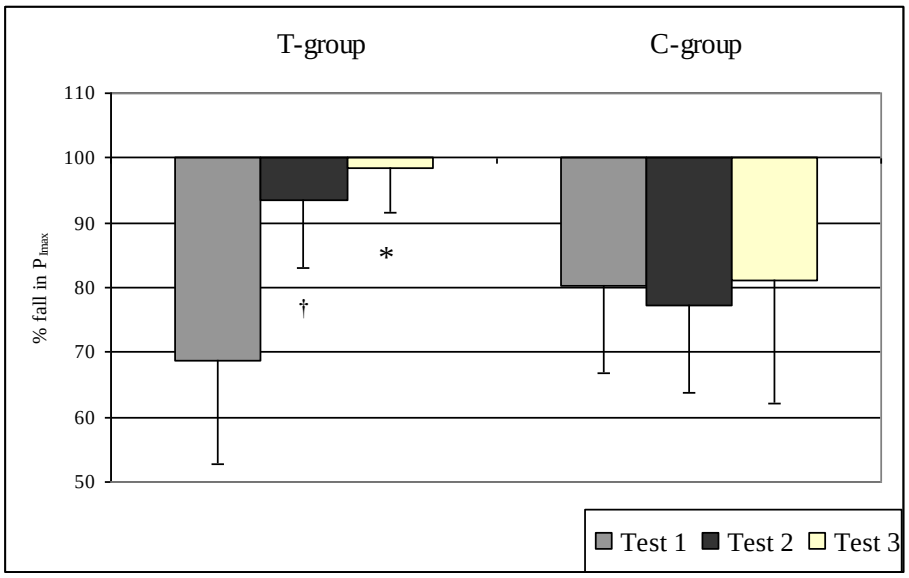


Figure 7.4 Relative changes in %IMF for both the T-group and C-group.

Note: $P_{Imax,}$ maximal inspiratory pressure; IMF (%), the percent of fatigue in the inspiratory muscles following 2 km ergometer row. *, significantly different to Test 1 ($p \leq 0.05$). †, significantly different compared to C-group ($p \leq 0.05$).

7.3.4: ROWING ERGOMETER PERFORMANCE TIME

Subject 4 rowed a 6:30 on his 2 km rowing ergometer time trial at the National Indoor Championships; this score was unrepresentative of his usual 2 km ergometer performance time, which was due to fainting during the final meters of the race. In an attempt to maintain statistical power, we corrected for this by calculating the percentage of improvement in 2 km rowing ergometer time trials for the whole of the T-group and then calculated a time based on a percentage of his post-IMT time trial performance (6:04.35). Table 7.4 provides a summary of 2 km ergometer performance times for individual participants. The limits of agreement for within subject changes is

summarised in Table 7.5. As shown below, the T-group's Test 1 value was an average 14.3 s (3.8%) faster than the C-group (P = 0.005).

Table 7.4 Summary of rowing ergometer performance times for both groups.

	Test 1 (n=12)	**Test 2** (n=12)	**Test 3** (n=12)
T-group			
1	06:00.0	05:57.0	06:02.5
2	05:58.7	05:54.2	06:00.9
3	06:00.1	05:56.0	06:01.4
4	06:04.3^	06:01.7	06:09.7
5	05:58.1	05:57.3	05:59.5
6	05:58.8	05:59.2	05:56.3
Mean	**06:00.0**	**05:57.6***	**06:01.7**
SD	**00:02.1**	**00:02.6**	**00:04.5**
C-group			
1	06:05.0	06:02.0^	06:07.5
2	06:12.1	06:03.0^	06:08.4
3	06:06.0	06:05.0^	06:03.9
4	06:13.1	06:11.0^	06:11.2
5	06:18.7	06:13.0^	06:18.5
6	06:31.0	06:33.0^	06:25.9
Mean	**06:14.3**	**06:11.2**	**06:12.6**
SD	**00:08.8**	**00:11.6**	**00:08.2**

Note: Rowing ergometer times are recorded in minutes: second. decisecond (m:s.ds). *, significantly different compared to the Test 1 ($p \leq 0.05$). Note: C-group Test 2 times were predicted from a 5 km time trial and T-group Test 3 times were derived from a test performed immediately following on-water training. ^ = estimated (see text for details).

Table 7.5 Absolute and ratio limits of agreement for rowing ergometer performance.

Measurements

Variable	N	Mean (SD)1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Absolute	SE	95% CI	Absolute	SE	95% CI for Lower L of A	95% CI for Upper L of A
T-group											
Test 1 - Test 2	6	360.0 (2.3)	357.6 (2.6)	2.4 (1.9)*	2.43	0.78	0.72 to 4.14	3.73	1.35	-4.25 to 1.66	3.20 to 9.12
Test 2 - Test 3	6	357.6 (2.6)	361.7 (4.5)	-4.2 (4.0)*	-4.15	1.61	-7.70 to -0.60	7.75	2.8	-18.05 to -5.75	-2.55 to 9.75
Test 1 - Test 3	6	360.0 (2.3)	361.7 (4.5)	-1.7 (2.6)	-1.72	1.04	-4.01 to 0.57	4.99	1.8	-10.68 to -2.75	-0.69 to7.24
C-group											
Test 1 - Test 2	6	374.3 (9.6)	371.2 (11.6)	3.2 (3.9)	3.15	1.57	-0.31 to 6.61	7.55	2.72	-10.39 to 1.59	4.71 to 16.69
Test 2 - Test 3	6	371.2 (11.6)	372.6 (8.2)	-1.4 (5.1)	-1.4	2.08	-5.97 to 3.17	9.98	3.6	-19.30 to -3.46	0.66 to 16.50
Test 1 - Test 3	6	374.3 (9.6)	372.6 (8.2)	1.8 (2.7)	1.75	1.09	-0.65 to 4.15	5.23	1.89	-7.64 to 0.67	2.83 to 11.14

Log transformed measurements

Variable	N	Mean (SD)1	Mean (SD) 2	Difference (SD)	Bias			Random Error			
					Ratio	SE	95% CI	Ratio	SE	95% CI for Lower L of A	95% CI for Upper L of A
T-group											
Test 1 - Test 2	6	5.9 (0.0)	5.8 (0.0)	0.0 (0.0)	1.007	0.002	0.002 to 0.012	1.01	0.003	0.990 to 1.003	1.011 to 1.024
Test 2 - Test 3	6	5.8 (0.0)	5.9 (0.0)	-0.0 (0.0)	0.989	0.004	-0.021 to -0.002	1.022	0.006	0.954 to 0.981	0.997 to 1.023
Test 1 - Test 3	6	5.9 (0.0)	5.9 (0.0)	0.0 (0.0)	0.995	0.003	-0.011 to 0.002	1.014	0.004	0.973 to 0.990	1.001 to 1.018
C-group											
Test 1 - Test 2	6	5.9 (0.0)	5.9 (0.0)	0.0 (0.0)	1.009	0.004	-0.001 to 0.018	1.02	0.006	0.976 to 1.001	1.017 to 1.042
Test 2 - Test 3	6	5.9 (0.0)	5.9 (0.0)	0.0 (0.0)	0.996	0.005	-0.016 to 0.008	1.027	0.007	0.954 to 0.987	1.006 to 1.039
Test 1 - Test 3	6	5.9 (0.0)	5.9 (0.0)	0.0 (0.0)	1.005	0.003	-0.002 to 0.011	1.014	0.004	0.982 to 0.999	1.010 to 1.027

*, significant difference ($p \leq 0.05$)

The T-group was 2.43 s faster at Test 2 compared to Test 1 (P = 0.026). Test 3 was not part of T-group's scheduled testing sessions and was performed as an 'in-training' test, immediately after a component of their regular water training; there was no improvement in 2 km performance on this time trial compared to Test 1 or Test 2 (P = 0.159 and P = 0.05, respectively).

