EFFECT OF ABDOMINAL BINDING ON CARDIORESPIRATORY FUNCTION IN PARALYMPIC ATHLETES WITH CERVICAL SPINAL CORD INJURY

A thesis submitted for the degree of Doctor of Philosophy

By

Christopher Roy West

Centre for Sports Medicine and Human Performance, School of Sport and Education,

Brunel University

May 2011

ABSTRACT

Spinal cord injury (SCI) causes a lesion-dependent impairment in cardiorespiratory function that may limit exercise capacity. The aims of this thesis were to describe cardiorespiratory function in highly-trained athletes with low-cervical SCI, and to investigate whether abdominal binding enhances cardiorespiratory function at rest and during exercise in this population. Using body plethysmography, bilateral phrenic nerve stimulation and transthoracic ultrasound, it was demonstrated that Paralympic athletes with cervical SCI exhibit a restrictive pulmonary defect, impaired diaphragm and expiratory muscle function, and low left ventricular mass and ejection fraction compared to able-bodied controls. Using the same methods, it was shown that abdominal binding improves resting cardiorespiratory function by reducing operating lung volumes, and increasing vital capacity, twitch transdiaphragmatic pressure, expiratory muscle strength and cardiac output. A further finding was a positive relationship between binder tightness and cardiorespiratory function. During a field-based assessment of fitness, abdominal binding reduced the time taken to complete an acceleration/deceleration test and increased the distance covered during a repeated maximal 4-min push test. During laboratory-based incremental wheelchair propulsion, abdominal binding altered breathing mechanics by reducing operating lung volumes and attenuating the rise in the pressure-time index of the diaphragm. Furthermore, abdominal binding increased peak oxygen uptake and reduced peak blood lactate concentration, despite no change in peak work rate. Peak oxygen uptake in the laboratory was related to the distance covered during the maximal 4-min push, suggesting that the improvement in field-based performance with binding was due to an improvement in aerobic capacity. In conclusion, this thesis demonstrates that abdominal binding significantly enhances cardiorespiratory function at rest, improves exercise performance in the field, and improves operating lung volumes, breathing mechanics and peak oxygen uptake during incremental treadmill exercise. Thus, abdominal binding provides a simple, easy-to-use tool that can be used to enhance cardiorespiratory function at rest and during exercise in highly-trained athletes with cervical SCI.

ACKNOWLEDGEMENTS

It gives me great pleasure to thank the many people who have made this thesis possible.

First, I would like to thank my two supervisors, Dr. Lee Romer and Prof. Ian Campbell. It is difficult to explain how grateful I am for the opportunity you afforded me three years ago. Lee, I cannot express how many times I thought I could see the light at the end of the tunnel, before realising it was just you with a torch, insisting I need to show the data in an ever-so-slightly different way! But on a more serious note, I thank you for your belief, encouragement and many invaluable conversations throughout my PhD. It has been a truly incredible learning experience and I have thoroughly enjoyed every minute of it. Ian, I am thankful for your support, enthusiasm and willingness to read over my work. I always enjoyed our meetings and found your 'bigger picture' look at my research very insightful and useful.

I would like to acknowledge all of my colleagues. In particular, I thank Bryan Taylor for his expert tuition in placing balloon catheters; Prof. Rob Shave and Eric Stöhr for their guidance with echocardiography; Stuart, Noel, Charlie, Danielle and Nicola for discussion and friendship; and Julie, Gary and Nalin for their help and support whenever it was needed. Special thanks also go to Vicky Tolfrey's team at Loughborough University for allowing me to use their laboratory and equipment, and UK Sport for part-funding this PhD.

I also express my thanks to the Great Britain wheelchair rugby squad, who swallowed balloons and allowed me to prod, poke, scan and stim them numerous times. Without you this thesis would not have been possible and I wish you every success in London 2012.

I also thank my friends and family. Mum, you have always believed in me and supported me in my decisions and for that I thank you. Finally, but by no means least, I give special thanks to Christine Voss. Christine, not only does your heart appear in this thesis, but your unconditional love and support has been invaluable over the last few years and I cannot express how thankful I am for everything you have done for me.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

A	Left ventricular inflow velocity during late diastole
A´	Myocardial tissue velocity during late diastole
a-$\bar{v}O_2$	Arterio-venous oxygen difference
AB	Able-bodied
ACE	Arm-crank ergometry
AD	Autonomic dysreflexia
ASIA	American Spinal Injuries Association
ATS	American Thoracic Society
BAMPS	Bilateral anterior magnetic phrenic nerve stimulation
C	Cervical
C_{dyn}	Dynamic lung compliance
CI	Confidence interval
CMS	Cervical magnetic stimulation
CN	Cranial nerve
CV	Co-efficient of variation
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
E	Left ventricular inflow velocity during early diastole
E´	Myocardial tissue velocity during early diastole
EDV	End-diastolic volume
EELV	End-expiratory lung volume
EF	Ejection fraction
EIAH	Exercise-induced arterial hypoxaemia
EILV	End-inspiratory lung volume
EMG	Electromyogram
ERS	European Respiratory Society
ERV	Expiratory reserve volume
ESV	End-systolic volume
FEV_1	Forced expiratory volume in 1 s
f_R	Respiratory frequency
FRC	Functional residual capacity
HR	Heart rate
IC	Inspiratory capacity
IFR	Inspiratory flow reserve
IRV	Inspiratory reserve volume
IVSd	Inter-ventricular septal thickness in diastole
IVSs	Inter-ventricular septal thickness in systole
IWRF	International wheelchair rugby federation
$[La^-]_B$	Blood lactate concentration
L	Lumbar
LVIDd	Left ventricular internal diameter in diastole
LVIDs	Left ventricular internal diameter in systole
LVM	Left ventricular mass
LVPWd	Left ventricular posterior wall thickness in diastole
LVPWs	Left ventricular posterior wall thickness in systole
MAP	Mean arterial pressure
MMEF	Mean mid-expiratory flow
MVV	Maximum voluntary ventilation
NEP	Negative expiratory pressure
OH	Orthostatic hypotension

P_{di}	Transdiaphragmatic pressure
$P_{E,max}$	Maximal expiratory pressure
PEF	Peak expiratory flow
PET_{CO2}	End-tidal partial pressure of carbon dioxide
P_{ga}	Gastric pressure
$P_{I,max}$	Maximal inspiratory pressure
PIF	Peak inspiratory flow
PCO_2	Partial pressure of carbon dioxide
PO_2	Partial pressure of oxygen
P_{oe}	Oesophageal pressure
$(P_{oe}/P_{I,max})/(V_T/VC)$	Rate of neuromechanical uncoupling
PTI_{di}	Diaphragm pressure-time index
$\dot{Q}$	Cardiac output
*R*aw	Airway resistance
RER	Respiratory exchange ratio
RPE	Ratings of perceived exertion
RV	Residual volume
S	Sacral
S´	Myocardial tissue velocity during systole
SBP	Systolic blood pressure
SCI	Spinal cord injury
SD	Standard deviation
SE	Standard error
SpO_2	Arterial oxygen saturation estimated from pulse-oximetry
s*R*aw	Specific airway resistance
SV	Stroke volume
T	Thoracic
T_I/T_{TOT}	Fractional inspiratory time
TLC	Total lung capacity
T_{TOT}	Total breath time
tw	Twitch
$\dot{V}CO_2$	Carbon dioxide output
$\dot{V}_E$	Minute ventilation
$\dot{V}_{ECAP}$	Ventilatory capacity
$\dot{V}O_2$	Oxygen uptake
VC	Vital capacity
V_T	Tidal volume
V_T/T_I	Tidal inspiratory flow
WCE	Wheelchair ergometry
WERG	Wheelchair ergometer

CHAPTER ONE:

GENERAL INTRODUCTION

Spinal cord injury (SCI) is a devastating and life changing event that results in substantial motor and sensory paralysis. There is also a lesion dependent impairment in cardiorespiratory and autonomic function, whereby function is reduced as the level of SCI moves cranially. In low cervical SCI (C5-C7), the muscles of the diaphragm, scalenes and sternocleidomastoids remain fully or partially innervated. However, the loss of innervation to other muscles of inspiration (inspiratory intercostals) and the major muscles of expiration (abdominals and expiratory intercostals) causes respiratory muscle weakness (Hopman *et al.*, 1997; Mateus *et al.*, 2007), a decrease in lung and chest wall compliance, and an increase in abdominal compliance (Estenne and De Troyer, 1986; Goldman *et al.*, 1988). Respiratory muscle weakness results in a restrictive pulmonary defect (Anke *et al.*, 1993; Schilero *et al.*, 2009), and forces the inspiratory muscles to work at an increased fraction of their pressure- and flow-generating capability (Gross *et al.*, 1980; Sinderby *et al.*, 1996a). Furthermore, intercostal muscle dennervation causes a paradoxical inward motion of the upper anterior rib cage during inspiration (Estenne and De Troyer, 1986; Urmey *et al.*, 1986), resulting in chest wall distortion (Fugl-Meyer and Grimby, 1971a; Mortola and Sant'Ambrogio, 1978). During exercise, these impairments manifest in a tachypnic breathing pattern and dynamic hyperinflation (Taylor *et al.*, 2010), which may predispose the respiratory muscles to fatigue, increase dyspnoea and impair exercise performance.

Cervical and high thoracic SCI also results in reduced cardiovascular function. Injury above T6 causes sympathetic dennervation of the large arteries in the splanchnic bed (Claydon *et al.*, 2006), which have been demonstrated to have a critical role in blood pressure control (Rowell *et al.*, 1972). Hence, these individuals suffer extreme alterations in blood pressure, ranging from episodes of very low blood pressure during orthostatic stress, to life-threatening bouts of high blood pressure known as autonomic dysreflexia (Krassioukov, 2004; Krassioukov *et al.*, 2006). There is also a loss of venous muscle pump activity below the lesion and a reduction in circulating blood volume (Houtman *et al.*, 2000), which compromises venous return, stroke volume and cardiac output, thereby leading to cardiac atrophy (de Groot *et al.*, 2006; Kessler *et al.*, 1986). In cervical SCI, cardiovascular function is further impaired due to a loss of sympathetic innervation to the myocardium and splanchnic bed resulting in bradycardia and splanchnic venous pooling (Krassioukov, 2009; Teasell *et al.*, 2000). During exercise, dennervation of the sympathetic chain

ganglia impairs the redistribution of blood from the lower limbs and splanchnic region (Thijssen *et al.*, 2009). Thus, for people with SCI above T6, the combination of a small active muscle mass and an inability of the cardiorespiratory system to compensate for the stress of exercise results in a low exercise capacity compared to the able-bodied population (Van Loan *et al.*, 1987).

Based on the aforementioned processes, it is reasonable to assume that improving cardiorespiratory function will improve exercise capacity in people with cervical or high thoracic SCI. Abdominal binding represents an acute intervention that has been demonstrated to improve resting pulmonary function (Estenne *et al.*, 1998; Hart *et al.*, 2005; McCool *et al.*, 1986), and attenuate orthostatic intolerance in patients with SCI (Huang *et al.*, 1983). However, the effect of abdominal binding on respiratory muscle function, cardiac function and exercise capacity in SCI are less well understood. For an intervention to increase exercise capacity, the intervention must increase the performance of a system that is normally limited during exercise. In the low- to moderately-trained cervical SCI population, the small untrained active muscle mass is unlikely to impose a sufficient stress on the cardiorespiratory system for it to be limited during maximal exercise (Hopman *et al.*, 1998a). In highly-trained Paralympic athletes with cervical SCI, however, the large volume of training undertaken over many years is likely to improve the strength and endurance of the arm muscles, such that a greater stress will be placed on the cardiorespiratory systems during exercise. Thus, highly-trained Paralympic athletes with cervical SCI may be limited during exercise by their cardiorespiratory system and therefore represent the ideal sample on which to test whether abdominal binding can improve cardiorespiratory function during exercise. Accordingly, the primary aim of this thesis was to determine whether abdominal binding improves cardiorespiratory function at rest and during exercise in highly-trained athletes with cervical SCI. General aims and hypotheses are provided at the end of the literature review.

The next chapter provides a review of the literature pertaining to cardiorespiratory function at rest, during exercise and in response to a number of different acute interventions in individuals with cervical SCI. In the third chapter, the general methodology employed in this thesis is described. The experimental studies are presented in chapters four, five, six and seven. Finally, the overall findings of this thesis are discussed in chapter 8.

CHAPTER TWO:

LITERATURE REVIEW

2-1 Overview

2-1.1 Spinal cord anatomy

The spine is a flexuous column of 33 vertebrae excluding those which form the skull. The spine is divided into the cervical (C), thoracic (T), lumbar (L), sacral (S) and coccygeal regions of which there are 7, 12, 5, 5 and 4 bones, respectively (Gray, 2008). The spinal cord begins at the caudal end of the medulla oblongata and leaves the cranial vault by extending through the foramen magnum into the vertebral canal. The spinal cord terminates at the intervertebral disc between the first and second lumbar vertebrae. Thus, the spinal cord does not extend the entire length of the vertebral canal. Accordingly, there is a progressive lengthening of the spinal nerve roots within the vertebral canal (Goshgarian, 2010). Each spinal nerve root exits the spinal cord through the intervertebral foramina above the vertebra with the corresponding number (see also Fig 2-1). There are two enlargements of the spinal cord due to the nerve plexus' that innervate the upper and lower limbs. The cervical enlargement, between C5 and T1, is due to the brachial plexus which innervates the upper limbs. The lumbar enlargement, between L1 and S2, is due to the lumbar plexus (L1-L4) and the sacral plexus (L4-S2) that innervate the lower limbs. The spinal cord serves as the major conduit for motor, sensory and autonomic neural information.

A spinal cord injury (SCI) affects the conduction of neural signals across the site of the injury. Injury to the cervical spinal cord, also commonly termed tetraplegia or quadriplegia, results in the loss of motor and / or sensory function in the trunk, pelvic area and legs, due to damage of the neural elements within the spinal canal (Maynard *et al.*, 1997) (see also Fig 2-1). Tetraplegia arises from the Greek roots of *tetra,* meaning four, and *plegia,* meaning paralysis. Tetraplegia is also used interchangeably with quadriplegia, which arises from the Latin *Quadri*, meaning four; however, the inappropriateness of combining Latin and Greek roots has precluded the use of quadriplegia in medical terminology. Accordingly, the terms tetraplegia or cervical SCI will be used in this thesis. Injury to the thoracic spinal cord, otherwise known as paraplegia, can be distinguished from tetraplegia as it does not cause impairment of the upper limbs. There is,

however, a lesion-dependent impairment of the trunk, pelvic area and legs. Both tetraplegia and paraplegia can be further classified as complete (without sensory and motor function below the neurological level of injury) or incomplete (partial preservation of sensory or motor function below the neurological level). An overview of the neural innervations to key muscle groups is provided in Fig 2-1.

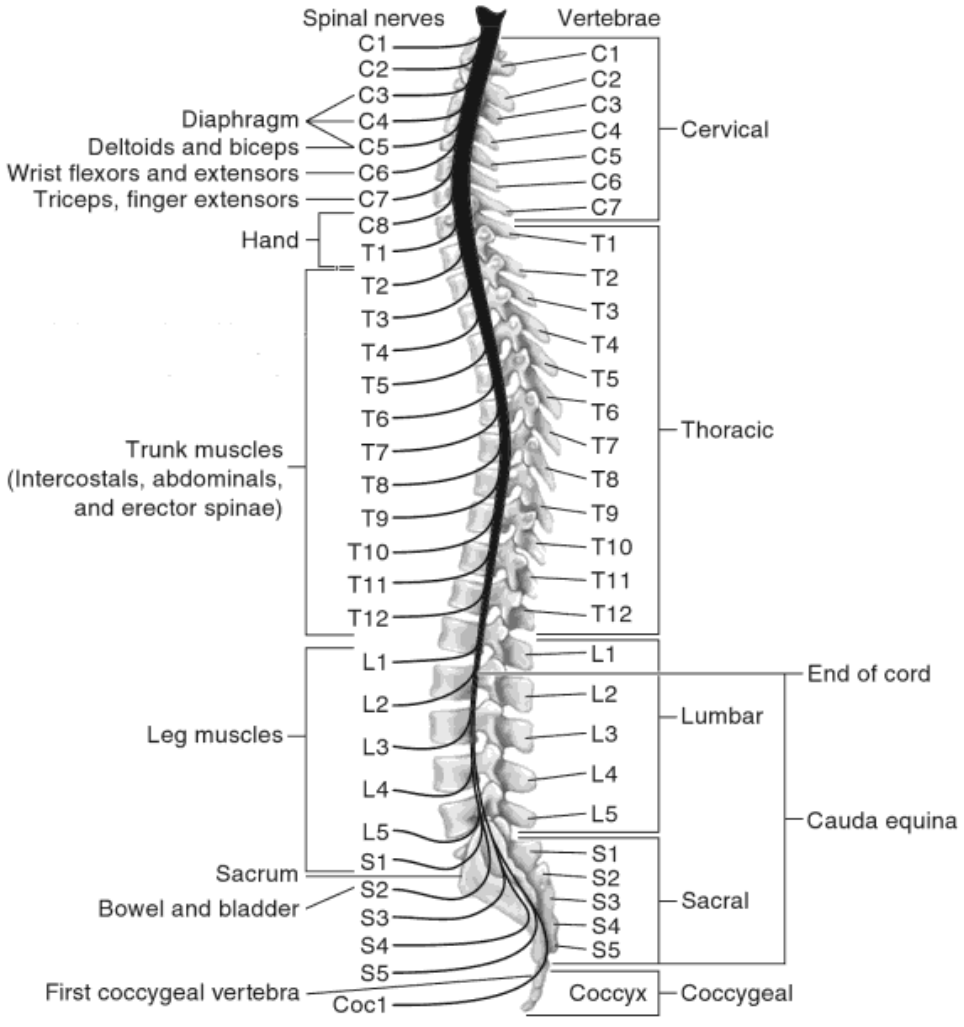

Fig 2-1 Neural innervation of key muscles throughout the body.
Reproduced from Tweedy & Diaper (2010).

2-1.2 Incidence and prevalence of spinal cord injury worldwide

The most comprehensive databases of SCI are stored and maintained in the USA and are well described in the literature (DeVivo *et al.*, 2002; Stover *et al.*, 1999). There is a large worldwide variation in the reported incidence of SCI, from 8 new cases per million population per year in the Netherlands (van Asbeck *et al.*, 2000), to 56 new cases per million population per year in Hualien, Taiwan (Lan *et al.*, 1993). Although approximately 30 studies have reported the annual incidence of SCI, most cover a period of less than 10 years, and were conducted over a decade ago. The relatively few studies that have examined the incidence of SCI post 2000, have been conducted in Australia (O'Connor, 2005a, 2006), China (Ning *et al.*, 2011) and Canada (Pickett *et al.*, 2006), where annual incidence rates vary from 23 to 42 individuals per million population.

Compared with the incidence of SCI, the prevalence of SCI is rarely reported. In 1980, the prevalence of SCI was estimated to be 906 individuals per million inhabitants in the USA (DeVivo *et al.*, 1980). More recently, the prevalence of SCI was estimated to be 681 individuals per million inhabitants in Australia (O'Connor, 2005b), and 280 individuals per million inhabitants in Helsinki (Dahlberg *et al.*, 2005). The large variation in global incidence and prevalence is likely due to several factors, including differences in population demographics, sampling bias with respect to most studies being conducted in specific rehabilitation centres, the definition of SCI, and the survival rate post SCI, which is likely to be much less in the developing world due to subordinate healthcare provision.

2-1.3 Characteristics of spinal cord injury

Approximately half of the studies that have examined the incidence and/or prevalence of SCI also reported age, sex, level of injury and completeness of injury in their cohort. The majority of studies report that 28 to 35 y males are most likely to suffer a SCI, after which the rate starts to decline with increasing age (Acton *et al.*, 1993; Dryden *et al.*, 2003; Ning *et al.*, 2011; Pickett *et al.*,

2006; Price *et al.*, 1994; Woodruff and Baron, 1994). The ratio between complete and incomplete, and cervical and thoracic SCI is more variable; some studies report that cervical SCI accounts for as little as 30% of all SCIs (Gehrig and Michaelis, 1968; Hart and Williams, 1994), whilst others report that cervical SCI accounts for over 70% of all SCIs (Lan *et al.*, 1993; Shingu *et al.*, 1994). The cause of SCI also appears to vary across continents and is heavily influenced by the racial and ethnic composition of the study demographic (Goshgarian, 2010). In the developed world, the most common cause of SCI is motor vehicle accident, followed in no particular order by violence, falls and sport (Goshgarian, 2010).

2-1.4 Mortality

Despite significant improvements in medical care following SCI, mortality rates are significantly elevated compared to the healthy able-bodied (AB) population (DeVivo *et al.*, 1999; DeVivo and Stover, 1995; Garshick *et al.*, 2005). Individuals with high cervical injury (C1-C5) have a relative risk of mortality in the acute period post injury that is over 5 times that of the AB population; for low cervical injuries (C5-C7) the relative risk is approximately 2.5 times that of the AB population; and for thoracic SCI the relative risk is just 1.5 times that of the AB population (DeVivo and Stover, 1995). The most common cause of mortality in acute SCI is pneumonia or other respiratory complications (Lidal *et al.*, 2007; Soden *et al.*, 2000). Hence, the difference in relative risk of mortality among lesion levels is likely due to the greater preservation of respiratory function as the level of SCI moves caudally (section 2-2). In chronic SCI, individuals with cervical and thoracic SCI have a life expectancy that is approximately 70% and 85% of the healthy AB population, respectively (Yeo *et al.*, 1998). The most common cause of mortality in the chronic SCI population regardless of lesion level is cardiovascular disease (Garshick *et al.*, 2005).

2-1.5 Summary

Spinal cord injury is a rare, but debilitating disease with an incidence rate between 8 and 56 new cases per million inhabitants per year. Males between the ages of 18 and 35 y are most at risk of suffering a SCI, and road traffic accidents are the primary cause. Spinal cord injury results in a lesion dependent loss of motor and / or sensory function. Injury to the cervical spinal cord also causes profound alterations in both respiratory and cardiovascular function that significantly reduce life expectancy, and may predispose these individuals to many secondary health conditions. Accordingly, the following two sections provide an overview of respiratory (section 2-2) and cardiovascular (section 2-3) function in individuals with cervical SCI.

2-2 Respiratory function

2-2.1 Introduction

The respiratory system is a complex and highly organised system that is responsible for pumping air in and out of the lungs and maintaining blood gas tensions within acceptable limits. The respiratory muscles are striated skeletal muscles that have either an inspiratory or expiratory action. Injury to the cervical spinal cord causes paralysis of the majority of respiratory muscles and significantly reduces the capacity of the respiratory system. Since individuals with cervical SCI are the focus of subsequent investigations throughout this thesis, this section provides an overview of respiratory function following cervical SCI only. The potential respiratory limitations to exercise that may occur in this population are then considered. Several excellent reviews are available on the respiratory system in AB individuals (Dempsey *et al.*, 1996; Romer *et al.*, in press) and in SCI (Brown *et al.*, 2006; Schilero *et al.*, 2009; Winslow and Rozovsky, 2003).

2-2.2 Respiratory muscle structure and function

2-2.2a Inspiratory muscle structure and function

Diaphragm

The diaphragm is the primary muscle of inspiration and contributes to ~70% of the resting tidal volume (Campbell, 1958; Wade, 1954). The diaphragm is a thin musculo-fibrous structure which separates the thorax from the abdomen. The muscular fibres of the diaphragm are divided into two parts: 1) the vertebral (or crural), which arises from the crura of the lumbar vertebrae and the arcuate ligament; and 2) the costal, which arises from the costal margin (Campbell, 1958). The crural and costal diaphragm receive neural innervation from separate branches of the phrenic nerves situated between C3-C5 (Pickering and Jones, 2002). Individuals with a SCI above C3 suffer complete paralysis of the diaphragm and require mechanical ventilation or diaphragm pacing to sustain life, whilst those with an injury caudal to C5 retain full neural innervation of the diaphragm. Accordingly, this review will focus on individuals with low cervical SCI (C5-C7) only.

Two studies have quantified the maximal pressure generating capacity of the diaphragm ($P_{di,max}$) in response to a maximal Müeller (inspiratory) manoeuvre in individuals with low cervical SCI (Sinderby *et al.*, 1996a; Taylor *et al.*, 2010). Both studies reported a lower $P_{di,max}$ compared to values typically reported in the AB population, suggesting that despite retaining full innervation of the diaphragm, the pressure generating capacity of this muscle is impaired in individuals with cervical SCI. One drawback of assessing $P_{di,max}$ during a Müeller manoeuvre is the reliance upon participant motivation to achieve a maximal response. The emergence of effort independent nerve stimulation techniques has allowed the pressure generating capacity of the diaphragm to be studied non-volitionally. Using cervical magnetic stimulation of the phrenic nerves, Hart *et al.* (2005) reported that twitch transdiaphragmatic pressure ($P_{di,tw}$) was 21 cmH_2O in individuals with cervical SCI. Similar values have also been reported in response to bilateral anterolateral magnetic stimulation of the phrenic nerves (Taylor *et al.*, 2010). Although neither study included

a control group, both studies reported values for SCI that are approximately one third lower than those typically reported in the AB population (Mador *et al.*, 2002). Thus, based on the $P_{di,max}$ and $P_{di,tw}$ values obtained to date, it appears that the force generating capacity of the diaphragm is impaired in SCI, most likely due to an anterio-caudal shift of the abdominal contents, which will shorten diaphragm length, reduce the radius of curvature, and alter the geometric shape of the diaphragm within the thorax, resulting in a reduction in insertional and appositional forces of the diaphragm through the zone of apposition, and a reduction in the force generating capacity of the diaphragm (Decramer, 1997). These factors are considered in more detail in the chest wall mechanics section (2-2.4). The neural innervations of the key inspiratory and expiratory muscles are displayed in Fig 2-2.

Scalenes

The scalenes are a group of three muscles, including the scalenus anticus, scalenus medius and scalenus posticus. The scalenes are located deeply at the side of the neck; they originate from the transverse process of the lower five cervical vertebrae and insert onto the upper surface of the first rib. The scalenes are innervated by branches of the lower cervical nerves situated between C2 and C7. Indwelling needle electrode studies have demonstrated that the scalenes are a primary muscle of inspiration and are active in both the supine and seated position in AB individuals (Raper *et al.*, 1966). Research by De Troyer *et al.* (1984) enhanced our understanding of the function of the scalenes by demonstrating that scalene activity is required to achieve full lung inflation, and to achieve complete movement of the rib cage along its relaxation characteristic. Individuals with low cervical SCI have varying degrees of residual function of the scalene depending on the level of their injury (Short *et al.*, 1991).

Sternocleidomastoids

The sternocleidomastoids descend from the mastoid process to the manubrium sterni and the medial clavicle. The sternocleidomastoids are not involved in resting breathing but contribute to inspiration during voluntary maximal inspiratory efforts and when ventilation is increased (Campbell, 1958). The sternocleidomastoids are innervated by the spinal accessory nerve (cranial nerve 11) and the second cervical nerve, and therefore are fully innervated in all individuals with a low cervical SCI. The respiratory action of the sternocleidomastoids has been determined in cervical SCI by DeTroyer *et al.* (1986b), who demonstrated that isolated contraction of the sternocleidomastoids causes a cranial displacement of the sternum and a large increase in upper rib-cage expansion.

External Intercostals

The intercostals are thin planes of muscular fibres that occupy the intercostal spaces. The external intercostals extend from the tubercles of the ribs dorsally to the costochrondal junctions ventrally. The external intercostals slope caudally and ventrally from the rib above to the rib below, such that contraction of these muscles raises the ribs. The parasternal intercostals form part of the internal intercostals, which usually slope caudal and distally from the rib above to the rib below such that they deflate the ribs; however, the close location of the parasternal intercostals to the sternum causes the rib cage to raise when the parasternal intercostals contract (De Troyer, 2005). Although there may be some limited contraction of the parasternal intercostals during quiet inspiration in healthy AB humans, the intercostals are considered accessory muscles of inspiration that are responsible for raising the rib cage during deep or vigorous breathing (Taylor, 1960). The intercostal muscles are innervated by the intercostal nerves (T1-T11) and are therefore not innervated in individuals with complete cervical SCI. The primary consequence of intercostal dennervation in cervical SCI is impaired chest wall mechanics (see section 2-2.4). There is some evidence that individuals with cervical SCI have reflex function

of the external intercostals, especially in the lower (eighth) intercostal muscle (Silver and Lehr, 1981). The authors also reported that the degree of reflex function appeared to be dependent on time since injury, suggesting that an improvement in reflex function of the intercostals may be partly responsible for the improvement in lung function over time in this population (see also section 2-2.5).

Other inspiratory muscles

The trapezius (innervated by cranial nerve 11 and cervical nerves C3-C4), erector spinae (C6-T10) and the serrati (C5-C7) all contract during large inspiratory efforts; however, their specific respiratory functions remain to be determined. The current understanding is that in healthy AB individuals these muscles primarily provide postural support during inspiration (De Troyer, 2005). For example, the upper fibres of the trapezius act to fixate the head whilst contraction of the sternocleidomastoids causes a marked increase in upper rib cage dimensions (Winslow and Rozovsky, 2003). For individuals with cervical SCI, contraction of the trapezius has been documented during deep inspirations (De Troyer *et al.*, 1986b).

2.2-2b Expiratory muscle structure and function

Abdominal muscles

The primary muscles of expiration are those of the ventrolateral abdominal wall; they include the rectus abdominis, internal oblique, external oblique and the transversus abdominis. Contraction of the abdominal muscles causes an inwards pull of the abdominal wall, which forces the diaphragm cranially, thereby increasing pleural pressure and decreasing lung volume. Contraction of the abdominal muscles also pulls the rib cage caudally. Thus, the abdominal muscles are considered the primary muscles of expiration. The expiratory muscles are innervated by thoracic and sacral nerves situated between T6 and L1. Consequently, expiratory function is severely reduced in individuals with cervical SCI. Indeed, early studies suggested that expiratory muscle

dennervation resulted in complete loss of expiratory function (Siebens *et al.*, 1964). More recently, however, it has been demonstrated that there is residual abdominal muscle function in individuals with cervical SCI, as evidenced by a significant increase in gastric pressure in response to thoracic stimulation of the abdominal nerve roots (Estenne *et al.*, 2000). The degree of increase was ~60% less than in AB individuals most likely due to muscle fibre atrophy in the abdominal muscles of individuals with cervical SCI (Estenne *et al.*, 2000). Further evidence that there may be some residual abdominal muscle function in individuals with SCI above T6 stems from a study that used fine-wire indwelling electrodes in the abdominal muscles (Bjerkefors *et al.*, 2009). Those authors demonstrated EMG activity in four abdominal muscles in response to a maximal voluntary contraction and balance perturbations in an individual with a complete T3 SCI.

Internal intercostals

The internal intercostals slope caudally and distally from the rib above to the rib below. In this respect, contraction of the expiratory intercostals causes the rib cage to deflate. Although some activity of the internal intercostals muscles has been documented during quite breathing, these muscles primarily assist expiration during deep and vigorous breathing (Taylor, 1960). Similar to the external intercostals, the internal intercostals are innervated by the intercostal nerves (T1-T11), and hence are dennervated in individuals with cervical SCI. To the author's knowledge there have been no studies assessing reflex activity of the internal intercostals in individuals with cervical SCI.

Triangularis sterni

The triangularis sterni lies deep to the parasternal intercostals. Studies in a canine model have shown that contraction of the triangularis sterni causes a marked caudal displacement of the ribs, a slight elevation of the sternum and a reduction in lung volume (De Troyer *et al.*, 1986a; Ninane *et al.*, 1989). In humans, the triangularis sterni has been shown to be active during cough (De

Troyer *et al.*, 1987). However, the expiratory action of the triangularis sterni on the lung is much smaller than that of the internal intercostals (Wilson *et al.*, 2001). The triangularis sterni receives innervation from the intercostal nerves and is therefore not innervated in cervical SCI. To the author's knowledge there have been no studies assessing reflex activity of the triangularis sterni in individuals with cervical SCI.

Pectoralis major

The pectoralis major originates from the body of the sternum, the cartilages of ribs 2-6, and the inferior and medial portion of the clavicle. The pectoralis major is innervated by the pectoral nerve between C5 and T1, hence the upper (clavicular) portion of the pectoralis major is innervated in individuals with lower cervical SCI. A series of experiments by DeTroyer and co-workers have shown that the clavicular portion of pectoralis major contributes to expiration in individuals with cervical SCI (De Troyer *et al.*, 1986a; Estenne and De Troyer, 1990). Although contraction of the pectoralis major may be sufficient to cause the upper rib cage to collapse, the lungs to empty, and dynamic compression of the intrathoracic airways (Estenne *et al.*, 1994), it cannot fully compensate for abdominal muscle paralysis. Hence, expiratory muscle strength is still much lower in cervical SCI compared with AB (section 2-2.3).

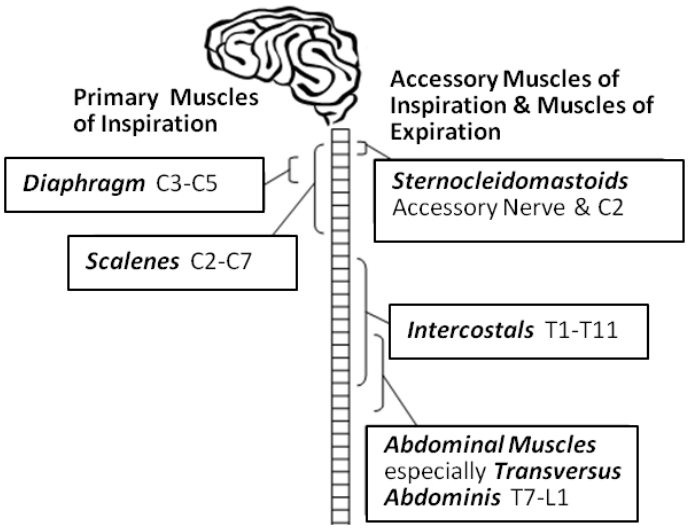

Fig 2-2 Neural innervation of key inspiratory and expiratory muscles.
Adapted from Sheel et al. (2008a).

2-2.3 Respiratory muscle strength

Respiratory muscle strength is most commonly measured via maximal static inspiratory or expiratory mouth pressures ($P_{I,max}$ and $P_{E,max}$, respectively). Although this technique is unable to partition the relative contribution of each inspiratory or expiratory muscle group to pressure development, the method does provide a good surrogate measure of global inspiratory or expiratory muscle strength (Green *et al.*, 2002). For individuals with SCI there is a lesion-dependent impairment in respiratory muscle strength, whereby respiratory muscle strength decreases as the level of injury moves cranially (Baydur *et al.*, 2001; Mateus *et al.*, 2007). For low cervical SCI, there is a greater preservation of inspiratory muscle strength relative to expiratory muscle strength due to the neural innervations of the respective muscles (Fig 2-2). Hence, $P_{I,max}$ is consistently reported to be higher than $P_{E,max}$ (Fugl-Meyer and Grimby, 1971a, b; Gounden, 1997; Mateus *et al.*, 2007).

2-2.4 Chest wall mechanics

In AB individuals, there is a synchronous relationship between the upper rib cage, lower rib cage and abdominal region of the thoracic cavity. In individuals with cervical SCI, however, dennervation of the intercostal and abdominal muscles alters this relationship such that the three parts of the thoracic cavity become uncoupled and act independently of each other. The majority of studies that have assessed chest wall mechanics in SCI were conducted in the early 1970s and 1980s. The first study to assess respiratory mechanics in individuals with cervical SCI used spirometry, and an electromagnet fixed to the body surface to assess changes in abdominal and rib cage compartment volumes during resting breathing in the seated, supine and various recumbent positions (Fugl-Meyer and Grimby, 1971a). The authors reported that in some individuals with cervical SCI, the antero-posterior movement of the rib cage was out of phase during resting breathing in the seated position, whereby the diameter of the upper rib cage

decreased at end inspiration and increased at end expiration – a phenomenon now recognised as a paradoxical breathing pattern. These findings have since been corroborated by others, who have also demonstrated evidence of a paradoxical breathing pattern in the upper rib cage during quiet breathing in the seated position (Mortola and Sant'Ambrogio, 1978; Urmey *et al.*, 1986).

Individuals with cervical SCI also demonstrate severe chest wall distortion and a subsequent reduction in the rib-cage contribution to ventilation during resting breathing (Grimby *et al.*, 1968). To determine the degree of rib cage distortion, changes in the size of the rib cage compartment are measured during passive lung inflation. Rib cage distortion during resting breathing is then assessed by quantifying the degree of deviation from the relaxation curve. Using inductive pneumography, Urmey *et al.* (1986) studied upper and lower rib cage distortion during quiet breathing in individuals with cervical SCI. The authors reported evidence of severe upper rib cage distortion in all individuals, whereas lower rib cage distortion was evident in only 50% of the individuals. This pattern of rib-cage distortion is suggestive of isolated contraction of the diaphragm, which expands the lower rib cage but reduces the antero-posterior dimensions of the upper rib cage (Danon *et al.*, 1979). A summary of the rib-cage and abdominal contributions to ventilation during diaphragmatic or neck and intercostal breathing is provided in Fig 2-3.

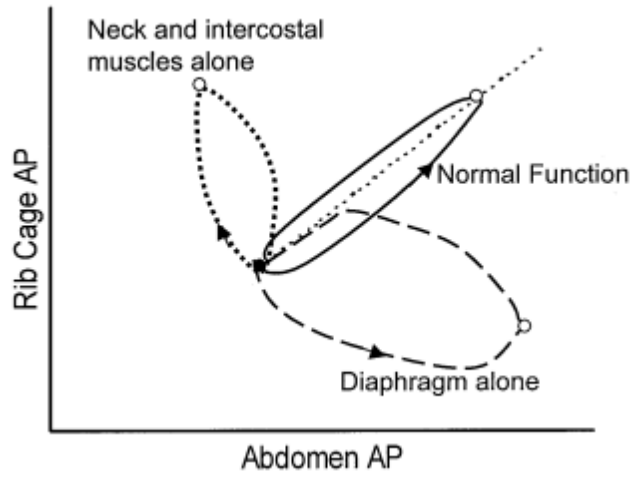

Fig 2-3 Konno-mead plot of abdominal versus rib cage motion during isolated breathing with the neck and intercostal muscles, or the diaphragm.
Note the increase in abdominal dimensions and reduction in upper rib cage dimensions during isolated diaphragmatic breathing. Note also the reduction in lower rib cage dimensions during isolated neck and intercostal muscle breathing. AP, anteroposterior; arrows indicate direction of motion; closed circles indicates end expiration; open circles indicate end inspiration. For the purpose of this figure, abdomen refers to the lower rib cage and abdominal compartment combined, and rib cage refers to the upper rib cage only. Reproduced from Winslow and Rozovsky (2003).

Studies that have assessed abdominal and rib cage compliance have enhanced our understanding of chest wall mechanics in individuals with cervical SCI. A number of studies have demonstrated that abdominal compliance is significantly elevated, whereas rib cage compliance is lower in individuals with cervical SCI compared to AB (Estenne and De Troyer, 1986; Goldman *et al.*, 1986a; Moulton and Silver, 1970; Scanlon *et al.*, 1989). In the seated position, an increase in abdominal compliance causes the abdominal contents to migrate antero-caudally, which reduces the intra-abdominal pressure rise as the diaphragm contracts. Intra-abdominal pressure can be regarded as the fulcrum for providing the elevating action of the diaphragm through the zone of apposition. Thus, a reduction in intra-abdominal pressure rise during inspiration directly reduces the expanding effect of the diaphragm on the lower rib cage through the zone of apposition. It should also be noted that an antero-caudal shift of the abdominal contents may shorten the diaphragm and reduce the radius of curvature, both of which will contribute to a reduction in the pressure-generating capacity of the diaphragm (De Troyer, 1997). The elevated abdominal

compliance is offset by a stiffer rib cage that is most likely caused by ankylosis in the joints arising from intercostal dennervation (Estenne and De Troyer, 1986). The current understanding of respiratory mechanics in individuals with cervical SCI in the seated position is summarised in Fig 2-4.

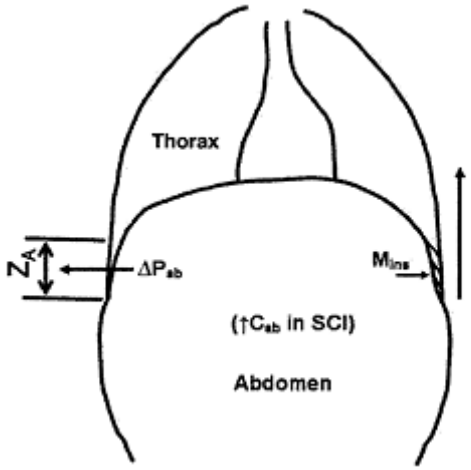

Fig 2-4 Interaction between the diaphragm and chest wall.
At the lower rib cage the diaphragm is in direct apposition to the rib cage (zone of apposition: ZA). In AB individuals, as the diaphragm contracts, intra-abdominal pressure increases to allow the diaphragm to elevate the lower rib cage through the ZA. In individuals with cervical SCI, a large increase in abdominal compliance reduces the curvature of the diaphragm, and therefore the size of the ZA. Furthermore, the increase in abdominal compliance for SCI causes a reduction in the intra-abdominal pressure rise during inspiration. Together, these two factors compromise the action of the diaphragm through the ZA, and hence reduce the effectiveness to which the diaphragm can expand the lower rib cage. The increase in abdominal compliance also allows the diaphragm to descend unimpeded into the abdominal compartment and the associated reduction in pleural pressure causes a paradoxical inwards movement of the upper rib cage during inspiration. Reproduced from Brown et al. (2006).

2-2.5 Pulmonary function

Numerous studies have investigated pulmonary function in individuals with chronic cervical SCI. The most simple and commonly reported measure of pulmonary function is vital capacity (VC), which can be measured via spirometry. Over the past 40 years it has been consistently reported that individuals with cervical SCI have a lower VC compared to AB predicted values, suggesting a restrictive pulmonary defect [e.g. (Baydur *et al.*, 2001; Estenne *et al.*, 1993; Forner, 1980; Fugl-Meyer and Grimby, 1971b; Jain *et al.*, 2006; Linn *et al.*, 2001; Maloney, 1979; Stepp *et al.*, 2008)].

The variation in VC between studies is mainly dependent upon the level of lesion within the cervical spinal cord; however, differences may also be due to body position, time since injury and age. For example, VC is higher in the supine versus seated posture in individuals with cervical SCI due to the effects of gravity compressing the abdomen (Estenne and De Troyer, 1987), a lengthening of muscle fibres at end-expiration that places the diaphragm in a more favourable position for tension development, or both (Baydur *et al.*, 2001). Furthermore, VC is severely reduced to approximately 30% of AB predicted in the acute phase following cervical SCI (Ledsome and Sharp, 1981). However, during the first three months post injury, VC increases to approximately 60% of AB predicted (Ledsome and Sharp, 1981). The improvement in respiratory function during recovery from SCI has been attributed to improvements in diaphragm performance (Oo *et al.*, 1999), reflex activity in the intercostal muscles (Silver and Lehr, 1981) and enhanced performance of the neck muscles (Frisbie and Brown, 1994).

In the healthy AB population, a low VC is commonly used to infer pulmonary restriction. However, it has been demonstrated that whilst a normal VC rules out restriction in all but 3% of individuals, a low VC is only accurate in determining pulmonary restriction less than 60% of the time (Aaron *et al.*, 1999). In cervical SCI, the usefulness of VC in determining a restrictive pulmonary defect may be further compromised as severe expiratory muscle weakness may result in a disproportionate reduction in VC (an expiratory manoeuvre) compared with total lung capacity (TLC; a combination of inspiratory and expiratory function). Thus, it is preferential to study TLC rather than VC to define pulmonary restriction in cervical SCI. The measurement of TLC, however, requires specialist equipment that is not readily available in most hospital or rehabilitation settings. Thus, TLC tends to be measured for research purposes only. The studies that have documented TLC and associated lung volumes in individuals with cervical SCI all show that TLC is below the lower 5th percentile of AB predicted (Anke *et al.*, 1993; Estenne *et al.*, 1993; Fugl-Meyer and Grimby, 1971b; Hart *et al.*, 2005; Huldtgren *et al.*, 1980; Roth *et al.*, 1997; Stepp *et al.*, 2008), which is considered evidence for pulmonary restriction based on current American

Thoracic Society (ATS)/European Respiratory Society (ERS) guidelines (Pellegrino *et al.*, 2005). Those same studies also reported that residual volume (RV) is elevated compared to AB predicted, whilst FRC is similar. The increase in RV is likely due to reduced expiratory muscle strength, but may also be due to premature airway closure, elevated airway resistance, or both (Stepp *et al.*, 2008). The relatively normal FRC can be explained by an elevated RV and small expiratory reserve volume (ERV). In addition to impairments in lung capacities and volumes, peak expiratory flow (PEF) and mean mid-expiratory flow (MMEF) are also significantly reduced following cervical SCI (Baydur *et al.*, 2001; Estenne *et al.*, 1998; Fujiwara *et al.*, 1999; Lin *et al.*, 2006; Mueller *et al.*, 2008). These reductions are most likely due to the dennervation of the abdominal muscles and the subsequent reduction in expiratory muscle strength (section 2-2.3).

In comparison to a restrictive pulmonary defect, an obstructive pulmonary defect was not thought to be present in the majority of individuals with cervical SCI. According to the current ATS/ERS guidelines, an obstructive defect is defined as a FEV_1-to-VC ratio below the lower 5^{th} percentile of the AB predicted value (Pellegrino *et al.*, 2005). However, this method is dependent upon lung volume history, such that a low VC may result in a normal FEV_1-to-VC ratio. Technological advances now allow airway resistance to be measured during resting tidal breathing. Indeed, using both body plethysmography and flow oscillometry it is well documented that individuals with cervical SCI have an obstructive pulmonary defect (Mateus *et al.*, 2006; Radulovic *et al.*, 2008; Schilero *et al.*, 2005; Singas *et al.*, 1996). The primary mechanism purported for the obstructive defect is overriding parasympathetic (cholinergic) tone, secondary to the loss of sympathetic innervation to the lungs (Mateus *et al.*, 2006). A secondary cause of the obstructive pulmonary defect may be related to loss of regular stretch of the airway smooth muscle, resulting from the restrictive pulmonary defect (Singas *et al.*, 1996).

2-2.6 The respiratory system during exercise

For individuals with cervical SCI who regularly engage in structured exercise, the aforementioned respiratory (dys)function may cause ventilatory constraint and thereby impair exercise capacity. In healthy AB individuals, the respiratory system must increase alveolar ventilation by up to 20 times that of rest to regulate alveolar partial pressure of oxygen (PaO_2) and carbon dioxide ($PaCO_2$) during high intensity exercise (Guenette and Sheel, 2007). Although it is generally accepted that the normal healthy respiratory system has ample capacity to cope with the increased demands placed upon it (Olafsson and Hyatt, 1969), large increases in the metabolic cost and work of breathing may conspire to limit exercise capacity in certain populations. For example, the respiratory system has been demonstrated to become constrained during lower-body exercise in male and female endurance trained athletes (Guenette *et al.*, 2007; Johnson *et al.*, 1992), patients with asthma (Haverkamp *et al.*, 2005; Haverkamp *et al.*, 2007), and patients with chronic obstructive pulmonary disease (O'Donnell and Laveneziana, 2007; Vogiatzis *et al.*, 2004). Three examples of how the respiratory system may limit exercise capacity are respiratory muscle fatigue, ventilatory constraint and exercise-induced arterial hypoxaemia (EIAH). This section will briefly review the evidence for each of the three examples during lower and upper body exercise in the AB population, and during upper-body exercise in the cervical SCI population. Numerous reviews on the respiratory system as a limiting factor for exercise performance in AB individuals are available [e.g., (Dempsey *et al.*, 2008a; Romer and Polkey, 2008)].

Respiratory muscle fatigue in able-bodied

The majority of research that has assessed respiratory muscle fatigue in AB individuals has concentrated on inspiratory muscle fatigue. As the diaphragm is the primary inspiratory muscle, fatigue of this muscle provides the most appropriate index of inspiratory muscle fatigue. Using electrical and magnetic nerve stimulation techniques, definitive evidence of inspiratory (diaphragm) muscle fatigue has been demonstrated following high intensity (>80% max) lower

body constant-load exercise (Johnson *et al.*, 1993; Mador *et al.*, 1993). Diaphragm fatigue is quantified by assessing the pre-to-post exercise decline in the twitch transdiaphragmatic pressure ($P_{di,tw}$) response to bilateral phrenic nerve stimulation. Using electrical anterolateral phrenic nerve stimulation it has been demonstrated that $P_{di,tw}$ is reduced following high intensity exercise, and $P_{di,tw}$ did not return to pre-exercise values until > 60 min post-exercise (Johnson *et al.*, 1993; Mador *et al.*, 1993). Although the link between diaphragm fatigue and exercise performance is still not fully understood in the AB population, diaphragm fatigue may contribute to impaired exercise performance by way of an inadequate ventilatory response, an alteration in breathing mechanics, and / or an increase in dyspnoea (Romer and Polkey, 2008). However, the most likely mechanism by which exercise-induced diaphragm fatigue reduces exercise performance is through activation of the respiratory muscle metaboreflex, whereby group III and IV phrenic afferents cause a global increase in sympathetically mediated vasoconstriction (Derchak *et al.*, 2002; St Croix *et al.*, 2000), which compromises limb locomotor blood flow (Harms *et al.*, 1997), exacerbates peripheral fatigue (Romer *et al.*, 2006b), and impairs exercise performance (Harms *et al.*, 2000).