Test 2 for the C-group was derived from a 5 km rowing ergometer time trial test. In order to compare 5 km to 2 km performance time, the 5 km rowing ergometer performance was used to estimate a time for the 2 km ergometer time trial. The calculation was based on the methods of BIRO that follows the assumption that a 2 km performance of 6 min is equivalent to 16 min for a 5 km performance, therefore for every 2.5 s ± difference from 16:00 for 5 km, is equal to 1 s ± difference from 6:00 for 2 km. The values from Test 2 are provided as an estimate of the 2 km ergometer time for the C-group.

As shown in Figure 7.5, the C-group showed a similar improvement to the T-group in 2 km performance time from Test 1 to Test 2 (3.1 s faster; P = 0.102) and Test 3 (P = 0.530).

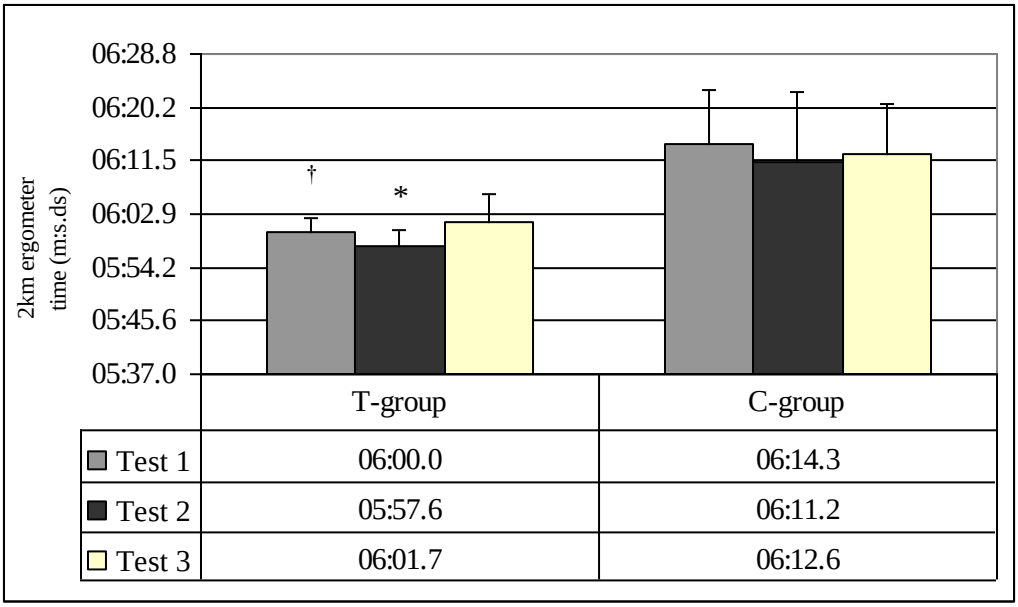


Figure 7.5 Comparison of 2 km rowing ergometer performance times between groups. Note: 2 km ergometer time is in m:s.ds. *, significantly different compared to Test 1 ($p \leq 0.05$). †, significantly different compared to C-group ($p \leq 0.05$).

Bivariate correlations were performed to determine if there was a relationship between the changes over time in IMF, PreEx-P_{Imax} and 2 km ergometer performance time; no significant relationships were detected in any parameter for either group.

7.4: DISCUSSION

7.4.1: MAIN FINDINGS

The primary aim of this study was to determine whether 2 km rowing ergometer time trial performance was influenced by IMT in 'World-Class' rowers. Related to this, we hypothesised an improvement in the maximal strength and shortening velocity of the inspiratory muscles, as well as attenuation of exercise-induced IMF following IMT. The results indicate significant IMF following 2 km rowing performance in both

groups. However after 4 and 11 wk IMT, the T-group demonstrated improvements in inspiratory muscle strength and a decrease in IMF. Following the initial 4 wk IMT, the T-group showed an improvement in 2 km rowing performance. However, inconsistencies in the conditions under which data were collected render it impossible to determine whether changes in rowing performance were due to IMT, or the athletes' other training.

At the time of this research (2002-2003), there was limited research supporting the strength benefits of IMT in elite athletes (e.g., Olympic standard), particularly as Coast et al. (1990) suggested that whole body training sufficiently trained the respiratory muscles. This study was one of the first to assess IMF in elite open-class oarsmen and to measure the impact of IMT on improvements in inspiratory muscle strength.

7.4.2: CHANGES IN PULMONARY AND INSPIRATORY MUSCLE FUNCTION

PULMONARY FUNCTION MEASURES

No change was found in pulmonary function. These findings are consistent with previous results in similar IMT studies (Edwards & Cooke, 2004; Romer et al., 2002a), except PIF, which has been shown to increase following IMT (Romer et al., 2002c; Romer & McConnell, 2003; see Ch. 4). The absence of an effect of IMT upon PIF in the present study may due to differences in breathing pattern during training between studies. If inspiratory flow rate during training is not sufficiently high, training-specificity may dictate that adaptations are biased towards improvements in strength and not shortening velocity (Romer & McConnell, 2003). Since it was not

possible to supervise the training of the athletes in the present study, it is possible that this arose.

INSPIRATORY MUSCLE STRENGTH

The improvement in P_{Imax} (~ 31%) in the T-group compares favourably with similar studies that have observed increases from 8-45% in inspiratory muscle strength following resistive loading (see table 2.1). More recent studies investigating IMT in elite oarsmen observed similar improvements of ~28% (Riganas et al., 2008) and 20-34% in P_{Imax} (Klusiewicz et al., 2008) following 6 - 11 wk of threshold loading IMT. In Klusiewicz et al.'s (2008) study with Polish elite rowers, the athletes performed a similar IMT protocol to the present study and tested inspiratory muscle strength following 6 and 11 wk of IMT. The only difference to the present study is that they used a mean load of 62.3% P_{Imax} during the first 6 wk and 77.5% P_{Imax} for the remaining weeks. They demonstrated a 20 ± 10% increase in P_{Imax} after 6 wk, with a further increase up to 34 ± 19% at 11 wk. The authors stated they initially attempted this study with a 50% P_{Imax} load and were unable to achieve significant changes in P_{Imax}; hence, they decided to increase the load and training time, which resulted in significant improvements. This is consistent with the findings from the previous study (see Ch. 6) investigating load magnitude, which suggested that inspiratory training loads ranging from 60-70% P_{Imax} correspond to the 30 RM and are sufficient to initiate a metaboreflex response.