Ventilatory constraint in able-bodied

Expiratory flow limitation may provide a source of ventilatory constraint during high intensity exercise. Expiratory flow limitation is most common in conditions which alter airway and lung elastic properties such as chronic obstructive pulmonary disease (Diaz *et al.*, 2000; Koulouris *et al.*, 1997), but has also been demonstrated in cyclists during high intensity exercise (McClaran *et al.*, 1999). The presence of expiratory flow limitation may cause a significant alteration in breathing pattern during exercise, whereby end-expiratory lung volume (EELV) increases back to, or above, resting levels (i.e. dynamic hyperinflation). Dynamic hyperinflation occurs to permit an increase in expiratory flow rate; however, this is at the expense of an increased elastic work of breathing during inspiration. Dynamic hyperinflation also impairs the pressure generating

capacity of the diaphragm by reducing the diaphragm's operating length, altering the geometric arrangement of the diaphragm within the thorax, reducing the zone of apposition, and reducing the insertional component of diaphragmatic action (Decramer, 1997). The net effect of hyperinflation is an increase in the elastic work of breathing and increased susceptibility to inspiratory muscle fatigue.

Exercise-induced arterial hypoxaemia in able-bodied

During exercise the alveolar to arterial pressure gradient progressively widens as exercise intensity increases. If this occurs without a compensatory hyperventilation and with an acid (or temperature) induced rightward shift of the haemoglobin dissociation curve, arterial oxygen desaturation will prevail and individuals will experience EIAH (Dempsey *et al.*, 2008b). The most commonly accepted methods for defining EIAH are a 10 mmHg or greater decrement in PaO_2 from rest to maximal exercise and/or a reduction in SaO_2 of 3-4% below rest (Dempsey and Wagner, 1999; Prefaut *et al.*, 2000). Although the incidence of EIAH may be dependent upon the site of temperature correction used (Scroop and Shipp, 2010), there is general agreement that when PaO_2 is corrected for oesophageal temperature (which is considered the most appropriate correction as it closely reflects the temperature of pulmonary venous blood), ~50% of highly fit male AB individuals exercising at sea-level experience EIAH (Powers *et al.*, 1988; Scroop and Shipp, 2010). The exact causes of EIAH are unknown, but may relate to a ventilation-to-perfusion mismatch, a right-left shunt or to an alveolar-to-capillary diffusion disequilibrium (Dempsey and Wagner, 1999). The effect of EIAH on exercise performance is pronounced; for every 1% drop in oxygen saturation below 95%, there is a 1-2% reduction in $\dot{V}O_{2peak}$, impaired exercise performance and exacerbated limb fatigue (Amann *et al.*, 2006; Harms *et al.*, 2000; Romer *et al.*, 2006a).

Upper body exercise in able-bodied

Compared with lower body exercise, little is known about the respiratory system as a limiting factor for exercise performance during upper body exercise. To the author's knowledge no study has assessed respiratory muscle fatigue or EIAH during upper body exercise, and only two studies have measured changes in operating lung volumes (i.e., EELV, and end-inspiratory lung volume; EILV) during upper body exercise in healthy AB individuals (Alison *et al.*, 1998; Cerny and Ucer, 2004). Both studies measured EELV by asking their participants to complete two inspiratory capacity (IC) manoeuvres at the end of each exercise stage, such that EELV could be calculated from the subtraction of IC from VC. Neither study, however, measured oesophageal pressure, nor attempted to place a tidal flow-volume loop within the maximal flow-volume loop to quantify whether expiratory flow limitation was present. These methodological shortcomings perhaps explain the discrepancy in findings, as one study reported a smaller reduction in EELV during leg versus arm exercise (Cerny and Ucer, 2004) whereas the other study reported a smaller reduction in EELV during arm versus leg exercise (Alison *et al.*, 1998). A smaller reduction in EELV during arm versus leg exercise seems intuitive due to the increased need for the abdominal muscles to stabilise the torso during upper body exercise. Support for this postulate stems from studies that have documented the tidal volume (V_T) and respiratory frequency (f_R) response to incremental upper body exercise (Pendergast, 1989; Takano, 1993). In these studies the authors reported that upper body exercise resulted in a smaller V_T than lower body exercise, suggesting that the dual use of the respiratory muscles in upper body exercise reduces the degree to which the respiratory muscles can increase V_T.

Respiratory muscle fatigue in cervical spinal cord injury

The respiratory system as a limiting factor for exercise performance has rarely been studied in the cervical SCI population. For individuals with cervical SCI who rely on upper body exercise, dual use of the respiratory muscles for respiratory activity and torso stabilisation may be exacerbated

due to abdominal muscle paralysis. Indeed, Sinderby *et al.* (1992) demonstrated an increase in electromyographic activity of the diaphragm during a simple postural challenge in individuals with cervical SCI. Thus, it is reasonable to believe that the combined respiratory and postural demands placed upon the diaphragm during exercise, combined with the impaired pressure generating capacity of the diaphragm at rest, may increase the susceptibility to diaphragm fatigue in the cervical SCI population. This postulate was first tested by Sinderby *et al.* (1996b) who found a significant reduction in the centre frequency of the diaphragm electromyogram (EMG) following a bout of maximal arm-cranking exercise in 10 individuals with cervical SCI, suggesting evidence of diaphragm fatigue in this population. However, the aetiology of spectral shifts with EMG to detect fatigue is controversial and may relate more to action potential transmission than fatiguing processes in the sarcomere (Supinski *et al.*, 2002). Conversely, using bilateral anterior magnetic stimulation of the phrenic nerves (BAMPS), Taylor *et al.* (2010) found no evidence of diaphragm fatigue following high intensity constant load exercise to exhaustion in highly-trained athletes with cervical SCI.

Ventilatory constraint in cervical spinal cord injury

The first study to assess whether the respiratory system is constrained during exercise in cervical SCI compared the peak minute ventilation ($\dot{V}_E$) achieved during exercise to that achieved during a maximum voluntary ventilation (MVV) test (Wicks *et al.*, 1983). That study showed that peak $\dot{V}_E$ was less than 50% of the value achieved during the MVV test. However, the validity of the MVV test as a true measure of ventilatory capacity has been criticised, mainly on the basis that the MVV only lasts 12-15 s and the pressures and flows generated during an MVV are not representative of those produced at peak exercise (Johnson *et al.*, 1999a). Furthermore, the MVV does not take into account dynamic changes in lung volumes that occur during exercise, and is a voluntary assessment of breathing capacity, whereas the hyperpnoea of maximal exercise is reflexively driven (Johnson *et al.*, 1999a). The MVV, therefore, provides only limited information

regarding the ventilatory capacity and does not provide information on breathing strategy or the degree of inspiratory or expiratory flow constraint.

Using the more sophisticated method of placing an exercise tidal flow-volume loop within a maximal flow-volume loop, Taylor *et al.*(2010) demonstrated that highly-trained athletes with cervical SCI exhibit a sudden and sustained dynamic hyperinflation from the onset of exercise and a tachypnic breathing pattern. That study also demonstrated a hyperventilatory response at the end of exercise that is similar to that in AB individuals. However, the peak ventilation achieved at end exercise was less than 50% of that typically achieved by AB individuals at the end of exercise. Thus, there appears to be a severe hyperventilatory response during exercise in the cervical SCI population for a relatively small increase in ventilation. A further constraint to ventilation during exercise may be caused by the increased rib-cage stiffness in individuals with cervical SCI. A stiff rib-cage may increase the relative pressure required to inflate the thoracic cavity. The relationship between pressure generated by the respiratory muscles (effort) and change in thoracic dimensions (displacement) during inspiration can be estimated by calculating the rate of neuromechanical uncoupling (effort-to-displacement ratio), which is defined as the ratio between tidal oesophageal pressure (P_{oe}) swings expressed relative to $P_{I,max}$ (effort) and the tidal volume (V_T) expressed relative to VC (O'Donnell *et al.*, 1997). This index has mostly been studied in individuals with chronic obstructive pulmonary disease (O'Donnell *et al.*, 2006; O'Donnell and Laveneziana, 2007), and has never been assessed in the cervical SCI population. An elevated index of neuromechanical uncoupling could contribute to exercise limitation in the cervical SCI population by increasing dyspnoea and susceptibility to respiratory muscle fatigue.

Exercise-induced arterial hypoxaemia in cervical spinal cord injury

There is debate as to whether inadequate pulmonary gas exchange and EIAH occurs in individuals with cervical SCI. One early study that did report EIAH during exercise in individuals with cervical SCI suggested that a paradoxical breathing pattern may reduce the expansion of the upper lobes

of the lung, increase dead space ventilation and thereby create an imbalance in ventilation that may explain the EIAH (Bergofsky, 1964). However, a more recent study that also found evidence of EIAH documented relatively normal values of ventilatory equivalents for oxygen and carbon dioxide (Klefbeck *et al.*, 1998). This suggests that dead space ventilation is not increased in individuals with cervical SCI, and other factors must explain the EIAH. In comparison to studies in untrained individuals, a study in highly-trained athletes with cervical SCI found no evidence of EIAH (Taylor *et al.*, 2010), suggesting perhaps that improved cardiorespiratory function in the trained SCI population may attenuate EIAH. A criticism of all the aforementioned studies, however, is that pulse oximetry was used to infer changes in arterial oxygen saturation. To date, no study has directly measured PaO_2 or arterial oxygen saturation during exercise in the SCI population. One study has reported that $\dot{V}O_{2peak}$ is increased during ACE when participants with cervical SCI were exposed to a hyperoxic (50% FiO_2) gas mixture (Hopman *et al.*, 2004), suggesting that oxygen supply may limit $\dot{V}O_2$. A limited oxygen supply during exercise in the tetraplegic population does not appear to be due to impaired oxygen extraction as the calculated arterio-venous oxygen difference ($a\text{-}\bar{v}\,O_2$) is similar between cervical SCI and AB (Van Loan *et al.*, 1987). Thus, a limited oxygen supply is more likely the result of an inability to increase $\dot{Q}$ sufficiently during exercise due to a limited increase in stroke volume and heart rate in the cervical SCI population (Dela *et al.*, 2003). These factors, along with an overview of the cardiovascular system and how it may limit exercise performance in the cervical SCI population is considered in more detail in section 2-3.6.

2-2.7 Summary

Cervical SCI causes profound alterations in respiratory muscle function, chest wall compliance and abdominal compliance. Together, these alterations cause severe chest wall distortion and a paradoxical inwards movement of the upper rib cage during resting breathing. The combined result of these alterations is a restrictive and obstructive pulmonary defect, and a predisposition to respiratory complications. During exercise there is evidence of an immediate and sustained dynamic hyperinflation, which may contribute to an inefficient breathing pattern, heightened exercise induced dyspnoea, and ultimately impaired exercise performance. A summary of the respiratory consequences of cervical SCI and how they may affect exercise performance is provided in Fig 2-5.

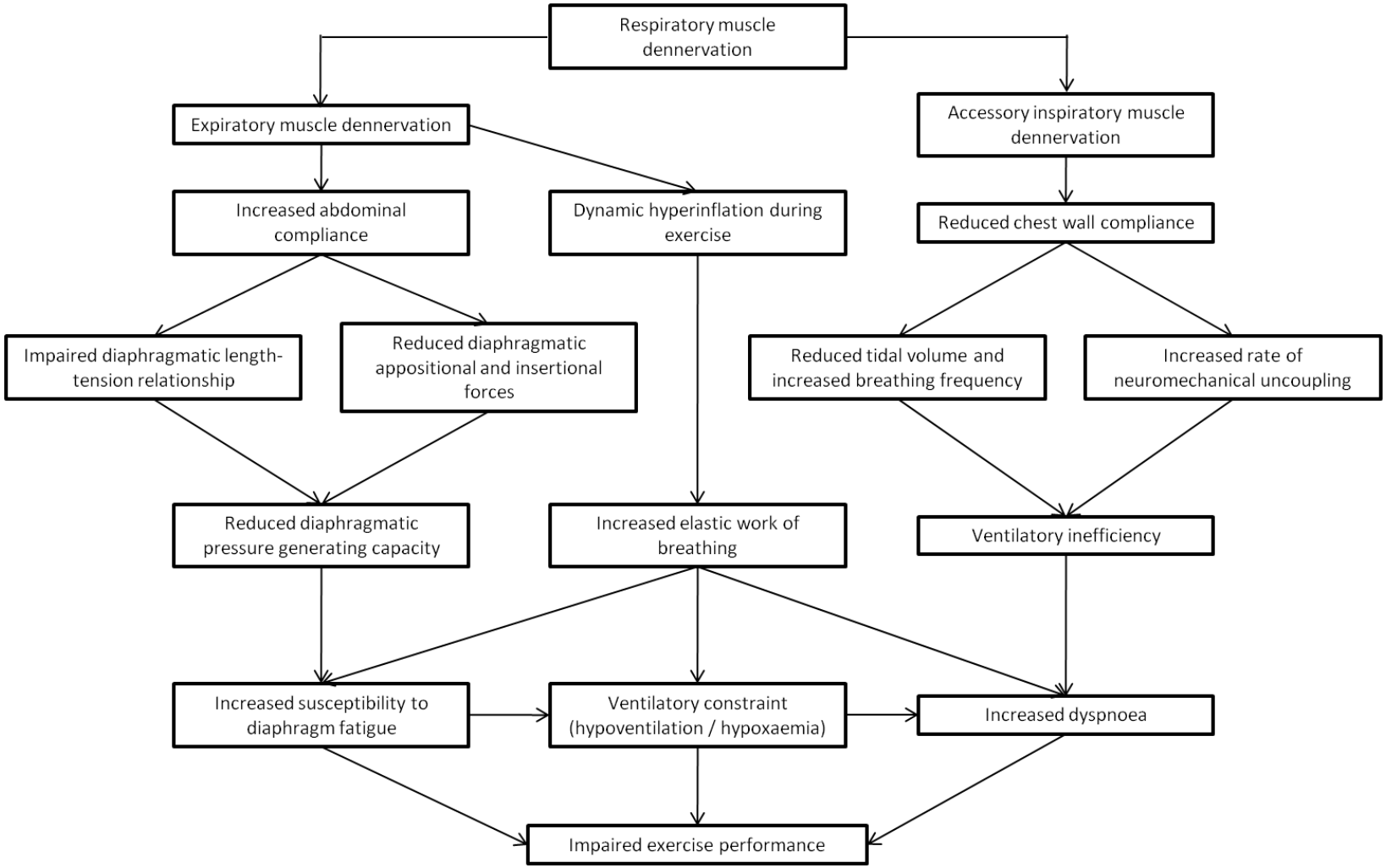

Fig 2-5 Respiratory consequences of cervical SCI and how they may contribute to impaired exercise performance.

2-3 Cardiovascular function

2-3.1 Introduction

Improvements in medical care between the 1970s and 1980s have increased life expectancy in individuals living with SCI, such that the leading cause of mortality in the chronic SCI population independent of lesion level is cardiovascular disease (CVD) (Hartkopp *et al.*, 1997). In the AB population, physical inactivity is one of the major modifiable risk factors for the development of CVD (Press *et al.*, 2003). Given the chronic physical deconditioning of patients with SCI, it is perhaps unsurprising that there is both an early onset of, and increased prevalence of CVD in SCI (DeVivo *et al.*, 1999; Yekutiel *et al.*, 1989). Furthermore, injury to the cervical spinal cord disrupts sympathetic autonomic pathways, predisposing these individuals to a range of clinical complications such as hypotension and autonomic dysreflexia (Krassioukov, 2009). Together, these cardiovascular consequences of cervical SCI lead to profound alterations in cardiac and vascular function, and a reduced exercise capacity (Haisma *et al.*, 2006). Due to the integral role of the autonomic nervous system in the cardiovascular system, this section will first provide an overview of the autonomic nervous system, and then will consider cardiac and vascular function following cervical SCI. This review will conclude by examining the pathophysiology of cardiovascular control during exercise in individuals with cervical SCI.

2-3.2 Overview of the autonomic nervous system

The autonomic nervous system is divided into the sympathetic and parasympathetic nervous system. The sympathetic nervous system is considered 'stimulatory', whilst the parasympathetic nervous system is 'suppressive'; by working together these systems provide balanced autonomic control. Both divisions of the autonomic nervous system innervate the majority of visceral organs, including the bronchopulmonary tree and the heart (Krassioukov, 2009). Despite functional differences, the organisation of the two systems is similar. For example, both divisions

of the autonomic nervous system have two neuronal populations. The first neuronal population is the preganglionic neuron, with the cell body in the gray matter of the brain or spinal cord. Axons of the preganglionic neurones travel in the anterior roots of the spinal cord and synapse on the second group of neurones, the postganglionic neurones, that are located in the autonomic ganglia in the peripheral nervous system (Alexander *et al.*, 2009). The axons of the postganglionic neurones then synapse with the target organs.

Preganglionic neurones of the sympathetic nervous system are located in the spinal gray matter in the thoracic (T1-T12) and upper lumbar segments of the spine (L1-L2). Axons of the sympathetic preganglionic neurones exit through the anterior roots of the spinal cord and synapse onto postganglionic sympathetic neurones in the sympathetic chain ganglia and prevertebral ganglia (celiac, superior and inferior mesenteric ganglia). Sympathetic postganglionic fibres are mostly adrenergic and release noradrenaline, with the exception of sympathetic fibres innervating sweat glands, which are cholinergic and release acetylcholine. The sympathetic innervation of various body structures is well described in medical textbooks, but in brief, the segmental innervations that are particularly important for understanding cardiovascular control in individuals with cervical SCI are the sympathetic innervation of the heart at T1-T4, the blood vessels of the upper limbs at T1-T4, and the blood vessels of the splanchnic bed and lower limbs at T6-L2 (see also Fig 2-6).

Parasympathetic neurones are located within four cranial nerves (CN III, VII, IX, X) of the brainstem and within the sacral spinal segments (S2-S4) (Alexander *et al.*, 2009; Krassioukov, 2009). In contrast to the sympathetic nervous system, there is no parasympathetic innervation of the peripheral vasculature. The heart and pulmonary tree, however, are under parasympathetic control through the vagus nerve (CN X; see also Fig 2-6), which exits the brainstem and synapses with the sino-atrial node and the nerve cells in the enteric nervous system. Hence, the upper portion of the gastrointestinal tract also receives parasympathetic innervation (Alexander *et al.*, 2009).

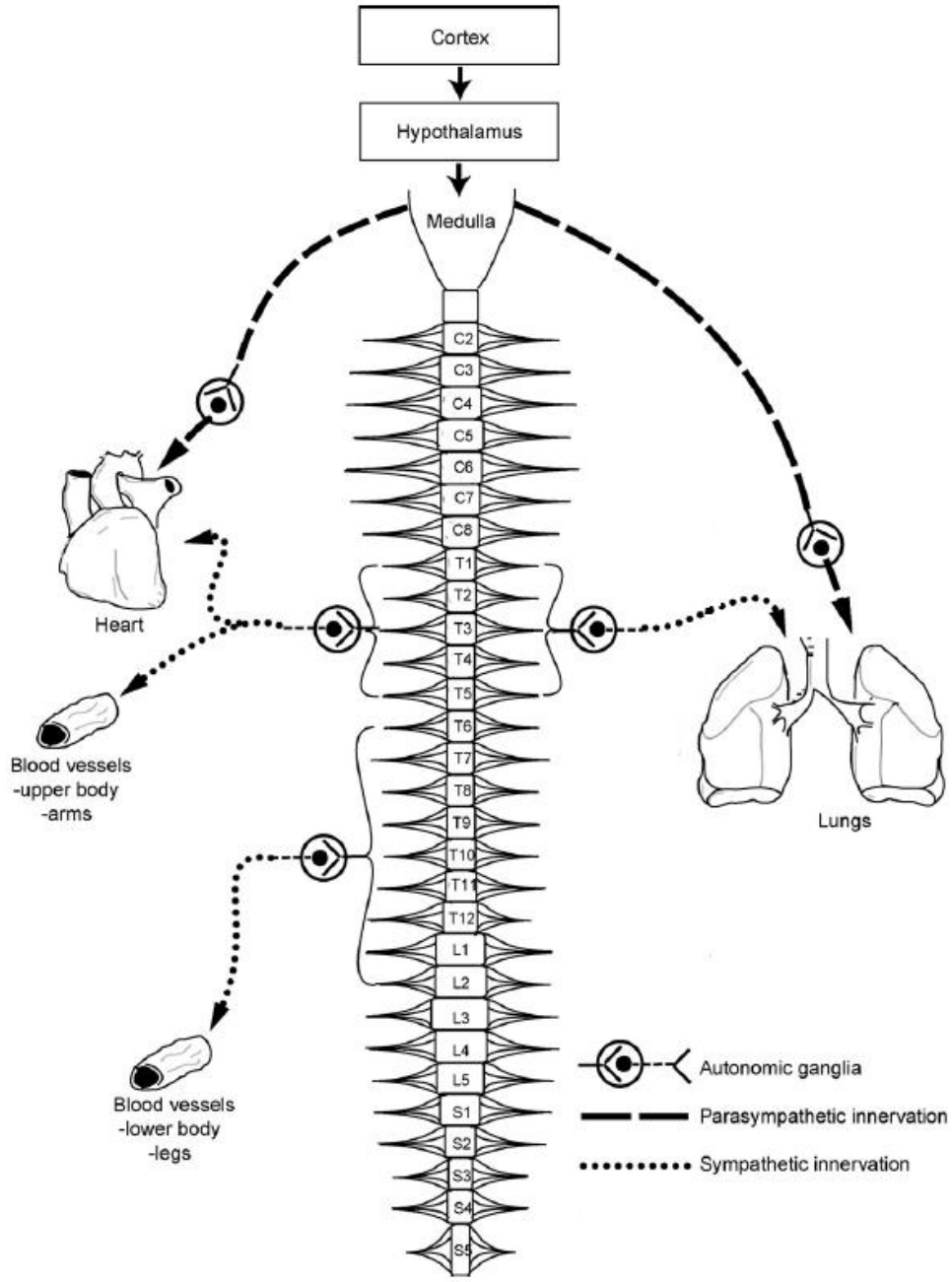

Fig 2-6 Sympathetic (dotted lines) and parasympathetic (dashed lines) pathways that are important for understanding cardiovascular control following cervical SCI.

Note that the parasympathetic preganglionic neurones exit the brainstem via the vagus nerve (CN X), whereas sympathetic preganglionic neurones exit the spinal cord through the ventral roots of spinal segments T1-L2. Adapted from Krassioukov (2009).

2-3.3 Clinical cardiovascular consequences of cervical spinal cord injury

Neurogenic and spinal shock

The acute period after cervical SCI is characterised by profound bradycardia and hypotension (see below), which are common components of neurogenic shock. Neurogenic shock is reported to last up to 5 wk (Piepmeier *et al.*, 1985; Winslow *et al.*, 1986), and pharmacologic intervention via vasopressive therapy is usually required to maintain arterial blood pressure during this period (Hadley, 2002; Piepmeier *et al.*, 1985). In addition to neurogenic shock, individuals with cervical SCI also experience spinal shock. Spinal shock is characterised by a marked reduction in tendon reflex function caudal to the site of injury and typically lasts 4 to 6 wk (Ditunno *et al.*, 2004). Individuals are considered to have recovered from spinal shock when tendon reflex function or bladder voiding reflex function return.

Hypotension

Hypotension is highly prevalent in acute SCI, and is directly related to the level of lesion (Lehmann *et al.*, 1987; Teasell *et al.*, 2000), whereby hypotension is more common in cervical versus thoracic SCI, most likely due to the sympathetic dennervation of the large blood vessels in the splanchnic bed (Teasell *et al.*, 2000). In addition to resting hypotension, most individuals with cervical SCI exhibit evidence of orthostatic hypotension when transferring from supine to seated position (Grigorean *et al.*, 2009; Krassioukov *et al.*, 2009; Lehmann *et al.*, 1987; Mathias and Frankel, 1983; Teasell *et al.*, 2000). Orthostatic hypotension is defined by the American Autonomic Society and the American Academy of Neurology (1996) as a decrease in systolic blood pressure of 20 mmHg, or diastolic pressure of 10 mmHg on assumption of an upright posture. In the AB population, it is well known that on assumption of the upright posture there is a baroreflex-mediated vasoconstriction via an increase in sympathetic outflow to maintain blood pressure and cerebral perfusion (Brown and Hainsworth, 2000; Bush *et al.*, 2000). In light of this, it is likely that both sympathetic dennervation and altered baroreceptor sensitivity are the primary causes of

orthostatic hypotension following cervical SCI (Munakata *et al.*, 2001; Wallin and Stjernberg, 1984). However, a lack of skeletal muscle pump (Faghri and Yount, 2002), cardiovascular deconditioning (Vaziri, 2003), and/or altered salt and water balance (Frisbie, 2004) have also been hypothesised to contribute to hypotension.

Autonomic dysreflexia

Patients with cervical SCI regularly experience life threatening periodic bouts of extreme hypertension (up to a systolic blood pressure of 300 mmHg) accompanied by pronounced bradycardia, a disorder known as autonomic dysreflexia (AD). Autonomic dysreflexia is most common in individuals with a lesion above T6 (Krassioukov, 2004), which corresponds to the neural innervation of the major splanchnic sympathetic outflow (see also Fig 2-6). During AD, noxious or non-noxious stimuli, such as bowel or bladder distension, cause exaggerated activity in the spinal circuits caudal to the injury which projects to the sympathetic pre-ganglionic neurones. In turn, this triggers a sympathetically-mediated vasoconstriction in the muscle, skin and vascular beds causing extreme hypertension below the lesion (Krassioukov *et al.*, 2006; Teasell *et al.*, 2000). Above the lesion, a parasympathetic induced bradycardia occurs via a baroreflex-mediated response to the hypertension in the lower body (Teasell *et al.*, 2000). Although there is still no consensus on the exact pathophysiology explaining AD, it is generally accepted that loss of supraspinal input to the spinal sympathetic circuits, reduced overall sympathetic activity, disruption of spinal reflexes, and plastic changes in the spinal cord and peripheral autonomic circuits all contribute to the development of AD (Krassioukov, 2009).

2-3.4 Cardiac structure and function

Individuals with cervical SCI suffer severe physical deconditioning due to prolonged bed rest and a wheelchair bound lifestyle. Chronic cervical SCI results in cardiac atrophy, whereby a 25% reduction in left ventricular (LV) mass has been reported (de Groot *et al.*, 2006; Eysmann *et al.*,

1995; Kessler *et al.*, 1986). Relatively few studies have examined cardiac structure and function in individuals with cervical SCI. Kessler *et al.* (1986) found evidence of impaired systolic function in individuals with cervical SCI compared to AB controls, as evidenced by a reduced LV mass (LVM), left atrial (LA) dimensions, and cardiac output ($\dot{Q}$). These findings were extended by Eysmann *et al.* (1995) who assessed diastolic function in young and old individuals with cervical SCI and compared them with age-matched AB controls. Using pulsed wave Doppler to measure LV filling velocities, the authors reported no difference in early or late filling velocities between SCI and AB controls, suggesting individuals with cervical SCI have 'normal' diastolic function. In the only study to assess both systolic and diastolic function in seven individuals with cervical SCI, de Groot *et al.* (2006) confirmed the findings of both previous studies by reporting impaired systolic, but normal diastolic function. In particular, the authors reported reduced dimensions of the LV, LA and inferior vena cava, and reduced LVM in individuals with cervical SCI compared with AB controls. However, both pulsed wave Doppler and tissue Doppler imaging (TDI) revealed no differences in diastolic filling velocities or diastolic mitral annular velocities (de Groot *et al.*, 2006).

The impaired systolic function in cervical SCI is likely caused by a reduction in the pressure and volume load imposed on the heart due to hypotension (Teasell *et al.*, 2000) and a reduction in blood volume (Houtman *et al.*, 2000). For AB individuals, the venous muscle pump, respiratory muscle pump and a large venous pressure gradient promote efficient venous return. In individuals with cervical SCI, however, the venous muscle pump in the lower limbs is abolished, the respiratory muscle pump is likely to be impaired, and the venous pressure gradient between the lower limbs and left atrium is likely to be reduced due to a large increase in abdominal compliance. Consequently, venous return and cardiac preload are likely to be reduced, ultimately resulting in a reduction in end-diastolic volume (EDV), stroke volume (SV) and $\dot{Q}$. In agreement with this postulate, de Groot *et al.* (2006) reported a trend towards a reduction in EDV, SV and $\dot{Q}$ in individuals with cervical SCI compared with AB controls; however, a small sample size resulted in low statistical power and therefore statistical significance was not reached. The similar

diastolic function between individuals with cervical SCI and AB individuals is surprising. In AB individuals who are subjected to prolonged inactivity via bed rest, there is a reduction in both systolic and diastolic function (Dorfman *et al.*, 2008; Perhonen *et al.*, 2001). The studies that documented normal diastolic function in SCI were conducted on individuals with chronic cervical SCI, suggesting there may be a chronic adaptation that serves to normalise diastolic function. However, longitudinal studies that track changes in diastolic function from acute to chronic SCI are required to confirm this hypothesis.

2-3.5 Vascular function

Aside from blood pressure, only two studies have examined vascular function in individuals with cervical SCI. Using Doppler, Nash *et al.* (1996) reported that both trained and untrained individuals with cervical SCI exhibited a lower arterial inflow to the common femoral artery and a smaller cross sectional area of the common femoral artery compared to untrained AB controls. However, blood pressure was not measured in that study, so vascular resistance in the legs could not be calculated. Leg vascular resistance has been measured in individuals with cervical SCI using venous-occlusion plethysmography (Casiglia *et al.*, 1999). The authors reported that over a 24 h period leg vascular resistance was consistently lower in individuals with cervical SCI compared to AB controls. Together, these studies suggest a reduction in blood flow and vascular resistance below the lesion in individuals with cervical SCI, due most likely to a smaller oxygen requirement of the lower limbs and the loss of sympathetic innervation to the arterial walls.

2-3.6 The cardiovascular system during exercise

During exercise in healthy AB individuals, heart rate (HR) increases linearly with work rate up to a maximum of 180-220 bpm. The initial exercise-induced tachycardia (up to ~ 110 bpm) is due to vagal inhibition of the cardiac pacemaker (Fagraeus and Linnarsson, 1976), after which further

increases in HR are due to an augmented sympathetic activity. The sympathetic nervous system acts to increase HR during exercise in two ways; first, by a direct stimulatory effect on the sino-atrial node and ventricular muscle from sympathetic fibres exiting the spinal cord between T1 and T4 (see also Fig 2-7); and second, from the stimulatory effect of noradrenaline and adrenaline on β_1-adrenoreceptors, which increases heart rate (cardiac chronotropy) and the force of myocardial contraction (cardiac inotropy). During exercise in cervical SCI, HR rarely exceeds 120 bpm (Coutts *et al.*, 1983; Figoni, 1993; Gass *et al.*, 1980; Goosey-Tolfrey *et al.*, 2006; Hopman *et al.*, 1998a; Lasko-McCarthey and Davis, 1991; Lewis *et al.*, 2007; Schmid *et al.*, 1998; Takahashi *et al.*, 2007; Wicks *et al.*, 1983). The reduction in maximal heart rate is due to a lack of sympathetic innervation to the myocardium (innervated at T1-T4) and a reduction in circulating adrenaline (Mathias *et al.*, 1975; Schmid *et al.*, 1998). There is some evidence of an exercise-induced increase in noradrenaline in individuals with cervical SCI (Dela *et al.*, 2003; Schmid *et al.*, 1998), likely due to spillover into the systemic circulation from post-ganglionic sympathetic nerve endings. However, the increase in noradrenaline during exercise in cervical SCI is less than in low thoracic SCI, and much less than in AB individuals (Dela *et al.*, 2003; Schmid *et al.*, 1998). Thus, the reduced maximum HR in cervical SCI reflects a reliance on vagal inhibition of the sino-atrial node, and a small degree of circulating catecholamines.

In AB individuals at rest, a large proportion of the resting blood volume is stored below the heart, mainly in the large venous beds of the splanchnic region (Rowell, 1973; Rowell *et al.*, 1965). During exercise in AB individuals, a sympathetically-mediated redistribution of blood volume occurs from the splanchnic bed to the periphery to supply the working muscle with oxygen (Rowell, 1973). In individuals with cervical SCI, elevated abdominal compliance and sympathetic dennervation may result in a greater degree of venous pooling in the splanchnic bed, and an impaired redistribution of this blood volume during exercise. Only two studies have investigated this hypothesis. Thijssen *et al.* (2009) used ultrasound to compare portal vein flow in response to arm exercise in individuals with high (above T6) and low (below T7) SCI compared to

AB controls. Individuals with high SCI showed no change in portal vein flow in response to exercise, suggesting an inability to redistribute blood from the splanchnic bed to the working muscles. The redistribution of blood during exercise has also been assessed by Dela *et al.* (2003), who measured blood flow to the legs of tetraplegics, paraplegics and AB controls during electrically-induced cycling. They found an exercise-induced increase in flow to the legs in all groups; however, this was least pronounced in the tetraplegic group, which presumably reflects an inability to mobilise the splanchnic blood reservoir (Dela *et al.*, 2003).

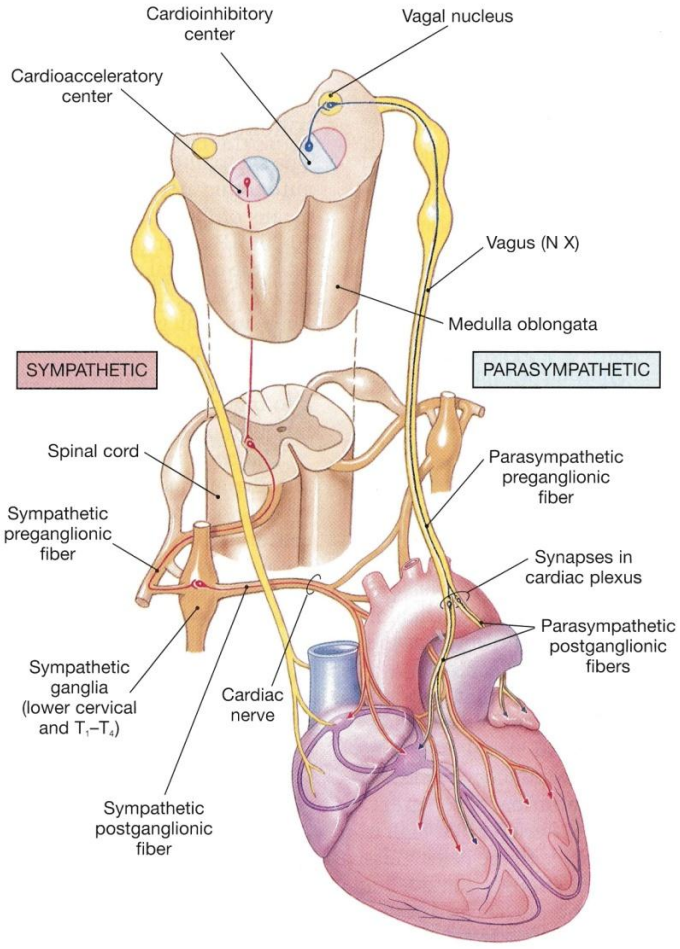

Fig 2-7 Summary of the autonomic innervation of the heart.
Note the heart is under control from both the parasympathetic (vagus nerve; CN X) and sympathetic (post-ganglionic sympathetic neurones; T1-T4) nervous systems. Reproduced from Martini (2001).

2-3.7 Thermoregulation

Individuals with SCI exhibit a lesion-dependent impairment in thermoregulatory function, whereby the degree of thermoregulatory impairment increases as the level of SCI moves cranially (Guttmann *et al.*, 1958). Consequently, tetraplegic individuals demonstrate an increased core temperature and a reduced sweating rate in response to heat exposure at rest compared to paraplegics or AB individuals (Guttmann *et al.*, 1958; Petrofsky, 1992). The primary mechanisms underpinning these differences are a loss of afferent feedback to the thermoregulatory centre in the hypothalamus and a loss of vasomotor control below the level of injury in cervical SCI (Price, 2006).

The majority of studies that have investigated core temperature during exercise in the SCI population have concentrated on paraplegic athletes and are reviewed elsewhere (Price, 2006). In the first study to investigate changes in core body temperature in tetraplegic athletes, Gass *et al.* (1992) reported that the largest individual increase in core body (rectal) temperature during a simulated 5 km time-trial on a wheelchair treadmill in normothermic conditions (23 deg) was 0.5 deg. Similarly, Ready *et al.* (1994) reported only small increases in both core (rectal) and skin (multiple sites) temperature in response to sub-maximal (75% peak) arm-crank exercise in normothermic conditions (17-21 deg). These small increases in core body temperature suggest that either the small active muscle mass is such that only minimal metabolic heat is produced during exercise, or that rectal temperature is not an accurate measure of core body temperature in the SCI population. In this regard, greater increases in core body temperature have been noted with oesophageal versus rectal temperature during treadmill propulsion in paraplegic athletes (Gass *et al.*, 1988). Thus, it is still unclear whether hyperthermia during exercise in normothermic conditions is a concern for tetraplegic athletes.

In the only two studies to investigate changes in core body temperature during recovery from exercise in normothermic conditions, it was reported that core body temperature increased during the first 2-5 min post exercise in tetraplegic individuals; whereas both paraplegic and AB individuals demonstrated a reduction in core body temperature following exercise (Gass *et al.*, 1992; Price and Campbell, 1997). This lesion-level dependent disparity in findings suggests the

possibility that convective cooling currents from the limb movements during exercise may be a mechanism by which individuals with cervical SCI are able to dissipate heat during exercise(Price and Campbell, 1997).

Compared to the responses in normothermic conditions, during prolonged (>40 min) exercise in the heat (>31 deg, 43% humidity), individuals with cervical SCI exhibit an increase in core body (aural) temperature that exceeds that of paraplegic and AB individuals (Price and Campbell, 2003; Webborn *et al.*, 2005). The finding of an accentuated exercise-induced increase in core-body temperature in tetraplegic athletes has led some researchers to investigate the potential use of cooling strategies. Hand (Goosey-Tolfrey *et al.*, 2008), foot (Hagobian *et al.*, 2004) and ice-vest (Webborn *et al.*, 2005) cooling have all been shown to significantly reduce core-body temperature in tetraplegic athletes during exercise in the heat (>32 deg). Despite these findings, only two studies have investigated the potential ergogenic benefit of various cooling techniques in tetraplegic athletes: One study reported that pre- and during-exercise cooling via an ice-vest increased time-to-exhaustion during arm-crank exercise by 31 and 45%, respectively (Webborn *et al.*, 2008); whereas the other study reported that hand cooling did not improve simulated 1 km time-trial performance (Goosey-Tolfrey *et al.*, 2008). In conclusion, the findings to date suggest that hyperthermia contributes to exercise limitation in tetraplegic athletes, especially when exercising in the heat; however, further studies are required to confirm this postulate.

2-3.8 Summary

Cervical SCI results in damage to the descending sympathetic pathways between the lower brainstem and sympathetic preganglionic neurones. This disruption in autonomic cardiovascular control leads to disordered blood pressure control, predisposing individuals with cervical SCI to life threatening episodes of autonomic dysreflexia. Disrupted autonomic control also leads to impaired vascular function, which combined with a lack of physical activity, predisposes individuals with cervical SCI to an early onset of cardiovascular disease. Autonomic dysfunction also results in venous pooling and impaired venous return, leading to a reduction in left ventricular filling and a subsequent reduction in stroke volume and $\dot{Q}$. Chronically, these reductions in pressure and volume loading of the heart cause cardiac atrophy. Alterations in autonomic function also limit the maximal achievable heart rate during exercise, which combined with a reduction in stroke volume and $\dot{Q}$ significantly reduces exercise capacity. A summary of the cardiovascular consequences of SCI and how they may contribute to exercise limitation in cervical SCI is provided in Fig 2-8.

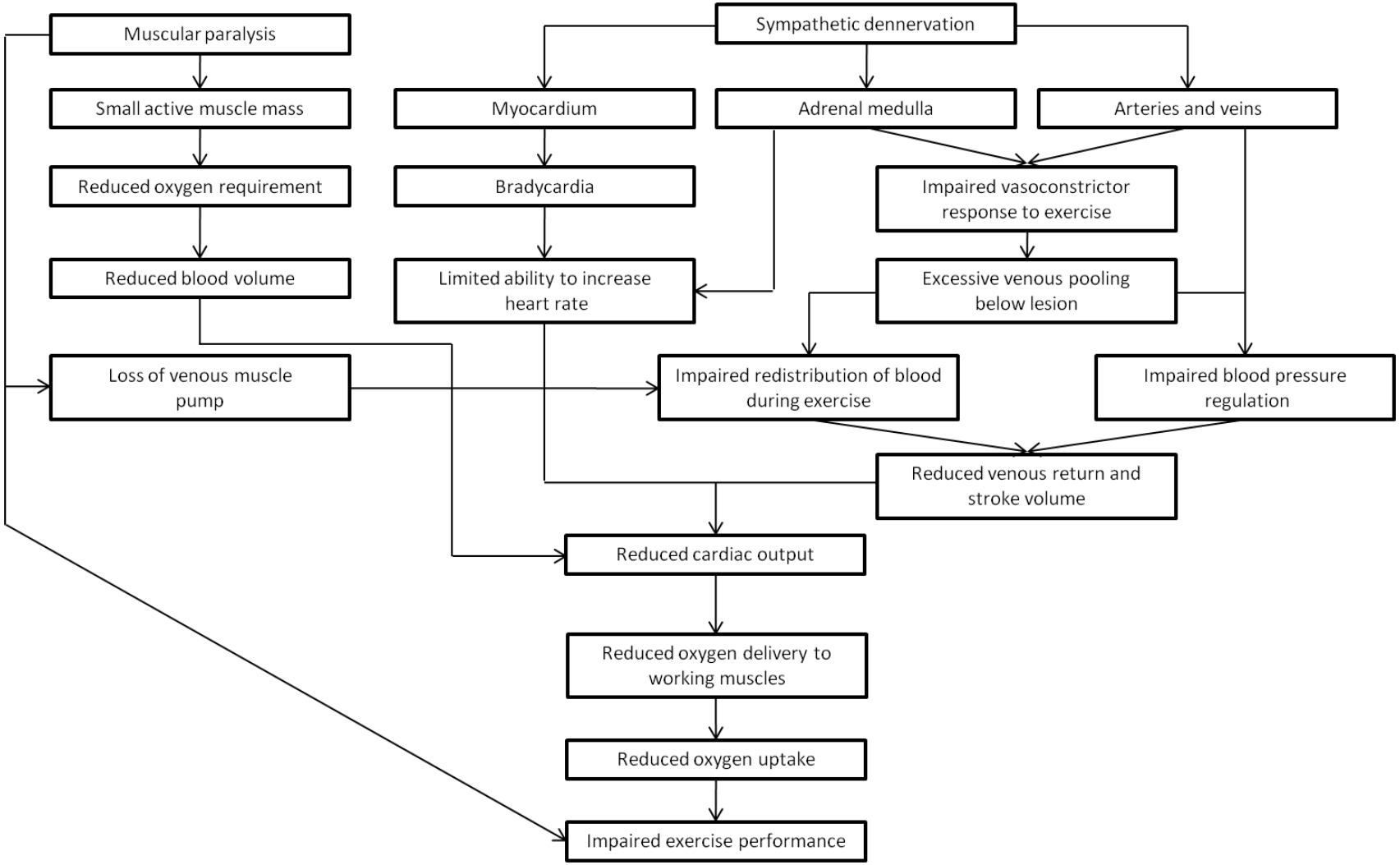

Fig 2-8 Cardiovascular consequences of cervical SCI and how they may contribute to impaired exercise performance.

2-4 Peak metabolic responses to exercise

Since the introduction of competitive sport for disabled war veterans in 1948 by Sir Ludwig Guttman, there has been a growing interest in the exercise responses of individuals with SCI. In 1988, the first data were published comparing trained and untrained individuals with cervical and thoracic SCI (Eriksson *et al.*, 1988). The authors reported that trained individuals with cervical SCI had a 22% higher peak oxygen uptake ($\dot{V}O_{2peak}$) than their untrained counterparts. This difference in $\dot{V}O_{2peak}$ increased as the SCI moved caudally, such that trained individuals with a thoracic SCI had a 38% higher $\dot{V}O_{2peak}$ than untrained individuals with thoracic SCI (Eriksson *et al.*, 1988). A higher $\dot{V}O_{2peak}$ in trained versus untrained individuals with cervical SCI has been substantiated by Bhambhani *et al.* (1995). In light of these findings and due to subsequent investigations in this thesis focusing on highly trained athletes with cervical SCI, a review of the exercise capacity of untrained individuals with cervical SCI, and those with thoracic SCI, is beyond the scope of this literature review. Reviews of the physical capacity of untrained individuals with SCI have been published elsewhere (Davis, 1993; Figoni, 1993; Haisma *et al.*, 2006), and normative values have been documented (Janssen *et al.*, 2002). For comparative purposes, Fig 2-9 shows data on the weighted mean $\dot{V}O_{2peak}$ for arm-crank ergometry (ACE) and wheelchair ergometry (WCE) in untrained individuals with thoracic and cervical SCI (Haisma *et al.*, 2006).

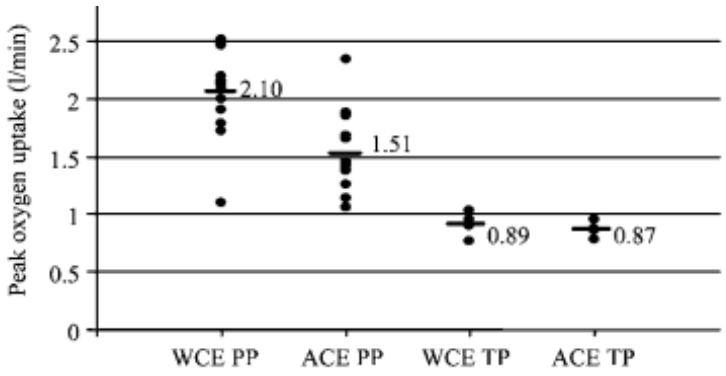

Fig 2-9 Summary of multiple studies (filled circles) investigating peak oxygen uptake in untrained individuals with SCI.
Values represent weighted mean peak oxygen uptake during wheelchair ergometry (WCE) and arm-crank ergometry (ACE) in patients with paraplegia (PP) and tetraplegia (TP). Reproduced from Haisma et al. (2006).

There are four modalities by which highly-trained individuals with cervical SCI have been assessed for exercise capacity; ACE, wheelchair propulsion on a treadmill (here on in referred to as wheelchair propulsion), WCE, and field-based exercise testing. In one of the largest studies to date in the highly-trained cervical SCI population, 13 athletes with a lesion between C6 and C7 from a range of sports were assessed for $\dot{V}O_2$ and $\dot{V}_E$ during ACE and wheelchair propulsion (Wicks *et al.*, 1983). The authors reported that the group mean $\dot{V}O_{2peak}$ during both ACE and wheelchair propulsion did not exceed 1 $L \cdot min^{-1}$ (15 $ml \cdot kg^{-1} \cdot min^{-1}$), and $\dot{V}_E$ did not exceed ~50 $L \cdot min^{-1}$ during either exercise modality. Slightly higher values for $\dot{V}O_{2peak}$ (1.08 – 1.14 $L \cdot min^{-1}$) during ACE and wheelchair propulsion have been published for trained wheelchair racers with cervical SCI (Lakomy *et al.*, 1987; Ready, 1994). To the author's knowledge the highest $\dot{V}O_{2peak}$ reported for individuals with cervical SCI was in a relatively large group (n = 8) of highly-trained competitive wheelchair marathon racers with cervical SCI (Bhambhani *et al.*, 1995). That study reported an average $\dot{V}O_{2peak}$ of 1.43 $L \cdot min^{-1}$ during maximal incremental WCE, which is substantially higher than that reported previously, and most likely reflects the superior endurance training undertaken by the marathon racers in that study.

Since 1992, when the sport of wheelchair rugby was officially recognised by the International Stoke Mandeville Wheelchair Sports Federation, research investigating highly-trained athletes with cervical SCI has focused primarily on the physical capabilities of trained wheelchair rugby players. The first study to assess wheelchair rugby players (n = 13) during exercise was conducted by Abel *et al.* (2003) who assessed $\dot{V}O_2$, HR and blood lactate during ACE, and HR and blood lactate during WCE and a field based exercise test. The authors reported a $\dot{V}O_{2peak}$ of ~1 $L \cdot min^{-1}$, a peak HR of 124 bpm and a peak blood lactate of 4.07 $mmol \cdot L^{-1}$ during ACE. HR was reported to be higher during WCE (133 bpm) and higher still during the field test (141 bpm). The field test also elicited a higher blood lactate (5.63 $mmol \cdot L^{-1}$) than ACE and WCE. However, no details were given regarding the protocol used for any of the three assessments; hence, these results are difficult to interpret. More recently, $\dot{V}O_{2peak}$ during ACE has been reported

to average 1 L·min^{-1}, and HR to average ~130 bpm in highly-trained athletes with cervical SCI who compete in wheelchair rugby or wheelchair tennis (Goosey-Tolfrey *et al.*, 2006). In 7 elite (Paralympic) wheelchair rugby players, Taylor *et al.* (2010) reported a $\dot{V}O_{2peak}$ during ACE of ~1.2 L·min^{-1} which is approximately 20% higher than that reported previously, but is still substantially lower than that reported in highly-trained wheelchair racers (Bhambhani *et al.*, 1995). Thus, marathon wheelchair racers appear to have superior aerobic fitness compared to court based athletes with cervical SCI. It has also been reported that wheelchair marathon racers have a high ventilatory threshold (Bhambhani *et al.*, 1995), suggesting an improved ability to work at a higher proportion of their $\dot{V}O_{2peak}$. The superior responses of the wheelchair racers with cervical SCI compared to court based athletes is likely due to the volume and nature of their training, but may also be related to a different posture during wheelchair racing that might facilitate venous return, thus allowing wheelchair racers to reach higher levels of $\dot{Q}$ and $\dot{V}O_2$.

In summary, the current literature suggests that exercise training is able to improve aerobic exercise capacity. However, a limitation with cross sectional research is that it cannot rule out the possibility that these individuals compete in wheelchair sport because of their enhanced exercise capacity. In the only study to assess the change in $\dot{V}O_2$ in response to an exercise training intervention, Gass *et al.* (1980) reported a 34% increase in $\dot{V}O_{2peak}$ and exercise time after 7-wk of upper-body exercise training in individuals with high level SCI. Thus, it is likely that the enhanced aerobic exercise capacity in highly-trained individuals with cervical SCI is due to the training undertaken, rather than a consequence of selection bias. Although the data presented in this review provides evidence that $\dot{V}O_{2peak}$ is higher in trained versus untrained individuals with cervical SCI, $\dot{V}O_{2peak}$ is still significantly lower compared to AB individuals (e.g., Eriksson *et al.*, 1988), and may be lower than that required to prevent the onset of CVD. This has lead to a number of investigators studying whether acute interventions such as electrical stimulation of the lower limbs, lower body positive pressure, compression stockings, or abdominal

binding can improve exercise capacity in the cervical SCI population. This will be the focus of the next section of this literature review.

2-5 Acute methods to improve cardiorespiratory function

2-5.1 Electrical stimulation

The most widely studied method to improve cardiorespiratory function during exercise in the cervical SCI population is lower-body functional electrical stimulation (FES). FES uses bursts of electrical pulses applied over motor points to elicit action potentials that propagate along axons to the target muscle. To achieve muscle contraction the FES system must be capable of producing at least 20 action potentials per second, otherwise the muscle will just twitch (Popovic *et al.*, 2001).

FES stimulation was first made possible from equipment that was developed as an offshoot of the cardiac pacemaker. To the author's knowledge, the first reported use of FES in the SCI population was by Kantrowitz (1960), who stimulated the quadriceps and gluteal muscles of a T3 paraplegic to allow a standing posture for a few minutes. A number of studies have further investigated the use of FES as a method for ambulation in the SCI population, a review of the literature pertaining to these studies is provided elsewhere (Glaser, 1985). FES stimulation has been successfully applied to the calf and thigh muscles of paraplegic individuals at rest, and increases in stroke volume and $\dot{Q}$ have been reported (Glaser *et al.*, 1987). Figoni and colleagues (1990) studied the cardiovascular adjustments to passive and FES-induced leg cycling in 17 individuals with cervical SCI and reported an increase in $\dot{V}O_{2peak}$, $\dot{Q}$, a-$\bar{v}$ O_2 difference, HR and $\dot{V}_E$ during FES-induced cycling compared to passive cycling. However, the group mean value obtained for $\dot{V}O_{2peak}$ was 0.72 L·min^{-1} which is substantially lower than that achieved during ACE (see section 2-4). In agreement with Figoni *et al.* (1990) a number of other studies have reported similar values for $\dot{V}O_2$ during FES cycle exercise in individuals with cervical SCI (Hjeltnes *et al.*,

1997; Hooker *et al.*, 1992; Pollack *et al.*, 1989). One study, however, reported values in excess of 1.4 $L \cdot min^{-1}$ during FES cycle exercise in individuals with cervical SCI (Dela *et al.*, 2003). That study also demonstrated a plateau in stroke volume and $\dot{Q}$ during FES leg cycling in cervical SCI, suggesting a central limitation on oxygen delivery. Two of the aforementioned FES cycling studies included a FES cycle intervention and reported improved $\dot{V}O_2$ following FES cycle training (Hooker *et al.*, 1992; Pollack *et al.*, 1989). However, even the improved $\dot{V}O_2$ was still lower than that typically reported during ACE. To date, the literature suggests that whilst FES-induced leg exercise may aid venous return and stroke volume, other exercise modalities may be more beneficial to increase cardiovascular fitness in cervical SCI.

Lower body FES can be combined with upper body exercise (referred to as hybrid exercise) to increase the size of the active muscle mass. To the author's knowledge only one study has assessed cardiovascular responses to hybrid exercise in individuals with cervical SCI (Hooker *et al.*, 1992). The authors reported a marked increase in stroke volume, $\dot{Q}$ and $\dot{V}O_2$ during hybrid exercise compared to arm or leg exercise alone (Hooker *et al.*, 1992). In comparison to the lack of data in individuals with cervical SCI, a number of studies report beneficial haemodynamic effects in hybrid compared with either arm or leg exercise alone in paraplegics [e.g. (Davis *et al.*, 1990; Mutton *et al.*, 1997; Verellen *et al.*, 2007)].

Despite the potential haemodynamic benefits associated with FES (and hybrid FES), the potential use of FES as a training aid has received mixed reviews. FES training has been shown to increase oxidative enzymes, and to beneficially alter muscle fibre type from type II to type I (Harridge *et al.*, 2002; Peckham *et al.*, 1973). However, long term FES use may cause burns to the skin from the high level of current required for transcutaneous stimulation (Gruner *et al.*, 1983), and may lead to unwanted spinal cord reflex activity as both afferent and efferent nerves can respond to the stimulation (Glaser, 1985). There are also practical issues to consider, such as the time required placing the electrodes during each training session, and skin irritation from repeated electrode placement. Furthermore, for individuals who compete regularly in wheelchair

sport, transcutaneous FES stimulation would be impossible to conduct during competition, and is likely to increase muscle mass in the legs. In turn this may reduce the power-weight ratio, which is likely to be an important determinant of exercise performance in cervical SCI.

2-5.2 Lower body positive pressure

Lower body positive pressure (LBPP), via anti-gravity (anti-G) suits or compression stockings have been shown to provide beneficial haemodynamic alterations at rest and during exercise in SCI. An anti-G suit applies pulsating pressure to the lower body and abdomen via five inflatable bladders (one on each calf, one on each thigh and one on the abdomen), whilst compression stockings apply positive pressure to the legs only. LBPP is most commonly used in the cervical SCI population as a non-pharmacological tool to improve mean arterial pressure (MAP) and prevent orthostatic hypotension (Gillis *et al.*, 2008). The first study to show that LBPP increases MAP was conducted by Valbona *et al.* (1963). Those authors reported an increase in MAP with an anti-G suit during 60° head up tilt in 12 individuals with cervical SCI. An increase in MAP during 20 and 45° head up tilt has also been reported in 27 acutely injured (47 days post injury) tetraplegics with and without pneumatic leg splints inflated at 65 mmHg (Huang *et al.*, 1983).

Two studies have investigated the use of LBPP as a method to improve exercise haemodynamics in SCI. In individuals with a low thoracic SCI (T6-T12) undertaking ACE with and without an anti-G suit, HR was significantly lower at 40 and 60% of peak power with the anti-G suit, whilst $\dot{V}O_2$ and $\dot{Q}$ were not statistically different between conditions, suggesting an enhanced stroke volume with an anti-G suit (Hopman *et al.*, 1992). That study also included an AB control group and found no difference in the $\dot{Q}$ or $\dot{V}O_2$ response to sub-maximal exercise between the SCI and AB groups. This suggests that unlike individuals with cervical SCI, individuals with low thoracic SCI appear to have a normal $\dot{Q}$ and $\dot{V}O_2$ response to exercise. It is, therefore, perhaps unsurprising that there was no increase in $\dot{Q}$ or $\dot{V}O_2$ during sub-maximal exercise with an anti-G suit in the SCI group. The effect of an anti-G suit on exercise haemodynamics has also been

studied in individuals with cervical SCI (Pitetti *et al.*, 1994). Those authors reported that LBPP increased stroke volume and $\dot{Q}$ during sub-maximal wheelchair propulsion, and increased $\dot{V}O_{2peak}$ by 16% during maximal ACE and wheelchair propulsion. The improvements noted by Pitetti *et al.* (1994) are much more pronounced than those reported by Hopman *et al.* (1992), despite similarities in the protocol and degree of pressure applied by the anti-G suit. It is likely that the greater loss of supraspinal cardiovascular control in individuals with cervical SCI explains the improvement in $\dot{V}O_{2peak}$ and $\dot{Q}$ in response to the anti-G suit.

2-5.3 Abdominal binding

The use of abdominal binding to improve resting haemodynamics in the acute period post injury dates back to as early as 1954 (Guttman, 1973). The first study to assess the effect of abdominal compression on resting cardiorespiratory function in individuals with cervical SCI was by Huang *et al.* (1983), who assessed the effect of an inflated abdominal cuff on $\dot{V}O_2$, BP and breathing pattern in the supine, head-up (20° and 45°) and head-down (20°) tilt positions. The authors reported that the inflated cuff increased $\dot{V}O_2$ in all positions, and attenuated the fall in systolic and diastolic BP during both head-up tilt conditions. However, that study was delimited to individuals with acute cervical SCI (47 days post injury) who may still be recovering from neurogenic shock sustained at the time of injury (see section 2-3.3). To the author's knowledge, no other studies have assessed the effect of an abdominal binder on BP at rest in individuals with cervical SCI.

The majority of studies that have investigated abdominal binding in individuals with cervical SCI have assessed the effect on resting pulmonary function. The first study to assess changes in pulmonary function with and without abdominal binding in individuals with cervical SCI was by Maloney *et al.* (1979). Those authors noted no significant changes in pulmonary function with binding. Conversely, a number of studies have since reported an increase in VC in the seated position with abdominal binding in cervical SCI (Boaventura *et al.*, 2003; Bodin *et al.*, 2005; Estenne *et al.*, 1998; Goldman *et al.*, 1986b; Hart *et al.*, 2005). The reason for the lack of increase

in VC in the study by Maloney *et al.* (1979) is unclear, but may be due to the rigid abdominal corset used. A rigid abdominal corset may overlie the lateral excursions of the lower rib cage and prevent rib cage expansion, thereby preventing any increase in VC.

Compared to VC, relatively few studies have assessed the effect of abdominal binding on absolute lung volumes. Those that have are generally in agreement that RV and FRC are reduced with binding (Bodin *et al.*, 2005; Estenne *et al.*, 1998; Hart *et al.*, 2005). The effect of abdominal binding on TLC remains controversial, with one study reporting an increase (McCool *et al.*, 1986), one study reporting a decrease (Bodin *et al.*, 2005) and two studies reporting no change (Estenne *et al.*, 1998; Hart *et al.*, 2005). The reason for this discrepancy in findings is unclear but may be related to an inadequate degree of abdominal compression, a large inter-individual variation in resting pulmonary function, the method used for measuring TLC, or the rest time between conditions. For example, in the only study to report an increase in TLC with abdominal binding, McCool *et al.* (1986) made measurements continuously such that TLC was first measured in the bound condition, after which the binder was released and TLC was immediately measured in the unbound condition. Thus, participants would not have had time to become accustomed to breathing without the binder before the unbound TLC measurements were made. There was also no mention of randomisation within the study design. It is possible, therefore, that inspiratory muscle fatigue may have occurred from repeated maximal inspirations in the bound condition, which may explain the subsequent reduction in TLC in the unbound condition. The effect of abdominal binding on pulmonary function in SCI was the focus of a recent meta-analysis (Wadsworth *et al.*, 2009). The authors reported that the mean weighted scores for all studies to date suggests that abdominal binding increases VC and RV, reduces FRC, whilst TLC remains relatively unchanged (see Fig 2-10). The improvements in VC with abdominal binding were attributed to improved chest wall mechanics and enhanced diaphragm performance. These two proposed mechanisms are now explained.

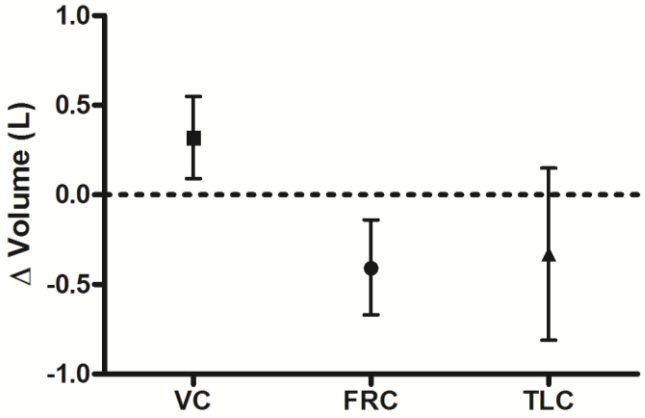

Fig 2-10 Summary of the meta-analysis conducted by Wadsworth *et al.* (2009).
Data shown represent weighted mean ± 95% confidence interval for differences (Δ) in vital capacity (VC; squares), functional residual capacity (FRC; circles) and total lung capacity (TLC; triangles) in bound versus unbound condition whilst seated.

The first study to assess the effect of abdominal compression on chest wall mechanics was conducted by Urmey *et al.* (1986). Using a large pneumatic cuff to apply external abdominal compression, upper and lower rib cage deformation was assessed via inductance pneumography. The authors reported that during tidal breathing with the cuff inflated, the area of the lower rib cage increased more per unit change in lung volume than it did with the cuff deflated. Also, abdominal compression abolished the paradoxical inwards motion of the upper rib cage during inspiration that was noted in the unbound condition. In the same year McCool *et al.* (1986) conducted a comprehensive assessment of upper and lower rib cage mechanics with and without abdominal binding in individuals with cervical SCI and AB controls. Chest wall mechanics were assessed using three pairs of magnetometers placed over the upper rib cage, lower rib cage and transversely in the anterior axillary line at the sixth intercostal space. Measurements were taken in the supine, seated and head-up tilt (37°) positions with and without an elasticised nylon abdominal binder placed below the lateral excursions of the lower rib cage. The authors reported that abdominal binding enhanced lower and upper rib cage dimensions in the seated and head-up tilt positions. The improvements in chest wall mechanics with abdominal binding in both studies were attributed to an enhanced expanding effect of the diaphragm through the zone of apposition.

The effect of abdominal binding on diaphragm function was first assessed in cervical SCI by Goldman *et al.* (1986b), who assessed sniff P_{di}, VC and $P_{I,max}$ with and without two types of abdominal binding (elastic and inelastic) in the seated, supine and 70° head up tilt positions. The authors reported that both types of abdominal binding increased VC in the seated and 70° head-up tilt, whereas only the elasticised binder increased sniff P_{di} in the seated and 70° head-up tilt position. The authors also reported that only the elasticised binder caused an increase in end-expiratory P_{ga}, suggesting that an improvement in diaphragm function may be dependent on an increase in end-expiratory P_{ga}. Thus, the results of that study confirm the previous suggestions of Urmey *et al.* (1986) and McCool *et al.* (1986) that abdominal binding does improve diaphragm function. The most comprehensive assessment of pulmonary function, respiratory muscle function and chest wall mechanics with and without abdominal binding in individuals with cervical SCI was provided by Hart *et al.* (2005). The authors reported that $P_{di,max}$, $P_{di,tw}$, and the pressure-time product of the diaphragm were significantly increased with abdominal binding, as was IC and VC; whereas FRC, abdominal compliance and perception of respiratory effort were all reduced with abdominal binding. The authors also noted an improved rib-cage contribution to tidal inspiration with abdominal binding. Together, these findings were attributed to an improved mechanical advantage of the diaphragm, and an optimisation of operating lung volumes. Whilst the findings of that study provide strong evidence that abdominal binding improves diaphragm function, the authors used cervical magnetic stimulation, which is not specific to the diaphragm as it also causes contraction of other muscles which may stabilise the rib cage during inspiration (Wragg *et al.*, 1994). Thus, although the evidence to date strongly suggests that an improvement in diaphragm function and chest wall mechanics is the mechanism by which pulmonary function is improved with abdominal binding in cervical SCI, further studies are needed to confirm this postulate.

Two studies have investigated the effect of abdominal binding on expiratory function and cough effectiveness. Estenne *et al.* (1998) assessed expiratory flow and oesophageal pressure

during a series of VC manoeuvres at different intensities of effort with and without abdominal binding. Isovolume-pressure flow curves were also created to assess whether abdominal binding improved the degree of dynamic airway compression, and therefore cough. The authors reported that abdominal binding resulted in small increases in flow and pressure throughout the VC manoeuvre, but a small sample size (n = 8) prevented statistically significant differences from being detected. The authors also concluded that abdominal binding did not improve dynamic airway compression and was therefore unlikely to improve cough in this population. More recently, Lin *et al.* (1998) assessed cough (by assessing PEF during a VC manoeuvre) in individuals with cervical SCI during three conditions: unassisted, with abdominal binding, and with abdominal binding and thoracic stimulation. The authors reported that abdominal binding alone had no significant impact on PEF during the VC; however, the combination of abdominal binding and thoracic stimulation improved PEF.

Compared to resting pulmonary function, there are limited data on the effect of abdominal binding on cardiac function and exercise capacity. In the only study to investigate the effect of abdominal binding on cardiac function, McCool *et al.* (1986) reported that right atrial size was increased with abdominal binding in 2 individuals with cervical SCI. Unfortunately, no data were provided regarding the extent of the improvement with binding, and the small sample size precluded the use of statistics to quantify any changes. Thus, further studies are required to investigate the effect of abdominal binding on cardiac function. The only study to investigate the effect of abdominal binding on peak exercise capacity in the SCI population was conducted in paraplegics (T3-T6) (Kerk *et al.*, 1995). That study found no effect of abdominal binding on $\dot{V}O_{2peak}$, HR or selected biomechanical indices. The authors had hypothesised that abdominal binding would improve exercise capacity by way of improving venous return, and redistributing blood from the abdomen to the arms, thereby aiding oxygen delivery to the working muscles. However, whilst high thoracic SCI is likely to compromise venous return and impair the redistribution of blood from the abdomen to the limbs during exercise, sympathetic innervation to the

myocardium would be partially (or fully) intact. Thus, $\dot{Q}$ and oxygen delivery could be augmented by increasing HR. It is, therefore, prudent to question the hypothesis of that study, as abdominal binding would only be expected to improve $\dot{V}O_{2peak}$ if it were limited during exercise. A summary of all of the studies that have investigated abdominal binding in SCI is provided in Table 2-1.

To understand the limiting factors to exercise performance in cervical SCI, cardiovascular function during exercise was assessed in response to a number of acute interventions (seated, supine, FES of the lower limbs, and the combination of an abdominal binder with lower body pressure stockings) in low- to moderately-trained individuals with cervical SCI during sub-maximal ACE at 20, 40 and 60% of peak power (Hopman *et al.*, 1998b). The authors reported improvements in stroke volume at 60% of peak power in response to FES of the lower limbs, and in response to abdominal binding and lower body pressure stockings. However, it is difficult to partition the degree to which the improvements are due to the abdominal binding or the pressure stockings. An extension of that study was carried out by the same research group to examine whether any of the aforementioned conditions could improve peak exercise responses during maximal incremental ACE in low- to moderately-trained individuals with cervical SCI (Hopman *et al.*, 1998a). The authors reported no changes in peak power or the cardiorespiratory responses to peak exercise with any intervention. Together, these two studies suggest that abdominal binding with lower body pressure stockings is able to elicit beneficial sub-maximal alterations in exercise haemodynamics; however, these do not translate to an increase in peak power. These findings imply that the limits to exercise performance during ACE in the low- to moderately-trained cervical SCI population are located peripherally (small active muscle mass) rather then centrally.

2-5.4 Summary

A number of studies have investigated acute methods of improving resting cardiorespiratory function in cervical SCI. Lower body FES stimulation or an anti-G suit has been shown to improve both resting and exercise haemodynamics in individuals with cervical SCI. However, neither intervention provides a viable option for highly-trained individuals with cervical SCI competing in wheelchair sport. Abdominal binding provides a means by which resting pulmonary function and respiratory muscle function can be improved; however, the mechanisms that underlie these alterations are not completely understood. The effects of abdominal binding on resting cardiovascular function and/or exercise capacity in cervical SCI also remain to be determined.

Table 2-1 Studies that have used abdominal binding as an acute method to improve cardiorespiratory function in individuals with cervical SCI

Author; Year; Total sample size	**Methods**	**Outcome in bound vs. unbound condition**
Maloney *et al.,* 1983 N = 15	**Population:** 15 tetraplegics (C4-T1); age range, 19-36 y; time since injury, 1-18 y **Binder:** Rigid abdominal corset **Conditions:** Supine, seated **Measures:** Spirometry	**1.** No significant effect of binding on spirometric indices
Huang *et al.,* 1983 N = 27	**Population:** 13 tetraplegics (C6-C7), 14 tetraplegics (C4-C5); mean age, 32 y; time since injury, 47 days **Binder:** Inflatable abdominal cuff **Conditions:** Supine, head-up tilted (20^{o} and 45^{o}) and head-down tilt (20^{o}) **Measures:** Spirometry, oxygen uptake and blood pressure	**1.** Increased oxygen uptake in all conditions ($p < 0.01$) **2.** Increased systolic and diastolic blood pressure in both 20^{o} and 45^{o} head-up tilt ($p < 0.01$)
Urmey *et al.,* 1986 N = 6	**Population:** 6 tetraplegics; age range: 20 – 65 y **Binder:** Inflatable abdominal cuff **Conditions**: Supine, head-up tilted (37^{o}) and seated **Measures**: Spirometry and pneumography	**1.** Increased lower and upper rib cage expansion during inspiration
McCool *et al.,* 1986 N = 13	**Population:** 13 tetraplegics (C4-C7), 9 AB controls; mean age, 30 y **Binder:** Nylon elasticised binder **Conditions:** Supine, head-up tilt (37^{o}) and seated **Measures**: Helium dilution and spirometry, magnetometers	**1.** Increased IC in all conditions ($p < 0.01$) **2.** Decreased FRC in all conditions ($p < 0.01$) **3.** Increased TLC in head up tilt and sitting conditions ($p < 0.05$) **4.** Increased rib cage dimensions at TLC

Author; Year; Total sample size	Methods	Outcome in bound vs. unbound condition
Goldman *et al.,* 1986 N = 7	**Population:** 7 tetraplegics (C5-C7), 20 AB controls; mean age, 33 y **Binder:** Elasticised and rigid **Conditions:** Seated, supine and head-up tilt (70^{o}) **Measures:** Spirometry, diaphragm function, maximal inspiratory mouth pressure	**1.** Increased VC with both binders in seated condition ($p < 0.01$) **2.** Increased sniff transdiaphragmatic pressure with both binders in 70% tilt condition ($p < 0.05$)
Kerk *et al.,* 1995 N=6	**Population:** 6 paraplegics (T1-T6); mean age, 21.8 y; mean body mass, 56.7kg **Binder:** Elasticised binder **Conditions:** Seated and during wheelchair propulsion **Measures:** Spirometry, maximal inspiratory mouth pressure, biomechanical and cardio-respiratory responses to exercise	**1.** Increased VC in 5/6 individuals at rest ($p > 0.05$)
Estenne *et al.,* 1998 N = 8	**Population:** 8 tetraplegics (C5-C8); age range, 21-41 y; time since injury, 6-200 months **Binder:** Elasticised binder **Conditions:** Seated **Measures**: Helium dilution and spirometry	**1.** Increased VC ($p < 0.002$) **2.** Decreased FRC and RV ($p < 0.001$)
Boaventura *et al.,* 2003 N = 10	**Population:** 10 tetraplegics (C4-C7); age range, 16-49 y; time since injury, > 12 months **Binder:** Elasticised binder **Conditions:** Seated and supine **Measures**: Spirometry, maximal inspiratory and expiratory mouth pressure	**1.** Increased VC in seated condition ($p < 0.05$) **2.** Increased maximal expiratory mouth pressure in seated condition ($p < 0.05$)

Author; Year; Total sample size	Methods	Outcome in bound vs. unbound condition
Bodin *et al.,* 2005 N = 20	**Population:** 20 tetraplegics (C5-C8); mean age, 39 y; mean time since injury, 13 y **Binder:** Elasticised binder **Conditions:** Seated, seated while breathing with expiratory resistance, and seated while breathing with inspiratory and expiratory resistance **Measures**: Body plethysmography and spirometry	**1.** Decreased FRC in all 3 conditions ($p < 0.001$) **2.** Decreased RV in seated condition ($p < 0.01$) **3.** Decreased TLC in seated condition ($p < 0.01$) **4.** Increased VC in seated condition *(*$p < 0.01$**)**
Hart *et al.,* 2005 N = 10	**Population:** 7 tetraplegics (C5-C7), 3 paraplegics (T1-T6); mean age, 36 y; time post-injury, 3-27 months **Binder:** Rigid binder **Conditions:** Seated position **Measures**: Gas dilution and spirometry, inductance plethysmography, diaphragm function	**1.** Decreased respiratory effort ($p = 0.02$) **2.** Increased IC and VC ($p < 0.02$ and $p = 0.012$, respectively) **3.** Decreased FRC ($p = 0.006$) **4.** Increased rib-cage contribution to resting breathing ($p = 0.02$) **5.** Increased diaphragm pressure-time product ($p = 0.01$) **6.** Increased maximal and twitch transdiaphragmatic pressure ($p = 0.03$ and $p = 0.02$, respectively)

Definition of abbreviations: C, cervical; T, thoracic; IC, inspiratory capacity; FRC, functional residual capacity; TLC, total lung capacity; VC, vital capacity; FVC, forced vital capacity

2-6 Aims and hypotheses

Based on the presented literature, the overall aim of this thesis was to determine whether abdominal binding improves cardiorespiratory function at rest and during exercise in highly-trained athletes with cervical SCI. Four studies were carried out with the following aims:

1. to describe resting cardiorespiratory function in a group of highly-trained Paralympic athletes with cervical SCI and to compare the data with an AB control group.
2. to determine if there is a dose-dependent change in resting cardiorespiratory function with abdominal binding in highly-trained athletes with cervical SCI.
3. to determine whether abdominal binding improves field-based measures of fitness in highly-trained athletes with cervical SCI.
4. to determine whether abdominal binding improves cardiorespiratory function during maximal incremental treadmill exercise in highly-trained athletes with cervical SCI.

The corresponding hypotheses were:

1. highly-trained athletes with cervical SCI will exhibit lower cardiorespiratory function compared to able-bodied individuals.
2. there will be a dose-dependent improvement in resting cardiorespiratory function with abdominal binding.
3. abdominal binding will improve performance in field-based tests that rely on cardiorespiratory function and/or postural support.
4. abdominal binding will reduce the degree of dynamic hyperinflation, increase the diaphragmatic contribution to inspiration and increase peak oxygen uptake.

CHAPTER THREE:

GENERAL METHODS

3-1 Pre-test preparation

3-1.1 Ethical approval

Ethical approval was obtained for each of the studies in this thesis from the School of Sport and Education Ethics Committee, Brunel University, which is a sub-committee of the Brunel University Research Ethics Committee. In addition, chapters 6 and 7 were conducted in collaboration with researchers from Loughborough University. Accordingly, a copy of the research ethics approval letter from Brunel was supplied to the Loughborough University Research Ethics Committee. Copies of the ethical approval letters for each study are supplied in Appendix A-1.

3-1.2 Participants

Prior to commencing each of the studies, the prospective participants were supplied with a detailed participant information sheet that outlined the specific testing protocols. All participants were required to give written informed consent. Participants with spinal cord injury (SCI) were also required to complete two health questionnaires, one specific to their disability (Appendix A-2), and one general pre-exercise health questionnaire (Appendix A-3: chapters 6 and 7 only). Any participant who reported acute or chronic cardiopulmonary disease was omitted from the studies. Prior to each of the studies, the participants with SCI were requested to provide their American Spinal Injuries Association (ASIA) classification and their classification within their respective sport. All participants with SCI had a low traumatic cervical SCI (C5 – C8). All participants with SCI were at least 3 y post injury and competed at national or International level within their chosen sport. SCI participants were requested to provide an example training log detailing their usual weekly training regime. SCI participants undertook at least 15 hr of training per week, consisting of push sessions, match play and resistance training. In chapters 4 and 5, an able-bodied (AB) recreationally active control group was included. All participants were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 2 h postprandial, and to avoid strenuous

exercise in the 24 h preceding testing. Each participant was asked to refrain from caffeine and alcohol for 12 h and 24 h before testing, respectively.

3-2 Apparatus and Procedures

The following section details the general methods used in this thesis. Chapter specific apparatus and procedures are contained within the relevant chapters. This section is ordered chronologically, such that each method appears in this section in the order it first appears in this thesis.

3-2.1 Pulmonary function

In chapters 4, 5 and 7, pulmonary volumes, capacities and flows were assessed using whole-body plethysmography and spirometry (Zan 530; Oberthulba, Würzburg, Germany). The Zan flow measurement system consists of a 'Zan flow handy', which incorporates a pneumotacograph for the measurement of pulmonary volumes and flows and a shutter for the measurement of plethysmographic intrathoracic gas volume (Fig 3-1). Prior to assessment, the pneumotachograph was calibrated for volume using a 3 L syringe and the plethysmograph was calibrated for pressure using the in-built manufacturer's calibration procedure, whereby a small sinusoidal volume of air was pumped into the inside of the cabin and the change in cabin pressure was calculated. Pulmonary function data were analysed using dedicated software (GPI 3.00, Zan; Oberthulba, Würzburg, Germany).

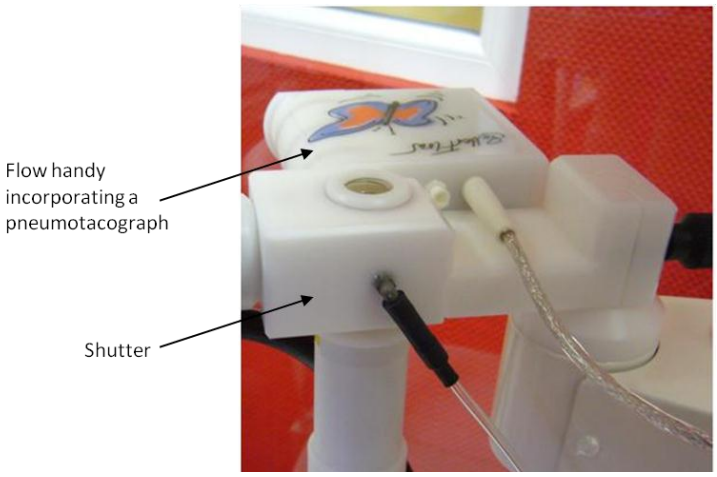

Fig 3-1 The pneumotacograph and shutter used in the body plethysmograph.

Prior to all plethysmographic measurements, participants with an SCI transferred from their wheelchair to a standard chair with a back support and arm rests. AB participants also sat in the same chair. All measurements were made with the participants seated upright and the nares occluded with a nose clip. The door of the cabin was closed and participants were instructed to breathe regularly for 5-6 breaths for the determination of airway resistance. Airway resistance was measured between flow rates of ± 0.5 $L \cdot s^{-1}$ (Lord and Edwards, 1978). Next, participants were instructed to breathe into the flow handy for 5-6 breaths whilst supporting their cheeks with their hands. Once a stable end-expiratory level was achieved the shutter was automatically activated and the participants were required to 'pant' against the shutter at a frequency of 0.5-1.0 Hz for the determination of plethysmographic intrathoracic gas volume, also termed functional residual capacity (FRC). Immediately after the shutter opened the participants were required to complete a slow expiration to residual volume (RV), followed by a maximal slow inspiratory vital capacity manoeuvre to total lung capacity (TLC; see Fig 3-2). The FRC measurement was performed a maximum of 5 times, or until 3 measurements of FRC varied by less than 5%, and the mean was reported (Wanger *et al.*, 2005). TLC, inspiratory capacity (IC) and RV were derived from the plethysmographic measurements.

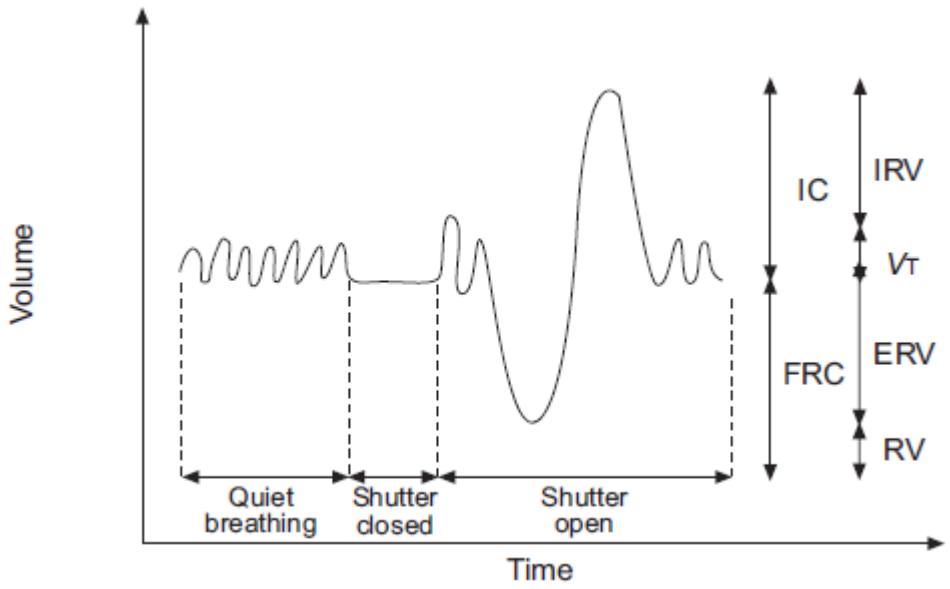

Fig 3-2 Method used to determine pulmonary volumes and capacities.
IC, inspiratory capacity; FRC, functional residual capacity; IRV, inspiratory reserve volume; VT, tidal volume; ERV, expiratory reserve volume; RV, residual volume. Reproduced from Wanger et al. (2005).

Following the determination of FRC, participants completed forced flow-volume loops using spirometry. Participants were instructed to inhale deeply from FRC to TLC and begin the forced exhalation to RV with minimal hesitation. Following exhalation, the participants were required to complete a maximal inhalation to TLC. This procedure was repeated a minimum of 3 and a maximum of 8 times, and all spirometry manoeuvres were required to conform to specific American Thoracic Society / European Respiratory Society acceptability criteria (Miller *et al.*, 2005b). For AB individuals, these were: 1) extrapolated volume was <5% of vital capacity; 2) the expiratory time was >6 s and a flow-plateau was evident in the volume-time curve; 3) the manoeuvre was free from artifacts such as coughing; and 4) at least three reproducible efforts were made, of which the two largest values of vital capacity (VC) and forced expiratory volume in 1 s (FEV_1) did not differ by more than 0.15 L. For SCI individuals, the acceptability criteria were modified to allow excessive back-extrapolation volume and an expiratory effort of less than 6 s duration if a flow plateau was evident for at least 0.5 s at RV (Kelley *et al.*, 2003). All participants were instructed to maintain a stable head position (Amodie-Storey *et al.*, 1996) and keep their hands in their lap during testing. Following examination of all of the acceptable spirometry manoeuvres, the largest VC and FEV_1 were reported even if they were from different expiratory

efforts. All other flow and volume indices were reported from the manoeuvre which had the largest combined VC and FEV_1, with the exception of PEF which was reported as the highest value from any of the acceptable manoeuvres (Miller *et al.*, 2005b). Pulmonary function indices were expressed as absolute and percent predicted values based on AB prediction equations (Quanjer, 1983; Quanjer *et al.*, 1993). The following indices were derived: VC, IC, FEV_1, inspiratory reserve volume (IRV), expiratory reserve volume (ERV), PEF, mean mid-expiratory flow (MMEF) and peak inspiratory flow (PIF).

Participants also completed a maximal voluntary ventilation (MVV) test, for which they were instructed to make an airtight seal around the mouthpiece and ventilate as rapidly and deeply as possible for 12 s. The volume of air exhaled was extrapolated and expressed over 60 s. The MVV test was repeated until two measurements did not differ by more than 20%, and the highest value was reported (Miller *et al.*, 2005b). MVV_{12} was expressed as absolute and percent predicted values based on AB prediction equations (Grimby and Sóderholm, 1963).

3-2.2 Maximal volitional pressures

In chapters 4 and 5, maximal inspiratory mouth pressure ($P_{I,max}$) and maximal expiratory mouth pressure ($P_{E,max}$) were assessed in response to maximal Müeller and Valsalva manoeuvres, respectively. All measurements were made using a handheld pressure metre (MicroRPM; Micro Medical Ltd, Kent, UK). A 2 mm orifice was incorporated into the pressure metre to prevent glottic closure during the $P_{I,max}$ manoeuvre, and buccal muscle recruitment during the $P_{E,max}$ manoeuvre . An investigator supported the participant's cheeks during the $P_{E,max}$ manoeuvre to further reduce the recruitment of buccal muscles and to prevent leeks around the mouthpiece. The nose was occluded with a clip and participants breathed through a flanged mouth piece. $P_{I,max}$ and $P_{E,max}$ were assessed from FRC and TLC, respectively. Each manoeuvre lasted approximately 5-6 s and was repeated a maximum of eight times in 30 s intervals, or until the three highest values varied by less than 10% (Green *et al.*, 2002). The highest value was reported and compared to AB predicted values (Bruschi *et al.*, 1992). In a sub-group of the sample in chapter 4, and in all

participants in chapter 5, gastric pressure (P_{ga}), oesophageal pressure (P_{oe}) and transdiaphragmatic pressure (P_{di}) were continually monitored during the maximal manoeuvres to partition the relative contributions of the diaphragm and chest wall muscles to maximal pressure generation.

3-2.3 Pressure measurements

In chapters 4, 5 and 7, P_{ga} and P_{oe} were measured using two latex balloon-tipped catheters (no. 47-9005; Ackrad Labs, Cooper Surgical, Berlin, Germany). Each catheter was 86 cm long and featured a series of small holes in the distal 5-7 cm, which were encased inside a 9.5 cm long latex balloon to stop the holes being occluded by oesophageal tissue (Benditt, 2005). To aid balloon placement the catheters incorporated a metallic guide wire that was removed once the balloons were situated in the oesophagus or stomach. The catheters were placed according to the methods of Baydur *et al.* (1982). Following local anaesthesia to the nasal mucosa and the pharynx with 2% lidocaine gel, a balloon catheter was passed pernasaly into the pharynx. Once the balloon reached the posterior pharynx, participants were directed to take small sips of water through a straw, and the naturally occurring peristalsis carried the balloon into the oesophagus and then into the stomach. A second catheter was also placed into the stomach in the same way. A glass syringe was used to inject a small amount of air into each balloon to maintain a column of air around the catheter, such that changes in the pressure of the structures surrounding the balloon were reflected by changes in pressure inside the balloon. The oesophageal and gastric balloons were filled with 2 ml of air, after which 1 ml of air was removed to leave 1 ml of air in each balloon. In chapter 7, 2 ml of air was left in the gastric balloon to prevent the balloon collapsing under high expiratory pressures (Polkey *et al.*, 1999). The proximal end of each catheter was coupled to a differential pressure transducer (DP45; Validyne, Northridge, CA, USA; range ± 229 cmH2O) that was calibrated across the physiological range (± 225 cm·H_2O) using an electro-manometer (model C9553; JMW Ltd., Harlow, UK). All pressure signals were amplified (model 1902; Cambridge Electronic Design, Cambridge, UK), digitised at sampling rates of 150 Hz (chapter 4 and 5) or 100

Hz (chapter 7) with an analogue-to-digital converter (micro 1401 mkII; Cambridge Electronic Design) and acquired using computer software (Spike 2 version 7; Cambridge Electronic Design).

Once both pressure traces were visible in the software, one of the catheters was withdrawn until a negative pressure deflection was evident, and then removed a further 10 cm to ensure placement in the lower one-third of the oesophagus. Thus, one catheter measured P_{oe} and the other measured P_{ga} (Fig 3-3). A virtual channel was set up in the software to calculate transdiaphragmatic pressure real-time. To validate the position of the oesophageal catheter, mouth pressure was measured while the participants performed a series of progressive static Müeller manoeuvres against a semi-occluded airway using a three-way valve (2100 Series 3-Way Stopcock, Hans Rudolph, MO, USA). The optimum position of the oesophageal balloon was defined as the position where mouth pressure most closely matched oesophageal pressure (Baydur *et al.*, 1982). Both balloons were fixed to the nose using standard medical tape.

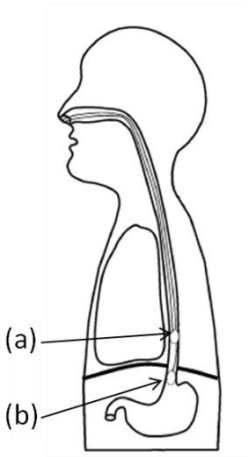

Fig 3-3 Oesophageal (a) and gastric (b) balloon placement.
Note the oesophageal balloon is placed cephalad to the diaphragm, and the gastric balloon is placed caudal to the diaphragm. This allows transdiaphragmatic pressure to be measured. Reproduced from Benditt (2005).

To ensure differences in the volume of air injected into the oesophageal and gastric balloons did not affect the pressure measurements, a pressure-volume curve was constructed for the balloon-tipped catheters prior to the first study (chapter 4); whereby air was introduced into

the balloon in 0.5 ml steps. The range of volumes over which the balloon pressure did not change was between 0.5 and 2 ml (Fig 3-4).

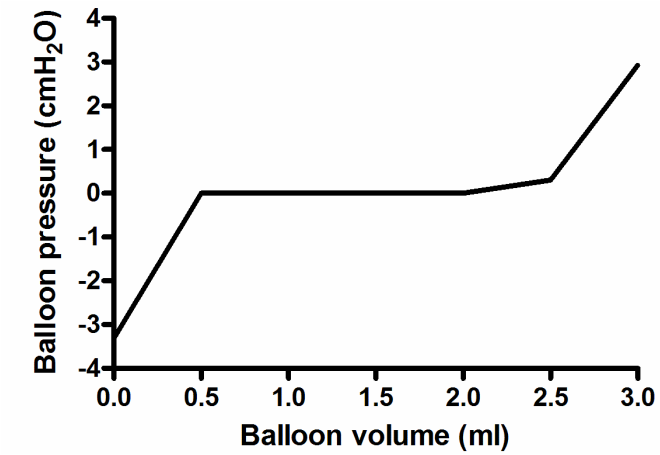

Fig 3-4 Pressure-volume curve for the balloon-tipped catheters.
Note that despite increases in volume from 0.5-2 ml, balloon pressure remained unchanged.

3-2.4 Magnetic stimulation

Magnetic stimulation is based on the principles set out by Michael Faraday who found that when a moving or time-varying magnetic field is applied to a conductive structure, it creates an electric field around it (Jalinous, 2001). Magnetic nerve stimulators generally consist of a high current pulse generator and stimulating coil which consists of one or more tightly wound copper coils that produce magnetic pulses (Fig 3-5). If the pulse is of sufficient magnitude it will stimulate neuromuscular tissue in a similar way to that of conventional electrical stimulation.

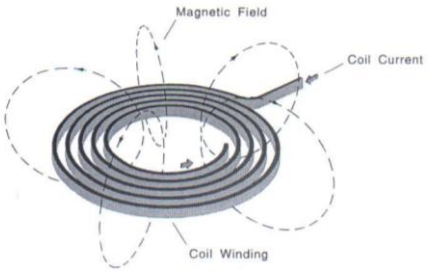

Fig 3-5 Magnetic stimuli are produced by passing strong electric current pulses through a coil of wire.
Reproduced from Jalinous (2001).

Phrenic nerves

In chapters 4 and 5, bilateral anterior magnetic phrenic nerve stimulation (BAMPS) was achieved via two 45-mm figure-of-eight coils, each powered by a separate Magstim 200 (Magstim 200; Magstim, Whitland, Wales). A digital output from the computer was used to discharge both stimulators simultaneously. The coils were positioned either side of the neck at the posterior border of the sternocleidomastoid muscle at the level of the cricoid cartilage (Mills *et al.*, 1996). Stimulations were performed at the end of a tidal expiration against an occluded airway. The optimum position of the coils was defined as the coil position which evoked the largest twitch transdiaphragmatic pressure ($P_{di,tw}$). The coil positions were marked using indelible ink and used for all subsequent stimulations. Three single twitches were obtained at 60, 70, 80, 85, 90, 95, and 100% of each stimulator's maximum power output to determine whether depolarization of the phrenic nerves was maximal (Fig 3-6). Each of the stimulations was separated by 30 s to avoid twitch potentiation. The incremental protocol was applied after 10 min of rest and at least 20 min before the first assessment of respiratory muscle function. All subsequent twitches were performed at 100% of each stimulator's power output. Three stimulations were delivered to the phrenic nerves in 30 s intervals against an occluded airway at FRC such that three unpotentiated $P_{di,tw}$ were obtained. Baseline-to-peak amplitude of the pressure responses ($P_{di,tw}$, $P_{ga,tw}$, $P_{oe,tw}$) was analysed for each stimulation. Excellent reliability of BAMPS for the measurement of $P_{di,tw}$, $P_{ga,tw}$, $P_{oe,tw}$ pressure in the SCI population has been published in our laboratory [within day, between trial co-efficient of variation 4.5%; Taylor *et al.* (2010)].

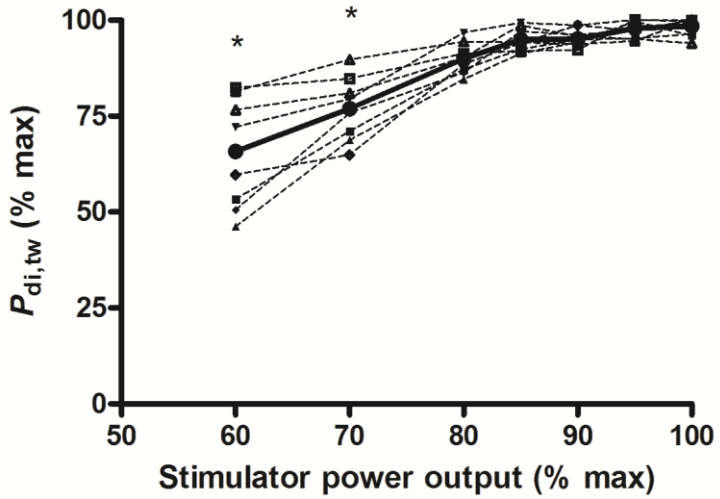

Fig 3-6 Individual participant (dotted line) and group mean (solid line) twitch transdiaphragmatic ($P_{di,tw}$) response to bilateral anterior magnetic stimulation of the phrenic nerves during the incremental stimulation protocol.

*Data shown are for the sub-group of 8 individuals with cervical SCI who were assessed for respiratory muscle function in chapter 4. Note that the $P_{di,tw}$ response started to level off at ~80-85% of the stimulator's power output. *p < 0.05, different from 100% of stimulator's power output.*

Bilateral anterior magnetic stimulation of the phrenic nerves was chosen over cervical magnetic stimulation (CMS) in this thesis as BAMPS directly stimulates the phrenic nerve trunks that innervate the diaphragm. Although CMS also evokes bilateral diaphragmatic contraction, it does so via stimulation of the cervical roots. Thus, CMS also induces co-contractions of various other muscle groups innervated by the cervical roots [e.g., deltoid, trapezius and rhomboid muscles (Similowski *et al.*, 1989)]. Further to the direct advantage of stimulating the phrenic nerves, BAMPS uses two figure-of-eight coils, rather than a single circular coil which is used in CMS. The main advantage of the figure-of-eight coil is that the induced tissue current is highest where the two windings meet, whereas for the single coil the current is highest directly under the windings (Fig 3-7). Thus, the figure-of-eight coil results in improved accuracy of stimulation.