The lack of further increase in P_{Imax} following IMT Phase 2 in our athletes was most likely due to their inconsistent training. Notwithstanding this, previous studies looking at the effects of IMT have shown a plateau effect usually occurs after 4-6 wk of IMT

training (Romer & McConnell, 2003; Volianitis et al., 2001c), which is consistent with the findings of this study.

One research group has specifically studied the benefits of IMT using threshold loading on highly trained male and female rowers. Their recent abstracts using highly trained rowers investigated the effect of IMT on end-stage arterial oxygen saturation (SaO_2) following 6 wk of IMT (Riganas et al., 2007; Vrabas et al., 2007). These studies showed a marked improvement in both P_{Imax} (39.3% in males and 53.7% in females) and breathing endurance (shown as an increase in maximal voluntary ventilation) in this population. Thus providing support that whole body endurance training alone is not sufficient to increase or sustain inspiratory muscle strength, even in highly trained athletes whose inspiratory muscles are engaged in locomotion. It seems that even highly trained rowers can achieve an increase in P_{Imax} following IMT of 20-39% in males (Klusiewicz et al., 2008; Riganas et al., 2007, 2008; personal studies), with female rowers achieving even greater improvements of up to 45-54% (Volianitis et al., 2001c; Vrabas et al., 2007). The magnitude of the increase in inspiratory muscle strength will depend upon the training intensity and duration of the training programme.

INSPIRATORY MUSCLE FATIGUE

This was the first study to demonstrate 'World-Class' male rowers of this calibre (i.e. Olympic standard) were susceptible to IMF as demonstrated by a ~29% decrease in post-exercise P_{Imax}. Inspiratory muscle fatigue (IMF) was defined as the percentage decrease in P_{Imax} occurring between two to three min of exercise cessation. The magnitude of IMF (>23%) observed in the present study is consistent with the results

of previous studies following short-term high intensity exercise (Caine & McConnell, 1998c; Lomax & McConnell, 2003; Mador et al., 1993; Volianitis, McConnell, Koutedakis & Jones, 1999, Volianitis et al., 2001c).

As discussed previously, IMF induces a variety of negative effects upon exercise performance, including a decrease in exercise tolerance, increased perception of breathing effort, and possibly a sympathetically mediated vasoconstriction of the exercising limb vasculature (Romer & Polkey, 2008). As reviewed in Ch. 2, a decrease in limb blood flow has been shown to cause skeletal muscle fatigue ultimately decreasing exercise tolerance (Harms et al., 2000; McConnell & Lomax, 2006); however, the existence of IMF does not implicitly indicate a metaboreflex activation (McConnell & Lomax, 2006). For rowers, RMF may have additional deleterious effects by compromising the postural stability of the thorax (Hodges et al., 2001). Rowers use the same abdominal and intercostal muscles for ventilation, postural stability and to assist in the transmission of propulsive forces (Steinacker et al., 1993). Consequently, IMF may potentially contribute to the breakdown of rowing technique through inefficient locomotor-respiratory coupling, loss of postural stability and injury. Thus, the maintenance of inspiratory muscle force generating capacity elicited by IMT in the present study may impact positively in a number of subtle ways.

A small number of IMT studies that have observed significant improvements in time trial performance, have also noted post-IMT amelioration, or even complete ablation, of IMF (Romer et al., 2002a, 2002c; Volianitis et al., 2001c; see Ch.4). For example, Volianitis et al. (2001c) demonstrated improvements in rowing performance in both

the 6 min all-out (6MAO) effort and the 5 km time trial after 4 wk of IMT in well-trained oarswomen in which IMF was decreased by 8.2% in the training group. Similarly, Romer et al. (2002b) studied the effects of IMT on time trial performance in trained male cyclists. After 6 wk of IMT, the training group showed an ablation of IMF after the 20 and 40 km cycling time trials as well as an increase in performance around 4%. The findings from the previous study (Ch. 4) investigating the effectiveness of IMT and EMT in competitive club-level oarsmen also, showed a significant decrease in IMF following 4 wk of IMT accompanied by a 2.7% increase in mean power during a 6MAO effort. On the face of it, these data might be interpreted as indicating that attenuating IMF is the mechanism underlying improvements in performance, and that this occurs because of the link between fatiguing inspiratory muscle work and inspiratory metaboreflex activation (Romer & Polkey, 2008). However, McConnell & Lomax (2006) found that after IMT, it was possible for IMF to be present (following inspiratory loading), but for metaboreflex activation to be absent.

Consistent with the observation of McConnell & Lomax (2006) are the findings of studies that have observed a decrease in exercise-induced IMF following RMT, but lacked any improvement in exercise performance (Riganas et al., 2008; Verges, Lenherr, Haner, Schulz & Spengler, 2007a). For example, the training group in Riganas et al.'s (2008) study substantially improved P_{Imax} and reduced post-exercise IMF, but exhibited no change in 5MAO or 2 km rowing ergometer performance. Similarly, a recent study by Verges et al. (2007a) showed a significant reduction in exercise-induced RMF following 4-5 wk of voluntary isocapnic hyperpnoea (VIH; decrease of ~17%), but no improvement in cycling time to the T_{lim}. Thus, the role of

IMF (as assessed by a fall in P_{Imax} voluntary post-exercise or loading), in exercise limitation remains unclear.

7.4.3: TIME TRIAL PERFORMANCE

The results of this study are consistent with the findings of our previous study in competitive club-level oarsmen (see Ch. 4) and by Volianitis et al. (2001c), as the athletes in the T-group showed a significant improvement in their 2 km ergometer time trial performance following 11 wk of IMT (2.4 s; 0.7% improvement). We expected both the T and C-group to show some improvement in their rowing performance as all the athletes were simultaneously undertaking whole body, rowing specific training throughout the study.

The difference in baseline rowing ergometer performance between groups was expected as the athletes in the T-group were crew members of the elite squad; whereas the C-group athletes were BIRO reserve athletes. As such, it was recognised that the T-group was the 'fastest' at baseline and therefore closest to their physiological potential. At Test 2, the C-group demonstrated a larger improvement in performance time (3.1 s faster) compared to the T-group (2.4 s faster). However, the Test 2 value for the C-group was estimated based on a 5 km performance trial, not an actual 2 km performance; therefore the changes in performance are not directly comparable. Whilst the improvement in 2 km time trial performance in the T-group (0.7%) was small, the BIRO coaches stated that a mean 2.4 s improvement was greater than they would normally expect during this phase of the training programme. The lack of further improvement in performance times at Test 3 most likely reflects the fact that this test was not part of the crew selection process, but rather was

undertaken on the same day, and shortly after, a normal on water training session. This test may therefore have been affected negatively by both the athletes' motivational state and the effects of prior fatigue due to training.