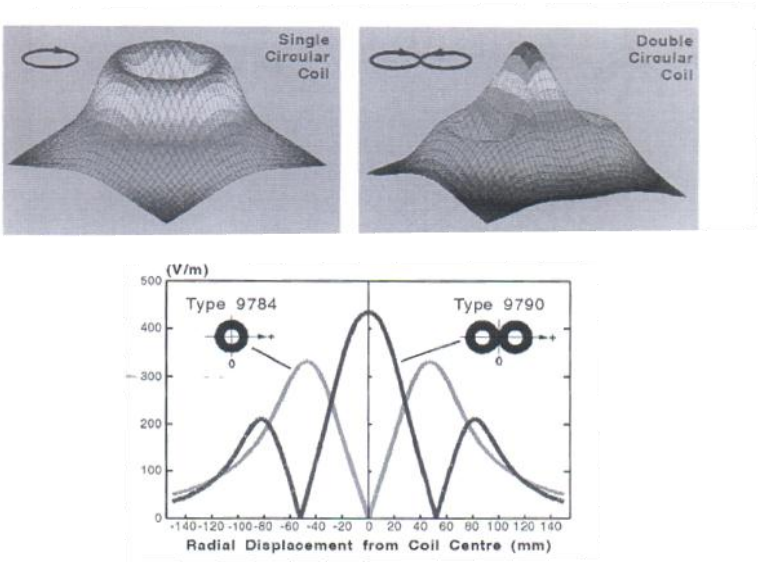

Fig 3-7 Comparison of the induced electric field profiles of a circular and figure-of-eight coil (double-circular coil).
Note the induced electrical current of the figure-of-eight coil is at its maximum where the two windings join, whereas the induced electrical field of the circular coil is zero under the centre and reaches its maximum directly under the winding. Adapted from Jalinous (2001).

3-2.5 Assessment of inspiratory muscle function

In chapter 4, baseline-to-peak amplitude of the P_{di}, P_{oe} and P_{ga} swings and ventilatory parameters (section 3-2.6) were assessed during 2 min of resting breathing. The diaphragm pressure-time index (PTI_{di}) was calculated as the product of the tidal P_{di} swing / $P_{di,max}$ and the fractional inspiratory time (T_I/T_{TOT}) (Bellemare and Grassino, 1982), where $P_{di,max}$ was the P_{di} (baseline-to-peak) recorded during a maximal Müeller manoeuvre. Additionally, in chapter 7, to estimate the relative contributions of the diaphragm and accessory muscles to the inspiratory volume excursion, the oesophageal and transdiaphragmatic pressure-time products (PTP) were calculated by integrating the area subtended by each transdiaphragmatic and oesophageal pressure curve over time during the period of inspiratory flow (Clanton *et al.*, 2002). The baseline used in the calculation of the PTP was set to zero such that the respiratory and bracing (isometric) work done by the diaphragm was included. The average PTP was multiplied by respiratory frequency and

reported in $cmH_2O \cdot s \cdot min^{-1}$. The inspiratory portion of the respiratory cycle was set using points of zero flow.

3-2.6 Ventilatory parameters

In chapter 4 and 5, minute ventilation ($\dot{V}_E$), tidal volume (V_T), respiratory frequency (f_R) and T_I/T_{TTOT} were assessed during 2 min of quiet breathing using an ultrasonic flow tube (Birmingham Flowmetrics Ltd; Birmingham, UK). Two ultrasonic transducers were mounted inside the flow tube on opposite sides and at a 40-degree angle to the airflow axis. The transducers measure the difference in the transit time of ultrasonic pulses propagating in and against the airflow axis. The resulting phase-shift of the received signal produces a voltage output that is linearly proportional to flow (Buess *et al.*, 1986). The flow metre is very fast (response time 1-2 ms), accurate (volume accuracy compared to room air ± 0.7%), has low noise (below 9 $ml \cdot s^{-1}$) and a flat frequency response up to 70 Hz (Buess *et al.*, 1986). The ultrasonic flow transducer was interfaced directly to the same software as the pressure measurements (Spike 2 version 7; Cambridge Electronic Design, Cambridge, UK). Tidal volume was calculated by the computer integration of the flow signal, and the expiratory portion of each breath was corrected to body temperature and pressure saturated (BTPS).

In chapter 7, ventilation was measured at rest and during exercise using a turbine flow transducer interfaced to an online gas analyser (Oxycon Pro; Jaeger, Höchberg, Germany). The flow transducer features a lightweight impeller (45 g) placed in the flow stream. As the impeller spins the number of interruptions of a light beam is counted and a voltage output that is linearly proportional to flow is created. The impellor had linearity of flow rates between 0 and 15 $L \cdot s^{-1}$, a very low resistance to flow (< 0.1 $kPa \cdot L^{-1} \cdot s^{-1}$ at 15 $L \cdot s^{-1}$) and was insensitive to moisture. A real-time analogue flow trace was exported from the Oxycon system (uDAQ; Eagle Technology, Cape Town, RSA), digitised at 100 Hz and imported into the same software as the pressure measurements (Spike 2 version 7; Cambridge Electronic Design, Cambridge, UK).

3-2.7 Echocardiography

In chapters 4 and 5, 2-D echocardiography was used to measure left ventricular (LV) volumes and dimensions, and Doppler was used to assess myocardial tissue velocity in systole, early diastole and late diastole. In chapter 4, trans-mitral filling velocity during early and late diastole was also measured.

General procedures for echocardiography

Participants with an SCI were required to transfer to a bed and rest in the left lateral decubitus position (chapter 4), or remain seated in their day wheelchair (chapter 5). AB individuals were always imaged in the same posture as SCI. Image acquisition was conducted on a commercially available ultrasound (Vivid 7; GE Medical Systems, Horton, Norway) machine using an M4S 2-5 MHz probe, with the frequency set at 1.7 MHz transmit and 3.6 MHz receive. During image acquisition participants were required to hold their breath at FRC while 5 consecutive cardiac cycles were recorded for off-line analysis. A 3-lead electrocardiogram recorded heart rate (HR) simultaneously to echocardiographic imaging.

LV dimensions and volumes

LV dimensions were calculated from the parasternal long-axis window. A motion-mode (M-mode) cut was taken perpendicularly to the septum and posterior wall of the LV at the level of the mitral leaflet tips. LV dimensions were measured between the endocardial border of the intraventricular septum and the posterior wall of the LV during systole and diastole (Fig 3-8). In chapter 4, end-diastolic volume (EDV) and end-systolic volume (ESV) were determined using the modified single plane Simpson's method, whereby the endocardial border was traced (Fig 3-9) at end-diastole and end-systole using dedicated software (EchoPac; GE Medical Systems, Horton, Norway). End-diastole was defined as the frame after mitral valve closure and end-systole was defined as the frame preceding mitral valve opening. The basal border of LV cavity was delineated by a straight line between the mitral valve insertions at the lateral and septal borders of the mitral annulus

(Lang *et al.*, 2005). Stroke volume (SV) was calculated from the difference between EDV and ESV, ejection fraction (EF) was calculated as SV/EDV × 100, and cardiac output ($\dot{Q}$) was calculated as the product of SV and HR.

In chapter 5, image quality in the seated position was insufficient to trace the endocardial border in several individuals with SCI; accordingly, EDV and ESV were estimated from the parasternal long-axis window using the formula of Teicholz *et al.* (1976). This formula is based on the standard cube formula (where the volume is approximately equal to the cube of the internal diameter) with a correction factor for the increasing spherical shape of the ventricle with increasing dilatation. Although the American Society of Echocardiography currently recommends the Simpson's biplane method for the determination of LV volumes (Lang *et al.*, 2005), this method requires tracing of the LV endocardial border in an apical four- and two-chamber view. In individuals with SCI, difficulty in image acquisition in the apical two-chamber view rendered image quality unacceptable for such an analysis. Furthermore, the Simpson's single plane has been shown to produce similar volumes to Simpson's biplane at end-systole and end-diastole (Wahr *et al.*, 1983), and although the Teichholz method may result in inaccuracies in absolute LV volumes (Lang *et al.*, 2005), it is only the relative change in each parameter (i.e., between condition comparison) that is of interest in chapter 5.

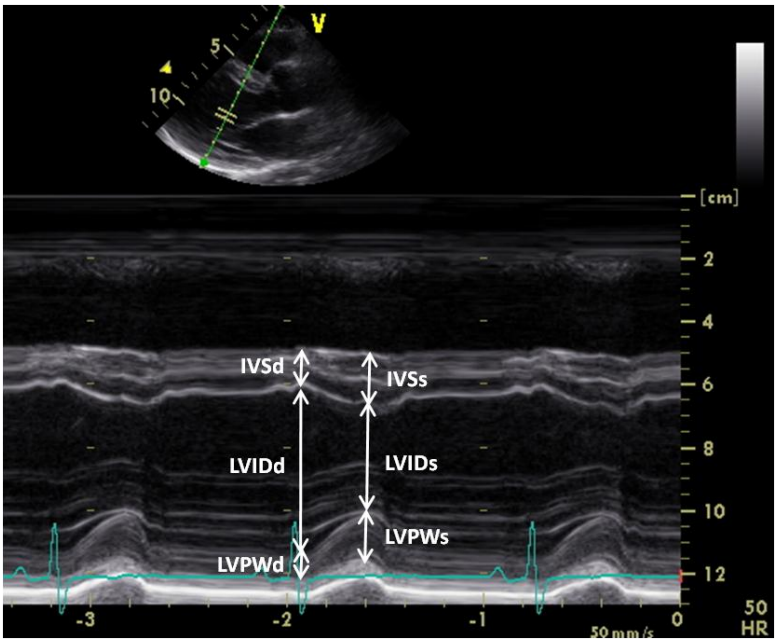

Fig 3-8 Example of an M-mode image and derived measurements at rest

The LV wall and cavity were imaged along one scan line from a parasternal long-axis view. IVSd, Intraventricular septum diastole; LVIDd, left ventricular internal diameter diastole; LVPWd, left ventricular posterior wall diastole; IVSs, intraventricular septum systole; LVIDs, left ventricular internal diameter systole; LVPWs, left ventricular posterior wall systole.

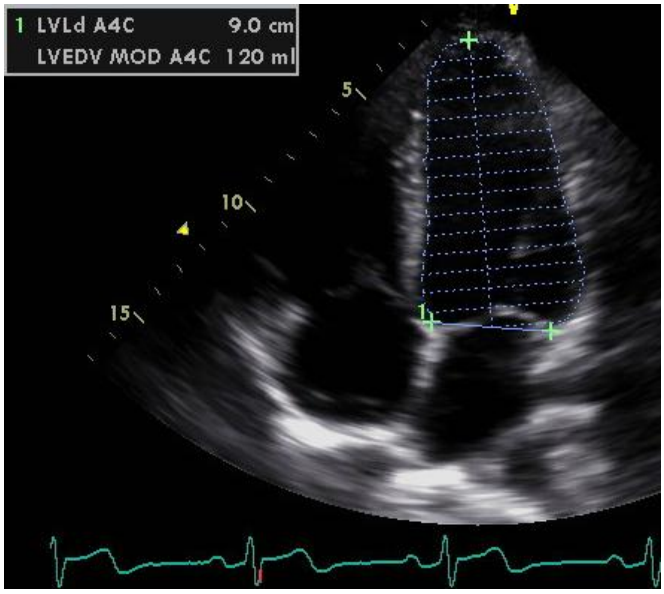

Fig 3-9 Example tracing of the endocardial border at end-diastole.

Note that the basal border of LV cavity was delineated by a straight line between the mitral valve insertions at the lateral and septal borders of the mitral annulus

LV function

Transmitral filling velocity (chapter 4) and myocardial tissue velocity (chapter 4 and 5) were assessed using Doppler. Doppler provides quantitative data on the direction and velocity of blood flow or myocardial tissue. As the source of the sound (blood or myocardial tissue) moves towards the transducer, the frequency of sound increases; whereas the opposite is true when the source of the sound moves away from the transducer. Thus, the movement of blood or myocardial tissue towards or away from the transducer allows for the calculation of velocity. In chapter 4, pulsed wave Doppler was used to measure mitral inflow velocities during early (E) and late (A) diastole. An apical four-chamber view was recorded and a pulsed wave sample volume was placed caudal to the mitral valve. The image was orientated to ensure the sample volume was placed vertically in line with the ultrasound beam (Fig 3-10).

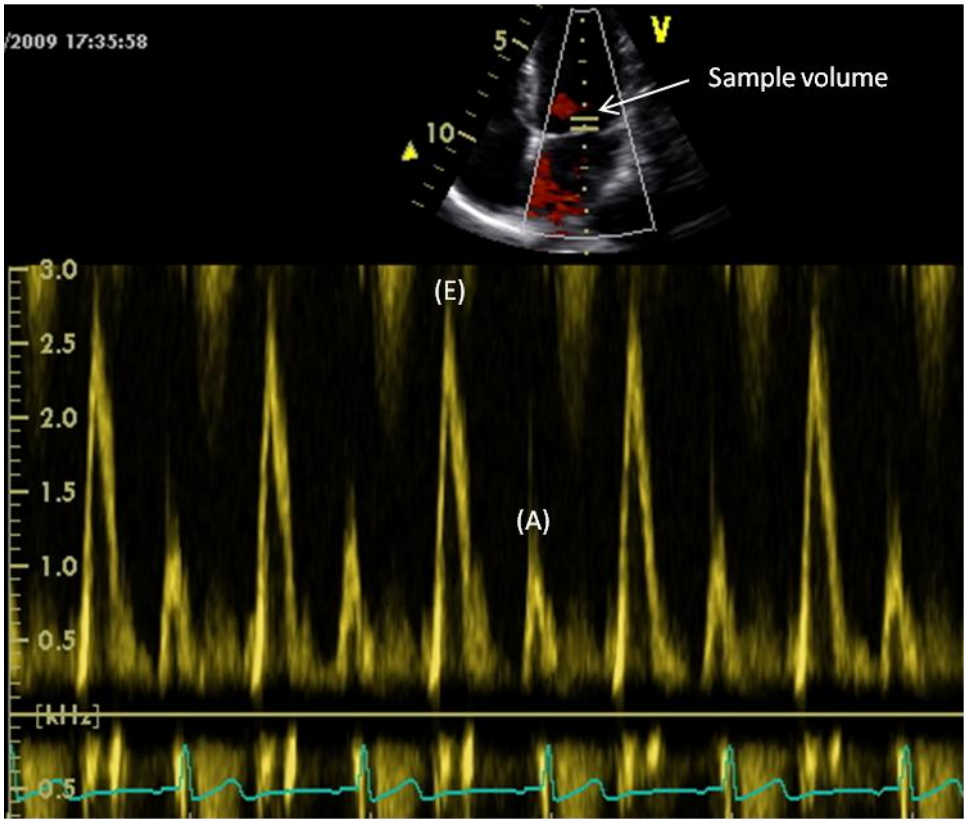

Fig 3-10 Example of an apical 4-chamber view and the measurement of mitral inflow velocities at rest.
A small pulsed wave sample volume is placed distal to the mitral valve for the measurement of early (E) and late (A) filling during diastole.

In chapters 4 and 5, myocardial tissue velocity was recorded via pulsed wave Doppler. A sample volume was placed in the mitral annulus during an apical four-chamber view. The width of the apical four-chamber view was reduced so that only the intraventricular septum and mitral annulus were in view, and the image was orientated such that the septal wall was as vertical as possible. The frame rate was set in excess of 200 frames per second and kept constant between conditions in chapter 5. Systolic (S'), early (E') and late (A') diastolic myocardial tissue velocity were measured (Fig 3-11).

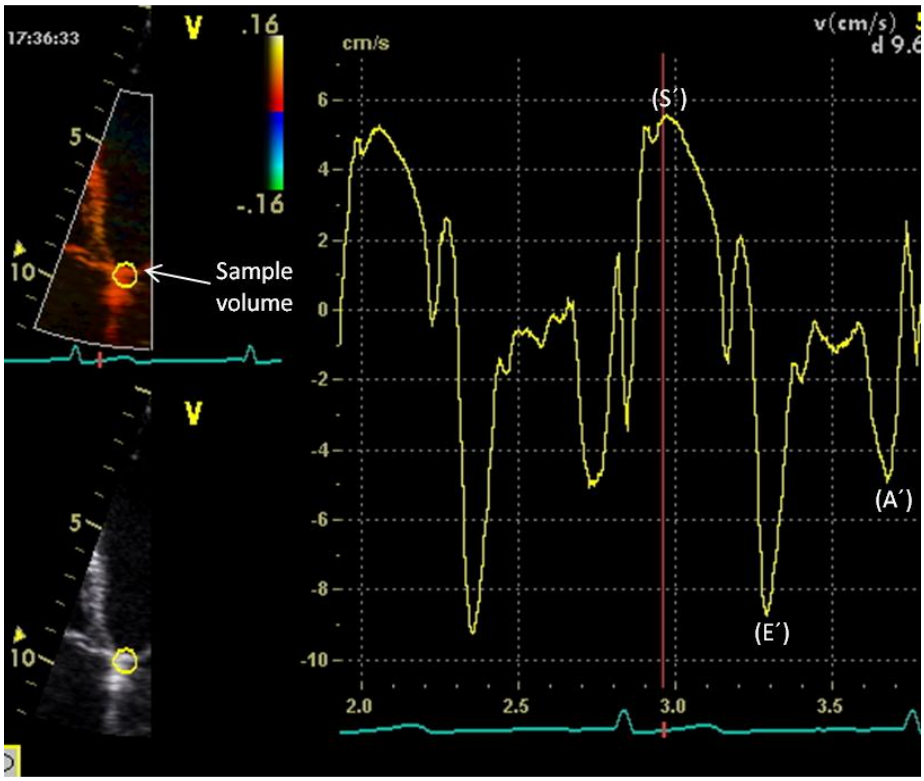

Fig 3-11 Example of an apical 4-chamber view, reduced to show the intraventricular septum and mitral annulus only.
A small pulsed wave sample volume was placed in the mitral annulus for the measurement of systolic (S´), and early (E´) and late (A´) filling during diastole.

Reliability

In chapters 4 and 5, image acquisition and image analyses were conducted by the author of this thesis. The within day, within participant co-efficient of variation (CV) of the sonographer was calculated from 7 AB participants (1 female). Participants rested in the seated position for 5 min

prior to the first set of images (trial 1). A series of 5 images were taken at FRC. Following a 10 min break image acquisition was repeated (trial 2). Data were analysed *post hoc* for LV dimensions, volumes (Teicholz method) and function. For each variable, 5 cardiac cycles were averaged and compared between trial 1 and trial 2 to calculate the CV (Table 3-1). The results demonstrate that the CV for LV dimensions, volumes and function ranged from 3-14%, which is in agreement with previously published data (Himelman *et al.*, 1988).

Table 3-1 Coefficient of variation for echocardiographic measures

LV index	Mean of trial 1 & 2	SD of trial 1 & 2	CV
EDV, mL	116	4	3.8
ESV, mL	38	4	9.1
SV, mL	77	4	5.9
EF, %	66	3	4.4
$\dot{Q}$, $L \cdot min^{-1}$	5.02	0.36	8.1
IVSs, mm	1.46	0.12	8.6
LVIDs, mm	3.09	0.13	4.2
LVPWs, mm	1.54	0.08	5.6
IVSd, mm	1.12	0.08	7.1
LVIDd, mm	4.95	0.07	1.4
LVPWd, mm	1.12	0.11	10.3
E, $cm \cdot s^{-1}$	7.53	0.24	3.2
A, $cm \cdot s^{-1}$	4.53	0.31	7.8
S', $cm \cdot s^{-1}$	5.43	0.29	7.1
E', $cm \cdot s^{-1}$	8.21	0.36	5.0
A', $cm \cdot s^{-1}$	3.60	0.47	14.7

Definition of abbreviations: EDV, end diastolic volume; ESV, end systolic volume; SV, stroke volume; EF, ejection fraction; $\dot{Q}$, cardiac output; IVSs, inter-ventricular septal thickness in systole; LVIDs, left ventricular internal diameter in systole; LVPWs, left ventricular posterior wall thickness in systole; IVSd, inter-ventricular septal thickness in diastole; LVIDd, left ventricular internal diameter in diastole; LVPWd, left ventricular posterior wall thickness in diastole; E, early transmitral filling velocity; A, late transmitral filling velocity; S', systolic myocardial tissue velocity; E', early diastolic myocardial tissue velocity; A', late diastolic myocardial tissue velocity.

3-2.8 Exercise testing

In chapter 6, participants completed a 5 min sub-maximal push on a wheelchair ergometer (WERG; Bromakin, Loughborough, UK; length: 1.14 m; circumference: 0.48 m) at 1.4 $m \cdot s^{-1}$. The

front of each participant's sports wheelchair was fitted onto the WERG using a customised bracket, and the rear wheels were mounted on the roller (Fig 3-12). Propulsion velocity was recorded at 100 Hz using a photo-optic sensor mounted on the flywheel connected to the roller via a fan-belt and a two-cog mechanism (Fig 3-12). The flywheel is a small disk with alternating black and white squares; as each square passes through a light-emitting diode and a photo-transistor a pulse in generated. Mean velocity was displayed continuously throughout the sub-maximal exercise test using a laptop computer (Compaq Armada1520, Series 2920A; Compaq Computer Corporation, Taiwan).

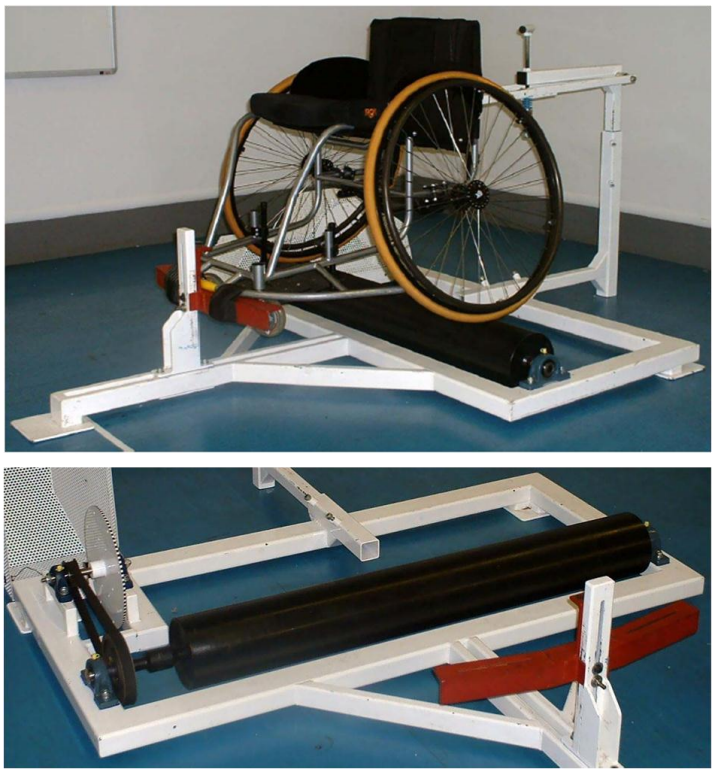

Fig 3-12 Wheelchair ergometer set up (top panel) and the flywheel (bottom panel).
Note the front of the wheelchair was fitted to the ergometer using a customised bracket and straps. Note also that the flywheel is connected to the roller via a fan-belt and a two-cog mechanism. Images reproduced from Lenton (2008).

During the final two minutes of sub-maximal exercise the participants breathed through a low resistance two way valve (Hans-Rudolph Inc, Kansas City, MO), and oxygen uptake ($\dot{V}O_2$) and carbon dioxide output ($\dot{V}CO_2$) were assessed using the Douglas bag technique (Douglas, 1911). The concentrations of oxygen and carbon dioxide were measured using a paramagnetic oxygen and

infrared carbon dioxide gas analyser (Servomex 5200 multipurpose analyser; Crowborough, UK), and $\dot{V}_E$ was assessed using a dry gas metre (Harvard Apparatus Ltd; Edenbridge, Kent, UK). Heart rate (HR) was assessed via telemetry (Polar Vantage NV; Polar Electro Oy, Kempele, Finland). Prior to each test the gas analysers were calibrated using low (100% Nitrogen) and high calibration gases for oxygen and carbon dioxide (20.93 and 9.98%, respectively). Energy expenditure was calculated from oxygen uptake and carbon dioxide output using the formula of Brouwer (1957). Gross efficiency was calculated from measures of energy expenditure and work rate (see section 3-2.9 for calculation of work rate).

Following completion of the sub-maximal exercise test, participants completed a 30 s Wingate test. Participants were instructed to propel the wheelchair as fast as they could for the full 30 s period. To prevent difficulty in overcoming the initial inertia of the WERG, the participants were given a rolling start at a speed of 1 $m \cdot s^{-1}$ (Goosey-Tolfrey, 2005). The test was initiated following a countdown from the investigator. Velocity data were collected at 100 Hz and power output was calculated in 1 s intervals throughout the test (section 3-2.9). Athletes were assessed for peak and mean power, peak and mean heart rate, and fatigue [(peak speed – minimum speed) / peak speed] during the Wingate test.

In chapter 7, participants completed incremental sub-maximal and maximal wheelchair propulsion on a motorised treadmill (HP Cosmos Saturn 300/125; Nussdorf-Traunstein, Germany). The treadmill was calibrated by the manufacturer prior to data collection. The wheelchair was lifted onto the treadmill using a mechanical lift, and the wheelchair was fitted securely by a metal rod that was connected to a sliding bracket on the side-bar of the treadmill (Fig 3-13). This rod allowed the participant to move forward and backwards in their wheelchair, but prevented sideways movement. A set of safety springs prevented the wheelchair from rolling off the back of the treadmill. All participants had previously completed maximal exercise test on the treadmill as part of their regular assessments.

Fig 3-13 Method of securing the wheelchair to the treadmill.
The wheelchair was secured to the treadmill via a metal rod that was connected to a sliding bracket on the side-bar of the treadmill. A set of safety springs (top of picture) prevented the wheelchair from falling off the back of the treadmill.

The sub-maximal treadmill test started at 1.2 $m \cdot s^{-1}$, incremented by 0.4 $m \cdot s^{-1}$ every 4 min, and was terminated when the participant's respiratory exchange ratio consistently exceeded 1. For the peak exercise test, the speed was kept constant at the speed equivalent to the penultimate stage of the sub-maximal exercise test, and the gradient was progressively increased (0.2% gradient every 40 s) until volitional exhaustion. The maximal exercise test was terminated when the participant's wheelchair hit the safety springs (Fig 3-13). For both exercise tests, the participants were allowed to self-select their push cadence. Ventilatory and pulmonary gas exchange indices were assessed during sub-maximal and maximal wheelchair propulsion using an online gas analysis system (Oxycon Pro, Jaeger, Höchberg, Germany). For the sub-maximal exercise test, ventilatory and pulmonary gas exchange indices were averaged over the final 30 s of resting breathing and the first 30 s of the final minute of each exercise stage. For the maximal exercise test, peak data were expressed as the highest 30 s rolling average. Additional measurements included: arterial oxygen saturation (S_pO_2) by earlobe pulse oximetry (PalmSAT 2500; Nonin Medical, Plymouth, Minnesota); HR by telemetry (Polar Vantage NV; Polar Electro Oy, Finland); and intensity of perceived arm and respiratory discomfort [Borg's modified category ratio 10 (CR-10) scale (Borg, 1998)].

In chapter 7, prior to the first exercise test participants were familiarised with Borg's modified CR-10 scale (Borg, 1998), and its endpoints were anchored such that 0 represented 'no respiratory or arm discomfort' and 10 represented 'the most severe respiratory (arm) discomfort you have experienced or could imagine experiencing'. The numbers on the scale were verbalised in sequence and the participant indicated their number with a predetermined signal. Participants rated their intensity of respiratory and arm discomfort at rest, at the end of each exercise stage, and at the symptom-limited end of exercise.

3-2.9 Calculation of power output

In chapter 6, power output on the WERG was calculated according to Thiesen and co-workers (1996). The equations were adapted according to Lenton (2008) to account for a single roller and flywheel system. Power output was calculated from the torque applied to the wheel and the angular velocity. The torque applied to the wheel is a function of the moment of inertia of the rear wheels, the moment of inertia of the roller, the participant's angular velocity and the internal torque of the wheelchair system (van der Woude *et al.*, 2001). The internal torque was measured using a deceleration test, where the participants were asked to accelerate the wheelchair for 5 s, and then allow the wheelchair to decelerate to a complete standstill while adopting a relaxed posture with their hands in their lap. Time and velocity data were collected throughout the deceleration period. Linear regression was applied to the data across the range of velocities performed in the testing. The slope of the regression line represents the acceleration from which the angular acceleration of the roller (α_R) can be obtained. The instantaneous hand torque delivered by the participant to the rear wheels was then calculated (equation 1), and multiplied by the corresponding angular velocities (equation 2). External power output is then the average (mean) of all the instantaneous power outputs (equation 3). The equations used were as follows:

$$T_H = (I_R \times [W_r \div R_r] + 2 \times I_W \times [R_r \div W_r]) \times \alpha_R + (I_F \times [W_r \div R_r]) \times \alpha_F \quad (1)$$

$$PO_i = TH \times \omega_W \quad (2)$$

$$PO = \bar{X}\, PO_i \quad (3)$$

, where T_H, instantaneous hand torque; I_R, roller inertia; I_W, wheel inertia; I_F, flywheel inertia; Wr, wheel radius; Rr, roller radius; α_R, angular acceleration of the roller; α_F, angular acceleration of the flywheel; ω_W, instantaneous angular velocity of wheels; Poi, instantaneous power output; PO, external power output.

In chapter 7 of this thesis, the calculation of power output was approached by considering the wheelchair-user combination as a free body that moves at a given speed and which encounters a number of drag forces, including: air resistance, rolling friction, internal friction and gravitational effects when going up/down a slope (van der Woude *et al.*, 2001). The sum of drag forces can be reliably determined using a standard drag test (Fig 3-14). The wheelchair was connected to a strain gauge via a cable, while the treadmill was raised to different inclination levels (0-4.5%) at a constant velocity (1.1 $m \cdot s^{-1}$). The drag force was determined at each inclination level. Power output was then estimated as follows:

$$PO = F_{drag} \cdot v$$

, where PO is power output; F_{drag} is the sum of all of the drag forces determined from a deceleration test; v is velocity of the treadmill.

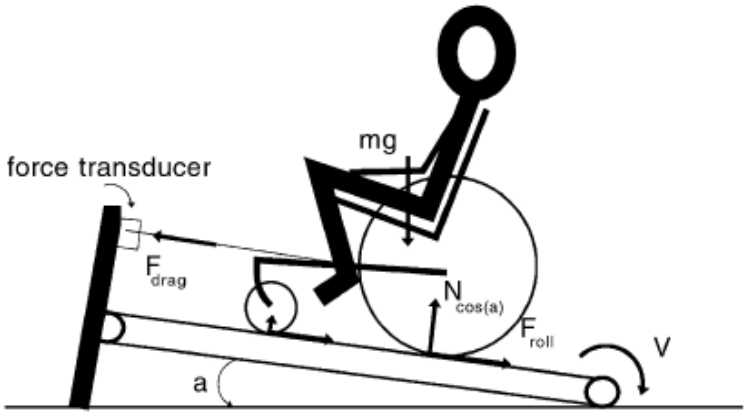

Fig 3-14 Drag test conducted on the treadmill.
Reproduced from Van der Woude et al. (2001).

3-2.10 Kinematic analysis

In chapter 6, 2-D biomechanical analysis was carried out during the maximal 30 s push to obtain kinematic parameters of each athlete's push technique. One high speed camera (Casio Exilim EX-F1) was positioned 10 m from the WERG. Prior to data collection the cameras were calibrated using a 15-point calibration frame with known coordinates (1.0 x 1.4 m). One reflective marker (19 mm in diameter) was placed on the seventh cervical vertebrae; a second reflective marker was placed on the rear-wheel axel of the wheelchair, as the iliac crest was not visible due to the bucket seat design of the wheelchair rugby chair. Tetraplegic athletes with poor hand function evidence a backhand push technique (Vanlandewijck *et al.*, 2001), which prevents the placement of a marker on the metacarpophalangeal joint to signify hand contact and hand release during wheelchair propulsion; accordingly hand contact and hand release were determined by visual inspection. All markers were filtered using a low-pass second-order Butterworth filter with a 6-HZ cut-off frequency, and digitised using a dedicated motion analysis system (SIMI Reality Motion Systems, Unterschleissheim, Germany). Each push was analysed for propulsion time (time in contact with wheel rim), recovery time (time not in contact with wheel rim), total cycle time (sum of push time and recovery time), start angle (angle at start of push phase with respect to a vertical axis), end angle (angle at end of push phase with respect to a vertical axis) and push angle (end angle – start angle; see also Fig 3-15). Active and passive trunk motion was also calculated by determining the maximum and minimum degree of trunk flexion during the push phase and recovery phase of each cycle, respectively.

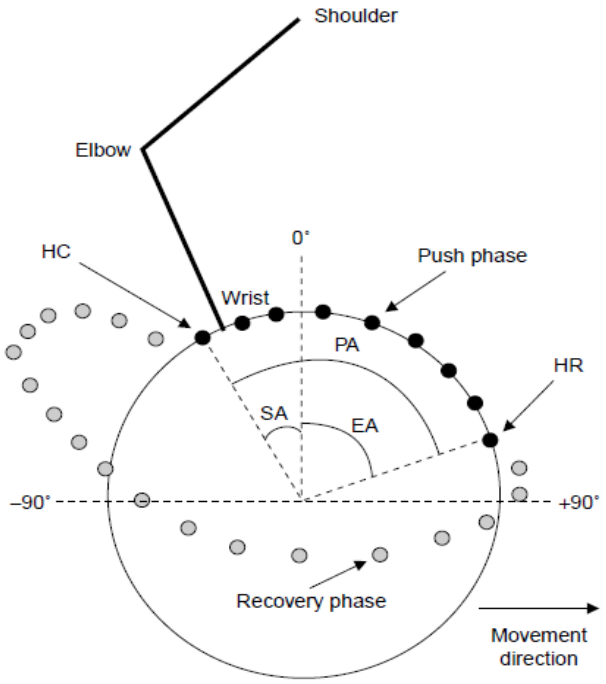

Fig 3-15 Kinematic variables assessed during each push.
HC, hand contact; SA, start angle; PA, push angle; EA, end angle; HR, hand release. Reproduced from Vanlandewijck et al. (2001).

3-2.11 Blood lactate sampling and analysis

In chapters 6 and 7, capillary blood samples were collected from a small incision on the right earlobe using a lancet. All samples were analysed in duplicate within 4 h of collection, and a mean of the duplicate values was reported for each sample. In chapter 6, blood was collected in 20 µl sodium heparinised capillary tubes and transferred immediately to small Eppendorf tubes which contained a lactate haemolysing solution. The tubes were immediately agitated and stored on ice for subsequent analysis in the laboratory using an automated desktop analyser (Biosen C_line Sport, EKF Diagnostic, Barleben, Germany). For analysis, immobilised L-lactate contained within the sample is converted enzymatically by lactate oxidase to pyruvate and hydrogen peroxide. Hydrogen peroxide is then subsequently detected by an electrode in the analyser and converted by linear regression to a corresponding blood lactate concentration. The analyser is reported by

the manufacturer to have a coefficient of variation of <1.5% at a blood lactate concentration of 12 mmol·L^{-1}. Based on the duplicate values collected in the unbound condition in chapter 6, the calculated CV was always <0.9%. The lactate analyser was calibrated using a 12 mmol·L^{-1} lactate standard at the start, and after every ten samples in line with the manufacturer's recommendations.

In chapter 7, blood was collected in capillary tubes, and approximately 25 µl of blood was injected into a desktop analyser (YSI 1550, Sport, YSI incorporated, Ohio, USA). This analyser uses a membrane sensor containing immobilised L-lactate oxidase that is placed between two membrane layers of polycarbonate and cellulose acetate. Hydrogen peroxide is then produced and oxidised on a platinum electrode resulting in an electrical signal that is directly proportional to the lactate concentration. The CV for this measurement technique was calculated from 12 repeated measurements on a 5 mmol·L^{-1} lactate standard and found to be 0.7%.

3-2.12 Operating lung volumes and ventilatory constraint

In chapter 7, the degree of ventilatory constraint was estimated by measuring dynamic changes in operating lung volumes [end-expiratory lung volume (EELV) and end-inspiratory lung volume (EILV)], the degree of flow limitation, the available inspiratory reserve (IRV) and the ratio of ventilation to a maximal estimated ventilation at a given lung volume (ATS/ACCP, 2003; Johnson *et al.*, 1999b). EELV was calculated immediately prior to exercise, during the final 30 s of every exercise stage and at the symptom-limited end of exercise by asking individuals to complete two IC manoeuvres separated by at least 15 s. Assuming that TLC remains unaltered during exercise (Stubbing *et al.*, 1980), then changes in IC reflect changes in EELV (TLC–IC). EILV was also measured at the same time points as EELV by [(TLC–IC) + V_T]. To ensure that a maximum effort was made with each IC, we confirmed the P_{oe} achieved during each IC was not different to pre-exercise baseline values.

To assess the degree of expiratory flow limitation, three maximal flow-volume manoeuvres were performed immediately prior to exercise and within 2 min of exercise cessation (Johnson *et al.*, 1999b). The maximal flow-volume loop used for analysis was the one which had the highest sum of VC and FEV_1 (Miller *et al.*, 2005b), whether it was before or after exercise. An average spontaneous flow-volume loop was obtained during resting breathing and during the first 30 s of the final minute of each exercise stage. To create the average flow-volume loop, the 30 s of data were first manually filtered to remove any breaths that did not fall within three standard deviations of the mean breath time (T_{TOT}) during the preceding 30 s. Each breath was then split into a certain number of time segments based on the mean T_{TOT} during the selected 30 s period with a resolution of 0.01 s. For example, if the mean T_{TOT} for the 30 s was 2.5 s then each breath was divided into 250 equal time segments. This method for dividing each breath based on the average T_{TOT} ensured the average flow-volume loop was created with a similar resolution between exercise stages. The degree of expiratory flow limitation was defined as the percent of the spontaneous flow-volume loop that encroached on, or exceeded, the descending expiratory limb of the maximal flow-volume loop.

Inspiratory flow reserve and the level of $\dot{V}_E$ relative to a theoretical maximal ventilatory capacity ($\dot{V}_{ECAP}$) was also determined at each time-point (Johnson *et al.*, 1999b). Inspiratory flow reserve (IFR) was expressed as the peak tidal inspiratory flow generated during tidal breathing relative to that achieved during the maximal flow- volume manoeuvre at the same lung volume. The V_{ECAP} was determined by integrating the area under the inspiratory and expiratory flow curves between EELV and EILV (Fig 3-16).

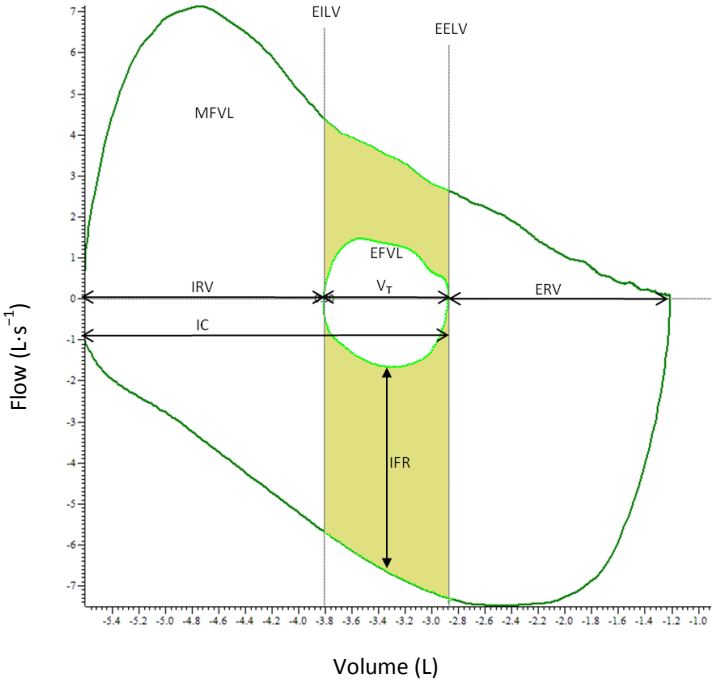

Fig 3-16 Method for calculating indices of ventilatory constraint.

An exercise flow-volume loop (EFVL) is placed within a maximum flow-volume loop (MFVL) and aligned at end-expiratory lung volume (EELV) using the inspiratory capacity manoeuvre (EELV = TLC-IC). End inspiratory lung volume (EILV) was calculated by the addition of V_T to EELV. Inspiratory flow reserve (IFR) was expressed as the peak tidal inspiratory flow generated during tidal breathing relative to that achieved during the maximal flow-volume manoeuvre at the same lung volume. The level of $\dot{V}_E$ relative to a theoretical maximal ventilatory capacity ($\dot{V}_E$:$\dot{V}_{ECAP}$) was also determined, where $\dot{V}_{ECAP}$ represents the total area under the inspiratory and expiratory flow curves between EILV and EELV (shaded area). IRV, inspiratory reserve volume; ERV, expiratory reserve volume.

3-3 Statistical analyses

All statistical analyses were conducted using SPSS version 16 (SPSS Inc., an IBM Company, Chicago, IL, USA). Alpha level was set *a priori* at 0.05 for all analyses. All data were screened for data entry errors and tested for the assumptions underlying parametric tests. Normality was tested via the Kolmogorov-Smirnov test and Q-Q plots. Homogeneity of variance was tested via Levene's test for equality of variances. In chapter 5, where data were analysed across three groups, data were also tested for sphericity via Mauchly's test. No assumptions underlying parametric testing were violated. Accordingly, parametric tests were conducted throughout this thesis. The individual statistical tests performed in each study are detailed in the relevant chapters.

CHAPTER FOUR:

CARDIORESPIRATORY FUNCTION IN PARALYMPIC ATHLETES WITH CHRONIC SPINAL CORD INJURY

4-1 Introduction

In individuals with cervical spinal cord injury (SCI) there is strong evidence for pulmonary restriction (Anke *et al.*, 1993), pulmonary obstruction (Radulovic *et al.*, 2008), and respiratory muscle weakness (Mateus *et al.*, 2007). Although inspiratory function is relatively well preserved in individuals with low cervical SCI (C5-C7), a reduction in chest wall compliance and an increase in abdominal compliance impair the ability of the diaphragm to generate pressure (Sinderby *et al.*, 1996a; Urmey *et al.*, 1986). Expiratory function, however, is more severely impaired due to dennervation of the abdominal muscles (Mateus *et al.*, 2007).

There is some evidence that respiratory function may be improved with short–term exercise training in individuals with SCI (Sheel *et al.*, 2008b). Two studies have reported small but significant increases in vital capacity (Silva *et al.*, 1998; Sutbeyaz *et al.*, 2005). However, the interventions were relatively short (6 wk) and individuals with cervical SCI were not studied. In the only investigation to have assessed respiratory function in athletes with cervical SCI who exercise chronically (Wicks *et al.*, 1983), vital capacity and forced expiratory volume in 1 s were higher than typically reported for untrained individuals with cervical SCI. It is unknown whether chronic exercise training elicits adaptations in other aspects of respiratory function in this population.

Abolition of the venous muscle pump and dennervation of the sympathetic chain ganglia in individuals with cervical SCI may increase venous blood pooling in the lower limbs and abdominal viscera (Krassioukov, 2009). The consequent decrease in venous return, in combination with a reduced circulating blood volume (Houtman *et al.*, 2000), contributes to the lower left-ventricular dimensions and mass as well as the lower stroke volume and cardiac output that have been reported for untrained individuals with cervical SCI compared to able-bodied (AB) individuals (de Groot *et al.*, 2006; Kessler *et al.*, 1986).

Only one study has assessed cardiac structure and function in response to exercise training in individuals with cervical SCI (Nash *et al.*, 1991). That study found a reversal of

myocardial atrophy, whereby left-ventricular mass increased by 35% after 6 months of electrically-stimulated cycling exercise (Nash *et al.*, 1991). In the AB population, chronic exercise training is associated with morphological changes in the heart, including increases in left-ventricular chamber size, wall thickness, and mass (Spirito *et al.*, 1994). It is unknown whether chronic upper-body exercise training provides sufficient stress to counteract the cardiac atrophy in individuals with cervical SCI.

Wheelchair rugby is the only high-intensity sport designed specifically for individuals with cervical SCI. As such, Paralympic wheelchair rugby players are an ideal population in which to assess the chronic effects of exercise training on cardiorespiratory function in cervical SCI. Furthermore, an understanding of cardiorespiratory function in wheelchair rugby players is of interest to those involved in disability sport and those who use exercise as a tool during rehabilitation. Thus, the aims of this study were to describe resting cardiorespiratory function in a group of highly-trained Paralympic wheelchair rugby players and to compare the data with an AB control group.

4-2 Methods

4-2.1 Participants

Twelve highly-trained athletes with traumatic cervical SCI (10 male) and 12 recreationally-active AB individuals (10 male) matched for age (mean ± SD 30.0 ± 5.2 vs. 28.2 ± 5.7 y), stature (1.75 ± 0.13 vs. 1.73 ± 0.07 m), and body mass (66.5 ± 15.1 vs. 71.3 ± 9.2 kg) volunteered to participate in the study. The participants with SCI were members of the Great Britain wheelchair rugby squad and were 9.4 ± 4.0 y post-injury. They had ≥3 years of competitive wheelchair rugby experience, and were undertaking ≥15 h per week of endurance and resistance training. The participants with SCI were classified using the International Standards for Neurological Classification of Spinal Cord Injury (ASIA, 2003) and the International Wheelchair Rugby Federation (IWRF) classification

system (Duffield and Hart, 2008). Of the 12 participants with SCI, 11 had complete tetraplegia (C5-C7, ASIA A) and 1 had incomplete tetraplegia (C5-C6, ASIA B). The following IWRF functional classifications were represented: 0.5 (n = 1), 1 (n = 3), 1.5 (n = 2), 2 (n = 2), and 2.5 (n = 4). None of the participants smoked or had a history of acute or chronic cardiopulmonary disease. The study was approved by the Brunel University Research Ethics Committee and all participants provided written informed consent.

4-2.2 Pre-test preparation

Participants were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 2 h postprandial, and to avoid strenuous exercise in the 24 h before testing. The participants were also asked to refrain from caffeine and alcohol for 12 h and 24 h before testing, respectively. On arrival at the laboratory, the participants with SCI were asked to void their bladder to minimize the risk of autonomic dysreflexia (Krassioukov, 2009). Participants with SCI self-reported stature and completed a medical questionnaire relating to their injury.

4-2.3 Experimental procedures

Pulmonary function: Pulmonary volumes, capacities, and flows were assessed using spirometry and body plethysmography (Zan 530; Oberthulba, Würzburg, Germany) as described in section 3-2.1. First, airway resistance (*R*aw) during regular tidal breathing was measured between flow rates of ± 0.5 $L \cdot s^{-1}$ (Lord and Edwards, 1978). Next, functional residual capacity (FRC), slow and forced vital capacity (VC), forced expiratory volume in 1 second (FEV_1), peak expiratory flow (PEF), peak inspiratory flow (PIF), and maximal voluntary ventilation in 12 s (MVV_{12}) were assessed according to American Thoracic Society/European Respiratory Society guidelines (Miller *et al.*, 2005b; Wanger *et al.*, 2005) and adapted for SCI (Kelley *et al.*, 2003). Total lung capacity (TLC), inspiratory capacity (IC), inspiratory reserve volume (IRV), expiratory reserve volume (ERV) and residual volume (RV) were derived from the pulmonary function measurements.

Maximal inspiratory pressure ($P_{I,max}$) and maximal expiratory pressure ($P_{E,max}$) were assessed using a hand-held pressure metre (MicroRPM; Micro Medical Ltd, Kent, UK) as described in section 3-2.2. A minimum of three and a maximum of eight manoeuvres were performed at 30 s intervals, and the maximum of three measurements that varied by <10% was recorded (Green *et al.*, 2002).

Inspiratory muscle function: Gastric pressure (P_{ga}) and oesophageal pressure (P_{oe}) were measured using two latex balloon-tipped catheters (no. 47-9005; Ackrad Labs, Cooper Surgical, Berlin, Germany) as described in section 3-2.3. Transdiaphragmatic pressure (P_{di}) was obtained online by subtracting P_{oe} from P_{ga}. Magnetic stimuli were delivered to the phrenic nerve roots using two 45 mm figure-of-eight coils, each of which was powered by a mono-pulse magnetic stimulator (Magstim 200; Magstim, Whitland, UK) as described in section 3-2.4. All twitches were performed from FRC at 100% of each stimulator's power output. Three stimulations, each separated by 30 s, were delivered to the phrenic nerves. $P_{di,tw}$, $P_{oe,tw}$ and $P_{ga,tw}$ amplitudes (peak minus baseline) were analyzed for each of the three stimulations and the mean was recorded.

Transdiaphragmatic pressure swings and ventilatory indices were assessed during 2 min of tidal breathing. The diaphragm pressure-time index (PTI_{di}) was calculated as the product of the tidal P_{di} swing / $P_{di,max}$ and the fractional inspiratory time (T_I/T_{TOT}) (Bellemare and Grassino, 1982), where $P_{di,max}$ is the P_{di} (baseline to peak) recorded during a maximal Müeller manoeuvre. Minute ventilation ($\dot{V}_E$), tidal volume (V_T), respiratory frequency (f_R), and inspiratory time (T_I) were assessed using an ultrasonic flow metre (Birmingham Flowmetrics Ltd., Birmingham, UK).

Cardiovascular function: 2-D echocardiography was performed by a single sonographer using a commercially available ultrasound system (Vivid 7; GE Medical, Horton, Norway) as described in section 3-2.7. Participants transferred to an echocardiography table and rested in the left-lateral decubitus position for 5 min. Left ventricular end-systolic volume (ESV), end-diastolic volume (EDV), ejection fraction (EF), and stroke volume (SV) were determined from the apical 4-chamber

view using the modified single plane Simpson's method. Heart rate (HR) was recorded simultaneously to echocardiographic images using a 3-lead electrocardiogram. Cardiac output ($\dot{Q}$) was calculated as the product of HR and SV. Left-ventricular inflow velocities during early (E) and late (A) diastole were assessed using pulsed-wave Doppler at the mitral leaflet tips. Mitral annular velocities during systole (S´), early diastole (E´), and late diastole (A´) were assessed using pulsed-wave tissue Doppler imaging of the septal wall at the level of the mitral annulus. Five consecutive cardiac cycles were recorded at the end of a tidal expiration and the mean value was recorded for each parameter. Left ventricular mass (LVM) was calculated according to Devereux *et al.* (1986).

Systolic (SBP) and diastolic (DBP) blood pressure were calculated from an arterial pressure wave measured continuously with a finger cuff (Finometer Pro; Finapress Medical Systems BV, Smart Medical, The Netherlands) during 5 min of rest in the seated position. The arterial wave form was sampled at 100 Hz (micro 1401 mkII; Cambridge Electronic Design, UK) and acquired using computer software (Spike 2 version 7; Cambridge Electronic Design, UK). Mean arterial pressure (MAP) was calculated as DBP plus one-third pulse pressure (SBP−DBP).

4-2.4 Statistics

Group mean differences for participant characteristics and absolute values for cardiorespiratory function were analyzed using independent samples *t*-tests. Associations between continuous outcome variables were assessed using Pearson's product-moment correlation. Statistical significance was set at $p < 0.05$. Data are reported as means ± SD. Statistical analyses were performed using SPSS 16.0 for Windows (SPSS Inc., an IBM Company, Chicago, IL, USA).

4-3 Results

4-3.1 Pulmonary function

Group mean values for pulmonary function are shown in Table 4-1. Values for AB were within normal limits. Values for TLC, IC, and VC were lower in SCI compared to AB ($p < 0.01$; see also Fig 4-1). ERV was lower ($p = 0.003$) whereas RV was higher ($p = 0.022$) in SCI, hence there was no difference in FRC ($p = 0.55$). Furthermore, FEV_1, PEF, PIF, and MVV_{12} were lower in SCI ($p < 0.013$). There were no differences in *R*aw between groups. $P_{I,max}$ was not different between groups ($p = 0.41$), but $P_{E,max}$ was lower in SCI ($p = 0.004$).

Table 4-1 Pulmonary function

	AB	SCI
TLC, L	6.40 ± 0.67	5.15 ± 1.15*
	(96 ± 6)	(76 ± 8)
FRC, L	3.43 ± 0.61	3.25 ± 0.87
	(106 ± 26)	(100 ± 19)
RV, L	1.17 ± 0.28	1.77 ± 0.88*
	(69 ± 16)	(108 ± 47)
IC, L	3.21 ± 0.57	2.32 ± 0.53*
	(90 ± 16)	(64 ± 10)
IRV, L	2.41 ± 0.50	1.73 ± 0.52*
ERV, L	1.10 ± 2.13	1.10 ± 0.35*
	(139 ± 23)	(65 ± 18)
RV/TLC	18.5 ± 4.4	33.9 ± 11.9*
	(70 ± 16)	(137 ± 48)
VC, L	5.17 ± 0.70	3.28 ± 0.82*
	(104 ± 8)	(64 ± 8)
FEV_1, L	4.28 ± 0.61	2.89 ± 0.72*
	(105 ± 10)	(69 ± 10)
FEV_1/VC, %	82.8 ± 5.7	85.3 ± 10.4
	(101 ± 8)	(103 ± 12)
PEF, $L \cdot s^{-1}$	8.4 ± 1.3	5.7 ± 1.6*
	(90 ± 13)	(58 ± 13)
PIF, $L \cdot s^{-1}$	7.1 ± 1.9	5.1 ± 1.1*
	(156 ± 40)	(104 ± 26)
MMEF,	4.5 ± 1.1	3.3 ± 1.1*
	(95 ± 22)	(66 ± 23)
MVV, $L \cdot min^{-1}$	157 ± 29	100 ± 26*
	(91 ± 15)	(58 ± 13)
*R*aw, $hPa \cdot s \cdot L^{-1}$	0.29 ± 0.09	0.33 ± 0.12
	(99 ± 31)	(114 ± 41)
s*R*aw, $hPa \cdot s \cdot L^{-1}$	0.95 ± 0.22	1.06 ± 0.50
	(103 ± 26)	(112 ± 45)
$P_{I,max}$, cmH_2O	−109 ± 26	−97 ± 34
	(104 ± 30)	(88 ± 29)
$P_{E,max}$, cmH_2O	109 ± 16	64 ± 22*
	(104 ± 30)	(47 ± 16)

Definition of abbreviations: TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; IC, inspiratory capacity; IRV, inspiratory reserve volume; ERV, expiratory reserve volume; VC, vital capacity; FEV_1, forced expiratory volume in 1 s; FEV_1:FVC, ratio of forced expiratory volume in 1 s to forced vital capacity; PEF, peak expiratory flow; PIF, peak inspiratory flow; MMEF, mean-mid expiratory flow; MVV, maximum voluntary ventilation in 12 s; *R*aw, airway resistance; s*R*aw, specific airway resistance; $P_{I,max}$, maximal inspiratory pressure; $P_{E,max}$, maximal expiratory pressure. Values in parentheses are percent of AB predicted values for lung function (Quanjer *et al.*, 1993), MVV (Grimby and Sóderholm, 1963), mouth pressures (Bruschi *et al.*, 1992), and airway resistance (Quanjer, 1983). Predicted values for ERV and IC were derived from differences between corresponding predicted values of FRC and RV, and between TLC and FRC, respectively (Quanjer *et al.*, 1993). Values are means ± SD for SCI (n = 12) and AB (n = 12).
*Significantly different vs. AB ($p < 0.05$).

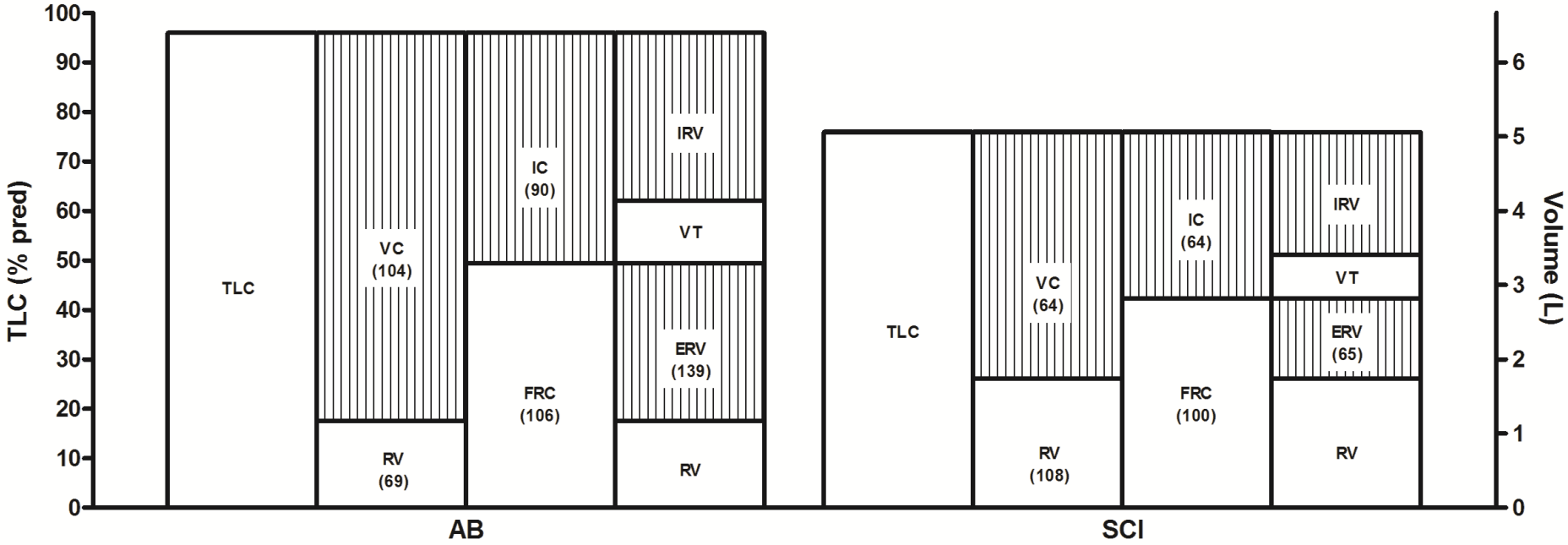

Fig 4-1 Static lung volumes and capacities for able-bodied (AB; n = 12) and cervical spinal cord injury (SCI; n = 12).
Values in parentheses are percent of AB predicted values for lung function (Quanjer et al., 1993). Note the elevated RV and smaller TLC, VC, IC, IRV, and ERV in SCI. TLC, total lung capacity; VC, vital capacity; RV, residual volume; IC, inspiratory capacity; FRC, functional residual capacity; IRV, inspiratory reserve volume; VT, tidal volume; ERV, expiratory reserve volume.

4-3.2 Inspiratory muscle function

Group mean values for inspiratory muscle function are shown in Table 4-2. $P_{di,max}$ and $P_{di,tw}$ were lower in SCI compared to AB ($p < 0.001$) primarily due to a lower P_{ga} contribution ($p < 0.001$). Tidal pressure swings were not different between groups ($p > 0.46$). Consequently, P_{di} / $P_{di,max}$ during resting breathing was elevated in SCI ($p < 0.015$). However, neither PTI_{di} nor ventilatory indices during resting breathing were different between groups ($p > 0.34$). In SCI, percent predicted TLC correlated with $P_{di,max}$ ($r = 0.74$, $p = 0.036$) but not $P_{I,max}$ ($r = 0.12$, $p = 0.78$).

Table 4-2 Respiratory muscle function and ventilatory indices

	AB	SCI
Respiratory muscle function		
P_{di} / $P_{di,max}$	0.08 ± 0.19	0.12 ± 0.36*
P_{di}, cmH_2O	9.8 ± 4.6	10.3 ± 1.5
P_{ga}, cmH_2O	6.4 ± 2.7	6.8 ± 2.1
P_{oe}, cmH_2O	–4.6 ± 1.7	–4.0 ± 1.3
$P_{di,max}$, cmH_2O	128 ± 20	93 ± 21*
PTI_{di}	0.051 ± 0.011	0.055 ± 0.025
C_{dyn}, $L{\cdot}cmH_2O^{-1}$	0.182 ± 0.059	0.142 ± 0.049
$P_{di,tw}$, cmH_2O	25.0 ± 2.6	15.5 ± 3.7*
$P_{ga,tw}$, cmH_2O	12.9 ± 0.8	6.1 ± 3.0*
$P_{oe,tw}$, cmH_2O	–12.1 ± 2.8	–9.4 ± 2.7
Ventilatory indices		
$\dot{V}_E$, $L{\cdot}min^{-1}$	7.5 ± 2.6	6.7 ± 1.0
f_R, $breaths{\cdot}min^{-1}$	13.7 ± 3.9	15.5 ± 4.7
V_T, L	0.57 ± 0.23	0.47 ± 0.17
T_I/T_{TOT}	0.50 ± 0.07	0.46 ± 0.08
V_T/T_I, $L{\cdot}s^{-1}$	0.30 ± 0.09	0.28 ± 0.04

Definition of abbreviations: P_{di}, transdiaphragmatic pressure; P_{ga}, gastric pressure; P_{oe}, oesophageal pressure; PTI_{di}, diaphragm pressure-time index; C_{dyn}, dynamic chest-wall compliance; $\dot{V}_E$, minute ventilation; f_R, respiratory frequency; V_T, tidal volume; T_I/T_{TOT}, fractional inspiratory time; V_T/T_I, tidal inspiratory flow; tw, twitch. Values are means ± SD for AB (n = 8) and SCI (n = 8). The participants were representative of their respective groups (i.e., no differences in descriptive characteristics or pulmonary function). *Significantly different vs. AB ($p < 0.05$).

4-3.3 Cardiovascular function

Group mean values for cardiovascular function are shown in Table 4-3. Participants with SCI demonstrated lower LVM (p = 0.030), left ventricular internal diameter during systole (p = 0.003) and diastole (p = 0.030), and EDV (p = 0.004). Consequently, $\dot{Q}$ (p = 0.004), SV (p = 0.006), and EF (p = 0.028) were lower in SCI. SBP (p < 0.001), DBP (p = 0.017), and hence MAP (p = 0.001), were also lower in SCI.

Table 4-3 Cardiovascular function

	AB	SCI
Dimensions		
IVSs, mm	14.5 ± 2.0	13.4 ± 2.1
LVIDs, mm	32.5 ± 3.4	28.1 ± 2.6*
LVPWs, mm	14.5 ± 2.2	13.8 ± 1.6
IVSd, mm	10.3 ± 2.0	10.4 ± 1.9
LVIDd, mm	50.6 ± 3.7	46.9 ± 3.8*
LVPWd, mm	9.5 ± 1.4	10.1 ± 2.8
EDV, ml	127 ± 18	103 ± 16*
ESV, ml	49 ± 8	46 ± 6
LVM, $g \cdot m^2$	103 ± 21	81 ± 22*
Systolic function		
$\dot{Q}$, $L \cdot min^{-1}$	4.3 ± 0.9	3.1 ± 0.9*
SV, ml	72 ± 9	56 ± 14*
EF, %	57 ± 6	51 ± 6*
S′, $cm \cdot s^{-1}$	5.9 ± 0.8	6.0 ± 1.0
Diastolic function		
E, $cm \cdot s^{-1}$	7.8 ± 1.1	7.9 ± 1.8
A, $cm \cdot s^{-1}$	4.2 ± 0.7	3.9 ± 0.4
E:A	1.9 ± 0.4	2.0 ± 0.5
E′, $cm \cdot s^{-1}$	10.1 ± 1.9	9.7 ± 1.7
A′, $cm \cdot s^{-1}$	6.1 ± 1.1	5.2 ± 0.8
Haemodynamics		
MAP, mmHg	84 ± 4	64 ± 14*
SBP, mmHg	128 ± 11	92 ± 18*
DBP, mmHg	62 ± 6	48 ± 13*
HR, bpm	57 ± 10	52 ± 9

Definition of abbreviations: IVSs, inter-ventricular septal thickness in systole; LVIDs, left ventricular internal diameter in systole; LVPWs, left ventricular posterior wall thickness in systole; IVSd, inter-ventricular septal thickness in diastole; LVIDd, left ventricular internal diameter in diastole; LVPWd, left ventricular posterior wall thickness in diastole; EDV, end diastolic volume; ESV, end systolic volume; LVM, left ventricular mass ($0.8\ (1.04\ ([LVIDd + LVPWd + IVSd]^3 - [LVIDd]^3)) + 0.6$) (Devereux *et al.*, 1986);$\dot{Q}$, cardiac output; SV, stroke volume; EF, ejection fraction; S′, systolic myocardial tissue velocity; E, early transmitral filling velocity; A, late transmitral filling velocity; E′, early diastolic myocardial tissue velocity; A′, late diastolic myocardial tissue velocity; MAP, mean arterial pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate. Values are means ± SD for SCI (n = 12) and AB (n = 12). *Significantly different vs. AB (p < 0.05).

4-4 Discussion

The aim of this chapter was to describe resting cardiorespiratory function in a group of highly-trained Paralympic athletes with cervical SCI and to compare the data with an AB control group. Compared to AB controls, highly-trained athletes with cervical SCI exhibited pulmonary restriction, but not obstruction, as evidenced by the lower TLC and similar *R*aw, respectively. Global inspiratory muscle strength ($P_{I,max}$) was similar between groups, but expiratory muscle strength ($P_{E,max}$) and diaphragm muscle function ($P_{di,max}$ and $P_{di,tw}$) were impaired in SCI. There was also evidence of cardiac atrophy and reduced systolic function in SCI, as demonstrated by the lower LVM and EF, respectively.

4-4.1 Pulmonary function

In line with previous findings in untrained individuals with cervical SCI [e.g., (Anke *et al.*, 1993)], TLC and VC were lower than normal in highly-trained athletes with cervical SCI. The percent predicted VC, however, was higher than typically reported for untrained individuals with low cervical SCI (Linn *et al.*, 2001). The higher VC in the current study was likely due to the superior expiratory muscle function, as demonstrated by the elevated $P_{E,max}$ and PEF. The enhanced expiratory muscle function is attributed to a training-induced increase in strength of the accessory muscles of expiration (Estenne *et al.*, 1989). Although the data suggest that pulmonary function improves with training, longitudinal data are needed to support this postulate.

To the author's knowledge, this is the first study to demonstrate normal levels of airway resistance (s*R*aw and *R*aw) in individuals with cervical SCI. Studies in untrained individuals with cervical SCI have documented elevated levels of airway resistance and have attributed this finding to sympathetic dennervation of airway smooth muscle (Radulovic *et al.*, 2008). That airway resistance was normal in the current study suggests that the hyperpnoea of chronic exercise training may have a protective effect on airway smooth muscle. In this regard, even small

amounts of airway stretch have been shown to disturb the bronchiolar latch state (i.e. reduced airway smooth muscle crossbridge formation) and promote relaxation of the airway smooth muscle (Fredberg *et al.*, 1997).

4-4.2 Inspiratory muscle function

The pressure generating capacity of the diaphragm in response to a maximal Müeller manoeuvre ($P_{di,max}$) and magnetic stimulation of the phrenic nerves ($P_{di,tw}$) was lower in SCI compared to AB controls, but similar to values reported for untrained individuals with cervical SCI (Hart *et al.*, 2005; Sinderby *et al.*, 1996a). The impaired pressure generating capacity of the diaphragm in cervical SCI appears to be due to elevated abdominal compliance, as evidenced by the significantly lower gastric contribution to P_{di}. In contrast, the P_{di} swing during tidal breathing was not different between groups. Accordingly, P_{di} / $P_{di,max}$ was higher in cervical SCI, implying that the relative pressure generating capacity of the diaphragm is elevated in this population. Nevertheless, there did not appear to be any functional consequences as the pressure-time index for the diaphragm did not exceed the "critical" threshold level for fatigue (Bellemare and Grassino, 1982) and none of the participants reported dyspnoea at rest. The similarity in diaphragm function between the highly-trained athletes in the current study and previously reported values for untrained individuals (Hart *et al.*, 2005; Sinderby *et al.*, 1996a) suggests that chronic exercise training does not improve the force generating capacity of the diaphragm. Rather, an elevated abdominal compliance is likely the overriding factor in determining the force generating capacity of the diaphragm in this population.

In the participants with cervical SCI, most of the variance in TLC was accounted for by $P_{di,max}$ (~55%) rather than $P_{I,max}$ (<2%). Thus, the pulmonary restriction observed in SCI was primarily due to weakness of the diaphragm than to weakness of the additional muscles of inspiration that contribute to $P_{I,max}$, such as the scalenes, sternocleidomastoids, and other neck muscles. In contrast, AB individuals progressively activate the scalenes and sternocleidomastoids

as lung volume increases to TLC (Raper *et al.*, 1966). The difference in response can be explained by the action of the neck muscles on the thoracic cavity. In AB, the neck muscles displace the upper rib-cage cranially thereby causing an increase in anteroposterior diameter (De Troyer and Estenne, 1984). In cervical SCI, a stiff rib-cage causes a decrease in tidal excursion of the upper rib-cage during inspiration and in some instances a paradoxical inwards motion of the upper rib-cage (Urmey *et al.*, 1986). Consequently, the increase in size of the thoracic cavity during a maximal inspiration to TLC in cervical SCI is primarily due to lower rib-cage expansion resulting from the appositional and insertional forces of the diaphragm acting through the zone of apposition (Urmey *et al.*, 1986).

4-4.3 Cardiovascular function

This is the first study to assess cardiac structure and function in athletes with cervical SCI. Cardiac dimensions and LVM were smaller in athletes with cervical SCI compared to AB controls, but similar to values reported in untrained individuals with cervical SCI (de Groot *et al.*, 2006; Kessler *et al.*, 1986). The similar LVM between trained and untrained SCI suggests that the volume of exercise training undertaken by the participants in the current study was insufficient to attenuate the degree of cardiac atrophy that is known to occur in this population (Kessler *et al.*, 1986). In this regard, it was recently reported that peak oxygen uptake was only 1.20 $L \cdot min^{-1}$ for highly-trained athletes with cervical SCI (Taylor *et al.*, 2010). Thus, the low LVM noted in the present study may represent an adaptation to the low metabolic demand in individuals with cervical SCI.

In addition to the aforementioned differences in cardiac dimensions and mass, EDV, $\dot{Q}$, SV, and EF were also lower in SCI compared to AB. There were, however, no differences in ESV or S' between groups; this suggests that the lower SV was explained by the lower EDV. The reason for the lower EDV in SCI is likely due to a reduction in cardiac preload, resulting from impaired sympathetic vasoconstrictor function below the lesion (Krassioukov, 2009) and reduced circulating blood volume (Houtman *et al.*, 2000). In agreement with findings from previous

studies in untrained individuals with cervical SCI (de Groot *et al.*, 2006; Eysmann *et al.*, 1995), diastolic function was not different between SCI and AB. The normal diastolic function in SCI may be explained by a reduction in left atrial size (i.e., cardiac atrophy), which in the face of a reduced venous return may result in similar left atrial pressures for SCI compared to AB. Although the current study did not include a measure of left atrial pressure, mitral inflow and mitral annular velocities during early diastole were similar in SCI versus AB. That a strong positive relationship exists between diastolic dysfunction and risk of heart failure (Grossman, 1990), suggests that the cardiac atrophy noted in the present study may be a morphological adaptation that serves to maintain normal diastolic function.

4-5 Conclusion

Compared to AB controls, highly-trained athletes with cervical SCI had a restrictive pulmonary defect, impaired diaphragm and expiratory muscle function, and low left ventricular mass and ejection fraction. These findings suggest that chronic exercise training does not 'normalise' cardiorespiratory function. However, the superior pulmonary function in highly-trained athletes compared to values typically reported in untrained individuals with cervical SCI implies that chronic exercise training has the potential to improve pulmonary function in this population.

CHAPTER FIVE:

CARDIORESPIRATORY EFFECTS OF ABDOMINAL BINDING IN SPINAL CORD INJURY

5-1 Introduction

Spinal cord injury (SCI) to the lower cervical cord causes paralysis of the accessory muscles of inspiration (inspiratory intercostals) and the primary muscles of expiration (abdominals and expiratory intercostals), thereby limiting the maximal inspiratory and expiratory pressure generating capacity of these muscles (Hopman *et al.*, 1997; Mateus *et al.*, 2007). During inspiration in the seated position the abdominal contents migrate anteriorly and caudally, due to gravity and increased abdominal compliance (Goldman *et al.*, 1986a). As such, the gastric contribution to tidal inspiration is reduced and the extent to which the diaphragm can exert its influence on the chest wall through the zone of apposition is diminished (Brown *et al.*, 2006; Loring and Mead, 1982). Consequently, individuals with cervical SCI exhibit a restrictive pulmonary defect (see chapter 4).

In addition to the aforementioned impairments in pulmonary function, cervical SCI causes hypokinetic circulation and impaired cardiovascular function. Unopposed parasympathetic tone below the level of injury causes hypotension and venous pooling in the lower-body and splanchnic bed (Teasell *et al.*, 2000). Another effect of the unopposed parasympathetic tone below the level of injury is an inability to redistribute blood effectively from the lower-body and splanchnic bed (Thijssen *et al.*, 2009), which, in combination with venous pooling and a reduction in circulating blood volume (Houtman *et al.*, 2000), is likely to reduce cardiac pre-load and end-diastolic volume (Hopman *et al.*, 1992; Kessler *et al.*, 1986). As a result, resting stroke volume and cardiac output are lower in cervical SCI compared to able-bodied (AB) individuals (see chapter 4).

A potential method of restoring cardiorespiratory function in SCI is to bind the abdomen. Abdominal binders are thought to 'mimic' the abdominal muscles by reducing abdominal compliance, thereby increasing the area of diaphragmatic apposition to the rib cage and the insertional component of diaphragm action on the rib cage (Urmey *et al.*, 1986). In this regard, abdominal binding has been shown to increase vital capacity (Boaventura *et al.*, 2003; Bodin *et al.*, 2005; Estenne *et al.*, 1998; Goldman *et al.*, 1986b; Hart *et al.*, 2005), and decrease functional

residual capacity and residual volume in patients with cervical SCI (Hart *et al.*, 2005). Abdominal binding has also been used in the acute phase after SCI to attenuate the degree of orthostatic hypotension and aid venous return (Huang *et al.*, 1983). However, a recent review of the literature concluded that whilst abdominal binding has the potential to improve cardiorespiratory function in individuals with SCI, the wide range of methodologies and participant inclusion criteria render conclusions difficult to interpret (Wadsworth *et al.*, 2009). As such, there is only limited evidence to support the use of abdominal binding in individuals with cervical SCI.

In AB individuals, augmented intra-abdominal pressure by predominant diaphragmatic breathing at rest has been shown to impede femoral venous blood flow (Miller *et al.*, 2005a). More recent evidence has shown a rapid emptying of blood from the splanchnic region to the heart during ramped increases in intra-abdominal pressure (Aliverti *et al.*, 2009). The degree of emptying, however, appears to be dose-dependent as a critical intra-abdominal pressure exists past which venous splanchnic outflow is reduced (Aliverti *et al.*, 2009). Thus, a dose-response relationship may exist between the magnitude of abdominal compression and the subsequent change in cardiovascular function. This postulate has been confirmed in a porcine model (Kitano *et al.*, 1999; Vivier *et al.*, 2006), but awaits verification in humans. A dose-response relationship may also exist for pulmonary function, as over-compression of the abdomen in AB humans has been shown to impair total lung capacity, functional residual capacity, and vital capacity (Bradley and Anthonisen, 1980; DiMarco *et al.*, 1981). It seems, therefore, that any improvements in cardiorespiratory function with abdominal binding may be critically dependent upon the degree of compression applied.

Accordingly, the primary aims of the present chapter were to determine if abdominal binding improves resting cardiorespiratory function in individuals with cervical SCI, and to assess whether any such changes are dependent on the magnitude of abdominal compression. It was hypothesised that abdominal binding would improve resting pulmonary function and

cardiovascular function, and that the magnitude of improvements would be dose-dependent. A secondary aim was to compare the responses in SCI to the 'normal' responses in AB individuals.

5-2 Materials and methods

5-2.1 Participants

After ethics committee approval and written informed consent, 13 individuals with traumatic SCI (12 males; mean ± SD age 33 ± 9 y, self-report stature 1.76 ± 0.12 m, body mass 70 ± 16 kg) and 8 recreationally active AB controls (6 males, age 32 ± 8 y, stature 1.75 ± 0.12 m, body mass 66 ± 14 kg) volunteered to participate in the study. For the SCI group, only individuals with paralysis of the abdominal muscles and preserved diaphragm function were recruited. The participants with SCI were clinically stable, 10 ± 4 y post-injury, and competed at national level in wheelchair sport (11 rugby and 2 tennis). Eleven of the participants with SCI had complete tetraplegia (C_5-C_7) and two had sensory incomplete tetraplegia (C_5-C_7). None of the participants in the SCI group had ever worn an abdominal binder as an outpatient. None of the participants had ever smoked or had a history of cardiopulmonary disease. All participants were instructed to arrive at the laboratory in a rested state, at least 2 h postprandial, and to avoid strenuous exercise in the 24 h before testing. The participants were also asked to refrain from caffeine and alcohol for 12 h and 24 h before testing, respectively.

5-2.2 Experimental design

During one visit to the laboratory, each participant performed three trials in each of three conditions [unbound (UB), loose (LB), tight (TB)]. The order of trials was sequential, whereas the order of conditions was randomised. TB was defined as the largest possible change in end-expiratory gastric pressure (P_{ga}). LB was defined as the end-expiratory P_{ga} midway between UB and TB. An inflatable rubber reservoir with a known volume of air was connected to a digital

pressure metre (model C9553; JMW Ltd., Harlow, UK), and placed between the binder and the anterior abdominal wall. The relationship between end-expiratory P_{ga} and abdominal surface pressure during trial 1 was used to reproduce a similar end-expiratory P_{ga} during trials 2 and 3 without the need for a gastric balloon. Measurements in all three conditions were taken with the participants seated without additional restrictive garments. The SCI group were assessed in their normal day-chair unless otherwise stated. On arrival at the laboratory, each participant with SCI was asked to void their bladder to minimize the risk of autonomic dysreflexia (Cunningham *et al.*, 1953).

5-2.3 Binder

A commercially available binder (493R Universal Back Support; McDavid Inc, Woodridge, IL, USA) that incorporated a semi-rigid neoprene back panel including six plastic stays (100% neoprene rubber), flexible mesh side panels (90% nylon, 10% Lycra), and a flexible neoprene panel with double Velcro fastening at the front was used in the current study. Three sizes of binder were used (small, medium, large). The binder was fitted with the upper edge below the costal margin such that the binder interfered minimally with rib cage movements.

5-2.4 Experimental procedures

Trial 1

Diaphragm function: Gastric pressure (P_{ga}) and oesophageal pressure (P_{oe}) were measured using two latex balloon-tipped catheters (no. 47-9005; Ackrad Labs, Cooper Surgical, Berlin, Germany) as described in section 3-2.3. Transdiaphragmatic pressure (P_{di}) was obtained online by subtracting P_{oe} from P_{ga}. Magnetic stimuli were delivered to the phrenic nerve roots using two 45 mm figure-of-eight coils, each of which was powered by a mono-pulse magnetic stimulator (Magstim 200; Magstim, Whitland, UK) as described in section 3-2.4. Supramaximal stimulation of

the phrenic nerves was achieved in all participants, as shown by a levelling off of $P_{di,tw}$ at ~80% of stimulator power output (see also Fig 3-6). All subsequent twitches were performed at 100% of each stimulator's power output. Three 1-Hz stimulations were delivered to the phrenic nerves in each of the three conditions. $P_{di,tw}$, $P_{oe,tw}$ and $P_{ga,tw}$ amplitudes (peak minus baseline) were analyzed for each of the three stimulations and the mean was recorded.

Measures during resting breathing: The participants rested for 10 min in each condition and recordings of transpulmonary pressures and airflow were evaluated for the final 2 min as described in sections 3-2.5 and 3-2.6, respectively. To estimate the relative contributions of diaphragm and accessory inspiratory muscle contractions to the inspiratory volume excursion, pressure-time products (PTPs) were calculated by integrating P_{di} and P_{oe} over time during the period of inspiratory flow (Clanton *et al.*, 2002). The following ventilatory measurements were also collected on a breath-by-breath basis during the same 2 min: minute ventilation ($\dot{V}_E$), tidal volume (V_T), respiratory frequency (f_R), fractional inspiratory time (T_I/T_{TOT}), mean tidal inspiratory flow (V_T/T_I). Breathing discomfort (dyspnoea) was rated using Borg's modified category ratio 10 scale (Borg, 1998).

Trial 2

Pulmonary function: Pulmonary volumes, capacities, and flows were assessed using spirometry and body plethysmography (Zan 530; Oberthulba, Würzburg, Germany) as described in section 3-2.1. Functional residual capacity (FRC), slow and forced vital capacity (VC), forced expiratory volume in 1 second (FEV_1), peak expiratory flow (PEF), peak inspiratory flow (PIF), and maximal voluntary ventilation in 12 s (MVV_{12}) were assessed according to American Thoracic Society/European Respiratory Society guidelines (Miller *et al.*, 2005b; Wanger *et al.*, 2005) and adapted for SCI (Kelley *et al.*, 2003). Total lung capacity (TLC), inspiratory capacity (IC), and residual volume (RV) were derived from the pulmonary function measurements.

In UB and TB only, maximal inspiratory and expiratory mouth pressures ($P_{I,max}$ and $P_{E,max}$) were assessed using a hand-held pressure metre (MicroRPM; Micro Medical Ltd, Kent, UK) as described in section 3-2.2. A minimum of three and a maximum of eight manoeuvres were performed at 30 s intervals, and the maximum of three measurements that varied by <10% was recorded (Green *et al.*, 2002).

Finally, each participant with SCI performed three forced expiratory vital capacity (VC) manoeuvres in UB and TB, from which isovolume-flow and -P_{oe} curves were constructed (Estenne *et al.*, 1998). Specifically, absolute lung volumes in each condition were calculated, and values of expiratory flow and P_{oe} were compared at 10% intervals between 80 and 20% of the largest bound VC.

Trial 3

Cardiovascular function: 2-D echocardiography was performed by the author of this thesis using a commercially available ultrasound system (Vivid 7; GE Medical, Horton, Norway) as described in section 3-2.7. Images were acquired in the seated position after 5 min of rest in each condition. Left ventricular end-diastolic volume (EDV), end-systolic volume (ESV), ejection fraction (EF), and stroke volume (SV) were estimated from the parasternal long-axis image (Teichholz *et al.*, 1976). A 3-lead electrocardiogram recorded heart rate (HR) simultaneously to echocardiographic imaging. Cardiac output ($\dot{Q}$) was calculated as the product of SV and HR. Mitral annular velocities during systole (S´), and early (E´) and late diastole (A´) were assessed using pulsed-wave tissue Doppler imaging of the septal wall at the level of the mitral annulus. For all echocardiographic parameters, five consecutive cardiac cycles were recorded at the end of a tidal expiration and the mean was calculated.

Systolic (SBP) and diastolic (DBP) blood pressure were calculated from the arterial pressure wave measured continuously with a finger cuff (Finometer Pro; Finapress Medical Systems BV, Smart Medical, The Netherlands). Mean arterial blood pressure (MAP) was calculated as DBP plus one-third pulse pressure (SBP−DBP).

5-2.5 Statistics

Differences in measures of cardiorespiratory function between conditions (UB, LB, TB) were analysed using separate one-way repeated measures ANOVA. Where applicable, Bonferroni corrected pairwise comparisons were carried out for *post hoc* analyses. When between-condition differences were assessed in the UB and TB conditions only (i.e., $P_{I,max}$ and $P_{E,max}$), paired samples *t*-tests were used. Isovolume-flow and $-P_{oe}$ data were analysed using a two-way analysis of variance, with one within-factor for condition (UB vs. TB) and one within-factor for lung volume (% tight-bound expired VC). Due to unequal sample sizes and a consequent low statistical power no comparisons were made between SCI and AB groups. Associations between selected variables were assessed using Pearson product-moment correlation. Data are reported as means ± SD. Statistical analyses were performed using SPSS 16.0 for Windows (SPSS Inc., an IBM Company, Chicago, IL, USA), and statistical significance was set at $p < 0.05$.

5-3 Results

5-3.1 Pulmonary function in spinal cord injury

Group mean values are shown in Table 5-1. FRC and RV decreased in TB vs. UB, whereas IC, IRV, and VC increased (see also Fig 5-1). For LB, there was a decrease in FRC as well as increases in IC, IRV and VC; these changes were in-between those for TB. PEF, FEV_1 and $P_{E,max}$ increased in TB vs. UB. Although $P_{I,max}$ was unchanged with abdominal binding, there was a trend towards an increase in $P_{di,max}$ during the $P_{I,max}$ manoeuvre in TB vs. UB (134 ± 24 vs. 121 ± 34, p = 0.11). There were also increases in maximal expiratory flow and P_{oe} throughout the VC during forced expirations in TB (p = 0.049 and 0.015, respectively; Fig 5-2).

Table 5-1 Pulmonary function with abdominal binding (unbound [UB], loose-bound [LB], tight-bound [TB]) in participants with spinal cord injury

	UB	LB	TB
TLC, L	5.29 ± 1.19	5.30 ± 1.04	5.18 ± 1.09
	(78 ± 10)	(76 ± 9)	(74 ± 9)
FRC, L	3.31 ± 0.85	2.97 ± 0.93*	2.56 ± 0.84*†
	(102 ± 17)	(90 ± 22)	(78 ± 19)
RV, L	1.77 ± 0.83	1.54 ± 0.76*	1.19 ± 0.78*†
	(106 ± 43)	(89 ± 41)	(68 ± 41)
IC, L	2.32 ± 0.53	2.55 ± 0.52*	2.79 ± 0.56*
	(66 ± 11)	(72 ± 14)	(79 ± 12)
IRV, L	1.69 ± 0.51	1.92 ± 0.52*	2.18 ± 0.47*†
ERV, L	1.09 ± 0.43	1.05 ± 0.42	1.06 ± 0.37
	(72 ± 33)	(69 ± 28)	(70 ± 22)
VC, L	3.41 ± 0.92	3.60 ± 0.85*	3.84 ± 0.85*†
	(68 ± 16)	(71 ± 16)	(76 ± 13)
FEV_1, L	2.95 ± 0.72	3.03 ± 0.65	3.21 ± 0.64*†
	(71 ± 17)	(77 ± 16)	(79 ± 13)
FEV_1/FVC, %	84.5 ± 10.3	85.0 ± 9.2	84.4 ± 8.9
	(103 ± 11)	(104 ± 10)	(104 ± 10)
PEF, $L \cdot s^{-1}$	5.8 ± 1.6	6.0 ± 1.5	6.1 ± 1.7*†
	(59 ± 18)	(63 ± 16)	(65 ± 17)
PIF, $L \cdot s^{-1}$	5.2 ± 1.1	5.5 ± 1.3*	5.2 ± 1.3
	(110 ± 36)	(121 ± 39)	(114 ± 39)
MVV, $L \cdot min^{-1}$	101 ± 26		103 ± 25
	(65 ± 17)		(64 ± 16)
$P_{I,max}$, cmH_2O	–95 ± 33		–100 ± 33
	(87 ± 29)		(92 ± 30)
$P_{E,max}$, cmH_2O	66 ± 23		80 ± 22*
	(50 ± 15)		(61 ± 16)

Definition of abbreviations: TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume; IC, inspiratory capacity; IRV, inspiratory reserve volume; ERV, expiratory reserve volume; VC, vital capacity; FEV_1, forced expiratory volume in 1 s; PEF, peak expiratory flow; PIF, peak inspiratory flow; MVV, maximum voluntary ventilation in 12 s; $P_{I,max}$, maximal inspiratory pressure at the mouth; $P_{E,max}$, maximal expiratory pressure at the mouth. Values in parentheses are percent of able-bodied predicted values for pulmonary volumes, capacities and flows (Quanjer *et al.*, 1993); MVV (Grimby and Sóderholm, 1963); and respiratory pressures (Bruschi *et al.*, 1992). Predicted values for ERV and IC were derived from differences between corresponding predicted values for FRC and RV, and between TLC and FRC, respectively (Quanjer *et al.*, 1993). Values are means ± SD for 13 participants. *Significantly different vs. UB ($p < 0.05$); † significantly different vs. LB ($p < 0.05$).

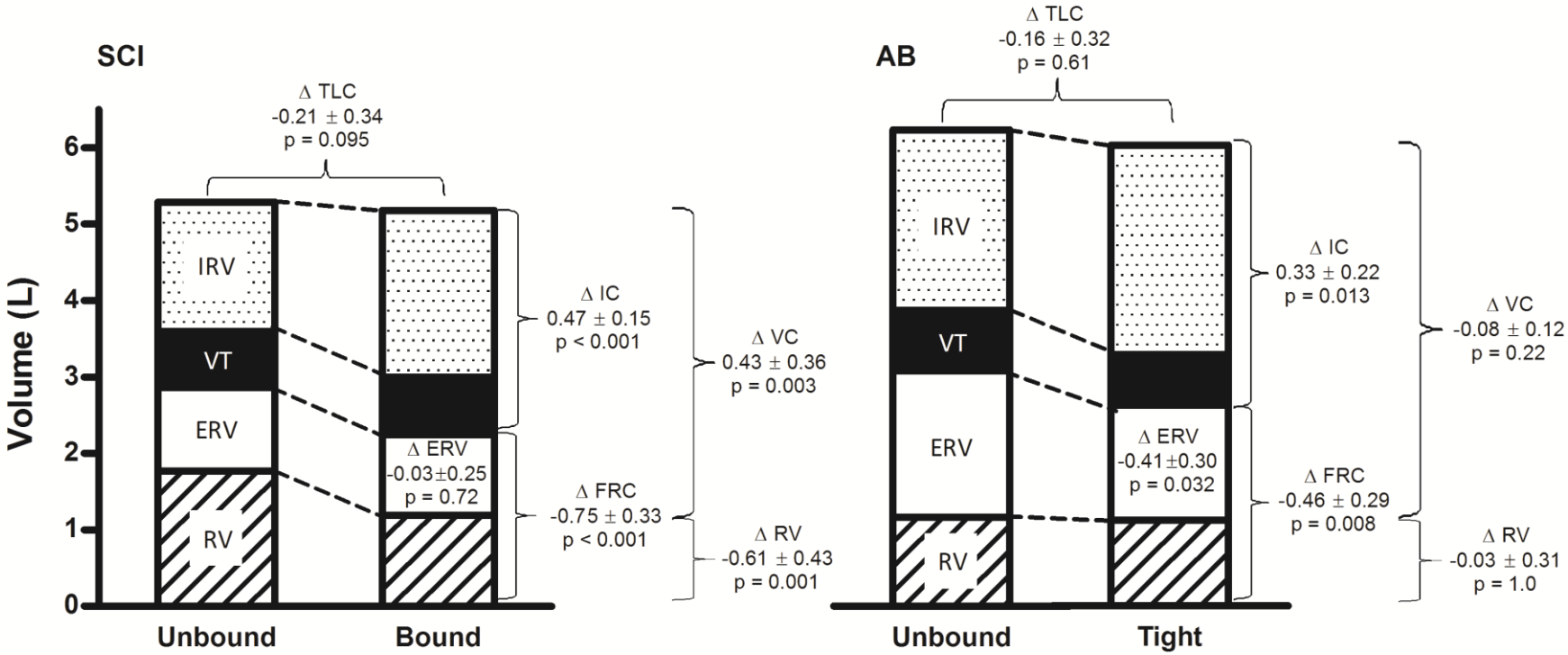

Fig 5-1 Static lung volumes and capacities in unbound and tight-bound conditions for SCI (left panel; n = 13) and AB (right panel; n = 8).
Note the decrease in RV and FRC, and the consequent increase in IC and VC in tight-bound vs. unbound for the SCI group. Also note the decrease in FRC and increase in IC in tight bound vs. unbound in the AB group; the increase in IC, however, was at the expense of a decrease in ERV, such that VC remained unchanged. SCI, spinal cord injury; AB, able-bodied; IRV, inspiratory lung volume; VT, tidal volume; ERV, expiratory reserve volume; RV, residual volume; TLC, total lung volume; IC, inspiratory capacity; FRC, functional residual capacity; VC, vital capacity; Δ, binding-induced change (i.e., mean difference ± SD between values in unbound and tight-bound).

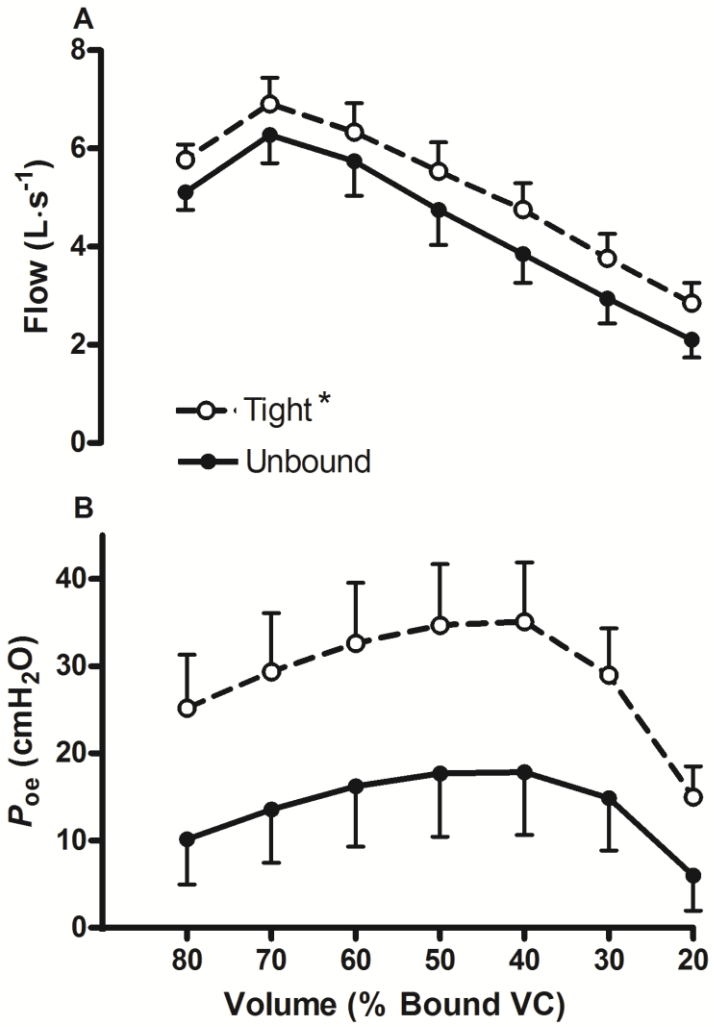

Fig 5-2 Group mean ± SD values of maximal expiratory flow (A) and oesophageal pressure (P_{oe}; B) at 10% intervals between 80 and 20% of the bound VC before and after tight binding for SCI (n = 13).
*SCI, spinal cord injury. *p < 0.05, significant main effect for flow and P_{oe}.*

5-3.2 Respiratory muscle function in spinal cord injury

Group mean values are shown in Table 5-2. Data are presented for 12 participants because one participant was unable to tolerate the balloon catheters. End-expiratory P_{ga} was 9.4 ± 0.3 cmH_2O in UB, 12.7 ± 5.6 cmH_2O in LB ($p < 0.001$ vs. UB), and 17.4 ± 5.2 cmH_2O in TB ($p < 0.001$ vs. UB and LB). Abdominal surface pressure was 185 ± 60 cmH_2O in LB and 340 ± 54 cmH_2O in TB ($p < 0.001$). Tidal inspiratory P_{di} and PTP_{di} increased in TB vs. UB and TB vs. LB. The increased tidal P_{di} was

primarily due to an increased gastric contribution, as demonstrated by the positive correlation between the percent change in tidal P_{di} and the percent change in tidal P_{ga} in TB vs. UB (r = 0.74, p = 0.004). $P_{di,tw}$ and its component parts ($P_{ga,tw}$ and $P_{oe,tw}$) were greater in TB vs. UB (see also Fig 5-3). Twitch pressures were also elevated in LB versus UB; these changes were approximately half those in TB. Ventilatory indices during resting breathing did not differ across conditions. None of the participants reported dyspnoea in any condition.