At the elite level, improvements in performance are not only harder to achieve, but also of greater impact, since the margins of success are also correspondingly smaller. At the Olympic Games in Athens, for which the athletes were preparing, the average difference between gold and silver in the A-finals was 0.41%, whilst the largest was 0.89%. The smallest was the Men's 4-, which was won by GB by a margin of just 0.04%. Thus, if only part of the 0.7% improvement observed in the T-group was attributable to IMT the effect is worthwhile at the elite level.

Several studies have observed a positive impact upon sport performance following IMT (see table 2.1); however, the ergogenic benefits associated with IMT on rowing performance remain equivocal (Feutz et al., 2006; Riganas et al., 2007, 2008; Volianitis et al., 2001c; Vrabas & Riganas, 2005; Vrabas et al., 2007). Volianitis et al. (2001c) were the first to investigate the effect of IMT, studying well-trained female rowers, and observing a 1.9% increase in distance covered in the 6MAO test and a decreased time to completion in the 5 km time trial by 2.2% compared to the control group. In contrast, a more recent study by Riganas et al. (2008) investigated the effect of IMT in the national Greek rowing squad (n = 19; 7 female); whilst they observed a significant increase following 6 wk IMT in both PreEx-P_{Imax} and PostEx-P_{Imax} immediately following a $\dot{V}O_{2max}$ test, they observed no change in 2 km rowing ergometer performance. It is possible that, similar to the present study, the 'World-Class' rowers in Riganas et al. (2008) study were already much closer to the limits of

their maximum potential, compared to the well-trained rowers studied by Volianitis et al. (2001c); hence it is more difficult to elicit and detect small, but functionally significant changes in performance. Furthermore, since the respiratory muscles form part of the system that stabilises and stiffens the trunk during rowing, it is possible that in highly trained and experienced rowers, the respiratory muscles are already well conditioned by the rowing stroke. Hence, any benefits that may be derived from IMT may have already been expressed. The persistence of IMF post-exercise could be interpreted as arguing against this, but a recent study suggests that loss of maximal inspiratory muscle force generating capacity (in response to specific loading) can be present without their having been activation of the inspiratory muscle metaboreflex post-IMT (McConnell & Lomax, 2006). A change in the threshold for activation of the metaboreflex occurs post-IMT (McConnell & Lomax, 2006; Witt et al., 2007) and has been suggested to be an important mechanism underlying the ergogenic effect of IMT. The observations of McConnell & Lomax (2006) suggest that loss of maximal force generating capacity does not imply metaboreflex activation, and cannot therefore be used as a means of predicting whether IMT is likely to improve performance.

As mentioned previously, the abstracts presented by Vrabas et al. (2007) and Riganas et al. (2007) in highly trained rowers also observed an increase in inspiratory muscle strength and distance rowed in the 5MAO for both the males (28 m, 1.9% improvement; Riganas et al., 2007) and females (34 m, 2.6% improvement; Vrabas et al., 2007) compared to control groups. However, only in the females did IMT elicit an increase in end-exercise SaO_2 levels (Vrabas et al., 2007), demonstrating a possible difference in mechanisms between genders. Similar results were observed in a

university collegiate rowing team (n = 13; 7 females) to determine the effects of 8 wk of IMT performed at 75% P_{Imax} (Feutz et al., 2006). The athletes showed significant increases in P_{Imax} (~46%), rowing distance (63 m; 4.1% improvement) and mean power output (27.2 W; 10.7% improvement) during a 6MAO rowing time trial; however no control group was used for comparison. Collectively, these studies support the use of specific threshold loading IMT as an ergogenic aid in well-trained rowers, but the data remain less clear for 'World-Class' rowers.

7.4.4: METHODOLOGICAL CONSIDERATIONS

The test selection process and allocation of athletes to the T-group was dictated by the BIRO national head coach and thus out of our control. Hence, the lack of comparable control data at Test 2 between groups and the post-training 2 km time trial at Test 3 for the T-group severely undermines the comparability (within and between groups) and the application of the findings. In addition, we cannot exclude the possibility that the lack of randomisation in the study design affected the outcome. As seen in Table 7.5, the T-group's 2 km ergometer performance time at Test 1 was an average 14.3 s faster than the C-group. The decision to have only the one squad perform IMT was agreed with the coaches, and was the only option open to us.

However, the overall pattern of performance change in the T-group was one of an improvement in the 2 km ergometer performance, inspiratory muscle strength and a reduction in IMF during the course of the study, compared with the C-group. In contrast to the T-group, the C-group's performance in all areas tested over the three testing periods remained relatively unchanged after Test 1. Admittedly, the data is inconclusive from a performance perspective, but there may be subtle benefits derived

from the improvement in strength and IMF resistance. This would tend to support the notion that IMT has a positive effect on 2 km ergometer performance and the IMF observed following high-intensity rowing exercise.

Alternatively, it could be argued that the T-group was initially rowing at faster speeds than the C-group (~4%), and this difference in ability may have actually limited the potential of improvement for the T-group. Thus, far from biasing the result in favour of IMT, the lack of randomisation may have limited the potential for improvement in the T-group. This being the case, it is possible that a greater influence of IMT may have been observed if the T-group had consisted of a mixture of subjects from the two squads.

Another important limitation to the study was a lack of control for differences in resistance training, ergometer and water based training regimens between the two groups. The absence of strict management in training differences between the two groups was due to restrictions placed on the study design by BIRO. Equally, both groups were in the pre-season phase of their season and would have adhered to broadly similar water and land based training programmes that were set by the head coach. As both groups improved rowing performance from Test 1 to Test 2 this would suggest improvements in their 2 km time trial performance were due to their whole body training.

7.5: CONCLUSION

These data support existing evidence that IMT increases inspiratory muscle strength (~27-29%) and attenuates exercise-induced IMF even in 'World-Class' oarsmen. The present study therefore provides some supportive evidence for the role of threshold

loading IMT as a beneficial adjunct to training in elite oarsmen.

The traditional training protocol of 1 set of 30 RM twice daily seems to provide significant benefits to inspiratory muscle strength, whilst decreasing IMF associated with high intensity exercise. However, a more rigorous study design is necessitated to determine the potential ergogenic benefit IMT has on 'World-Class' performers. Additional research investigating more sophisticated IMT protocols and appropriate training progression would prove beneficial to determine the extent IMT may have on sports performance.

CHAPTER EIGHT

DISCUSSION

8.1: MAIN FINDINGS

MAIN FINDINGS

The primary aims of this dissertation were to: 1) evaluate the application of respiratory muscle training (RMT) in competitive rowers and 2) explore methods to optimise the application of RMT to rowing. The project was divided into two sections: three empirical laboratory based studies and one applied field study working specifically with oarsmen. The key aims of the individual studies were to determine:

1) the functional effectiveness of inspiratory muscle training (IMT), expiratory muscle training (EMT) and subsequent combined IMT/EMT in competitive club-level oarsmen.
2) the effect of specific rowing postures upon respiratory pressure flow and volume generating capacity.
3) evaluation of different inspiratory loading protocols for IMT in rowers.
4) the functional effectiveness of IMT in 'World-Class' oarsmen.

The main findings of the study were:

1) IMT improved rowing performance (an increase of 2.7% in mean power and 0.92% improvement in distance rowed during a six minute all out {6MAO} rowing effort) in competitive club level oarsmen, but EMT and subsequent combined IMT/EMT did not. These data suggest that rowers need only train their inspiratory muscles, in addition to their whole body and rowing specific training, in order to improve their rowing performance.
2) There are no statistically significant differences in respiratory muscle strength (RMS) when comparing various simulated rowing postures. Ventilatory muscles appear to work effectively in all rowing-related postures, but they

seem to be optimal in the seated or more upright postures. No further investigation was warranted for posture-specific IMT.

3) IMT loads of 60-70% of P_{Imax} were equivalent to the widely used 30 repetition maximum (RM), which is higher than reported for non-rowers (Caine & McConnell, 1998a). Further, there is evidence of an inspiratory metaboreflex response to acute inspiratory loading at 60% P_{Imax}, as evidenced by a time-dependent rise in heart rate (70.1 ± 13.2 to 98.0 ± 22.8 bpm; $p < 0.05$) and mean arterial blood pressure (92.4 ± 8.5 to 99.7 ± 10.1 mmHg; $p < 0.05$).
4) IMT increases inspiratory muscle strength (~27-29%) and attenuates inspiratory muscle fatigue (IMF) induced by simulated racing in 'World-Class' oarsmen. However, practical limitations imposed upon the study design rendered the data as inconclusive with respect to the ergogenic effect of IMT upon rowing performance in elite male oarsmen.

8.2: APPLICATION OF RMT TO COMPETITIVE ROWING

8.2.1: INFLUENCE OF IMT UPON RESPIRATORY MUSCLE FUNCTION AND ROWING PERFORMANCE

One of the primary objectives of this dissertation was to determine the functional effectiveness of RMT for male rowers at various competitive levels. Overall, our findings suggest that IMT was effective in significantly increasing inspiratory muscle strength (20-29%) in competitive club-level and elite oarsmen. These findings are consistent with previous observations suggesting that whole-body exercise and rowing training performed simultaneously is insufficient to maximally strength train the inspiratory muscles (Klusiewicz et al., 2008; Riganas et al., 2008; Volianitis et al.,

2001c). Following 4 wk IMT, we also observed an attenuation of IMF (a fall of ~8-28%) following 2 km rowing ergometer time trial performance in both competitive club-level and 'World-Class' oarsmen. Thus, suggesting IMT is an effective means of increasing inspiratory muscle strength and reducing exercise-induced IMF in well-trained competitive oarsmen.

In club-level rowers, an increase in P_{Imax} was associated with improved 2 km rowing time-trial performance, as demonstrated by a 2.7% improvement in mean power output and a 0.92% increase in mean distance rowed. Although our heavyweight elite oarsmen demonstrated a smaller improvement in 2 km time trial performance following 4 wk IMT (2.4 s faster; 0.7%), it was impossible to determine if this improvement was due solely to IMT or to other training factors. The potential for adaptation in response to IMT in the 'World-Class' oarsmen may have been more limited than in the control group, as the former were closer to their potential for physiological adaptation (as demonstrated by faster 2 km time trial times). Thus, compared to the other competitive rowers assessed, the relatively small change in rowing performance in elite performers may have been due to the close proximity to their maximum potential. Although these findings suggest, at best, a minimal change in elite rowing performance after IMT, this needs to be judged in the context of elite competition, particularly elite rowing, in which medals are won by hundredths of a second. It is important to note that applied research such as these studies are reflective of a very competitive and elite sporting nature in which statistical significance is of less importance than the demonstration of a functionally meaningful change in performance (Hopkins, 2004).

It would have been interesting to assess whether the elite oarsmen had noticed a difference in their perception of breathlessness at submaximal intensities, as this may have implications for training quality. In retrospect, the study design would have been enhanced if the athletes had provided ratings of their breathing effort during the time trials, but due to the chaotic nature of 'boat crew selection testing' and the actual races when the testing took place with the elite oarsmen this would have been virtually impossible.

Collectively, the data from this dissertation support the hypothesis that IMT increases inspiratory muscle strength, attenuates exercise-induced IMF and improves 2 km time trial rowing ergometer performance in competitive club-level oarsmen; however the data remain equivocal as to the potential performance benefits for the 'World-Class' rower.

8.2.2: INFLUENCE OF EMT UPON RESPIRATORY MUSCLE FUNCTION AND ROWING PERFORMANCE

Previous research has demonstrated that the expiratory muscles are also subject to exercise-induced fatigue (Fuller et al., 1996; Derchak et al., 2002, Taylor et al., 2006; Taylor & Romer, 2008), initiate a metaboreflex response during exercise (Derchak et al., 2002), and that pre-fatigue impairs exercise performance (Taylor & Romer, 2008). Research has already demonstrated that P_{Emax} increases following EMT in COPD patients and individuals suffering from conditions that specifically weaken the respiratory muscles (Mota et al., 2007; Weiner et al., 2003). However, unlike IMT, the influence of EMT upon exercise tolerance in healthy individuals remains uncertain, particularly its influence upon sport performance.

Although not statistically significant, an 18% increase in expiratory muscle strength and a 5% reduction in post-exercise expiratory muscle fatigue (EMF) were observed following 4 wk EMT. The addition of a subsequent 6 wk period of combined IMT/EMT in the EMT group significantly increased expiratory muscle strength to 31% compared to baseline; but with no further changes to EMF. These findings suggest that EMT increases expiratory muscle strength and attenuates exercise-induced EMF, however; these adaptations occurred less consistently and appeared to require a longer period of training than those observed after IMT. It has been suggested that EMF may affect exercise performance negatively by increasing the sensation of dyspnoea and by sympathetically mediated vasoconstriction in limb blood flow (Taylor et al., 2006). This being the case, it is possible that EMT might improve exercise performance; however this was not evident in our subjects.

Based upon previous IMT research, an improvement > 15% P_{Imax} appears to represent a threshold level of adaption that is required in order to enhance performance (see table 2.1). It is therefore conceivable that a similar, much higher, threshold may exist for the expiratory muscles. There is limited research investigating the effects of EMT upon exercise performance in healthy individuals (see table 2.2); indeed, research identifying appropriate training protocols for EMT in healthy subjects is non-existent. The study in Ch.4 employed similar inspiratory and expiratory training loads (30 RM); however this load may not have been sufficient to elicit the same level of muscular adaptations in both sets of muscles. It is also possible that training for competitive rowing imparts unique adaptations to the expiratory muscles that render this population unresponsive to EMT. Future research into EMT is required to

determine the training loads necessary to elicit a training and/or physiological response, if any, during exercise in healthy sports participants.