Table 5-2 Respiratory muscle function and ventilatory indices with abdominal binding (unbound [UB], loose-bound [LB], tight-bound [TB]) in participants with spinal cord injury

	UB	LB	TB
Tidal pressure swings			
P_{di}, cmH_2O	9.8 ± 1.5	12.1 ± 2.3*	13.7 ± 3.0*
P_{ga}, cmH_2O	6.7 ± 2.0	8.8 ± 2.0	11.0 ± 3.1*†
P_{oe}, cmH_2O	4.3 ± 2.0	4.1 ± 2.7	3.8 ± 3.1
PTP_{di}, $cmH_2O{\cdot}s{\cdot}min^{-1}$	138 ± 19	158 ± 53	186 ± 59*†
PTP_{oe}, $cmH_2O{\cdot}s{\cdot}min^{-1}$	46 ± 13	37 ± 25	36 ± 35
PTP_{di} / PTP_{oe}	3.1 ± 0.8	3.0 ± 4.1	4.1 ± 3.1
Evoked twitches			
$P_{di,tw}$, cmH_2O	14.5 ± 4.6	17.8 ± 6.8*	20.5 ± 6.8*
$P_{ga,tw}$, cmH_2O	5.7 ± 3.2	6.6 ± 4.2	8.1 ± 3.7*
$P_{oe,tw}$, cmH_2O	–8.7 ± 3.8	–11.2 ± 5.7*	–12.8 ± 5.5*
Ventilatory indices			
$\dot{V}_E$, $L{\cdot}min^{-1}$	7.1 ± 1.7	6.7 ± 1.0	6.7 ± 1.3
V_T, L	0.57 ± 0.22	0.50 ± 0.14	0.49 ± 0.13
f_R, $breaths{\cdot}min^{-1}$	15.5 ± 4.2	15.3 ± 3.1	15.7 ± 3.1
T_I/T_{TOT}	0.48 ± 0.08	0.46 ± 0.12	0.46 ± 0.07
V_T/T_I, $L{\cdot}s^{-1}$	0.29 ± 0.05	0.28 ± 0.04	0.29 ± 0.04

Definition of abbreviations: P_{di}, transdiaphragmatic pressure; P_{ga}, gastric pressure; P_{oe}, oesophageal pressure; PTP_{di}, transdiaphragmatic pressure integrated over time during the period of inspiratory flow; PTP_{oe}, oesophageal pressure integrated over time during the period of inspiratory flow; $P_{di,tw}$, twitch transdiaphragmatic pressure; $P_{ga,tw}$, twitch gastric pressure; $P_{oe,tw}$, twitch oesophageal pressure; $\dot{V}_E$, minute ventilation; V_T, tidal volume; f_R, respiratory frequency; T_I/T_{TOT}, fractional inspiratory time; V_T/T_I, mean tidal inspiratory flow. Values are means ± SD for 12 participants. *Significantly different vs. UB ($p < 0.05$); † significantly different vs. LB ($p < 0.05$).

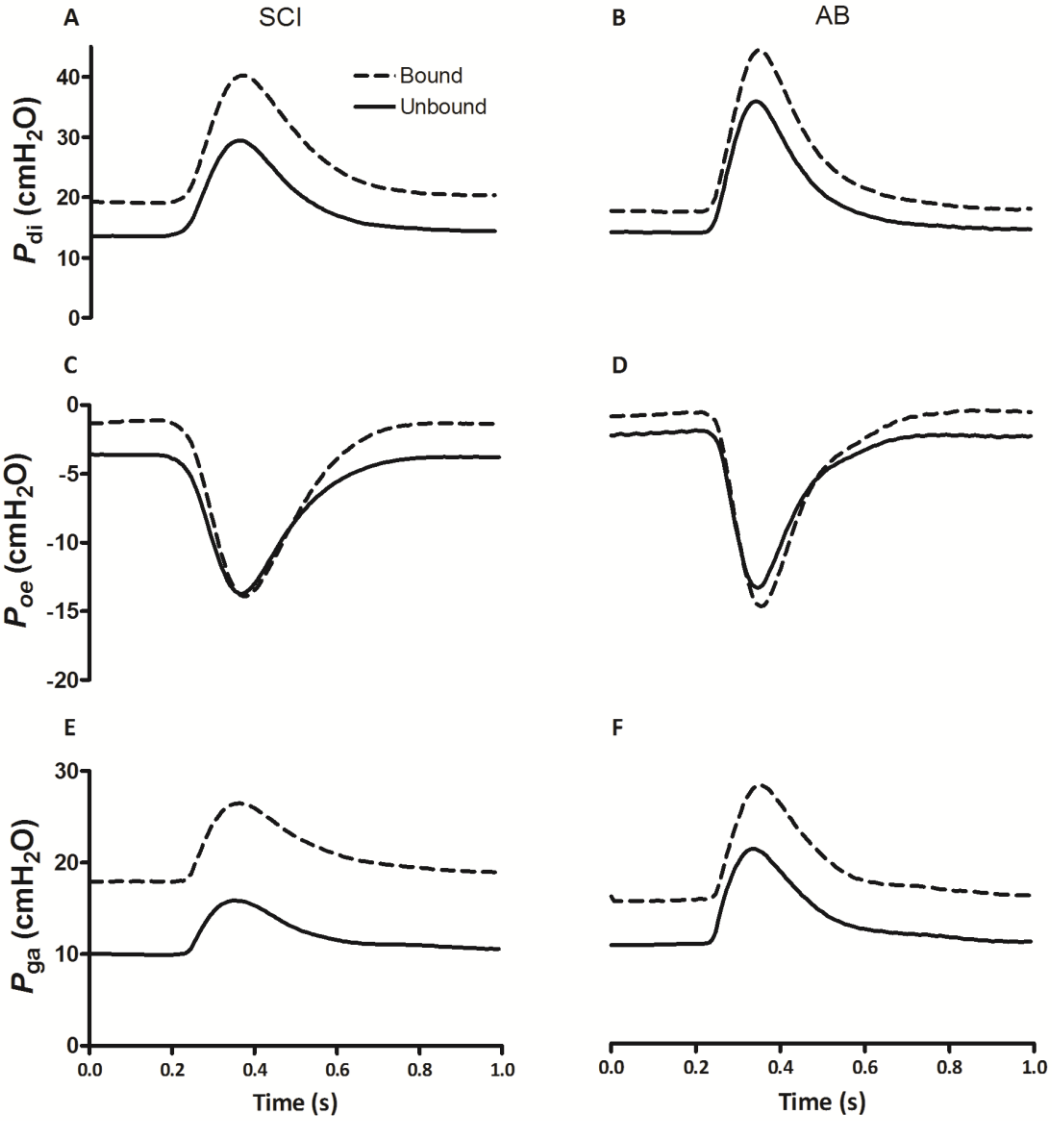

Fig 5-3 Group mean ensemble-average tracings for twitch transdiaphragmatic ($P_{di,tw}$; A and B), oesophageal ($P_{oe,tw}$; C and D), and gastric ($P_{ga,tw}$; E and F) pressure in tight-bound and unbound conditions for SCI (left panels; n = 12) and AB (right panels; n = 8).

All stimulations were delivered at 0.2 s. Note the increase in $P_{di,tw}$ (baseline-to-peak) for the SCI group in tight-bound vs. unbound, primarily as a result of the increase in $P_{ga,tw}$; whereas the increase in $P_{di,tw}$ in the AB group is primarily the result of the increase in $P_{oe,tw}$. SCI, spinal cord injury; AB, able-bodied.

5-3.3 Cardiovascular function in spinal cord injury

Group mean values are shown in Table 5-3. Cardiac data are presented for 11 participants because of poor image quality in 2 participants, likely due to the seated position adopted. There was a dose-response between binding tightness and increase in $\dot{Q}$, reaching statistical significance

in TB (p = 0.048). HR did not differ between conditions, but SV and EDV tended to increase in TB vs. UB (p = 0.11 and 0.17, respectively). Furthermore, S´ and A´ were significantly elevated in TB vs. UB (p = 0.037 and 0.049, respectively; see also Fig 5-4). There was a trend towards an increase in MAP with binding (p = 0.052).

Table 5-3 Cardiovascular function with abdominal binding (unbound [UB], loose-bound [LB], tight-bound [TB]) in participants with spinal cord injury

	UB	LB	TB
$\dot{Q}$, L·min^{-1}	3.4 ± 0.7	3.9 ± 0.8	4.3 ± 1.0†
HR, b·min^{-1}	60 ± 9	59 ± 7	61 ± 7
SV, ml	60 ± 11	67 ± 13	69 ± 14
EDV, ml	92 ± 18	97 ± 21	103 ± 23
ESV, ml	31 ± 9	31 ± 12	33 ± 12
EF, %	66 ± 6	68 ± 9	68 ± 7
S′, cm·s^{-1}	5.28 ± 0.92	5.42 ± 0.94	6.4 ± 1.47*
E′, cm·s^{-1}	−6.17 ± 1.10	−6.99 ± 1.75	−7.32 ± 2.16
A′, cm·s^{-1}	−3.49 ± 1.50	−4.57 ± 1.03*	−5.25 ± 2.08*
SBP, mmHg	91 ± 17	99 ± 18	98 ± 15
DBP, mmHg	48 ± 13	51 ± 13	50 ± 9
MAP, mmHg	71 ± 11	78 ± 11	74 ± 10

Definition of abbreviations: $\dot{Q}$, cardiac output; HR, heart rate; SV, stroke volume; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction; S′, systolic myocardial tissue velocity; E′, early diastolic myocardial tissue velocity; A′, late diastolic myocardial tissue velocity; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure. Values are means ± SD for 11 participants (13 participants for SBP, DBP and MAP). *Significantly different vs. UB (p < 0.05); † significantly different vs. LB (p < 0.05).

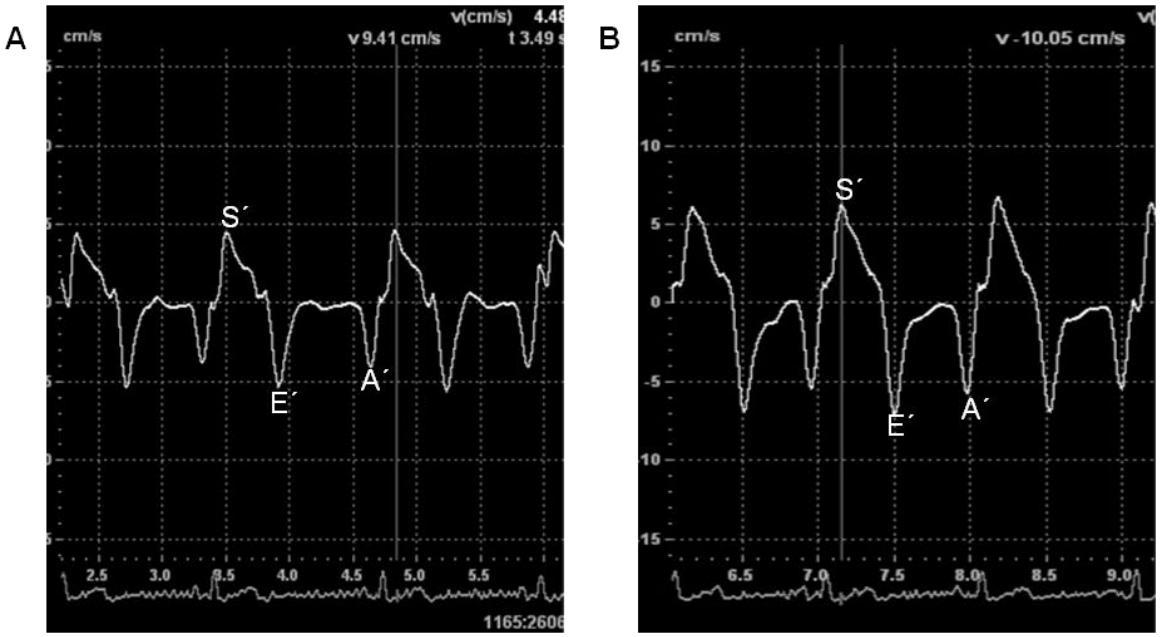

Fig 5-4 Representative image obtained from a single participant illustrating myocardial tissue velocity during systole (S´), early diastole (E´), and late diastole (A´) in the unbound (panel A) and tight-bound (panel B) condition.
Note the increase in S´ and A´ in tight-bound.

5-3.4 Able-bodied group

Age, stature, and body mass were not different between AB and SCI. End-expiratory P_{ga} was 10.3 ± 3.5 cmH_2O in UB, 11.1 ± 3.8 cmH_2O in LB (p = 0.41 vs. UB), and 15.0 ± 4.4 cmH_2O in TB ($p < 0.05$ vs. UB and LB). Tidal inspiratory P_{di} increased in TB vs. UB (12.2 ± 1.3 cmH_2O vs. 9.8 ± 2.5 cmH_2O, $p = 0.025$). In contrast to SCI, the increase in tidal P_{di} was primarily due to an increase in P_{oe} (6.1 ± 3.4 vs. 4.6 ± 1.7 cmH_2O, $p = 0.050$) rather than P_{ga} (7.3 ± 2.8 vs. 6.4 ± 2.7 cmH_2O, $p = 0.40$). Evoked $P_{di,tw}$ was also increased in TB vs. UB, again due to a significant increase in the oesophageal component (Fig 5-3). Pulmonary function in the UB condition was within normal limits. There was a significant increase in IC with TB, but this was at the expense of a reduction in ERV such that VC remained unchanged (Fig 5-1). Cardiovascular function did not change in either TB or LB (Table 5-4). The cardiorespiratory responses in LB were inconsistent, but always smaller than in TB.

Table 5-4 Cardiovascular function with abdominal binding (unbound [UB], loose-bound [LB], tight-bound [TB]) in able-bodied participants

	UB	LB	TB
$\dot{Q}$, L·min^{-1}	4.1 ± 0.6	3.8 ± 0.8	3.8 ± 0.7
HR, b·min^{-1}	62 ± 7	60 ± 8	63 ± 6.9
SV, ml	66 ± 11	64 ± 14	62 ± 12
EDV, ml	104 ± 18	104 ± 18	100 ± 17
ESV, ml	38 ± 8	41 ± 9	39 ± 8
EF, %	64 ± 3	61 ± 7	62 ± 5
S′, cm·s^{-1}	5.15 ± 1.20	5.22 ± 0.77	5.85 ± 1.02
E′, cm·s^{-1}	−7.27 ± 1.33	−7.58 ± 1.77	−7.56 ± 1.35
A′, cm·s^{-1}	−3.20 ± 1.00	−3.40 ± 0.87	−3.89 ± 1.18
SBP, mmHg	132 ± 16	137 ± 20	136 ± 15
DBP, mmHg	64 ± 8	67 ± 9	71 ± 10
MAP, mmHg	87 ± 9	91 ± 10	93 ± 11

Definition of abbreviations: $\dot{Q}$, cardiac output; HR, heart rate; SV, stroke volume; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction; S′, systolic myocardial tissue velocity; E′, early diastolic myocardial tissue velocity; A′, late diastolic myocardial tissue velocity; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure. Values are means ± SD for 8 participants.

5-4 Discussion

The aim of this chapter was to determine if there is a dose-dependent change in resting cardiorespiratory function with abdominal binding in highly-trained athletes with cervical SCI. The main findings were that elastic abdominal binding improved cardiorespiratory function in individuals with low-cervical chronic SCI by reducing FRC, and increasing VC, $P_{di,tw}$ and $\dot{Q}$. In contrast, abdominal binding in AB individuals did not change VC or $\dot{Q}$. It is shown for the first time that a dose-response relationship exists between the degree of abdominal compression and the resultant improvements in cardiorespiratory function in individuals with SCI. Specifically, the changes in cardiorespiratory function in TB were approximately double those in LB.

5-4.1 Pulmonary function

In agreement with previous studies in SCI, abdominal binding reduced FRC and RV (Bodin *et al.*, 2005; Hart *et al.*, 2005) and increased VC (Boaventura *et al.*, 2003; Bodin *et al.*, 2005; Estenne *et al.*, 1998; Goldman *et al.*, 1986b; Hart *et al.*, 2005). The percent increase in VC was most strongly correlated with the percent decrease in RV, which suggests that the increase in VC was primarily due to an improvement in expiratory, not inspiratory function. Further evidence for an improvement in expiratory function with binding is the significant increase in $P_{E,max}$ and PEF. Previous studies that have assessed the effect of abdominal binding on expiratory function found contradictory results. For example, some studies found an improvement in $P_{E,max}$ (Boaventura *et al.*, 2003) and PEF (Hart *et al.*, 2005) with abdominal binding, whereas other studies found no differences (Estenne *et al.*, 1998; Lin *et al.*, 1998). Potential reasons for the discrepancies include differences in the sample size and the degree of abdominal compression applied. The mechanism by which abdominal binding improves expiratory function is likely due to a reduction in abdominal compliance rather than an increase in lung elastic recoil, as TLC was unchanged with binding. If this were the case then AB participants, who would be expected to have smaller changes in abdominal compliance with binding, should demonstrate minimal changes in pulmonary function with binding. Indeed, IC was increased in the AB group; however, ERV was reduced, such that VC and TLC remained unchanged (Fig 5-1).

Another potential mechanism for the improvement in pulmonary function with abdominal binding in individuals with SCI is a change in sitting posture. In AB individuals, upright posture increases VC, FEV_1, and PEF when compared to slump sitting (Lin *et al.*, 2006). To determine whether a change in posture contributed to the respiratory changes with binding, diaphragm and pulmonary function was reassessed in the 11 participants who competed in wheelchair rugby while they sat in their competition chair. The competition chair features a steeply-angled seat ($53 \pm 7°$ from back support) that promotes a more upright sitting posture than the day chair's horizontal seat (i.e., 90°). There were, however, no differences in P_{di} swing, $P_{di,tw}$,

or VC between the two positions. Thus, the improvements in pulmonary function with abdominal binding noted in the present study were unlikely due to changes in sitting posture.

5-4.2 Respiratory muscle function

In agreement with one previous study in SCI (Hart *et al.*, 2005), tidal P_{di} and evoked $P_{di,tw}$ increased with binding. Collectively, these findings imply an improvement in diaphragm function with binding. In line with previous findings (Bellemare and Bigland-Ritchie, 1984; Koulouris *et al.*, 1989) binding also improved diaphragm function in AB participants. However, the mechanism by which abdominal binding improves diaphragm function appears to differ in both groups. Abdominal binding caused an increase in end-expiratory gastric pressure in both groups, presumably resulting in a lengthening and cranial displacement of the diaphragm and a subsequent increase in the area of the diaphragm in apposition with the rib cage. This, in turn, is expected to increase lower rib cage expansion through two mechanisms: first, by pushing outward at the zone of apposition (Mead, 1979); and second, by apposing descent of the diaphragm during contraction at the costal fibres such that the diaphragm is forced to lift the lower rib cage (De Troyer *et al.*, 1982). In the AB group there was an increase in rib cage expansion, as inferred by the increase in tidal P_{oe} swing. In the SCI group, however, the lack of increase in tidal P_{oe} swing with binding suggests that the diaphragm was unable to exert a further expanding effect, due perhaps to a stiff rib cage consequent to chronic spasticity of the intercostal muscles (Estenne and De Troyer, 1986). A more likely explanation for the improvement in diaphragm function in SCI was a decrease in abdominal compliance, as evidenced by the increased gastric contribution to the P_{di} swing and $P_{di,tw}$.

5-4.3 Cardiovascular function

A novel finding was that binding improved cardiac function in the SCI group such that values were similar to those at baseline in the AB group. Specifically, TB significantly increased $\dot{Q}$ through an increase in SV; these findings are in agreement with those in patients with autonomic dysfunction (Smit *et al.*, 2004). The increase in SV observed in the present study was likely due to additional engagement of the Frank-Starling mechanism, as evidenced by the 14% increase in EDV. Further evidence for an improvement in systolic function is provided by the significant increase in S´ (Fig 5-4). This latter finding implies an increased systolic wall velocity, likely related to an enhanced diastolic stretch. That A´ was increased with binding suggests a larger atrial contribution to EDV, and may reflect an increased volume of blood within the left atrium. Collectively, these findings suggest that abdominal binding has the potential to improve cardiac function in individuals with SCI by increasing venous return.

Aliverti *et al*. (2009) have recently suggested that external compression of the abdomen in AB individuals increases venous return by an increase in flow through the hepatic vein due to a translocation of blood from the splanchnic vasculature. This will increase blood pressure at the entry of the hepatic vein to the inferior vena cava, thereby abolishing the pressure gradient between the lower extremities and the right atrium. A reduced pressure gradient may explain why previous studies have found a cessation of femoral venous outflow with large increases in intra-abdominal pressure (Miller *et al.*, 2005a). In light of these recent findings (Aliverti *et al.*, 2009), an increase in venous return with abdominal binding seems intuitive; through reducing abdominal compliance and increasing intra-abdominal pressure, the binder compresses the splanchnic vasculature and translocates some of the blood pooled in this region back to the heart. However, a major difference between the current study and that of Aliverti *et al.* (2009) was that static rather than cyclical compression was used. With static compression one may expect an initial increase in $\dot{Q}$ followed by a transient decline due to activation of the baroreflex, as has been demonstrated in the AB population using an anti-G suit (Gray *et al.*, 1969; Seaworth *et al.*,

1985). There was no increase in $\dot{Q}$ with binding in the AB group, suggesting either that the increase in intra-abdominal pressure was insufficient to cause translocation of blood from the splanchnic region to the heart, or that $\dot{Q}$ initially increased and then transiently decreased through activation of the baroreflex. Likely reasons why $\dot{Q}$ was elevated in SCI but not in AB include a greater degree of abdominal compression (i.e., greater change in end expiratory P_{ga}), and an impaired baroreflex (Krum *et al.*, 1992).

5-4.4 Clinical relevance

A major pulmonary complication in many individuals with SCI is an inability to cough and clear mucous secretions due to abdominal muscle paralysis (Brown *et al.*, 2006; Schilero *et al.*, 2009). Individuals with cervical SCI use the pectoralis major and other accessory expiratory muscles to compress the upper rib cage and increase intrathoracic pressure during an expiratory manoeuvre (De Troyer *et al.*, 1986a). Binding might be expected to increase peak flow and the initial blast of turbulent flow at the start of a cough by way of an increase in intrathoracic pressure (McCool, 2006). In the present study, the increase in peak P_{oe} during a forced vital capacity manoeuvre (~14 cmH_2O) resulted in a significant 25% increase in maximal expiratory flow throughout most of a forced vital capacity (Fig. 5-2). In contrast, Estenne *et al.* (1998) found that binding did not significantly increase peak P_{oe} (~1.4 cmH_2O) or maximal expiratory flow throughout a forced vital capacity in participants with lesions similar to those in the present study. The reason for the discrepancies is not entirely clear, but may stem from a difference in intra-abdominal pressure with binding. The mechanism for the increase in expiratory flow noted in the present study may relate to an improved transmission of pleural pressure arising from an antagonist contraction of the diaphragm consequent to a decrease in abdominal compliance (Estenne and Gorini, 1992). In this regard, P_{di} throughout the expiratory portion of the vital capacity manoeuvre was significantly elevated with binding.

5-5 Conclusions

In line with the original hypothesis acute abdominal binding causes a significant improvement in pulmonary function, inspiratory and expiratory muscle function, and cardiovascular function in individuals with low-cervical SCI. It was also found that the magnitude of increase in cardiorespiratory function exceeded that in AB individuals, and was dependent upon the degree of abdominal compression applied. That is, the greater the degree of abdominal compression, the greater the improvement in cardiorespiratory function. Future studies are needed to determine the chronic effects of abdominal binder use in individuals with SCI.

CHAPTER SIX:

EFFECT OF ABDOMINAL BINDING ON FIELD-BASED FITNESS AND PUSH KINEMATICS IN PARALYMPIC ATHLETES WITH CERVICAL SPINAL CORD INJURY

6-1 Introduction

Spinal cord injury (SCI) results in a lesion dependent impairment in cardiorespiratory function, whereby cardiorespiratory function decreases as the level of injury moves cranially (Teasell *et al.*, 2000). In cervical SCI, an increase in abdominal compliance (Goldman *et al.*, 1986a), dennervation of the sympathetic chain ganglia (Krassioukov, 2009), abolition of the venous muscle pump, and a reduction in circulating blood volume (Houtman *et al.*, 2000), cause venous pooling below the lesion (Teasell *et al.*, 2000). Thus, venous return, stroke volume and cardiac output at rest are lower in cervical SCI compared with able-bodied (AB) individuals (see also chapter 4). The consequent decrease in cardiorespiratory function may be exacerbated during exercise as, in addition to the need for an increased oxygen delivery, many of the respiratory muscle are also involved in postural control and wheelchair propulsion (Sinderby *et al.*, 1992; Vanlandewijck *et al.*, 2001). The potential disequilibrium between increased demand and reduced capacity may conspire to limit exercise performance in this population.

Despite the potential disequilibrium of the cardiorespiratory system, chronic deconditioning of the arm musculature appears to explain the low peak oxygen uptake ($\dot{V}O_{2peak}$) in low- to moderately-trained individuals with cervical SCI (Hopman *et al.*, 1998a). In the elite athletic population with cervical SCI, however, the arm muscles are highly trained and both peak power and $\dot{V}O_{2peak}$ are higher compared with untrained individuals of a similar lesion level (Bhambhani *et al.*, 1995; Eriksson *et al.*, 1988). Thus, for highly-trained athletes with cervical SCI, inadequate haemodynamic adjustments to exercise may reduce $\dot{V}O_{2peak}$ such that these individuals become centrally- rather than peripherally-limited during exercise.

Few studies have examined methods to improve cardiorespiratory function during exercise in individuals with cervical SCI. The most commonly studied methods are electrical stimulation of the lower limbs and anti-gravity (anti-G) suits, both of which have been shown to improve cardiac output and $\dot{V}O_{2peak}$ during maximal incremental exercise (Figoni *et al.*, 1990; Pitetti *et al.*, 1994; see also section 2-5.1 and 2-5.2). Those methods, however, would not be

feasible to use during competitive sport and neither method would be expected to improve respiratory function or postural control. An alternative method for improving cardiorespiratory function and aiding postural control in cervical SCI is abdominal binding. At rest, abdominal binding has been shown to improve vital capacity, twitch transdiaphragmatic pressure and cardiac output (see chapter 5). In the only study to examine the effect of abdominal binding on cardiorespiratory function during exercise, Kerk *et al.* (1995) reported no effect on $\dot{V}O_{2peak}$ or selected biomechanical indices during sub-maximal and maximal wheelchair ergometry. That study, however, was delimited to individuals with high-thoracic SCI, who are able to increase cardiac output and oxygen delivery through the stimulatory effects of the sympathetic nervous system on the myocardium. Thus, it is perhaps unsurprising that abdominal binding did not increase $\dot{V}O_{2peak}$, as the higher cardiac output and oxygen delivery means $\dot{V}O_{2peak}$ is unlikely to be centrally limited in individuals with high thoracic SCI.

The majority of individuals with cervical SCI who take part in competitive sport participate in court-based sports such as wheelchair rugby or wheelchair tennis. For those individuals, laboratory tests are non-sports-specific, and the reliability and external validity may therefore be compromised (Ellis *et al.*, 2000; Franklin *et al.*, 1990). Accordingly, the primary aim of the present study was to determine whether abdominal binding affects field-based measures of fitness in highly-trained athletes with cervical SCI. It was hypothesised that binding would improve performance in tests that rely on cardiorespiratory function and/or postural support.

6-2 Methods

6-2.1 Participants

Ten highly-trained athletes (9 male) with traumatic (9.7 ± 4 yr post injury) cervical SCI [9 complete (C5-C7), 1 sensory incomplete (C6-C7)] were recruited for the study (mean ± SD age 31 ± 5 y, self-report stature 1.73 ± 0.13 m, and body mass 62 ± 8 kg). All athletes competed regularly for Great

Britain (GB) in international wheelchair rugby. For the purposes of this study, an athlete was defined as having played competitive sport for at least 2 y and who trained for at least 15 h/wk. All participants were free from acute and chronic cardiorespiratory disorders. Nine of the ten athletes had taken part in the study described in chapter 5 of this thesis. Prior to the study, all athletes were classified for wheelchair rugby functional class according to the International Wheelchair Rugby Federation Classification system (Duffield and Hart, 2008). This study was approved by the institutional research ethics committee and all participants provided written informed consent. Participants were instructed to arrive at the testing venue in a rested and fully hydrated state, at least 2 h postprandial, and to avoid strenuous exercise in the 24 h preceding each testing session. Each participant was also asked to refrain from caffeine and alcohol for 12 h and 24 h before testing, respectively. All participants were provided with an abdominal binder 4 weeks prior to the start of the experimental trials. Participants were encouraged to use the binder during training such that they would be familiarised with pushing while wearing the binder.

6-2.2 Experimental design

Procedures were performed during two field-based testing sessions, separated by at least 48 h but not longer than 1 wk apart. At the initial visit, participants performed three randomised experimental trials in two randomised and counterbalanced experimental conditions (bound and unbound). During trial one, athletes were assessed for time to complete an agility test and an acceleration test. During trial two, athletes were assessed for cardiorespiratory function and gross efficiency during a 5 minute sub-maximal push, and for peak power and push kinematics during a 30 s Wingate push on a wheelchair ergometer (WERG; Bromakin, Loughborough, UK; length: 1.14 m; circumference: 0.48 m). During trial three, athletes were assessed for time to complete ten 20 m repeated pushes. On a separate day, participants were assessed for the distance covered during two maximal 4-min pushes separated by a 4-min rest interval, with and

without abdominal binding in a randomised and counterbalanced order (trial 4). There was a 30 min rest between conditions in all trials except trial 1, where athletes were given a 10 min rest between conditions. All athletes were fully familiarised with each of the trials.

6-2.3 Binder

The same elasticised binder as described in chapter 5 was used. The binder was placed and tightened in the same way as described in section 5-2.2. Briefly, an inflatable rubber reservoir with a known volume of air was connected to a digital pressure metre (model C9553; JMW Ltd., Harlow, UK), and placed between the binder and the anterior abdominal wall. The binder was fastened until the pressure in the rubber reservoir matched that used in the tight-bound condition in chapter 5. For the one individual who had not taken part in chapter 5, the binder was fastened until the pressure within the rubber reservoir reached ~ 340 cmH_2O at end-expiration (i.e., the group mean end-expiratory surface pressure in chapter 5).

6-2.4 Experimental procedures

Trials one through three were performed on a wooden sprung floor in a large sports hall. Trial four was performed on an indoor 140 m straight synthetic running track. At the start of each testing session, participants performed a 15-min standardised warm-up comprising of a series of short pushes (self-selected pace) and some upper body static stretches as per their normal warm-up routine prior to a training session. At the start of each of the trials, the participants were allowed one practice at a slow pace (~20-30% effort). Each trial was explained to the athletes by the same investigator. For trials one and three, the participants started with their front castor on a line that was 30 cm behind the start line to prevent triggering of the timing gates.

Trial 1

Agility test: Time to complete the agility test and acceleration test was recorded to the nearest 0.01 s using infrared timing gates interfaced to a timing device (Brower TC timing system, Draper, UT, USA). The agility test was set up with timing gates in the centre of a figure-of-eight course; this allowed the time to complete a right and left turn to be separated from the total time (Fig 6-1). The timing gates were set to the lowest height possible, such that they were started and stopped when the front castors of the wheelchair passed through them. For each condition, participants completed the agility test 3 times with a 60 s rest between each attempt. The fastest time was recorded.

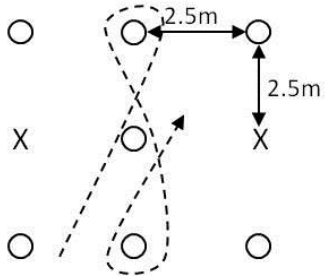

Fig 6-1 Experimental set up for the agility test.
X, timing gate; O, course marker; – – –, path followed by the athlete

Acceleration/deceleration test: The acceleration/deceleration test consisted of a 5 m forward push, 2.5 m backwards push and a 12.5 m forward push. For each condition, participants completed the agility test 3 times with a 60 s rest between each attempt. The fastest time was recorded.

Trial 2

Sub-maximal push: Participants completed a 5-min sub-maximal steady-state push at 1.4 $m \cdot s^{-1}$ on the WERG. This velocity was chosen based on prior exercise test data, whereby all of the current participants could push at 1.4 $m \cdot s^{-1}$ for 5-min with a respiratory exchange ratio <1. Heart rate was assessed throughout the test via telemetry (Polar Vantage NV; Polar Electro Oy, Kempele, Finland). Participants breathed through a low resistance two way valve, and oxygen uptake and

carbon dioxide output were assessed during the final 2 min using the Douglas bag technique (Douglas, 1911). The concentrations of oxygen and carbon dioxide were measured using a paramagnetic oxygen and infrared carbon dioxide gas analyser (Servomex 5200 multipurpose analyser, Crowborough, UK), and minute ventilation ($\dot{V}_E$) was assessed using a dry gas metre (Harvard Apparatus Ltd., Edenbridge, UK). Prior to each measurement, the gas analysers were calibrated using low (100% Nitrogen) and high (20.93 and 9.98%, respectively) calibration gases. Energy expenditure was calculated from oxygen uptake and carbon dioxide output using the formula of Brouwer (1957). Power output was calculated according to the methods of Theisen *et al.* (1996) as described in section 3-2.9. Gross efficiency was calculated as the ratio of work done to total energy expended (Whipp and Wasserman, 1969).

30 s maximal push: Within 2 min of completing the sub-maximal push, participants completed a 30 s maximal push. The push commenced from a rolling start (speed of $1.m \cdot s^{-1}$) to prevent the difficulty in overcoming the initial inertia of the WERG (Goosey-Tolfrey, 2005). Athletes were assessed for peak and mean power, peak and mean heart rate, and fatigue [(peak speed – minimum speed) / peak speed] during the Wingate test.

Kinematic analysis: As described in section 3-2.10, 2-D kinematic analysis of the push technique was carried out during the Wingate push. For each push, the following variables were measured: push time (time in contact with wheel rim), recovery time (time not in contact with wheel rim), total cycle time (sum of push time and recovery time), start angle (angle at start of push phase with respect to a vertical axis), end angle (angle at end of push phase with respect to a vertical axis) and push angle (end angle – start angle). Active and passive trunk motion was calculated by determining the maximum and minimum degree of trunk flexion during the push phase and recovery phase of each cycle, respectively. The first three pushes (Pushes 1,2 and 3) of the Wingate test, along with the mean of the first three pushes after 15 s of the Wingate test had

elapsed (maximal pushing), were analysed to determine the effect of abdominal binding on acceleration and maximal propulsion kinematics.

Trial 3

Repeated push: Time to complete ten 20 m repeated maximum pushes was recorded using a set of timing gates interfaced to a timing device. Further sets of timing gates were placed at 5 m and 15 m, such that 5 m sprint time could also be assessed. The athletes were given 20 s to complete each push, turn around and get ready for the start of the next push. Accordingly, alternate sprints were performed in the opposite direction. A fatigue index was calculated using the following formula:

Fatigue = (100 × [total push time / ideal push time]) – 100

*,w*here total sprint time is the sum of all sprint times, and ideal sprint time is the product of the number of sprints and the fastest sprint time *(Glaister et al., 2008).*

Trial 4

Four minute push: Participants completed the 4-min push twice with 4-min rest between pushes. Markers were placed every 2.5 m, to allow distance to be recorded to the nearest 2.5 m. At rest and immediately after each 4-min push, ratings of perceived respiratory discomfort (dyspnoea) and arm-discomfort were assessed using Borg's modified CR-10 scale (Borg, 1998). At the same time-points, an earlobe capillary blood sample was also taken to determine blood lactate concentration using an enzymatic method (Biosen C_line Sport; EKF Diagnostics, Barleben, Germany; see also section 3-2.11).

6-2.5 Reliability

Prior to the experimental days, the within-day reliability of the acceleration/deceleration test, agility test and the repeated 4-min push test was assessed in a sub-group of the cohort (6

athletes). Reliability was assessed using the coefficient of variation (CV) and ratio limits of agreement (Nevill and Atkinson, 1997). The tests were conducted as per the protocols listed above. The acceleration/deceleration and agility tests were conducted at baseline and then repeated after 10 min rest, the quickest times were used for the calculations. The 4-min push was conducted at baseline and then repeated after 30 min rest. The furthest distance at each time point was used for the calculations.

6-2.6 Statistics

Differences in push kinematics during the 30 s maximal push test were analysed using a two-way repeated-measure analysis of variance, with one within-factor for condition (Unbound vs. Bound) and one within-factor for time (push 1, push 2, push 3, maximal pushing). For all other dependent variables, paired samples t-tests were used to assess differences between conditions. Statistical analyses were carried out using SPSS 16.0 for Windows (SPSS Inc., an IBM Company, Chicago, IL, USA). Data are presented as mean ± SD.

6-3 Results

6-3.1 Trials 1-3

Time to complete the acceleration/deceleration, agility and repeated sprint tests, along with selected variables during the sub-maximal and Wingate push on the wheelchair ergometer are shown in Table 6-1. Abdominal binding significantly reduced the time taken to complete the acceleration/deceleration test (2%, $p = 0.005$; see also Fig 6-2a). The magnitude of improvement was correlated with the time taken to complete the test in the unbound condition ($r = 0.82$, $p = 0.004$; see also Fig 6-2b), such that the slowest participants improved the most. The repeated sprint test resulted in fatigue for all participants, but was unaltered with abdominal binding. During the sub-maximal push, abdominal binding had no effect on oxygen uptake or gross

efficiency. $\dot{V}_E$, however, was significantly reduced with abdominal binding (10%, $p = 0.040$) and there was a trend towards a reduction in $\dot{V}_E/\dot{V}O_2$ (10%, $p = 0.11$). During the Wingate push, abdominal binding had no effect on peak power, mean power or fatigue index. There were also no statistically significant alterations in push kinematics with abdominal binding (Table 6-2), although recovery time tended to be quicker ($p = 0.055$).

Table 6-1 Performance variables during trials 1-3

	UNBOUND	BOUND
Agility test time		
Left turn, s	4.85 ± 0.27	4.89 ± 0.29
Right turn, s	4.86 ± 0.30	4.88 ± 0.27
Acceleration/deceleration test time, s	10.25 ± 0.72	10.07 ± 0.59**
Repeated sprint test time		
Fastest 5 m, s	1.35 ± 0.13	1.35 ± 0.15
Fastest 20 m, s	6.39 ± 0.57	6.38 ± 0.55
Total sprint time, s	68.83 ± 6.47	68.46 ± 6.03
Fatigue index, %	7.72 ± 2.74	7.68 ± 2.80
Sub-maximal push		
Propulsion speed, $m \cdot s^{-1}$	1.39 ± 0.02	1.40 ± 0.01
$\dot{V}O_2$, $l \cdot min^{-1}$	0.69 ± 0.09	0.67 ± 0.07
$\dot{V}CO_2$, $l \cdot min^{-1}$	0.60 ± 0.06	0.55 ± 0.07
$\dot{V}_E$, $l \cdot min^{-1}$	27.3 ± 7.2	24.1 ± 5.0*
$\dot{V}_E/\dot{V}O_2$	40.1 ± 10.5	36.2 ± 6.4
$\dot{V}_E/\dot{V}CO_2$	45.7 ± 10.5	43.9 ± 5.8
Heart rate, bpm	105 ± 11	101 ± 13
Gross efficiency, %	9.6 ± 1.2	9.9 ± 1.4
Maximal Wingate 30 s push		
Peak power, W	66 ± 13	68 ± 13
Mean power, W	58 ± 11	60 ± 12
Time to peak power, s	8.1 ± 2.2	7.8 ± 2.4
Fatigue index, %	21.0 ± 8.0	19.6 ± 5.9
Peak heart rate, bpm	140 ± 14	135 ± 15

Definition of abbreviations: $\dot{V}O_2$, oxygen uptake; $\dot{V}CO_2$, carbon dioxide output; $\dot{V}_E$, minute ventilation. Values are means ± SD for 10 participants. * Significantly different vs. unbound ($p < 0.05$); ** significantly different vs. unbound ($p < 0.01$).

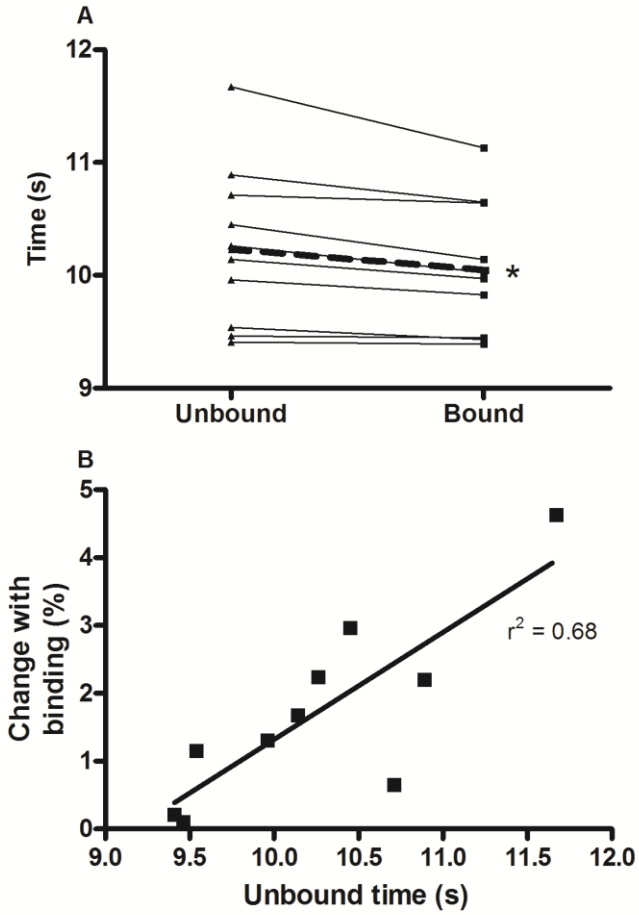

Fig 6-2 Individual (solid line) and group mean (dashed line) time to complete the acceleration/deceleration test in the unbound and bound condition (panel A). *Relationship between the percentage improvement in the acceleration/deceleration test with binding and the time to complete the acceleration/deceleration test in the unbound condition (panel B). Note that the athletes who improved the most with binding had the slowest unbound test times. *Significantly different vs. unbound ($p < 0.05$).*

Table 6-2 Kinematic analysis of wheelchair propulsion

	PUSH 1		PUSH 2		PUSH 3		MAXIMAL PUSHING	
	UNBOUND	BOUND	UNBOUND	BOUND	UNBOUND	BOUND	UNBOUND	BOUND
Temporal parameters								
Push time, s	0.32 ± 0.06	0.30 ± 0.06	0.25 ± 0.05	0.25 ± 0.06	0.21 ± 0.04	0.20 ± 0.03	0.13 ± 0.03	0.13 ± 0.04
Recovery time, s	0.30 ± 0.06	0.28 ± 0.04	0.27 ± 0.04	0.25 ± 0.05	0.28 ± 0.04	0.27 ± 0.05	0.32 ± 0.05	0.33 ± 0.04
Cycle time, s	0.62 ± 0.09	0.58 ± 0.07	0.52 ± 0.06	0.50 ± 0.06	0.49 ± 0.07	0.47 ± 0.06	0.46 ± 0.04	0.47 ± 0.04
Start angle, deg	-28.8 ± 7.4	-28.5 ± 12.6	-21.8 ± 13.2	-21.4 ± 14.5	-16.5 ± 14.9	-15.7 ± 14.1	1.4 ± 17.8	2.2 ± 22.5
End angle, deg	66.9 ± 13.3	68.1 ± 13.1	68.2 ± 11.2	67.4 ± 12.1	70.7 ± 10.5	67.5 ± 13.3	67.2 ± 11.6	68.5 ± 13.1
Push angle, deg	95.7 ± 15.4	96.7 ± 16.8	90.1 ± 16.3	88.9 ± 17.8	87.2 ± 13.7	83.2 ± 15.1	65.8 ± 18.4	66.3 ± 19.3
Active trunk motion								
Hand contact, deg	-7.2 ± 5.8	-6.0 ± 5.7	-2.0 ± 3.4	-1.5 ± 4.9	0.0 ± 3.4	-0.5 ± 3.5	4.2 ± 2.8	3.7 ± 3.8
Hand release, deg	-4.8 ± 2.8	-4.4 ± 3.2	-3.9 ± 2.5	-3.9 ± 2.9	-2.5 ± 2.2	-3.7 ± 3.5	0.3 ± 2.6	-0.2 ± 4.1
Maximum flexion, deg	-0.3 ± 3.4	-0.2 ± 3.5	0.7 ± 2.6	1.0 ± 3.2	1.7 ± 2.4	0.4 ± 3.4	4.3 ± 2.7	3.8 ± 3.8
Maximum extension, deg	-8.2 ± 4.7	-7.4 ± 4.5	-4.7 ± 2.6	-5.2 ± 3.1	-2.8 ± 2.1	-3.7 ± 3.5	0.3 ± 2.6	-0.2 ± 4.1
Active ROM, deg	7.8 ± 3.7	7.3 ± 2.7	5.4 ± 1.6	6.2 ± 2.4	4.5 ± 1.8	4.1 ± 1.4	4.1 ± 1.2	4.0 ± 1.2
Passive trunk motion								
Maximum flexion, deg	-1.9 ± 3.4	-1.1 ± 4.4	0.2 ± 3.3	-0.1 ± 3.2	1.8 ± 2.7	0.6 ± 3.7	4.4 ± 2.8	3.9 ± 3.7
Maximum extension, deg	-6.8 ± 3.6	-6.2 ± 3.9	-5.4 ± 2.7	-5.3 ± 3.0	-3.5 ± 2.2	-4.6 ± 3.6	-0.5 ± 2.6	-1.1 ± 4.1
Passive ROM, deg	4.9 ± 2.4	5.1 ± 1.9	5.6 ± 2.3	5.2 ± 1.4	5.3 ± 2.1	5.2 ± 1.9	4.9 ± 1.5	5.0 ± 1.2

Definition of abbreviations: deg, degrees; ROM, range of motion. Values are means ± SD for 10 participants.

6-3.2 Trial 4

The distance covered during each 4-min push, along with perceptual and metabolic indices immediately after each 4-min push, are shown in Table 6-3. Abdominal binding increased the distance covered during both the first and second 4-min push in 8 out of 10 individuals. The group mean increase in distance covered during the first and second 4-min push was 2% (p = 0.043) and 4% (p = 0.016), respectively (see also Fig 6-3a & 6-3b). The mean and cumulative distances covered were also increased with abdominal binding (both 3%, $p < 0.009$). Perceptual ratings of arm discomfort and blood lactate accumulation were also lower during the second 4-min push in the bound vs. unbound condition (8%, p = 0.022 and 13%, p = 0.010, respectively). Ratings of dyspnoea remained unchanged between conditions.

Table 6-3 Performance variables during trial 4

	UNBOUND	BOUND
4-Min push 1		
Distance, m	700 ± 75	713 ± 83*
$[La^-]_B$, mmol·l^{-1}	6.4 ± 1.6	5.8 ± 1.6
RPE, arm discomfort	7.3 ± 1.8	6.6 ± 1.6
RPE, dyspnoea	7.1 ± 1.5	7.5 ± 1.4
4-min push 2		
Distance, m	694 ± 81	722 ± 84*
$[La^-]_B$, mmol·l^{-1}	7.9 ± 2.0	6.8 ± 1.8*
RPE, arm discomfort	7.7 ± 1.7	7.0 ± 1.2*
RPE, dyspnoea	7.7 ± 1.2	7.3 ± 1.3
Best push distance, m	704 ± 77	726 ± 82**
Mean push distance, m	699 ± 78	718 ± 82**
Cumulative distance, m	1394 ± 155	1434 ± 166**

Definition of abbreviations: $[La^-]_B$, blood lactate concentration; RPE, ratings of perceived exertion. Values are means ± SD for 10 participants. * Significantly different vs. unbound ($p < 0.05$); ** Significantly different vs. unbound ($p < 0.01$).

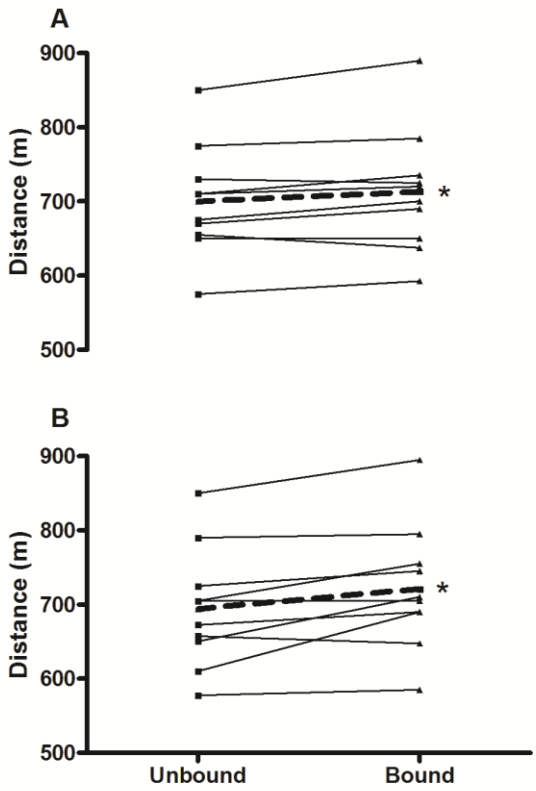

Fig 6-3 Individual (solid line) and group mean (dashed line) distance covered during the first (panel A) and second (panel B) 4-min push in the unbound and bound condition.