In summary, IMT and EMT increased inspiratory and expiratory muscle strength (26% and 18% respectively), however only those subjects performing IMT improved 2 km rowing ergometer time trial performance. Further significant increases in P_{Emax} and a decrease in EMF following combined IMT/EMT in the EMT group also failed to elicit any change in rowing performance. The EMT group performing the combined IMT/EMT showed only a slight increase in P_{Imax} (~13%), which also failed to improve rowing performance. Perhaps more important than the absolute increase in P_{Imax}, is the attenuation of IMF, as both the club-level and 'World-Class' oarsmen performing IMT showed a significant decrease in IMF following 2 km rowing ergometer time trial, and an increase in rowing performance. The attenuation of IMF has been shown to delay the activation of the inspiratory metaboreflex, which has been cited as a potential mechanism for the improved exercise tolerance associated with IMT (McConnell & Lomax, 2006; Witt et al., 2007). These results suggest that rowers need only train the inspiratory muscles to improve 2 km rowing time trial performance and that a minimum percentage of improvement in P_{Imax} may be required to elicit rowing performance enhancement.

8.*2.3: INFLUENCE OF RMT UPON PHYSIOLOGICAL VARIABLES DURING ROWING*

It has been suggested that the two most likely candidate mechanisms for the improved exercise tolerance associated with IMT are a decrease in respiratory effort (Suzuki et al., 1995; Romer et al., 2002a, 2002b; Volianitis et al., 2001c), and a modulation of

the inspiratory muscle metaboreflex (McConnell & Lomax, 2006; Witt et al., 2007). In Ch.4, heart rate (f_c), oxygen uptake ($\dot{V}O_2$), earlobe blood lactate concentration ($[La^-]_B$) and ratings of respiratory effort (RRE) were assessed during an incremental rowing 'step' test and six minute all out effort (6MAO). After 4 wk IMT and a subsequent 6 wk period of combined IMT/EMT, there was a significant reduction in $[La^-]_B$ and RRE following the 6MAO effort in the IMT group. Although non-significant, there was a trend towards a decrease in $[La^-]_B$, peak end-stage f_c and RRE during the incremental step test following IMT. However, despite a significant improvement in P_{Emax} and a reduction in EMF in the EMT-group following the combined IMT/EMT program, no improvements were evident in any of the physiological or performance variables tested. The influence of IMT upon peak end-stage f_c may reflect the absence or attenuation of metaboreflex mediated sympathetic drive.

8.3: OPTIMISING IMT FOR COMPETITIVE ROWERS

The second aim of this dissertation was to characterise, and to explore means of enhancing the IMT regimen for rowers. Two different studies were employed to investigate, 1) the effect of simulated rowing postures upon respiratory pressure flow and volume, and 2) differing inspiratory pressure loads for IMT protocols.

8.3.1: EFFECT OF POSTURE ON RESPIRATORY MUSCLE FUNCTION

As lung volume has been shown to influence respiratory muscle force generation (Black & Hyatt, 1969), it was expected that changes in lung volume observed with adjustments in body position and posture would also influence respiratory muscle strength (RMS), and vice versa. The study in Ch.5 assessed the influence of relevant

postures related to rowing upon P_{Imax}, P_{Emax} and flow volume loops. Although no changes were observed in RMS in trunk flexion or recumbent postures, the data suggested that respiratory pressures and lung function showed a tendency to decrease in recumbent postures and that P_{Imax} and P_{Emax} were highest in the most upright postures (90º and 110º). These findings are consistent with similar studies investigating the effect of sitting, standing, supine and lateral recumbent postures on RMS and lung volumes (Kera & Maruyama, 2001a, 2001b; Ogiwara & Miyachi, 2002). If a significant decrease had been observed in any of the postures tested, a further study would have been warranted to explore the possibility of training the respiratory muscles in these postures. Since no significant influences were found, this line of research was not pursued further. However, as the simulated catch position was performed with legs straight, future investigations may prove useful to determine if there are any significant changes to pressure and flow generating capacity when the abdominal wall is adjacent to the thighs, as occurs during rowing.

8.3.2: LOAD MAGNITUDE

In Ch.6, our principal aim was to characterise the breathing pattern and cardiovascular responses to inspiratory pressure threshold loads ranging from 50-90% P_{Imax}. Similar to previous research, task failure occurred when breathing at high inspiratory loads as demonstrated by a progressive decrease in T_{lim} as loading intensity increased (% P_{Imax}) (Gorman et al., 1999; McKenzie et al., 1977; Rohrbach et al., 2003; Sheel et al., 2002). The higher resistive loads ($\geq$ 70% P_{Imax}) sufficiently overloaded the inspiratory muscles leading to a significant decrease in $V_{T,}$ T_{lim} and repetitions performed compared to the 60% P_{Imax} load.

The estimated amount of work performed during acute inspiratory loading was highest at the 60% P_{Imax} load and lowest at the 80% P_{Imax} load. Although this may seem counterintuitive, the heavier inspiratory loads ($\geq$ 70% P_{Imax}) actually elicited less work due to the effect of the respiratory pressure volume relationship upon V_T. Thus indicating a possible threshold for the influence of load upon V_T arising at loads > 60% P_{Imax}.

The results demonstrated there was a similar relationship to the number of maximum repetitions achieved at each inspiratory training load (% P_{Imax}) compared to the recommended workloads at similar training intensities for whole body resistance training programmes. Moreover, loads of ~65% P_{Imax} were equivalent to a 30 RM in male rowers, which is slightly higher than the 50-60% P_{Imax} load reported previously in healthy, young untrained individuals (Caine & McConnell, 1998a). This most likely reflects the higher baseline training status of the rowers' inspiratory muscles.

Although previous research using flow resistive loading has examined the inspiratory muscle metaboreflex (Witt et al., 2007), to our knowledge this was the first study to examine the metaboreflex using pressure threshold loading. This is relevant in that, despite a differing duty cycle between the two loading methods, the pressure threshold loads required to elicit the metaboreflex are the same. We observed that inspiratory pressure loads of 60-70% were required to activate the inspiratory muscle metaboreflex, as demonstrated by a time dependent increase in f_c and MAP. These findings are consistent with limb muscle research, which proposes that loads must be of sufficient duration and intensity to amply fatigue the muscles, in order to initiate

the exercise pressor response (Augustyniak et al., 2001; Hunter, Duchateau & Enoka, 2004; O'Leary, Augustyniak, Ansorge & Collins, 1999).

8.3.3: IMPLEMENTING IMT INTO WHOLE BODY TRAINING PROGRAMS

ANECDOTAL OBSERVATIONS

Although no formal interview or questionnaire was used to obtain participant feedback about IMT, informal discussions regarding individual perceptions of IMT were conducted with all subjects. Almost all participants commented on the unexpectedly high degree of effort they had experienced when IMT was initiated; many did not appreciate that IMT would be similar to limb muscle resistance training, and would require similar levels of effort.