*Note that despite a large inter-individual variability in the unbound condition, 8 out of 10 athletes pushed further during both 4-min pushes with abdominal binding. *Significantly different vs. unbound ($p < 0.05$).*

6-3.3 Reliability

Limits of agreement for the agility, acceleration/deceleration and 4-min push field tests are shown in Table 6-4. For the acceleration/deceleration test, the within-subject CV was 0.4%, the systematic bias ratio for the 95% ratio limits of agreement was 0.998, and the random error was 1.010. For the 4-min push test, the CV was 0.5%, the systematic bias ratio for the 95% ratio limits of agreement was 0.997, and the random error was 1.012. Thus, in a worst case scenario a participant who completes 700 m in the first 4-min push test could theoretically cover as little as 686 m or as much as 707 m in the second test. The CV for the left and right turns during the agility test was 2.1% and 1.8%, respectively.

Table 6-4 Ratio limits of agreement for field-based exercise tests

Test	Bias			Random error			
	Ratio	SE	95% CI	Ratio	SE	95% CI lower	95% CI upper
Agility test							
Left turn time	1.032	0.002	0.026 to 0.036	1.032	0.004	0.991 to 1.008	1.057 to 1.073
Right turn time	1.024	0.003	0.018 to 0.029	1.038	0.005	0.977 to 0.996	1.053 to 1.072
Acceleration/deceleration test time	0.998	0.001	−0.004 to −0.001	1.010	0.001	0.985 to 0.991	1.005 to 1.011
4-min push distance	0.997	0.001	−0.005 to −0.002	1.012	0.002	0.982 to 0.988	1.005 to 1.011

Definition of abbreviations: SE , standard error; CI, confidence interval

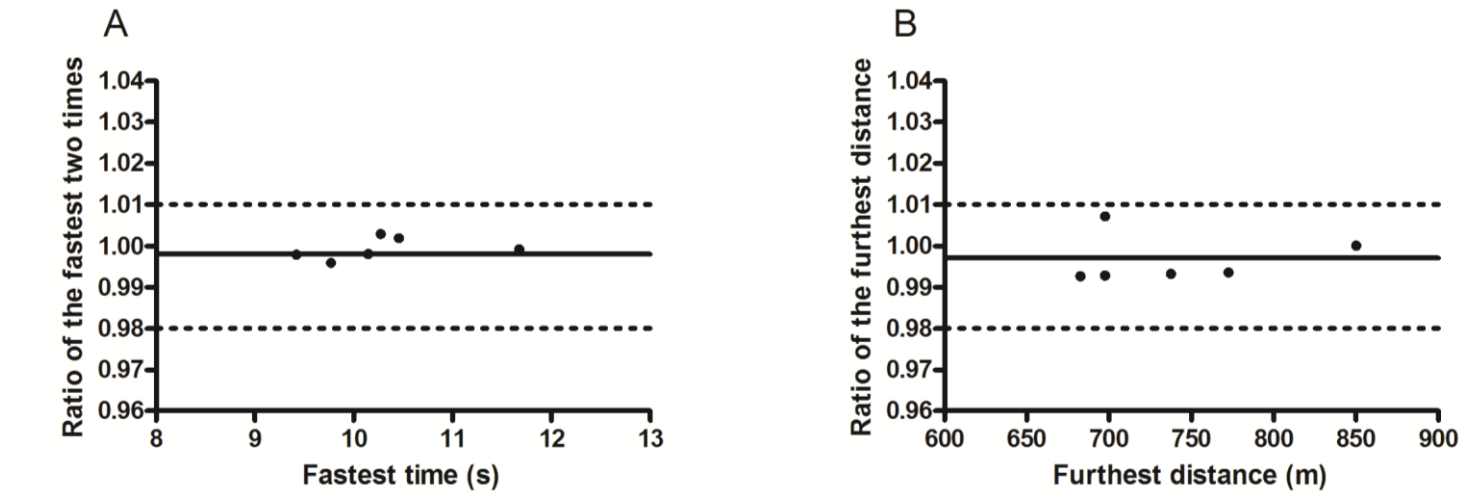

Fig 6-4 Within-day Bland-Altman plots for the acceleration/deceleration test (panel A) and the 4-min push test (panel B).
The continuous lines show the mean difference between the measures (systematic bias) and the dashed lines show the random error components.

6-4 Discussion

The aim of the chapter was to determine whether abdominal binding improves field-based measures of fitness in highly-trained athletes with cervical SCI. It is demonstrated for the first time that abdominal binding reduces the time taken to complete a field-based acceleration/deceleration test and decreases minute ventilation during sub-maximal wheelchair ergometry, but does not alter push technique. Another novel finding is that abdominal binding increased the maximal distance covered, as well as attenuated blood lactate accumulation and ratings of perceived limb discomfort in response to maximal repeated 4-min pushes.

Abdominal binding caused a significant 2% reduction in acceleration/deceleration test time. This improvement was outside the 95% limits of agreement and was ~ four times greater than the within-day CV (Table 6-4). Thus, the reduced acceleration/deceleration test time represents a meaningful improvement for these athletes. The key aspects to performance in the acceleration/deceleration test are acceleration itself as well as the speed with which an athlete can change from forward-to-backward propulsion and vice versa. As binding did not change the fastest 5 m push, it is unlikely that acceleration was improved. Instead, the strong trend towards an improvement in the recovery phase of the push during the kinematic analysis suggests that the binder may provide additional truncal support that allows individuals to recover to a more upright posture quicker. An enhanced postural support with binding may also be expected to improve the time taken to complete the agility test. There was, however, no difference in the time taken to complete an agility test with binding; suggesting that either binding induced improvements in truncal support occur in the coronal plane only, or that the higher CV for the agility test rendered any small changes with binding difficult to detect.

Abdominal binding significantly increased the distance covered during both 4-min push tests in 8 out of 10 highly-trained athletes with cervical SCI (Fig 6-3). The two athletes who did not show an improvement in 4-min push performance with binding had a sensory incomplete injury and reported discomfort under the rib cage from wearing the binder. No other participant

reported discomfort from wearing the binder. The mean increase in distance covered during the 4-min push tests with binding was 3% (or 20 m), which is ~6 times the within-day CV and is outside of the 95% ratio limits of agreement (Table 6-4). Thus, the increase in 4-min push distance with abdominal binding represents a meaningful change for these athletes. The 3% improvement in 4-min push performance is much smaller than that reported with 'boosting'. Boosting is the practice of voluntarily inducing autonomic dysreflexia (usually via bladder distension, sitting on sharp objects or the use of tight leg straps) prior to an athletic event to enhance athletic performance, and is commonly performed by athletes in wheelchair racing and wheelchair rugby (Bhambhani *et al.*, 2010). In the only study to specifically investigate the effect of boosting on athletic performance in tetraplegic athletes, Burnham *et al.* (1994) reported a 10% decrease in the time to complete a simulated 7.5 km time trial on a wheelchair ergometer. Due to the degree of performance improvement and due to the considerable danger of inducing autonomic dysreflexia, the International Paralympic Committee considers boosting a form of 'doping' and has banned its use in competition (IPC, 2000). Although blood pressure was not measured in the current study there was no evidence of dysreflexia at rest in any participant in chapter 5. Thus, athletes appear able to utilise the cardiorespiratory benefits of abdominal binding without inducing autonomic dysreflexia and within the rules of wheelchair sport.

Compared to the 4-min push, peak-power during the Wingate test and time to complete a repeated sprint test were unchanged with abdominal binding. This difference in response between the tests provides an insight into the mechanism by which abdominal binding may improve the distance covered during a 4-min push test. In the cervical SCI population, the distance covered during a 12-min maximal push is related to $\dot{V}O_{2peak}$ (Franklin *et al.*, 1990). Evidence in AB individuals that an 'all out' exercise test that lasts for 3 min is sufficient to elicit $\dot{V}O_{2peak}$ (Burnley *et al.*, 2006) suggests that the distance covered in a 4-min push may also be related to $\dot{V}O_{2peak}$ in the cervical SCI population. This is different to a Wingate or repeated sprint test, where performance is independent of $\dot{V}O_{2peak}$ and is more heavily reliant on anaerobic

exercise capacity and recovery (Bogdanis *et al.*, 1995; Gaitanos *et al.*, 1993; Smith and Hill, 1991). Thus, it is reasonable to believe that the improvement in 4-min push performance with abdominal binding in the current study represents an acute improvement in $\dot{V}O_{2peak}$.

An increase in $\dot{V}O_{2peak}$ with binding may arise from a translocation of blood from the abdomen to the heart, thereby increasing venous return, stroke volume, cardiac output and oxygen delivery. Evidence supporting this postulate is provided by the lower blood lactate concentrations with binding, suggesting a greater contribution to energy production from oxygen dependent pathways. An increase in $\dot{V}O_{2peak}$ contradicts the only study to assess the effect of abdominal binding on $\dot{V}O_{2peak}$ in SCI (Kerk *et al.*, 1995). That study, however, was delimited to individuals with thoracic SCI, who are unlikely to be limited by oxygen delivery in the same way as cervical SCI due to partial or full cardiac sympathetic innervation in thoracic SCI (see also intro). Thus, a binding-induced increase in $\dot{V}O_{2peak}$ with binding seems a plausible explanation for the improvement in 4-min push distance in athletes with cervical SCI; however, further studies that measure $\dot{V}O_2$ during a maximal 4-min push with and without abdominal binding are required to confirm this postulate.

A final finding of the current study was that abdominal binding reduced sub-maximal minute ventilation, but not $\dot{V}O_2$; accordingly, there was a strong trend towards a reduction in $\dot{V}_E/\dot{V}O_2$with binding. In the unbound condition, $\dot{V}_E/\dot{V}O_2$ exceeded 40 during sub-maximal steady state exercise, which is higher than that expected in healthy AB individuals and is suggestive of inefficient pulmonary gas exchange (Wasserman *et al.*, 2005). The high $\dot{V}_E/\dot{V}O_2$ is most likely due to the tachypnic breathing pattern in this population (Taylor *et al.*, 2010), which increases dead space ventilation and reduces ventilatory efficiency. Although the mechanism by which abdominal binding reduces $\dot{V}_E/\dot{V}O_2$ is not entirely clear, it is speculated that abdominal binding may increase tidal volume and reduce dead space ventilation. However, studies investigating alterations in breathing pattern during exercise with and without abdominal binding are required to confirm this hypothesis.

6-5 Conclusion

This is the first study to demonstrate that acute use of abdominal binding significantly increases the distance covered during a maximal 4-min push in the field, attenuates the rise in blood lactate accumulation during repeated 4-min pushes, and reduces the time taken to complete an acceleration/deceleration test in highly-trained athletes with cervical SCI. Although the mechanisms responsible for the improvements remain elusive, an improvement in $\dot{V}O_{2peak}$ and cardiorespiratory function during exercise may be implicated. That abdominal binding did not alter push kinematics suggests that athletes can utilise the potential cardiorespiratory benefits of abdominal binding without altering their push technique.

CHAPTER SEVEN:

EFFECT OF ABDOMINAL BINDING ON CARDIORESPIRATORY FUNCTION DURING EXERCISE IN PARALYMPIC ATHLETES WITH CERVIAL SPINAL CORD INJURY

7-1 Introduction

Individuals with cervical spinal cord injury (SCI) have a reduced aerobic exercise capacity due to a small active muscle mass, an inability to activate the venous muscle pump, and an absence of the sympathetically-mediated chronotropic and inotropic responses to exercise (Figoni, 1993). Furthermore, abolished supraspinal control of sympathetic effector organs, such as the smooth muscle of arteries and veins (Teasell *et al.*, 2000), results in an impaired redistribution of blood of the splanchnic bed during exercise (Thijssen *et al.*, 2009). Redistribution of the splanchnic reservoir is considered a key haemodynamic adjustment during exercise (Rowell, 1973; Thijssen *et al.*, 2009). Thus, perturbed cardiovascular control following cervical SCI may impair central haemodynamics, culminating in a reduced stroke volume and cardiac output at rest (Kessler *et al.*, 1986; Nash *et al.*, 1991) and during high intensity exercise compared to AB individuals (Dela *et al.*, 2003; Hopman *et al.*, 1992). In turn, impaired haemodynamics may place a central limitation on oxygen delivery, thereby reducing aerobic exercise capacity (Dela *et al.*, 2003; Hopman *et al.*, 2004).

In addition to the impairment in cardiovascular function, individuals with cervical SCI also have weak respiratory muscles (Mateus *et al.*, 2007) and exhibit a restrictive pulmonary defect (Baydur *et al.*, 2001; Schilero *et al.*, 2009). During exercise, individuals with cervical SCI exhibit dynamic lung hyperinflation and a tachypnic breathing pattern (Taylor *et al.*, 2010). The negative consequences of hyperinflation include a reduction in the mechanical advantage of the diaphragm, and a subsequent reduction in the appositional and insertional component of diaphragmatic action through the zone of apposition (De Troyer, 1997). Consequently, there is a reduction in the pressure generating capacity of the diaphragm in individuals with cervical SCI (section 4-3.2). This, in turn, may increase the effort (pressure) required to inflate the rib cage (i.e., effort/displacement ratio). An increased effort/displacement ratio (rate of neuromechanical uncoupling) has been shown to be elevated in able-bodied (AB) individuals exercising with chest

wall restriction (O'Donnell *et al.*, 2000) and in individuals with chronic airflow limitation (O'Donnell *et al.*, 1997).

Despite these cardiorespiratory limitations to exercise in cervical SCI, relatively few studies have investigated methods to improve cardiorespiratory function during exercise in this population. The method that has received most attention is the addition of lower-body electrical stimulation to arm-crank exercise, whereby improvements in cardiac output (Davis *et al.*, 1990) and peak oxygen uptake ($\dot{V}O_{2peak}$) (Hettinga and Andrews, 2008; Mutton *et al.*, 1997; Verellen *et al.*, 2007) have been reported. Another method that has been used in the SCI population is an anti-G suit. Two studies have assessed the effect of an anti-G suit on the cardiorespiratory responses to exercise; one study reported no change in $\dot{V}O_2$ during sub-maximal exercise, despite an enhanced stroke volume (Hopman *et al.*, 1992), whereas the other study reported an improvement in $\dot{V}O_{2peak}$ (Pitetti *et al.*, 1994). This discrepancy may have been accounted for by the participants' lesion level. Hopman *et al.* (1992) used low paraplegics (T6-T12), who would be expected to have full sympathetic innervation of the myocardium and partial innervation of the splanchnic bed and would be less likely, therefore, to have disordered cardiovascular control and reach a central limitation during exercise. In contrast, Pittetti *et al.* (1994) studied individuals with cervical SCI, who have a much greater loss of supraspinal cardiovascular control and would be more likely, therefore, to be centrally limited during exercise by way of an impaired oxygen delivery.

Another potential method for improving cardiorespiratory function during exercise in individuals with SCI is abdominal binding. At rest, abdominal binders are thought to mimic the abdominal muscles, thereby resulting in reduced functional residual capacity (FRC) and increased inspiratory capacity (IC), vital capacity (VC) and twitch transdiaphragmatic pressure ($P_{di,tw}$) (see also sections 5-3.2 and 5-3.3). Abdominal binders are also used as a non-pharmacological method of improving orthostatic intolerance in the acute stage following SCI (Gillis *et al.*, 2008; Huang *et al.*, 1983). Despite these positive findings at rest, only one study has assessed the effect of

abdominal binding on cardiorespiratory function during exercise in SCI. Kerk *et al.* (1995) reported no difference in physiological responses to wheelchair ergometry with abdominal binding. A limitation to that study, however, is that only individuals with thoracic SCI were studied and the degree of abdominal compression was not measured. In contrast, data from chapter 6 of this thesis showed that the distance covered during a maximal 4-min push was significantly enhanced with binding in highly-trained athletes with cervical SCI (see section 6-3.2). The mechanism underlying the improvement in 4-min push performance was unclear, but may be related to an improvement in cardiorespiratory function because there was no change in push kinematics with abdominal binding. Thus, the purpose of the present study was to investigate the mechanism(s) by which abdominal binding alters cardiorespiatory function during exercise in elite athletes with cervical SCI. Specifically, two hypotheses were tested: first, whether abdominal binding abolishes or delays the dynamic hyperinflation during exercise, and thereby improves the diaphragmatic contribution to inspiration and the ventilatory response to exercise; and second, whether abdominal binding improves peak oxygen uptake.

7-2 Methods

7-2.1 Participants

Eight athletes (7 male) with chronic SCI (mean ± SD 9 ± 3 y post injury) who were members of the Great Britain wheelchair rugby squad volunteered to participate in the study (age 29 ± 2 y, self-report stature 1.79 ± 0.10 m, body mass 67 ± 15 kg). Training diaries revealed that each of the athletes trained for at least 15 hr / wk. Training consisted of push sessions, rugby specific training drills and resistance exercise. Prior to the study, the participants were classified by an independent observer using the International Standards for Neurological Classification of Spinal Cord Injury (ASIA, 2003) and the International Wheelchair Rugby Federation classification system

(Duffield and Hart, 2008). Seven of the participants had complete tetraplegia (C5-C7, ASIA A) and one had incomplete tetraplegia (C5-C6, ASIA B). Seven of the eight participants had taken in the studies described in Chapter 5, and all participants had taken part in the study described in chapter 6. None of the participants smoked or had acute or chronic cardiopulmonary disease as assessed using a health questionnaire. The study was approved by the institutional research ethics committee and all participants provided written informed consent. Participants were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 2 h postprandial, and to avoid strenuous exercise in the 24 h preceding testing. Each participant was asked to refrain from caffeine and alcohol for 12 h and 24 h before testing, respectively.

7-2.2 Experimental design

During one visit to the laboratory, participants completed two experimental trials (sub-maximal [Trial 1] and maximal [trial 2] wheelchair exercise on a treadmill) in two conditions (bound and unbound) in their own sports wheelchair. During trial 1, participants were assessed for breathing mechanics, ventilatory constraint, and the cardiorespiratory and metabolic responses to discontinuous sub-maximal incremental wheelchair exercise on a motorised treadmill (0.4 $m \cdot s^{-1}$ every 4 min, starting speed 1.6 $m \cdot s^{-1}$, 30 s rest between stages). During trial 2, participants were assessed for the cardiorespiratory and metabolic responses to maximal incremental wheelchair exercise (0.2% every 40 s, constant speed between 2.2 to 2.7 $m \cdot s^{-1}$ depending on responses during trial 1). The order of conditions was randomised, and counterbalanced, whereas the order of the experimental trials was always sequential (i.e., trial 2 followed trial 1). Participants rested for 30 min between conditions and 60 min between trials.

7-2.3 Binder

The elasticised binder described in chapters 4 and 5 was used in the current study. The tightness of the binder was set the same way as during trials 2 and 3 in chapter 5 (see section 5-2.2). Briefly, an inflatable rubber reservoir with a known volume of air was connected to a digital pressure metre (model C9553; JMW Ltd., Harlow, UK), and placed between the binder and the anterior abdominal wall. The binder was fastened until the pressure in the rubber reservoir matched that used in the tight-bound condition in chapter 5. For the one individual who had not taken part in chapter 5, the binder was fastened until the pressure within the rubber reservoir reached ~ 340 cmH_2O at end-expiration (i.e., the group mean end-expiratory surface pressure in chapter 5).

7-2.4 Experimental procedures

Preliminary measures: Body mass was assessed using electronic beam-scales (Marsden MPWS-300, Marsden, Rotherham, UK), and stature was self-reported. Power output on a motorised treadmill (HP Cosmos Saturn 300/125; Nussdorf-Traunstein, Germany) was determined by a drag test (van der Woude *et al.*, 1986; see also section 3-2.9). Power output was calculated from the product of drag-force and velocity (van der Woude *et al.*, 2001).

Trial 1

Pressure measurements: Gastric and oesophageal pressure was measured using 2 balloon-tipped catheters as described in section 3-2.3. Airflow was sampled at the mouth using a turbine transducer connected to an online system (Oxycon Pro; Jaeger, Höchberg, Germany). A real-time analogue flow signal was taken directly from the online system to the data acquisition system via a commercially available device (uDAQ; Eagle Technology, Cape Town, South Africa) and digitised at 100 Hz (micro 1401 mkII; Cambridge Electronic Design, Cambridge, UK).

Ventilatory constraint and respiratory mechanics: The degree of ventilatory constraint was assessed by measuring dynamic changes in end-expiratory lung volume (EELV) and end-inspiratory lung volume (EILV) during exercise, as described in section 3-2.12. Total lung capacity (TLC) was taken for each participant as the values obtained in both conditions during chapter 5. The degree of expiratory flow limitation was defined as the percentage of the tidal flow-volume loop that met or exceeded the expiratory portion of the maximal flow-volume loop obtained at rest or within 2 min after exercise (ATS/ACCP, 2003; Johnson *et al.*, 1999b). Inspiratory flow reserve (IFR) was expressed as the peak inspiratory flow generated during tidal breathing relative to that achieved during the maximal flow- volume manoeuvre at the same lung volume. The degree of ventilatory constraint was estimated using the ratio of EILV-to-TLC, and the ratio of minute ventilation ($\dot{V}_E$) relative to a theoretical maximal ventilatory capacity ($\dot{V}_{ECAP}$) (Johnson *et al.*, 1999b).

Global respiratory mechanics were assessed by calculating the diaphragm and oesophageal pressure-time products (PTP_{di} and PTP_{oe}, respectively). Pressure-time products for each breath were obtained by integrating the area beneath the diaphragm and oesophageal waveforms down to zero, such that both the work done during inspiration and the isometric work done by the diaphragm at relaxation volume were included in the calculations. Due to the length of the testing protocol and the breaks required between trials, pressure-derived data and operating lung volume data were only collected during the sub-maximal exercise tests in both conditions.

Trials 1 and 2

Exercise responses: Both sub-maximal (trial 1) and maximal (trial 2) exercise tests were conducted using a motorised treadmill (HP Cosmos Saturn, Nussdorf-Traunstein, Germany). For both trials, ventilatory and pulmonary gas exchange indices were assessed using an online system (Oxycon Pro; Jaeger, Höchberg, Germany). Other measures included heart rate (HR) via telemetry (Polar

Vantage NV; Polar Electro Oy, Kempele, Finland), arterial oxygen saturation (SpO_2) via earlobe pulse oximetry (PalmSAT 2500, Nonin Medical, Minnesota, US), and ratings of perceived respiratory (dyspnoea) and arm-discomfort via Borg's modified CR-10 scale (Borg, 1998). Earlobe capillary blood lactate concentration ($[La^-]_B$), via an enzymatic method (YSI 1500 SPORT, YSI Incorporated, Ohio, USA) was also assessed prior to the start of each trial, at the end of each exercise stage during trial 1, and immediately after exercise in trial 2.

7-2.5 Data analyses

All breath-by-breath measurements at rest and during sub-maximal exercise were averaged over 30 s epochs. To avoid breath contamination from paired IC measurements the first 30 s of every 4th min of sub-maximal exercise was analysed. The 30 s of data used for analysis was filtered to remove outlying breaths, defined as any breaths deviating by more than three standard deviations from the mean total breath time (T_{TOT}) during the preceding 30 s. For the determination of peak cardiorespiratory responses, breath-by-breath data were interpolated to give second-by-second values and reported as the highest 30 s rolling average (ATS/ACCP, 2003; Robergs *et al.*, 2010). To determine the degree of expiratory flow limitation, an average breath was constructed for the selected 30 s period by splitting each breath into equal time segments. The number of time segments was based on the mean T_{TOT} with a resolution of 0.01 s; for example, if the mean T_{TOT} for the final 30 s was 2.5 s then every breath was split into 250 equal time segments. A flow-volume loop was then constructed from the average breath and placed at EELV inside the maximal flow-volume loop to determine the degree of expiratory flow limitation and available inspiratory reserve (Johnson *et al.*, 1999b). The diaphragm pressure-time index (PTI_{di}) was calculated as the product of the tidal P_{di} swing/$P_{di,max}$ ($P_{di,max}$ was taken as the highest P_{di} during a maximal Müeller manoeuvre) and the fractional inspiratory time (T_I/T_{TOT}) (Bellemare and Grassino, 1982). An index of neuromechanical uncoupling was calculated as the ratio of inspiratory effort ($P_{oe}/P_{I,max}$) to thoracic displacement (V_T/VC) (O'Donnell *et al.*, 1997).

7-2.6 Statistics

Two-factor repeated measures ANOVA was used to assess for differences in selected pressure- and flow-derived variables for time (rest, stages 2-5) and condition (unbound and bound). Where a significant interaction effect occurred, post-hoc analysis was carried out using Bonferroni corrected pairwise comparisons. Peak cardiorespiratory, metabolic and perceptual data were analysed using paired samples t-test. Statistical analyses were carried out using SPSS 16.0 for Windows (SPSS Inc., an IBM Company, Chicago, IL, USA). Data are presented as means ± SD.

7-3 Results

7-3.1 Sub-maximal exercise responses

Cardiorespiratory and perceptual

Group mean cardiorespiratory data are shown in Fig 7-1. There was a significant interaction effect for $\dot{V}O_2$ between condition and time ($p = 0.002$). Post-hoc analysis revealed that $\dot{V}O_2$ was significantly higher in the bound condition at stage 5 ($p = 0.021$). A significant interaction effect also occurred for $[La^-]_B$ between condition and time ($p = 0.010$). Post-hoc analysis revealed $[La^-]_B$ was significantly lower in the bound condition at stage 5 ($p = 0.024$). There was a linear increase in HR vs. $\dot{V}O_2$ throughout exercise in both conditions; however, the slope of the HR-$\dot{V}O_2$ response was less with binding (57.9 ± 34.3 vs. 69.2 ± 33.5 beats/ml/min, $p = 0.026$). Minute ventilation, f_R and V_T were unchanged with binding, as were all other cardiorespiratory indices and perceptual ratings ($p > 0.05$).

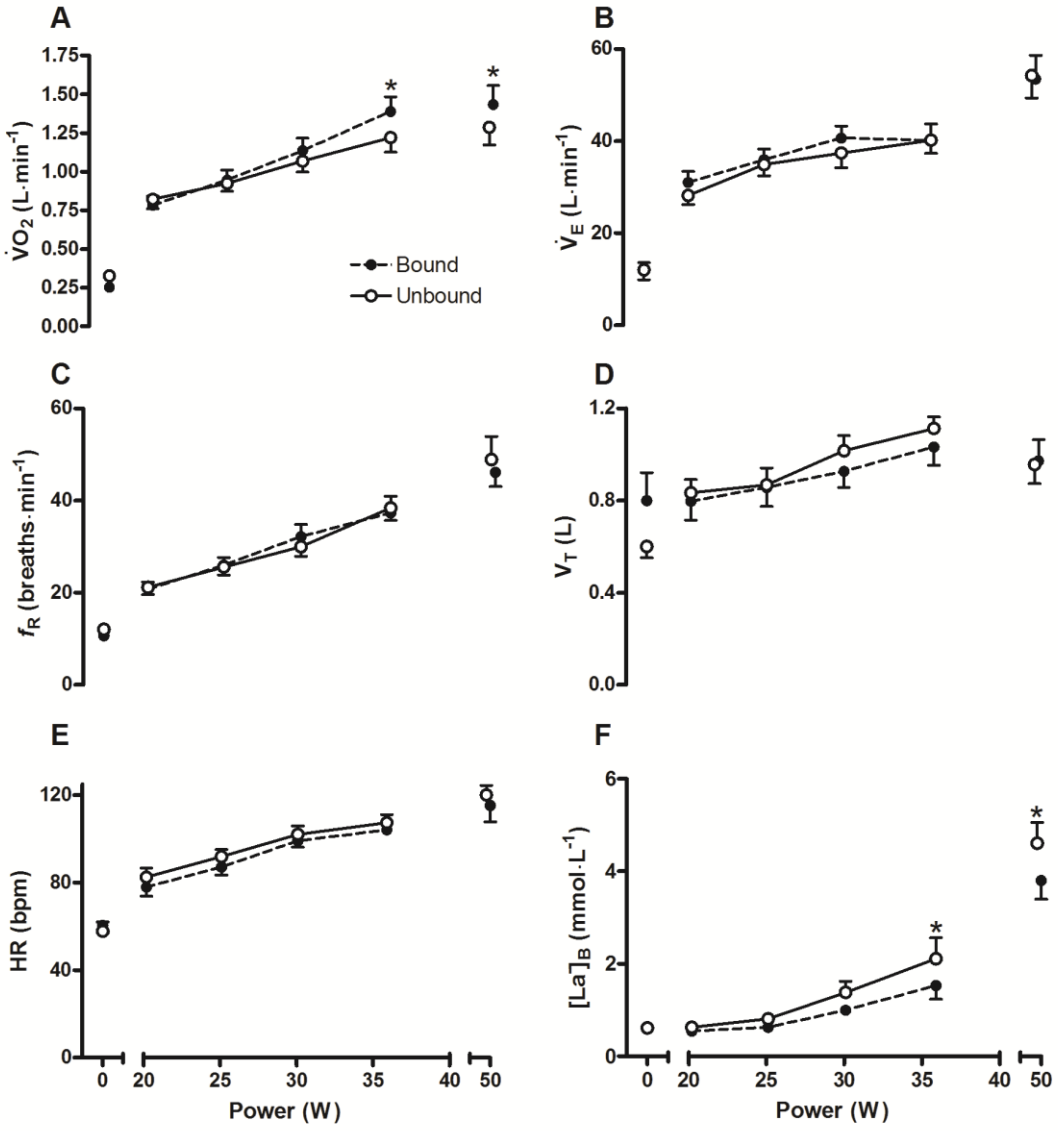

Fig 7-1 Oxygen uptake (A), minute ventilation (B), breathing frequency (C), tidal volume (D), heart rate (E) and blood lactate concentration (F) in response to sub-maximal and maximal wheelchair propulsion with (dashed lines, solid circles) and without (solid lines, open circles) abdominal binding.

Note the significant increase in oxygen uptake and concomitant reduction in blood lactate concentration during the final stage of sub-maximal exercise and at maximal exercise. Data are mean ± standard error for 8 participants. Significant difference between conditions at that time point ($p < 0.05$)*

Operating lung volumes and ventilatory constraint

Group mean measurements of operating lung volume and selected indices of ventilatory constraint are shown in Table 7-1. There was a significant reduction in EELV and EILV throughout exercise with binding (p = 0.017 and p = 0.035, respectively; see also Fig 7-2 and 7-3). There was also a significant interaction effect for EELV between condition and time (p = 0.019), whereby the increase in EELV throughout exercise was attenuated at stage 5 with binding (p = 0.002). Despite these reductions in EELV, binding was unable to offset the dynamic hyperinflation at the onset of exercise; consequently EELV increased throughout exercise in the unbound and bound conditions (p < 0.001 and p < 0.001, respectively). The $\dot{V}_E/\dot{V}_{ECAP}$ was unchanged with binding throughout exercise (p = 0.384), as was the rate of neuromechanical uncoupling (p = 0.211).

Pressure-derived indices and respiratory mechanics

Group mean pressure-and flow-derived measurements are shown in Table 7-2. Binding increased end-expiratory P_{di} and P_{ga} (p = 0.001 and p < 0.001, respectively) as well as end-inspiratory P_{di} and P_{ga} (p < 0.001 and p = 0.001, respectively) throughout exercise (see Fig 7-4). There was no change in tidal P_{di} swing during sub-maximal exercise with binding; accordingly the increase in end-expiratory P_{di} with binding resulted in an increase in PTP_{di} throughout exercise (p = 0.002). There was a significant interaction effect for PTI_{di} between binding and time (p = 0.049), whereby the increase in PTI_{di} throughout exercise was attenuated at stage 5 with binding (p = 0.006).

Table 7-1 Operating lung volume and indices of ventilatory constraint at rest and during sub-maximal incremental exercise with and without binding

	Effect		0 W	20 W	25 W	30 W	35 W
EELV, %TLC	†‡	UB	50 ± 9	55 ± 6	56 ± 7	59 ± 5	64 ± 10
		B	44 ± 9	52 ± 9	53 ± 8	54 ± 7	56 ± 8*
EILV, %TLC	†	UB	61 ± 9	73 ± 3	72 ± 5	79 ± 5	83 ± 11
		B	57 ± 13	67 ± 9	69 ± 7	71 ± 6	75 ± 5
IRV, % TLC	†	UB	39 ± 9	27 ± 3	28 ± 5	21 ± 5	17 ± 10
		B	43 ± 13	33 ± 9	31 ± 7	29 ± 6	26 ± 4
IFR, % capacity		UB	6 ± 2	20 ± 13	27 ± 15	26 ± 11	34 ± 18
		B	10 ± 2	20 ± 7	28 ± 15	29 ± 11	34 ± 14
$\dot{V}_E/\dot{V}_{ECAP}$, %		UB	7 ± 3	17 ± 5	25 ± 6	23 ± 7	34 ± 14
		B	12 ± 5	18 ± 10	28 ± 15	23 ± 8	31 ± 7

Definition of abbreviations: UB, unbound; B, bound; EELV, end-expiratory lung volume; TLC, total lung capacity; EILV, end-inspiratory lung volume; IRV, inspiratory reserve volume; IFR, inspiratory flow reserve; $\dot{V}_E$, minute ventilation; $\dot{V}_{ECAP}$, ventilatory capacity. Values are means ± SD for 7 participants. † Significant main effect for condition ($p < 0.05$); ‡ significant interaction effect ($p < 0.05$); * significant *post-hoc* pairwise comparison ($p < 0.05$).

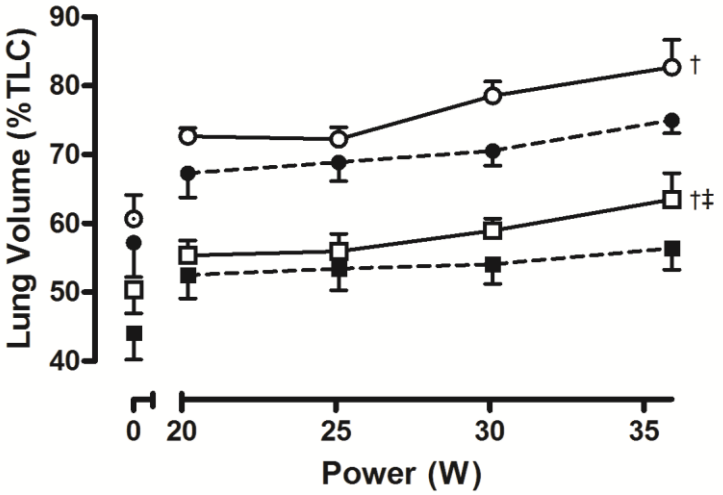

Fig 7-2 End-expiratory lung volume (EELV; squares) and end-inspiratory lung volume (EILV; circles), expressed as a percentage of total lung capacity (TLC) with (dotted line, solid symbols) and without (sold line, open symbols) abdominal binding.

*Note the significant reduction in both EELV and EILV throughout exercise with abdomianl binding. Data are mean ± standard error for 7 participants. † Significant main effect for condition (*p *< 0.05); ‡ significant interaction effect (*p *< 0.05).*

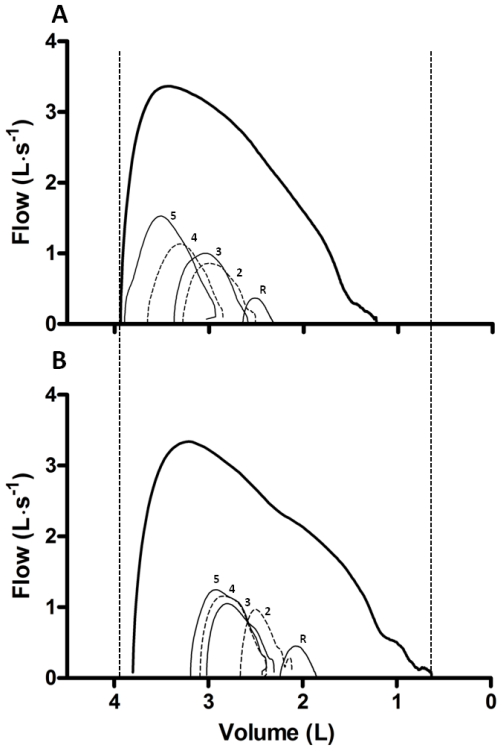

Fig 7-3 Representative flow-volume response to sub-maximal incremental treadmill exercise for a single subject in the unbound (A) and bound condition (B).

Each flow-volume loop represents an ensemble-averaged flow-volume loop, averaged over 30 s of rest (R) and over the first 30 s of the final min of each sub-maximal exercise stage (2-5). Note the progressive dynamic hyperinflation from the onset of exercise in both conditions. Note also the reduction in operating lung volumes and concomitant increase in inspiratory reserve volume at rest, and throughout all sub-maximal exercise stages in the bound condition. Vertical dotted lines indicate the changes in total lung capacity and residual volume in the bound condition.x

Table 7-2 Respiratory muscle function at rest and during sub-maximal incremental exercise with and without binding

	Effect		0 W	20 W	25 W	30 W	35 W
Tidal pressure swings							
P_{di}, cmH_2O	†	UB	16.0 ± 6.9	29.9 ± 12.8	33.5 ± 11.5	40.9 ± 12.8	44.4 ± 10.1
		B	20.5 ± 6.0	35.3 ± 16.7	39.0 ± 17.3	39.7 ± 14.2	43.9 ± 14.4
P_{ga}, cmH_2O	†	UB	13.3 ± 6.6	22.7 ± 10.8	25.4 ± 10.0	31.9 ± 10.6	35.5 ± 7.6
		B	16.7 ± 5.6	27.5 ± 14.5	29.5 ± 13.7	31.5 ± 11.5	35.4 ± 11.8
P_{oe}, cmH_2O		UB	−2.7 ± 0.7	−7.2 ± 2.5	−8.1 ± 2.6	−8.9 ± 3.0	−10.4 ± 4.3
		B	−3.8 ± 1.8	−7.9 ± 3.1	−9.5 ± 5.4	−8.9 ± 4.0	−9.9 ± 5.3
PTP_{di}, $cmH_2O{\cdot}s{\cdot}min^{-1}$	†	UB	564 ± 138	927 ± 311	1072 ± 296	1068 ± 304	1128 ± 235
		B	734 ± 234	1131 ± 383	1410 ± 476	1332 ± 338	1445 ± 231
PTP_{oe}, $cmH_2O{\cdot}s{\cdot}min^{-1}$		UB	−140 ± 44	−197 ± 40	−194 ± 42	−205 ± 50	−217 ± 42
		B	−178 ± 62	−173 ± 50	−231 ± 97	−239 ± 64	−232 ± 57
$C_{L,dyn,}$ $L{\cdot}cmH_2O^{-1}$		UB	0.18 ± 0.05	0.12 ± 0.06	0.10 ± 0.05	0.10 ± 0.05	0.10 ± 0.05
		B	0.16 ± 0.05	0.11 ± 0.06	0.11 ± 0.05	0.12 ± 0.08	0.12 ± 0.08
T_I/T_{TOT}	†	UB	0.45 ± 0.03	0.43 ± 0.05	0.49 ± 0.05	0.47 ± 0.05	0.50 ± 0.02
		B	0.46 ± 0.04	0.44 ± 0.07	0.44 ± 0.03	0.46 ± 0.06	0.46 ± 0.02
PTIdi,	‡	UB	0.07 ± 0.03	0.11 ± 0.04	0.13 ± 0.05	0.15 ± 0.06	0.20 ± 0.11
		B	0.07 ± 0.03	0.07 ± 0.04	0.12 ± 0.06	0.13 ± 0.05	0.16 ± 0.09*
$(P_{oe}/P_{I,max})/(V_T/VC)$		UB	0.19 ± 0.21	0.43 ± 0.29	0.56 ± 0.47	0.53 ± 0.52	0.56 ± 0.61
		B	0.21 ± 0.15	0.34 ± 0.18	0.43 ± 0.28	0.40 ± 0.32	0.46 ± 0.40

Definition of abbreviations: UB, unbound; B, bound; P_{di}, transdiaphragmatic pressure; P_{ga}, gastric pressure; P_{oe}, oesophageal pressure; $C_{L,dyn}$, dynamic lung compliance; T_I/T_{TOT}, fractional inspiratory time; PTI_{di}, diaphragm pressure-time index; $(P_{oe}/P_{I,max})/(V_T/VC)$, rate of neuromechanical uncoupling. Values are means ± SD for 7 subjects. † Significant main effect for condition ($p < 0.05$); ‡ significant interaction effect ($p < 0.05$); * significant *post-hoc* pairwise comparison ($p < 0.05$).

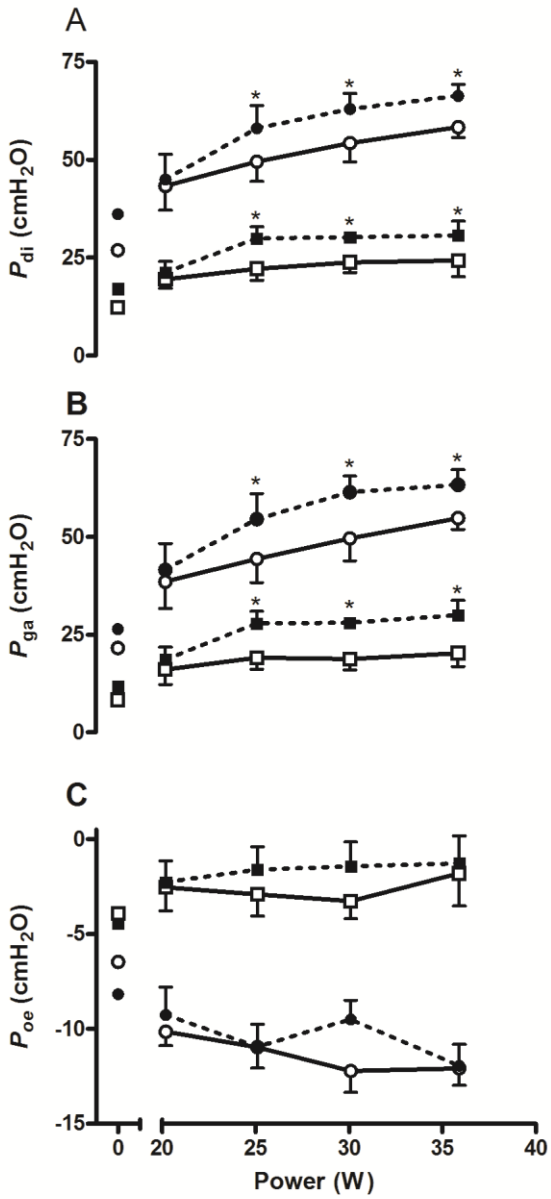

Fig 7-4 End-expiratory (squares) and end-inspiratory (circles) transdiaphragmatic pressure (A), gastric pressure (B) and oesophageal pressure (C) with (dotted lines) and without (solid lines) abdominal binding.

*Note the significant increase in both end-expiratory and end-inspiratory transdiaphragmatic and gastric pressure throughout sub-maximal exercise with abdominal binding. Data are mean ± standard error for 7 participants. * Significant* post-hoc *pairwise comparison (*p *< 0.05).*

7-3.2 Peak exercise responses

Peak cardiorespiratory and metabolic responses to incremental exercise are shown in Table 7-2. There was no difference in peak power between the two conditions; however, $\dot{V}O_{2peak}$ was increased ($p < 0.001$) and there was a strong trend towards a reduction in peak $[La^-]_B$ ($p = 0.052$) with binding (Fig 7-1). Minute ventilation, f_R and V_T were unchanged with binding ($p > 0.53$). There was, however, a significant reduction in $\dot{V}_E/\dot{V}CO_2$ ($p = 0.012$) and a trend towards a reduction in $\dot{V}_E/\dot{V}O_2$ ($p = 0.067$) with binding. For both conditions, PET_{CO2} remained similar to baseline values throughout exercise, but there was no evidence of arterial hypoxaemia at peak exercise in either condition, except in one participant for whom SpO_2 dropped from 97% at resting baseline to 89% at peak exercise in both conditions. All other cardiorespiratory indices were unchanged with binding.

Table 7-3 Peak responses to treadmill exercise

	Unbound	Bound
Peak power, W	49.1 ± 11.6	50.0 ± 13.2
$\dot{V}O_2$, l·min^{-1}	1.29 ± 0.33	1.43 ± 0.35*
$\dot{V}O_2$, ml·kg^{-1}·min^{-1}	19.0 ± 2.1	21.2 ± 2.8*
$\dot{V}CO_2$, l·min^{-1}	1.38 ± 0.36	1.54 ± 0.35
$[La^-]_B$, mmol·l^{-1}	4.6 ± 1.2	3.8 ± 1.0*
$\dot{V}_E$, l·min^{-1}	46.1 ± 8.7	48.9 ± 14.2
f_R, breaths·min^{-1}	54 ± 14	54 ± 15
V_T, l·min^{-1}	0.94 ± 0.21	0.92 ± 0.24
RER	1.08 ± 0.12	1.08 ± 0.13
$\dot{V}_E/\dot{V}O_2$	39.0 ± 10.2	33.0 ± 6.0
$\dot{V}_E/\dot{V}CO_2$	35.6 ± 6.1	30.6 ± 4.4*
PET_{CO2}, mmHg	37.5 ± 8.0	35.5 ± 5.8
SpO_2, %	95 ± 3	95 ± 3
HR, beats·min^{-1}	120 ± 12	122 ± 13
RPE (dyspnoea)	7.0 ± 2.7	7.1 ± 2.9
RPE (arm discomfort)	7.5 ± 2.0	7.4 ± 2.0

Definition of abbreviations: $\dot{V}O_2$, oxygen uptake; $\dot{V}CO_2$, carbon dioxide output; $[La^-]_B$, blood lactate concentration; $\dot{V}_E$, minute ventilation; f_R, respiratory frequency; V_T, tidal volume; RER, respiratory exchange ratio; PET_{CO2}, end-tidal partial pressure of carbon dioxide; S_pO_2, arterial oxygen saturation; HR, heart rate; RPE, ratings of perceived exertion. Values are means ± SD for 8 subjects. * Significant difference between conditions ($p < 0.05$).

7-4 Discussion

The aim of this chapter was to determine whether abdominal binding improves cardiorespiratory function during maximal incremental treadmill exercise in highly-trained athletes with cervical SCI. The main findings were that abdominal binding significantly increased peak oxygen uptake and reduced peak blood lactate concentration in response to maximal wheelchair propulsion in highly-trained athletes with cervical SCI, despite no change in peak work rate. Furthermore, abdominal binding altered respiratory mechanics during sub-maximal incremental exercise by reducing operating lung volumes and attenuating the rise in the pressure-time index of the diaphragm.

7-4.1 Cardiorespiratory responses to abdominal binding

It is shown for the first time that abdominal binding increases sub-maximal and peak $\dot{V}O_2$ and reduces peak blood lactate concentration in individuals with SCI. The 12% increase in $\dot{V}O_{2peak}$ is similar to that reported by Pitetti *et al.* (1994) in response to an anti-G suit. An anti-G suit applies rhythmical compression to the legs and abdomen in unison. Thus, a similar increase in $\dot{V}O_{2peak}$ with abdominal compression alone may seem surprising. It is possible, however, that the increased stroke volume and oxygen uptake in the study by Pitetti *et al.* (1994) may be due primarily to the translocation of blood from the abdominal viscera to the heart, rather than from the lower body to the heart. Evidence supporting this postulate is provided by a study that demonstrated a cessation of femoral venous outflow from the lower limbs with large increases in abdominal pressure (Miller *et al.*, 2005a), and from a study that demonstrated a large increase in splanchnic emptying with abdominal compression (Aliverti *et al.*, 2009).

An increase in $\dot{V}O_{2peak}$ with binding contradicts the only previous study to investigate the effect of abdominal binding on $\dot{V}O_{2peak}$ in the SCI population (Kerk *et al.*, 1995). In that study, Kerk *et al.* (1995) reported that $\dot{V}O_{2peak}$ was unchanged with abdominal binding in individuals with high-

thoracic SCI. This discrepancy is likely due to greater loss of supraspinal sympathetic control in the current study cohort, but may also be due to differences in exercise modality or the degree of abdominal compression applied by the binder. In the current study, the degree of abdominal compression was quantified by measuring the change in gastric pressure with binding, whereas in the study by Kerk *et al.* (1995) abdominal compression was only estimated via a change in abdominal girth.

The increase in $\dot{V}O_2$ with abdominal binding in the current study is attributed to improved central haemodynamics during the later stages of exercise. The large increase in end-expiratory gastric pressure with binding may increase the pressure gradient between the abdomen and the right atrium, and aid venous return and cardiac output through a greater engagement of the Frank-Starling mechanism (see also section 5-4.3). Although no measure of stroke volume or cardiac output was provided in this chapter, the slope of the HR-$\dot{V}O2$ response was less with binding suggesting a greater stroke volume. Furthermore, 7 of the 8 study participants also took part in the study described in chapter 5 of this thesis and demonstrated an increase in resting cardiac output with binding (see section 5-3.3). There is also no reason to believe that binding improves oxygen extraction. Thus, according to the Fick principle, any increase in oxygen uptake with binding is likely due to improved oxygen delivery, subsequent to an enhanced venous return, stroke volume and cardiac output.