The feedback from the 'World-Class' athletes regarding IMT were generally very positive. However, the subjects had differing opinions on IMT and how to incorporate IMT into their full time training schedule. All athletes stated that IMT was very helpful as a respiratory warm-up prior to races and ergometer time trials, but they felt that it was difficult to get into the habit of undertaking IMT twice daily. Participants provided the following suggestions on how they thought IMT could be incorporated into their regular training routine:

1) as a respiratory warm-up prior to daily rowing outings, possibly 1 set of 10 RM to be included into their stretching routine.
2) as a respiratory warm-up in preparation for an ergometer time-trial or race.
3) IMT might be most useful early in the season when they still experience 'lung burn' during the 2 km rows.
4) IMT might be incorporated as part of their weight training programme.

TRAINING RECOMMENDATIONS

Based on the participants' training logs, their informal feedback, and the experience of conducting IMT trials, it was apparent that athletes found long-term (> 4 wk) adherence to IMT challenging, and eventually, boring. Accordingly, a structured approach to IMT, where it is incorporated into a whole-body resistance training program may prove beneficial and improve training adherence, as well as functional outcomes.

There is currently, an incomplete understanding with respect to the mechanisms underpinning the ergogenic influence of IMT. This makes it difficult to make meaningful training recommendations. It is possible that the ergogenic benefits received after IMT are an 'all or nothing' phenomenon. If this were the case, maximising the potential benefit of IMT would be a simple process of optimising IMT to reduce breathing effort and/or increase the threshold for activation of the metaboreflex. Future research is required to assess whether the effect is 'all or none', and to focus on optimising the IMT regimen to elicit and maintain the changes found to be responsible for the ergogenic effect. However, based on the body of knowledge thus far, it appears that most rowers would benefit from a simple IMT programme of 4-6 wk, twice daily, at a minimum intensity of 30 RM. After which, they can be transferred to a maintenance programme of 2-3 days per week of 2 sets at 30 RM (Romer & McConnell, 2003). Figure 8.1 provides a suggested program for implementing IMT into a year long whole body program. The pre-season prescription is designed to increase baseline inspiratory muscle strength. Whereas the in-season prescription focuses on maintaining strength gains by performing 2 sets of IMT at least 2 days of the week and as a warm-up before training sessions and races. The

intent of the post-season IMT prescription is to stabilise performance gains and when needed as a respiratory warm-up.

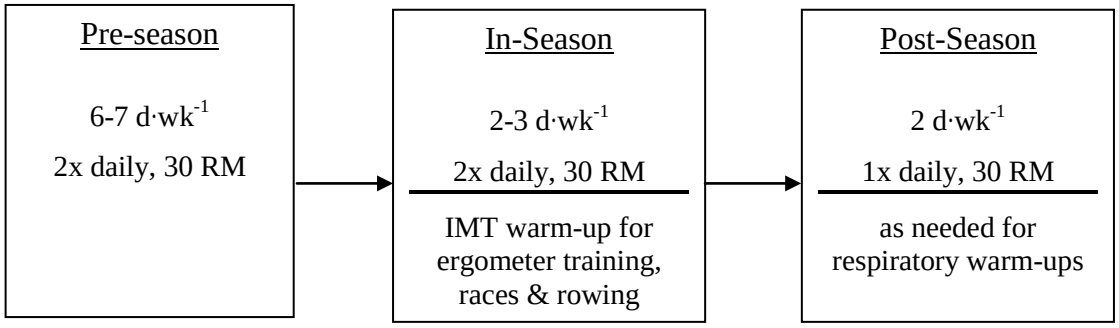


Figure 8.1 Implementation of IMT into a whole-body training program.

An increase in inspiratory muscle strength may alleviate some of the challenges associated with increased inspiratory muscle work during exercise at high altitudes (Dempsey, Amann, Romer & Miller, 2008). Previous research has demonstrated that 4 wk IMT significantly reduces $\dot{V}_E$ and increases arterial O_2 saturation during hypoxic exercise (Downey et al., 2007) thus suggesting athletes may benefit from an intensive 4-6 wk of IMT prior to altitude training. Further investigations to assess any influence of undertaking IMT at altitude may also be warranted. Moreover, IMT can be used by athletes as an effective tool for:

1) warm-up prior to racing, trials or training sessions.
2) part of the preseason whole body training programme to develop a foundation.
3) can be targeted for athletes with specific respiratory conditions including expiratory flow limitation, asthma and/or exercise induced asthma.
4) maintenance of inspiratory muscle function during rehabilitation from injury.

8:4: CONCLUSION

8.4.1: CONCLUSIONS

In conclusion, the findings from this dissertation suggests that pressure threshold IMT increases inspiratory muscle strength, attenuates IMF, and is an effective ergogenic aid for competitive (sub-elite) oarsmen. There appear to be no independent or additional benefits to EMT or combined IMT/EMT above that of IMT alone. The absence of impairment of respiratory mechanics due to rowing movements suggests that it is unlikely that posture-specific IMT would enhance breathing mechanics during rowing, but respiratory muscles do appear to function most effectively in upright postures. Finally, the moderate intensity pressure threshold loading protocol (~60-70% P_{Imax}) implemented in these studies (and previous research) activates the inspiratory muscle metaboreflex, and results in higher levels of inspiratory muscle work than 'heavier' loading.

8.4.2: FUTURE DIRECTIONS

Future research should continue to investigate the underlying mechanisms that lead to the improved exercise and sport performance associated with IMT. Suggested areas for future applied research:

1) Identify whether the ergogenic benefits received following IMT are an 'all or nothing' phenomenon. If so, identify the optimal IMT regimen to elicit and maintain these ergogenic effects.
2) Differentiating the effects of strength vs. endurance IMT protocols on respiratory muscle function and exercise/sport performance.
3) Investigate whether EMT enhances exercise tolerance in other types of sports. If so, identify the appropriate training load required to elicit a substantial

improvement in expiratory muscle strength and/or to significantly reduce EMF.

4) Investigate whether metaboreflex activation during IMT is an obligatory factor for increasing metaboreflex activation threshold after IMT.
5) Attempt to differentiate the respective contributions of reductions in effort perception and metaboreflex activation to the ergogenic effect of IMT.

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A-1

SAMPLE INFORMED CONSENT FORM

Department of Sport Sciences **Consent Form**

I _____consent to take part in_____ _____.

The full details of the tests have been explained to me by _____ _____. I confirm that I have understood what participation will involve and confirm that I have been made aware of the potential benefits and risks of participation.

I understand that I may withdraw from the study at any time and that I am not under any obligation to give reasons for my withdrawal.

I confirm that I have not experienced any of the following:

Chest pain, extreme shortness of breath, high blood pressure, dizzy spells, loss of consciousness

I confirm that I have never been advised to abstain from exercise by a medical practitioner and that I know of no reason why participation in these tests might present a risk to my safety.

I understand that all concerned will treat any information about myself that I have given, or that is obtained during the course of the tests, as confidential.

Signature **…….**

Date **……..……**

Supervisor Dr. Alison McConnell, Brunel University, Department of Sport Sciences, Tel. 01895-274000 ext 5798

Participant declaration of consent

I _____, being over eighteen years of age consent to being a participant in the research project entitled *'The efficacy of strength vs. endurance IMT on rowing performance'.*

I have been given a copy of a 'Participant information sheet' that I fully understand, describing the procedures to be followed and the consequences and risks involved in my participation as a participant.

I understand that the information provided to me about the study is confidential. I also understand that I am bound by this requirement for confidentiality.

I have read the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this activity, realising that I may withdraw from the study without prejudice at any time.

I agree that research data gathered from the study may be published provided my name is not used.

Name of participant _____

Signature of participant _____Date _____

Name of witness _____

Signature of witness _____ Date _____

Certifying that the terms of the form have been verbally explained to the participant, that the participant appears to understand the terms prior to signing the form.

Signature of researcher _____ Date _____

A-2

SAMPLE HEALTH QUESTIONNAIRE

Brunel University

Department of Sport Sciences

General Health Questionnaire

Name: .....
Address: .....
.....
.....
Phone: .....

Name of the responsible investigator for the study:

.....

Please answer the following questions. If you have any doubts or difficulty with the questions, please ask the investigator for guidance. These questions are to determine whether the proposed exercise is appropriate for you. Your answers will be kept strictly confidential.

1.	You are.....	Male	Female
2.	What is your exact date of birth? Day..... Month.....Year..19..... So your age is..... Years		
3.	When did you last see your doctor? In the: Last week..... Last month..... Last six months..... Year..... More than a year.....		
4.	Are you currently taking any medication?	YES	NO
5.	Has your doctor ever advised you not to take vigorous exercise?	YES	NO
6.	Has your doctor ever said you have “heart trouble”?	YES	NO
7.	Has your doctor ever said you have high blood pressure?	YES	NO
8.	Have you ever taken medication for blood pressure or your heart?	YES	NO
9.	Do you feel pain in your chest when you undertake physical activity?	YES	NO

10.	In the last month have you had pains in your chest when not doing any physical activity?	YES	NO
11.	Has your doctor (or anyone else) said that you have a raised blood cholesterol?	YES	NO
12.	Have you had a cold or feverish illness in the last month?	YES	NO
13.	Do you ever lose balance because of dizziness, or do you ever lose consciousness?	YES	NO
14.	a) Do you suffer from back pain b) if so, does it ever prevent you from exercising?	YES YES	NO NO
15.	Do you suffer from asthma?	YES	NO
16.	Do you have any joint or bone problems that may be made worse by exercise?	YES	NO
17.	Has your doctor ever said you have diabetes?	YES	NO
18.	Have you ever had viral hepatitis?	YES	NO
19.	If you are female, to your knowledge, are you pregnant?	YES	NO
20.	Do you know of any reason, not mentioned above, why you should not exercise?	YES	NO
21.	Are you accustomed to vigorous exercise (an hour or so a week)?	YES	NO

I have completed the questionnaire to the best of my knowledge and any questions I had have been answered to my full satisfaction.

Signed: .....

Date: .....

A-3

INSPIRATORY MUSCLE TRAINING DIARY

POWERbreathe Training Diary

NAME

NOTES

WEEK NUMBER

DAY 1	DAY 2	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7
EXPECTED	*EXPECTED*	*EXPECTED*	*EXPECTED*	*EXPECTED*	*EXPECTED*	*EXPECTED*
30 Breaths morning and evening	30 Breaths morning and evening	30 Breaths morning and evening	30 Breaths morning and evening	30 Breaths morning and evening	30 Breaths morning and evening	30 Breaths morning and evening
ACTUAL	*ACTUAL*	*ACTUAL*	*ACTUAL*	*ACTUAL*	*ACTUAL*	*ACTUAL*
am	am	am	am	am	am	am
pm	pm	pm	pm	pm	pm	pm

Please use the following coded responses to record actual training for the day:

A - Trained as expected
B - Less than expected (please indicate number of breaths)
C - Did not train (forgot)
D - Did not train (too busy)
E - Did not train (too difficult)
F - Did not train (lack of motivation)
G - Did not train (other reason, please specify)
H - Increased training load

If you need to contact me (Lisa Miller) call:

01895 274000 x5819 (Work)

Don't hesitate to call me if you have any difficulties with the training

A-4

CATEGORY RATIO (CR10) SCALE

Borg's CR10 Scale Instructions

Basic instruction: 10, "Extremely strong – Max P", is the main anchor. It is the strongest perception (P) you have ever experienced. It may be possible, however, to experience or to imagine something even stronger. Therefore, "Absolute maximum" is placed somewhat further down the scale without a fixed number and marked with a dot "•". If you perceive an intensity stronger than 10, you may use a higher number.

Start with a *verbal expression* and then choose a *number*. If your perception is "Very weak", say 1; if "Moderate", say 3; and so on. You are welcome to use half values (such as 1.5, or 3.5 or decimals, for example, 0.3, 0.8, or 2.3). It is very important that you answer what *you* perceive and not what you believe you ought to answer. Be as honest as possible and try not to overestimate or underestimate the intensities.

Scaling perceived exertion: We want you to rate your perceived (P) exertion, that is, how heavy and strenuous the exercise feels to you. This depends mainly on the strain and fatigue in your muscles and on your feeling of breathlessness or aches in the chest. But you must only attend to your subjective feelings and not to the physiological cues or what the actual physical load is.

1 is "very light" like walking slowly at your own pace for several minutes.

3 is not especially hard; it feels fine, and it is no problem to continue.

5 you are tired, but you don't have any great difficulties.

7 you can still go on but have to push yourself very much. You are very tired.

10 This is as hard as most people have ever experienced before in their lives.

- This is "Absolute maximum", for example, 11 or 12 or higher.

0	Nothing at all	“No P”
0.3		
0.5	Extremely weak	Just noticeable
1	Very weak	
1.5		
2	Weak	Light
2.5		
3	Moderate	
4		
5	Strong	Heavy
6		
7	Very strong	
8		
9		
10	**Extremely strong “Max P”**	
11		
⇓		
•	Absolute maximum	Highest possible

Borg CR10 scale

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ETHICS APPROVAL LETTER

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DUPLICATE LETTER

12th April 2010

Dear Lisa

RE49-07: A Comparison of Two Pressure Threshold Inspiratory Muscle Training Upon Rowing Performance

I am writing to confirm the Research Ethics Committee of the School of Sport and Education received your application connected to the above mentioned research study. Your application has been independently reviewed to ensure it complies with the University/School Research Ethics requirements and guidelines.

The Chair, acting under delegated authority, is satisfied with the decision reached by the independent reviewers and is pleased to confirm there is no objection on ethical grounds to the proposed study.

Any changes to the protocol contained within your application and any unforeseen ethical issues which arise during the conduct of your study must be notified to the Research Ethics Committee for further consideration.

On behalf of the Research Ethics Committee for the School of Sport and Education, I wish you every success with your study.

Yours sincerely

Dr Simon Bradford
Chair of Research Ethics Committee
School Of Sport and Education

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COPIES OF PUBLICATIONS ARISING FROM THIS DISSERTATION