An alternative explanation for the increase in $\dot{V}O_2$ with abdominal binding may be related to an increased diaphragmatic work. This appears unlikely, however, given that respiratory muscle work (PTP_{di}) was higher with abdominal binding throughout all sub-maximal exercise stages, yet $\dot{V}O_2$ was only different during the final sub-maximal exercise stage and at peak exercise. Furthermore, in AB individuals who reach substantial levels of ventilation, the total $\dot{V}O_2$ required by the respiratory muscles is 13-15% of whole-body $\dot{V}O_2$ (Aaron *et al.*, 1992). In the current study, binding caused a 13% increase in whole body $\dot{V}O_{2peak}$. Thus, an increase in

diaphragm work may account for a small proportion of the increase in $\dot{V}O_2$, however, it cannot account for the majority of the increase.

Despite an improvement in $\dot{V}O_{2peak}$ with binding, there was no change in peak power during the incremental treadmill test. This was surprising given that abdominal binding significantly increased the distance covered during a 4-min maximal push in the field (see section 6-3.2). The reason why binding did not improve peak power is unclear, but may relate to an inability of the treadmill protocol to elicit a true peak response, as evidenced by the substantially lower peak blood lactate concentrations in the current study compared to the maximal 4-min push field test (see also section 7-4.4). An alternative reason for the lack of difference in peak power with abdominal binding may have been due to fatigue from the sub-maximal exercise tests. This appears unlikely, however, as blood lactate had always returned to pre-exercise baseline values prior to the start of every exercise trial and the order of the conditions was counterbalanced.

In agreement with the sub-maximal data in chapter 6, there was a strong trend towards a reduction in $\dot{V}_E/\dot{V}O_2$ during maximal exercise in the current study. There was also a significant reduction in the $\dot{V}_E/\dot{V}CO_2$ at maximal exercise with binding. However, the cause of the reduction in ventilatory equivalents appears to differ between chapters. In chapter 6, it was hypothesised that binding may improve ventilatory efficiency by increasing V_T and reducing dead space ventilation. Although dead space ventilation was not measured in the current study there was no change in V_T or $\dot{V}_E$, suggesting binding does not improve ventilatory efficiency *per se*. Rather the reductions in the ventilatory equivalents in the current study appear to be a consequence of the increased $\dot{V}O_2$ and $\dot{V}CO_2$.

7-4.2 Operating lung volumes and ventilatory constraint

In agreement with previous findings during arm-crank exercise in highly-trained athletes with cervical SCI (Taylor *et al.*, 2010), participants in the current study exhibited a sudden and

sustained dynamic hyperinflation from the onset of wheelchair exercise in the unbound condition, despite no evidence of expiratory flow limitation. Whether this response is a consequence of abdominal muscle dennervation, or the 'normal' response to upper-body exercise is unclear as only two studies have documented changes in IC (and therefore EELV) during upper-body exercise in healthy AB individuals, and both reported contradictory results (Alison *et al.*, 1998; Cerny and Ucer, 2004). For individuals with cervical SCI, abdominal muscle dennervation results in the recruitment of accessory expiratory muscles (e.g., pectoralis major) to expire below FRC (De Troyer *et al.*, 1986a). Given that most of these accessory expiratory muscles are also prime movers during upper body exercise (Lin *et al.*, 2004; Vanlandewijck *et al.*, 2001) it is perhaps unsurprising that dynamic hyperinflation prevails from the onset of exercise in this population. Although abdominal binding was unable to prevent dynamic hyperinflation the degree of hyperinflation throughout sub-maximal exercise was attenuated with binding, a finding that is attributed to the binder compressing the abdomen and reducing FRC such that there is less air left in the thorax at end-expiration.

In the absence of a change in TLC with binding, the reduction in operating lung volumes with binding lowered the EILV-to-TLC ratio. A high EILV-to-TLC ratio causes ventilatory constraint by increasing the elastic work of breathing and predisposing the inspiratory muscles to fatigue (Johnson *et al.*, 1999b). In the current study, EILV averaged 83% of TLC during the final stage of sub-maximal exercise in the unbound condition, with three of the participants exceeding 90% of TLC. The reduction in EILV with binding was such that no participant's EILV exceeded 80% of their TLC. Thus, abdominal binding may reduce the inspiratory elastic work of breathing and the propensity for diaphragm fatigue. Diaphragm fatigue was not measured in the current study, however, the PTI_{di} during exercise in the unbound condition exceeded values known to elicit diaphragm fatigue in AB individuals (>0.15; Bellemare and Grassino, 1982), and individuals with cervical SCI (> 0.10; Nava *et al.*, 1996). With abdominal binding, however, the PTI_{di} was reduced

during the final stage of sub-maximal exercise, suggesting abdominal binding may reduce the propensity for diaphragmatic fatigue during high-intensity wheelchair propulsion.

The ventilatory and metabolic responses to wheelchair exercise in the unbound condition were higher than values typically reported in the literature for untrained cervical SCI during arm-crank exercise and wheelchair ergometry (Coutts *et al.*, 1983; Gass and Camp, 1979; Janssen *et al.*, 2002; Wicks *et al.*, 1983) and higher than athletes with cervical SCI during arm-crank exercise (Goosey-Tolfrey *et al.*, 2006; Taylor *et al.*, 2010). Despite these ventilatory and metabolic demands, there was no evidence of expiratory flow limitation in any individual during wheelchair propulsion in either condition. That EELV was reduced at any given work rate with abdominal binding, and the available expiratory flow reserve becomes less as RV approached, suggests that binding may increase the propensity for expiratory flow limitation. However, it was demonstrated in chapter 5 of this thesis that expiratory flow throughout a vital capacity manoeuvre is increased with binding. Thus, any negative effects of reducing EELV in terms of expiratory flow limitation may have been offset by an increase in the available expiratory flow at that EELV. Accordingly, the reduction in operating lung volumes with binding can be considered a beneficial mechanical alteration.

7-4.3 Pressure-derived indices and respiratory mechanics

End-inspiratory and end-expiratory P_{di} and P_{ga} were increased throughout sub-maximal exercise with abdominal binding (see Fig. 7-4). These findings are in agreement with a previous study in AB individuals who exercised with an abdominal corset (Hussain *et al.*, 1985). Unlike that study, however, there was no increase in tidal P_{di} during any exercise stage in the current study. Thus, the increase in PTP_{di} with binding noted in the present study reflects the increase in end-expiratory P_{di}, suggesting that binding does not increase the respiratory work of the diaphragm *per se*. Rather, binding increases the bracing work of the diaphragm to prevent the abdominal contents from entering the thoracic cavity.

The pressures developed by the diaphragm during inspiration in the unbound condition are ~50% higher than those reported in AB individuals during high intensity (70-80% of maximum work rate) lower limb cycling to exhaustion (Aliverti *et al.*, 1997; Hussain *et al.*, 1985). However, ventilation was substantially lower in the current study. There are two potential reasons for this latter finding; first, dennervation of the abdominal muscles may increase transdiaphragmatic pressure by increasing the postural work of the diaphragm (Sinderby *et al.*, 1992); and second, the decreased rib-cage compliance in cervical SCI (Estenne and De Troyer, 1986) may increase the respiratory pressure required to inflate the thoracic cavity (i.e., increased rate of neuromechanical uncoupling). Although the work of the diaphragm in maintaining posture could not be elucidated in the current study, the rate of neuromechanical uncoupling during exercise in both conditions was similar to that reported in AB individuals during lower body exercise (O'Donnell *et al.*, 2000). This rate, however, may be underestimated in cervical SCI due to the tachypnic breathing pattern minimising inspiratory pleural pressure generation such that changes in P_{oe} may underestimate the true neuromotor output of the respiratory muscles in this population (Lougheed *et al.*, 2002). Indeed, if the rate of neuromechanical uncoupling is calculated using the P_{di} swing and $P_{di,max}$, rather than the P_{oe} swing and $P_{I,max}$, then the rate of neuromechanical uncoupling is increased four-fold to 2.29 ± 2.20 in the unbound condition and 2.12 ± 1.96 in the bound condition, both of which are substantially higher than has been reported in AB individuals with chest wall restriction (O'Donnell *et al.*, 2000) and in individuals with COPD (O'Donnell *et al.*, 1997). It was also found in the current study that V_T increased to ~1 L and then fell during maximal exercise in both conditions. This pattern is similar to that seen in AB individuals during exercise with chest wall restriction, where the rate of neuromechanical uncoupling is increased (O'Donnell *et al.*, 2000). Thus, the large transdiaphragmatic pressure swings during exercise in the current study are likely the result of an impaired thoraco-abdominal configuration and represent a mismatch between the pressure required to inflate the thoracic cavity and the actual change in V_T during exercise.

7-4.4 Critique of methods

A protocol with constant speed and a progressively increasing gradient was used to elicit peak cardiorespiratory responses. This protocol has been shown to produce similar peak responses to those elicited by protocols that increase speed with a constant gradient, or that increase both speed and gradient simultaneously (Hartung *et al.*, 1993). However, Hartung *et al.* (1993) studied untrained individuals with low thoracic SCI, and retrospectively it appears that the smaller muscle mass in cervical compared to thoracic SCI may have rendered the current protocol insufficient to elicit a true peak response. That is, the gradient became too demanding for the small muscle mass of cervical SCI individuals. When the same individuals were assessed during a maximal 4-min push (chapter 6) they tended to lean far forward during wheelchair propulsion, but would regularly sit upright to take a deep breath; presumably because the leant forward posture impaired the pressure-generating capacity of the diaphragm. This propulsion technique cannot be fully replicated on a treadmill as a continuous push is required to prevent the wheelchair from falling against the springs on the side bar of the treadmill, which was considered the termination point of the laboratory-based protocol. Thus, the choice of protocol and the subsequent alteration in push technique may explain why peak power was not increased during the maximal incremental test.

Dynamic lung hyperinflation during exercise is a common finding in endurance athletes (Mota *et al.*, 1999), fit elderly (Johnson *et al.*, 1991) and individuals with COPD (O'Donnell *et al.*, 2001; Vogiatzis *et al.*, 2004). Hyperinflation in those populations occurs to permit an increase in expiratory flow (Ferguson, 2006). In the current study, there was no evidence of expiratory flow limitation in any participant. Thus, the question remains as to why EELV was not preserved at baseline values? The method used for detecting flow limitation in the current study depends critically on the placement of the tidal-flow-volume loop inside the maximal flow-volume loop and the selection of an appropriate maximal flow-volume loop (Johnson *et al.*, 1999b). Previous studies in healthy AB individuals have demonstrated a small bronchodilation post-exercise (Jensen

et al., 1980). Thus, a pre-exercise maximal flow-volume loop may underestimate ventilatory capacity during exercise. Conversely, in asthmatics there may be a reduction in the maximal flow-volume loop after ~15 min post-exercise (Beck *et al.*, 1994); in this instance a pre-exercise maximum flow-volume loop may overestimate ventilatory capacity during exercise. There are currently no guidelines on the appropriate selection of a maximal flow-volume loop in SCI; however, there was minimal evidence of exercise-induced bronchodilation in the current study (one participant exhibited a post-exercise VC that was 0.1 L greater than that recorded pre-exercise). This presumably reflects the low catecholamine release in the cervical SCI population (Dela *et al.*, 2003; Schmid *et al.*, 1998). Thus, in 6/7 participants the maximal flow-volume loop was selected from the highest VC prior to exercise. This may, however, still lead to an over estimation of the ventilatory capacity during exercise because many of the muscles typically used during a VC manoeuvre in this population (i.e., pectoralis major and latissimus dorsi) are also prime-movers during wheelchair propulsion (Lin *et al.*, 2004; Vanlandewijck *et al.*, 2001). Accordingly, the maximal flow-volume loop at rest may not reflect the maximum flow-volume that can be generated during exercise. It is possible, therefore, that individuals with SCI in the current study demonstrated expiratory flow limitation but that the current method of measurement was insufficient to detect its presence.

An alternative method for assessing expiratory flow limitation is the negative expiratory pressure (NEP) technique (Johnson *et al.*, 1999a; Koulouris *et al.*, 1995; Valta *et al.*, 1994). The NEP technique applies a small negative pressure at the mouth, and the flow curve is compared to that from the preceding breath. The NEP method may be advantageous in the SCI population as it circumvents the problem of placing a tidal flow-volume loop inside a maximal flow-volume loop. However, the NEP technique does not allow the degree of flow limitation to be quantified (Johnson *et al.*, 1999a). Thus, future studies should combine the NEP technique with IC measurements to assess expiratory flow limitation during exercise in this population.

7-5 Conclusions

It is shown for the first time that abdominal binding improves peak oxygen uptake and reduces the metabolic acidosis during maximal wheelchair propulsion on a treadmill. Another novel finding is that reducing operating lung volumes and the degree of ventilatory constraint with abdominal binding provides beneficial mechanical alterations to the respiratory system during exercise in highly-trained individuals with cervical SCI. Thus, abdominal binding provides a simple, easy-to-use tool that can improve cardiorespiratory function and increase $\dot{V}O_{2peak}$ during exercise in individuals with cervical SCI.

CHAPTER EIGHT:

GENERAL DISCUSSION

8-1 Introduction

This chapter reviews the primary findings of the thesis and provides a summary of mechanisms by which abdominal binding improves cardiorespiratory function at rest and during exercise in highly-trained athletes with cervical SCI. The chapter also provides an overview of how this research has helped to understand what factors limit exercise performance in this population. The clinical benefits of abdominal binding are also considered. The chapter concludes with recommendations for further study.

8-2 Main findings

8-2.1 Resting cardiorespiratory function in highly-trained individuals with spinal cord injury

The aim of chapter 4 was to describe resting cardiorespiratory function in a group of highly-trained athletes with cervical SCI and to compare the data with an able-bodied control group. Using body plethysmography, spirometry, phrenic nerve stimulation and transthoracic echocardiography, it was demonstrated that highly-trained athletes with cervical SCI had a restrictive pulmonary defect, impaired diaphragm and expiratory muscle function, and low left ventricular mass and ejection fraction compared to able-bodied controls. The values for pulmonary function and expiratory muscle strength were higher than those typically reported in the untrained cervical SCI population, most likely due to a training-induced increase in strength of the accessory muscles of expiration. A surprising finding from chapter 4 was that cardiac function in highly-trained individuals with cervical SCI was similar to that reported in untrained individuals with cervical SCI, suggesting that the volume of exercise training undertaken by the participants in the current thesis was insufficient to prevent the cardiac atrophy that is known to occur in cervical SCI.

8-2.2 Effect of abdominal binding on resting cardiorespiratory function

The aim of chapter 5 was to determine if abdominal binding improves resting cardiorespiratory function in individuals with SCI, and to assess whether any such changes are dependent on the magnitude of abdominal compression. To gain insight into the mechanisms that underlie any alterations in function, an able-bodied control group was also included in the study. Using body plethysmography, spirometry, phrenic nerve stimulation and transthoracic echocardiography, it was demonstrated that abdominal binding improved pulmonary capacities, increased diaphragm and expiratory muscle strength, and increased cardiac output in the SCI group. It was also demonstrated that a dose-response relationship exists between the degree of abdominal compression and the resultant improvements in cardiorespiratory function in individuals with cervical SCI. Specifically, the changes in cardiorespiratory function with tight binding were approximately double those with loose binding. In the AB group, binding caused small and inconsistent changes in cardiorespiratory function. The most likely mechanism responsible for the binding-induced improvements in cardiorespiratory function in individuals with cervical SCI was a reduction in abdominal compliance and an increase in abdominal pressure.

8-2.3 Effect of abdominal binding on field-based fitness and push kinematics

The aim of chapter 6 was to determine whether abdominal binding improves the performance of field-based exercise tests in highly-trained athletes with cervical SCI. Using four different field-based assessments, abdominal binding increased the maximal distance covered during repeated 4-min pushes, and attenuated blood lactate accumulation and ratings of perceived limb discomfort during these pushes. However, abdominal binding did not improve peak power or push kinematics during a 30 s maximal push, nor did it improve the time to complete an agility field test or a repeated 20 m push test. The mechanism underlying the improvement in 4-min push performance could not be elucidated, but was hypothesised to relate to an improvement in cardiorespiratory function during high-intensity exercise with binding.

8-2.4 Effect of abdominal binding on cardiorespiratory responses to exercise

The aim of chapter 7 was to investigate the mechanism(s) by which abdominal binding alters cardiorespiratory function during exercise in highly-trained athletes with cervical SCI. During one experimental visit to the laboratory, participants completed sub-maximal and maximal wheelchair exercise on a treadmill, with and without abdominal binding. During sub-maximal incremental treadmill propulsion, abdominal binding reduced operating lung volumes, attenuated the degree of dynamic hyperinflation, increased inspiratory reserve volume and increased the diaphragmatic contribution to inspiration. During maximal incremental exercise, abdominal binding increased $\dot{V}O_{2peak}$ and reduced peak blood lactate concentration. Based on the findings of this chapter it was concluded that the increase in $\dot{V}O_{2peak}$ was likely due to a translocation of blood from the splanchnic bed and subsequent increases in venous return, stroke volume, cardiac output and oxygen delivery. However, the exact mechanism responsible for the increased $\dot{V}O_{2peak}$ awaits verifiction.

8-3 Respiratory mechanisms

Section 2-2 provided an overview of the potential ways in which the respiratory system may limit exercise performance in cervical SCI. The following section summarises the mechanisms by which abdominal binding improves respiratory function at rest and how abdominal binding may attenuate the potential respiratory limitations to exercise in individuals with cervical SCI.

Resting mechanisms

The main respiratory outcomes of this thesis were that abdominal binding reduced FRC and increased VC, $P_{di,tw}$, $P_{di,max}$ and $P_{E,max}$. The changes in pulmonary capacities are in agreement with those that have been published in the untrained cervical SCI population (Fig 8-1).

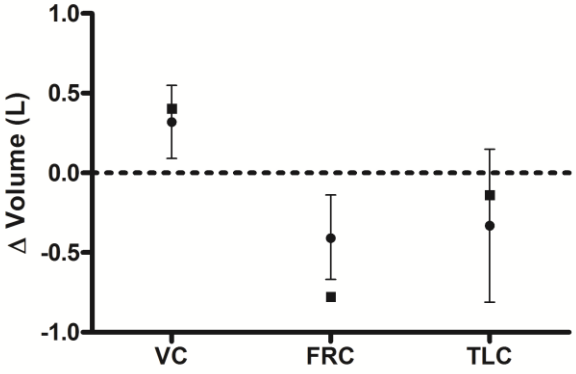

Fig 8-1 Comparison of pulmonary capacities between the data reported in chapter 5 (squares) and those reported in the meta-analysis by Wadsworth *et al.* (2009) (circles).

Fig 8-1 demonstrates that the binding-induced reduction in FRC reported in chapter 5 exceeded that of previous studies, most likely due to a greater degree of abdominal compression in chapter 5 compared to previous studies. The reduction in FRC results in subsequent breaths starting with less air in the thorax, and potentially leads to a reduced TLC, which increases the risk of atelectasis (Wadsworth *et al.*, 2009). However, these potential negative effects of reducing FRC were offset by a subsequent increase in IC, such that TLC remained unchanged with binding. In the absence of a change in TLC, the reduction in FRC and RV resulted in an increased VC with binding and suggests pulmonary function is enhanced with abdominal binding. The magnitude of change in pulmonary volumes and capacities is almost identical to that reported in cervical SCI individuals in the supine versus seated posture (Estenne and De Troyer, 1987). On assumption of the supine posture gravity compresses the abdomen in a similar way to that of binding in the seated posture. In this regard, abdominal binding can be considered to abolish the postural dependence of VC in individuals with cervical SCI.

A reduction in FRC is expected to cranially displace the diaphragm, resulting in diaphragmatic lengthening and an increased radius of curvature; both of which may explain the increased pressure generating capacity of the diaphragm reported in chapter 5. The magnitude of increase in $P_{di,tw}$ and $P_{di,max}$ with binding confirms previous research in cervical SCI (Hart *et al.*,

2005), and suggests that binding improves the mechanical advantage of the diaphragm. A cranial displacement of the diaphragm during inspiration is also expected to increase the zone of apposition and thereby increase lower rib cage expansion. A further consequence of increasing the zone of apposition is a reduction in the area of the upper rib cage exposed to the change in pleural pressure during inspiration (De Troyer, 2005). In unbound individuals with cervical SCI, a small zone of apposition results in a large area of the rib cage exposed to the change in pleural pressure during inspiration. In the absence of intercostal activity, the reduction in pleural pressure due to diaphragmatic contraction will result in a paradoxical inwards motion of the upper rib cage during inspiration (Mortola and Sant'Ambrogio, 1978; Urmey *et al.*, 1986). Thus, further to improving the mechanical advantage of the diaphragm, abdominal binding may improve breathing mechanics by increasing lower rib cage expansion and reducing the degree to which the upper rib cage moves paradoxically during inspiration (Urmey *et al.*, 1986).

Exercise mechanisms

The aforementioned reduction in FRC (EELV) with binding will also benefit respiratory function during exercise. In chapter 7, the degree of dynamic hyperinflation was attenuated throughout exercise with binding, resulting in a subsequent reduction in the EILV-to-TLC ratio and an increased IRV throughout exercise. Attenuation of dynamic hyperinflation is expected to reduce the elastic load imposed on the diaphragm, optimise the length-tension relationship of the diaphragm and reduce the propensity for diaphragmatic fatigue (ATS/ACCP, 2003; Decramer, 1997). Although no objective measure of diaphragmatic fatigue was included in the current thesis, the pressure-time index of the diaphragm was reduced during the final stage of sub-maximal exercise.

To establish the importance of the above mentioned alterations in respiratory function during exercise, it is necessary to establish whether the respiratory system becomes constrained during exercise. Section 2-2.6 proposes three mechanisms that may cause ventilatory constraint during exercise. In the current thesis, there was no evidence of expiratory flow limitation or

exercise-induced arterial hypoxaemia during maximal incremental treadmill exercise. In chapter 7, the pressure-time index of the diaphragm at maximal exercise was elevated to levels associated with fatigue in able-bodied individuals (Bellemare and Grassino, 1982). However, the only study to directly assess whether the diaphragm fatigues during exercise in the highly-trained cervical SCI population reported no difference in the $P_{di,tw}$ pre- to post-exercise (Taylor *et al.*, 2010). Furthermore, during maximal incremental treadmill exercise $\dot{V}_E$ rarely exceeded 50 L·min^{-1}, which is ~50% of that obtained during a maximal voluntary ventilation test and resulted in a $\dot{V}_E/\dot{V}_{ECAP}$ that rarely exceeded 40. Finally, ratings of perceived respiratory discomfort seldom exceeded that of arm discomfort during a maximal 4-min push (chapter 6) or during maximal incremental treadmill exercise (chapter 7). It is concluded, therefore, that the respiratory system does not become constrained during exercise in highly-trained individuals with cervical SCI. Thus, it is unlikely that the alterations in respiratory function with binding contributed to the enhanced $\dot{V}O_{2peak}$ or explained the increased distance covered during the maximal 4-min push.

The summary of potential respiratory limitations to exercise performance in individuals with cervical SCI provided in the literature review (Fig 2-5) is revisited in Fig 8-2. An emphasis is placed on the mechanisms by which abdominal binding may attenuate these limitations.

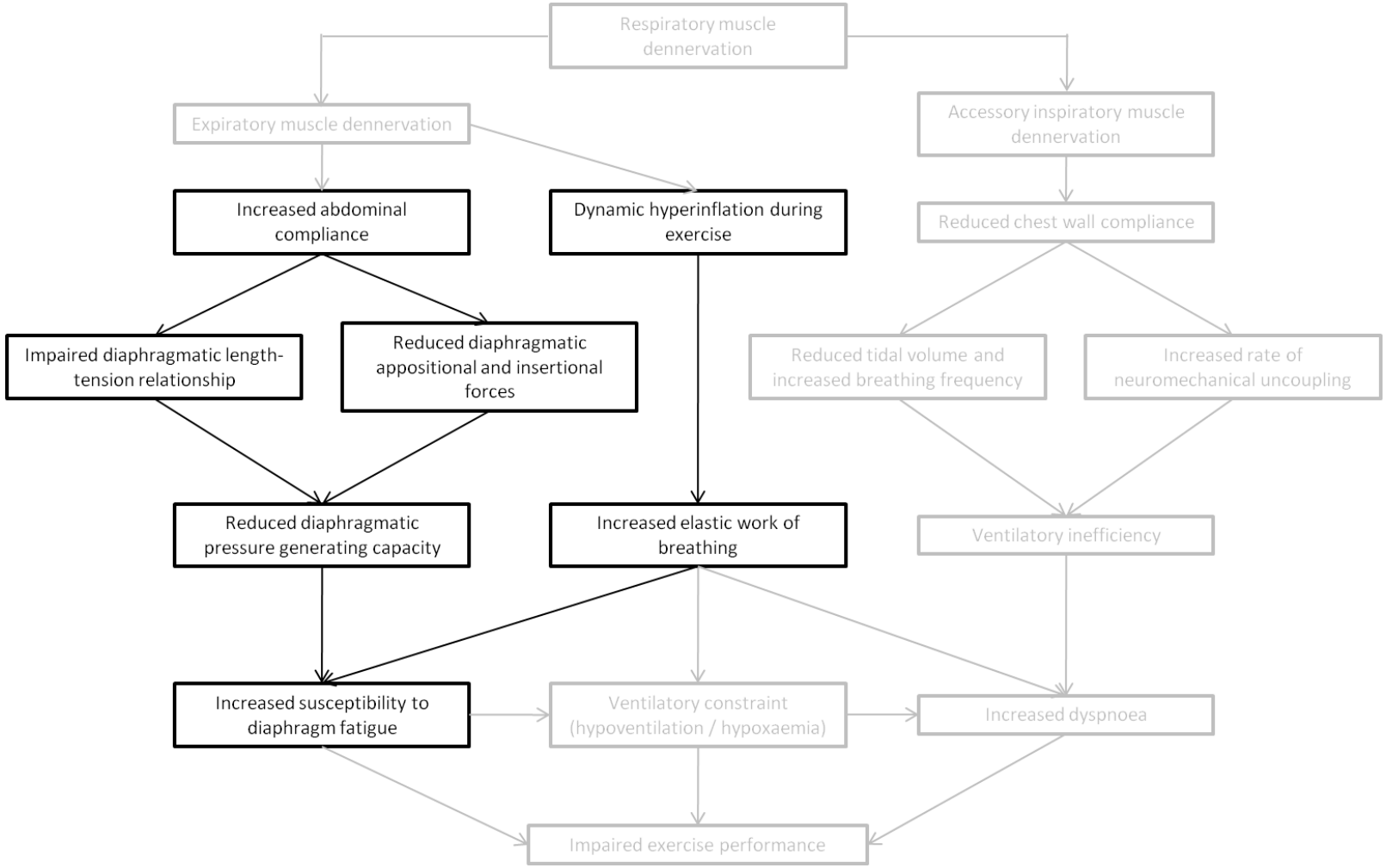

Fig 8-2 Respiratory consequences of cervical SCI and how they may contribute to impaired exercise performance.
An emphasis (black boxes) is now placed on the mechanisms by which abdominal binding may attenuate the respiratory limitations to exercise. See narrative for explanation.

8-4 Cardiovascular mechanisms

Section 2-3 provided an overview of the potential ways in which the cardiovascular system may limit exercise performance in the SCI population. The following section will summarise the mechanisms by which abdominal binding improves cardiovascular function at rest and how abdominal binding may attenuate the potential cardiovascular limitations to exercise in individuals with cervical SCI.

Resting mechanisms

The primary outcome of chapter 5 was that abdominal binding increased resting cardiac output. The postulated mechanism for the increased cardiac output is an enhanced abdomino-throacic pressure gradient, which facilitates a translocation of blood from the abdominal viscera to the heart and increases venous return. In turn, this will increase cardiac pre-load, left ventricular filling, stroke volume and cardiac output through a greater engagement of the Frank-Starling mechanism. Support for this postulate is provided by Aliverti *et al.* (2009) who demonstrated in able-bodied individuals that large increases in intra-abdominal pressure cause a translocation of blood form the abdomen to the thorax and increase cardiac output. The binding-induced increase in cardiac output reported in chapter 5 could also stem from an improved pump action of the diaphragm. This is unlikely, however, as both inspiratory tidal P_{oe} and tidal volume remained unchanged with binding. Thus, right atrial transmural pressure would be expected to be similar for both conditions.

In able-bodied humans, cardiac output is tightly regulated to metabolic demand. In this regard, any mechanical alteration that increases cardiac output without altering metabolism (e.g., lower body positive pressure) stimulates the baroreflex (Seaworth *et al.*, 1985), resulting in a sympathetically mediated vasodilatation and a reduction in HR, such that mean arterial pressure and cardiac output return to 'normal'. In the current thesis, resting energy expenditure was not measured in chapter 5, but was found to be similar for the bound and unbound conditions in chapter 7. Although the temporal response of cardiac output was not examined in the current thesis, the aforementioned increases in resting cardiac output with binding were detected after

10 min of rest. Thus, a prolonged augmentation of cardiac output in the absence of increased resting energy expenditure suggests that the cardiovascular system is unable to adjust in the 'normal' way, perhaps due to an impaired baroreflex (Krum *et al.*, 1992; Munakata *et al.*, 2001). In conclusion, the data presented in this thesis suggest that a translocation of blood from the abdominal viscera to the heart, coupled with an inability of the cardiovascular system to adapt to changes in cardiac output, explains the augmented resting cardiac output with binding.

Exercise mechanisms

The proposed mechanisms by which abdominal binding increased resting cardiac output would also be expected to increase cardiac output during exercise. These mechanisms may also explain the binding-induced increase in $\dot{V}O_{2peak}$ during maximal incremental wheelchair propulsion (chapter 7). To investigate this hypothesis further it is useful to examine the kinetic response of $\dot{V}O_2$ during maximal incremental exercise in both conditions (Fig 8-3, panel A).

Providing that a-$\bar{v}$ O_2 difference remains unaltered with acute abdominal binding, then the $\dot{V}O_2$ response shown in Fig 8-3 should mirror that of cardiac output. It is postulated that if stroke volume is the mechanism by which binding increases cardiac output and $\dot{V}O_2$, then an increased $\dot{V}O_2$ should only be visible once stroke volume becomes limited. Although no study has reported stroke volume changes during incremental exercise in cervical SCI, it is well documented in able-bodied individuals that stroke volume plateaus at 50% of $\dot{V}O_{2max}$ (Astrand *et al.*, 1964; Higginbotham *et al.*, 1986). In Fig 8-3 it is shown that the $\dot{V}O_2$ response to maximal incremental exercise with binding diverges from the $\dot{V}O_2$ response without binding at ~50% of $\dot{V}O_{2peak}$. There is also a slight reduction in HR throughout the maximal incremental exercise test (Fig 8-3, panel B). It is speculated, therefore, that the mechanism by which abdominal binding increases $\dot{V}O_{2peak}$ is via an increased cardiac output subsequent to an enhanced stroke volume. However, research that measures stroke volume during maximal incremental exercise with and without abdominal binding is required to confirm this postulate.

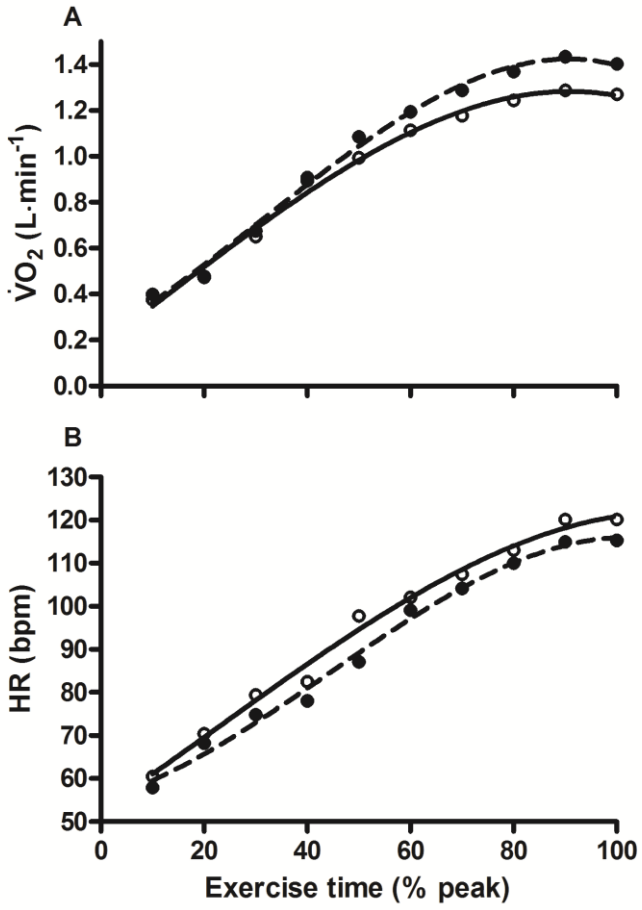

Fig 8-3 Group-mean oxygen uptake ($\dot{V}O_2$; panel A) and heart rate (HR; panel B) response to maximal incremental exercise.
Data have been fitted with a 4th order polynomial function in the unbound (solid lines, open circles) and bound (dotted lines, closed circles) conditions (all $r^2 > 0.99$). Note the divergent $\dot{V}O_2$ response in the bound condition above ~50% of $\dot{V}O_{2peak}$. Note also the trend towards a reduction in HR throughout exercise in the bound condition.

To establish the relevance of the above mentioned alterations in $\dot{V}O_{2peak}$ and cardiovascular function it is necessary to consider whether $\dot{V}O_{2peak}$ limits exercise capacity. In low- to moderately-trained individuals with cervical SCI cardiorespiratory system does not appear to become limited during maximal incremental exercise in the laboratory (Hopman *et al.*, 1998a). In the current study, the plateau in $\dot{V}O_2$ during maximal incremental wheelchair propulsion in the unbound condition (Fig 8-3) suggests that cardiac output and a-$\bar{v}$ O_2 difference reached a plateau, and provides evidence that the cardiovascular system was limited (Bassett and Howley, 2000). It follows, therefore, that the enhanced $\dot{V}O_{2peak}$ with abdominal binding should result in an improvement in exercise capacity. However, there was no change in the peak power achieved during incremental wheelchair propulsion with abdominal binding, suggesting that the

cardiovascular system does not limit exercise capacity during maximal incremental exercise in the laboratory.

Compared to laboratory based exercise tests, there are no available data on the cardiovascular responses to a maximal 4-min push field test in individuals with SCI. Thus, it is difficult to establish whether the cardiovascular system becomes limited during a field-based exercise test. In the current thesis, seven out of the eight participants in chapter 7 also took part in chapter 6, which allows some physiological correlates of 4-min push performance to be obtained. There was a significant correlation between $\dot{V}O_{2peak}$ in the laboratory and the maximal distance covered in the 4-min push ($r = 0.57$, $p = 0.034$), suggesting that the performance of the cardiovascular system is an important determinant of 4-min push performance. Thus, an improvement in 4-min push performance with abdominal binding suggests a central (cardiovascular) rather than peripheral limitation to exercise in the unbound condition. The difference in findings between the laboratory and field test with respect to a cardiovascular limitation to exercise is likely explained by an inability of the laboratory protocol to elicit a peak response, as well as the greater external validity of the 4-min push and the consequent differences in push technique (see section 7-4.4).

In conclusion, the data presented in this thesis suggest that the cardiovascular system becomes limited during maximal exercise in the highly-trained cervical SCI population. Accordingly, the mechanism by which abdominal binding improves 4-min push performance is likely through an improvement in cardiovascular function. Specifically, it is postulated that abdominal binding increases stroke volume, cardiac output and oxygen delivery subsequent to an enhanced venous return from the splanchnic bed.

The summary of potential cardiovascular limitations to exercise performance in individuals with cervical SCI provided in the literature review (Fig 2-8) is revisited in Fig 8-4. An emphasis is placed on the mechanisms by which abdominal binding may attenuate these limitations.

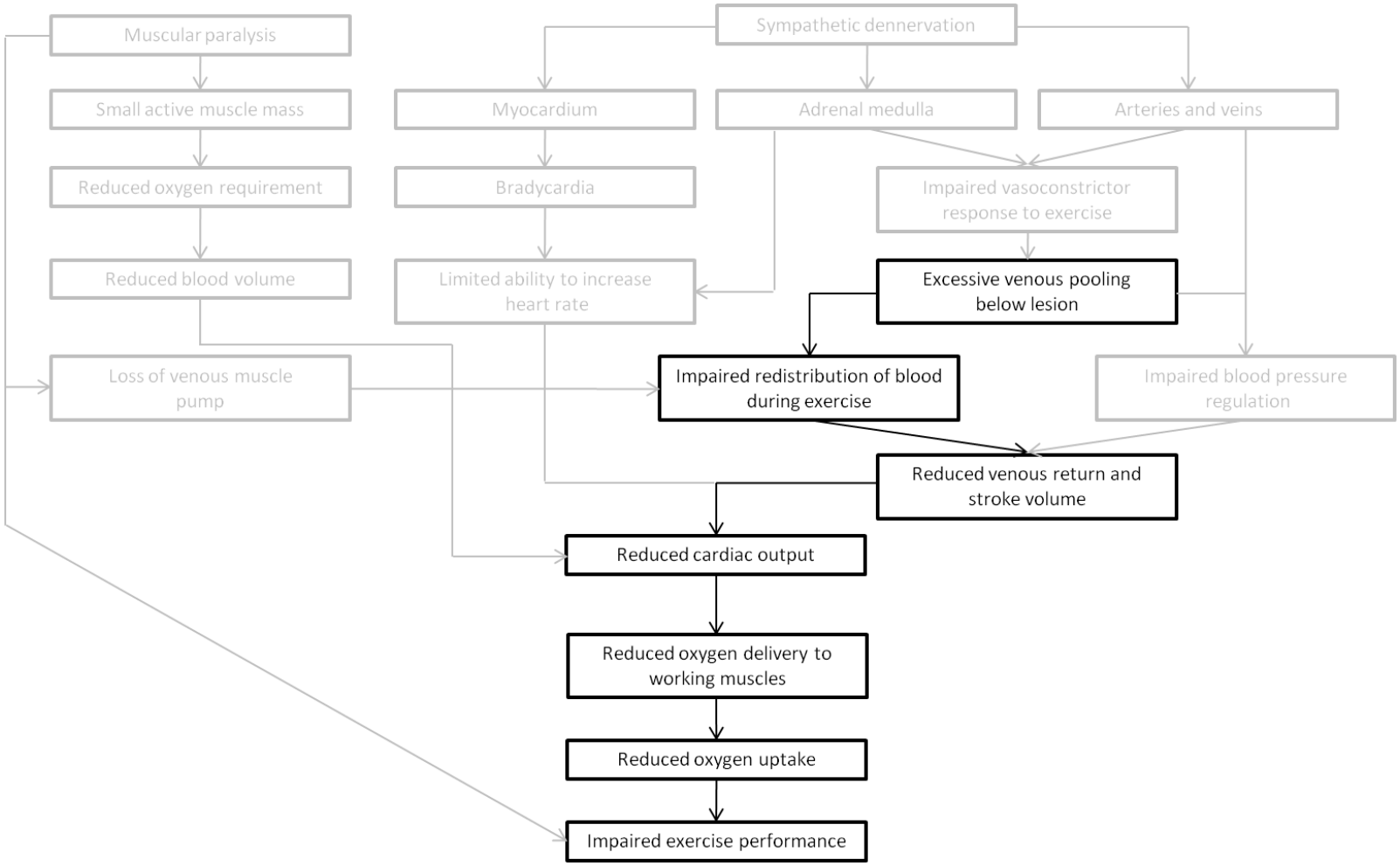

Fig 8-4 Cardiovascular consequences of cervical SCI and how they may contribute to impaired exercise performance.
An emphasis (black boxes) is now placed on the mechanisms by which abdominal binding may attenuate the respiratory limitations to exercise. See narrative for explanation

8-5 Practical implications for athletes

All of the athletes studied in this thesis were provided with a written report detailing their individual results. Based on the findings of chapters 5 through 7, the athletes who had complete cervical SCI were advised that the binder should be worn during training and competition to aid cardiorespiratory function and increase exercise intensity. The two athletes who had incomplete cervical SCI were advised not to wear the current binder due to the discomfort felt and the slight decrement in 4-min push performance. At the time of writing this thesis, UK sport had provided funding for each member of the Great Britain wheelchair rugby squad to have a customised abdominal binder made. To the author's knowledge, 75% (n = 9) of the squad are having an abdominal binder custom-made for them. The remaining 25% either have an incomplete cervical SCI or, despite the feedback provided, felt that they did not gain any benefit from wearing the binder.

The results of this thesis have been presented to UK Sport and to the coaches of court-based sports involving individuals with complete cervical SCI. There is a possibility that athletes who have a thoracic SCI between T1 and T6 may also benefit from the additional postural support afforded by the binder; however, the postural benefits of binding in the SCI population still need to be established. Based on the partial or full sympathetic innervations of the myocardium in high thoracic SCI (T1-T6), it is unlikely that binding will increase $\dot{V}O_{2peak}$. Outside of court-based sports, wheelchair racing is the only other high-intensity upper-body sport available to individuals with a cervical SCI. Wheelchair racing chairs are set-up very differently to court-based chairs, in that athletes have their legs bent underneath them and are heavily strapped around the abdomen due to the 'more horizontal' posture associated with wheelchair racing. Accordingly, it is very likely that those athletes are already 'abdominal binding' with their usual straps, and would therefore be unlikely to gain any further benefits from wearing the binder described in this thesis.

8-6 Clinical implications

8-6.1 Orthostatic hypotension

In section 2-3.3 the potential mechanisms underlying orthostatic hypotension were summarised. In healthy AB individuals, orthostatic hypotension is defended against by activation of the venous muscle pump and a baroreceptor-mediated increase in peripheral vasoconstriction and splanchnic venoconstriction. For cervical SCI, both of those mechanisms are impaired such that orthostatic hypotension is highly prevalent, especially during the acute period post injury (Claydon *et al.*, 2006). The large binding-induced increase in P_{ga} in chapter 5 suggests that compressing the abdomen and increasing intra-abdominal pressure may promote splanchnic venoconstriction and aid venous emptying. The subsequent increase in venous return and cardiac output may attenuate the reduction in blood pressure during orthostatic stress. Thus, abdominal binding may provide a useful tool to help manage orthostatic hypotension. However, future studies that specifically examine the effect of abdominal binding on blood pressure changes during passive tilting are needed to confirm this postulate.

8-6.2 Cough

For individuals with SCI above T6, paralysis of the primary expiratory muscles leads to increased abdominal compliance (Morgan *et al.*, 1985; Mortola and Sant'Ambrogio, 1978). During forced expiratory manoeuvres, the rise in intrathoracic pressure is dissipated through the flaccid diaphragm and causes a paradoxical expansion of the abdominal wall. Perhaps the single most debilitating respiratory consequence of this pressure dissipation is an impaired cough, which predisposes individuals with cervical SCI to mucous retention, atelectasis and pulmonary infections (Brown *et al.*, 2006; Schilero *et al.*, 2009). Although abdominal binding alone has been shown to cause no improvement in cough effectiveness (Estenne *et al.*, 1998; Lin *et al.*, 1998), the degree of abdominal compression may have been too low. In chapter 5, abdominal binding significantly increased expiratory flow, P_{oe} and P_{di} throughout a forced vital capacity manoeuvre. Thus, the mechanism for the increase in expiratory flow may relate to an improved transmission

of pleural pressure arising from an antagonist contraction of the diaphragm consequent to a decrease in abdominal compliance (Estenne and Gorini, 1992). In conclusion, the data collected in this thesis suggest that cough may be improved with binding; however, future research that specifically investigates dynamic airway compression with and without tight abdominal binding is required to confirm this postulate.

8-6.3 Exercise rehabilitation

Exercise is commonly used as a tool to improve both health and social outcomes following SCI. Although this thesis has investigated highly-trained athletes, the findings may have important implications for exercise in the untrained cervical SCI population. As described in section 2.3.1, there is an increased risk, and early onset of cardiovascular disease in the untrained population with cervical SCI due to the loss of active muscle mass, the loss of sympathetic nervous system function and the resultant sedentary lifestyle (DeVivo *et al.*, 1999; Yekutiel *et al.*, 1989). A major modifiable risk factor for cardiovascular disease is physical inactivity (Bassuk and Manson, 2005; Kohl, 2001; Press *et al.*, 2003). Upper-body exercise training is typically advocated to improve aerobic fitness during rehabilitation in individuals with SCI (personal communication from D. Tussler, National Spinal Injuries Centre, 22nd April 2011). However, the small active muscle mass and low $\dot{V}O_{2peak}$ may be insufficient to elicit health benefits (Hicks *et al.*, 2003). The improved circulatory benefits and $\dot{V}O_2$ associated with FES/hybrid exercise (Barstow *et al.*, 1996; Hettinga and Andrews, 2008; Hooker *et al.*, 1992; Pollack *et al.*, 1989; Thijssen *et al.*, 2005) may provide an alternative method by which exercise capacity can be increased. However, both FES and hybrid exercise require the use of a lower-body electrical stimulation unit and specialised exercise equipment. Conversely, abdominal binding is simple to implement, cost-effective, can be used during any form of upper body exercise and appears to afford individuals similar haemodynamic improvements during exercise compared to hybrid exercise (i.e., increases in stroke volume, cardiac output and $\dot{V}O_{2peak}$). However, before abdominal binding can be incorporated into rehabilitation programmes, studies that compare health outcomes and exercise responses

between upper-body exercise, hybrid exercise, and upper-body exercise with abdominal binding are needed.

8-7 Recommendations for future research

8-7.1 What are the mechanisms underpinning the improvement in cardiovascular function during exercise with abdominal binding?

The current thesis provides evidence that abdominal binding improves exercise performance by improving cardiovascular function. However, the exact mechanism(s) underpinning this improvement remain to be elucidated. Recent methodological advances have demonstrated that it is possible to measure portal vein flow during sub-maximal exercise (Thijssen *et al.*, 2009) and cardiac output during maximal exercise (Hostettler *et al.*, 2010) in the cervical SCI population. The former provides an index of splanchnic emptying during exercise, whilst the latter could be used to confirm that it is an increase in cardiac output, rather than an improvement in oxygen extraction, that results in the improvement in $\dot{V}O_{2peak}$ with abdominal binding. Thus, future research should consider making use of these methods to determine the mechanism(s) by which abdominal binding improves cardiovascular function during exercise.

8-7.2 What are the long-term benefits of abdominal binder use?

This thesis has documented the acute cardiorespiratory responses to abdominal binding. The increased $\dot{V}O_{2peak}$ and concomitant reduction in peak blood lactate accumulation, combined with the increased distance covered during a 4-min maximal push with abdominal binding, provides promising evidence that abdominal binding may enable highly-trained athletes with cervical SCI to exercise at higher intensities and for longer periods of time. In the healthy able-bodied population, a considerable body of evidence suggests that high-intensity exercise training is associated with improved gains in $\dot{V}O_{2max}$ and reductions in cardio-metabolic risk compared to

low-moderate intensity exercise training (e.g. Kemi *et al.*, 2005; Swain and Franklin, 2006). Accordingly, chronic use of abdominal binding may improve exercise performance due to the associated benefits of exercising at higher intensities for longer periods of time. Thus, longitudinal studies are needed to determine the degree to which long-term exercise training with abdominal binding impacts on exercise capacity and cardio-metabolic risk in cervical SCI.

8-7.3 Does abdominal binding improve posture in individuals with high spinal cord injury?

This thesis has examined the cardiorespiratory responses to abdominal binding. However, abdominal muscle dennervation and the consequent increase in abdominal compliance cause individuals with cervical and high thoracic SCI (above T6) to adopt a 'slump' posture. Previous research in able-bodied individuals and individuals with cervical SCI has demonstrated that the diaphragm is active during postural challenge (Gandevia *et al.*, 2002; Sinderby *et al.*, 1992). Highly-trained athletes with SCI who compete in wheelchair sport are subjected to numerous postural challenges during training and competition. Thus, it is reasonable to believe that there is an additional non-respiratory load placed upon the diaphragm during competitive sport. Future studies, therefore, should seek to quantify the postural load imposed on the diaphragm and determine whether abdominal binding attenuates the postural work of the diaphragm.

8-8 Conclusions

1) Compared to AB controls, highly-trained athletes with cervical SCI had a restrictive pulmonary defect, impaired diaphragm and expiratory muscle function, and low left ventricular mass and ejection fraction.

2) Abdominal binding caused a significant improvement in pulmonary function, inspiratory and expiratory muscle function, and cardiovascular function in individuals with cervical SCI. It was also found that the magnitude of increase in cardiorespiratory function exceeded that in AB individuals, and was dependent upon the degree of abdominal compression applied. That is, the greater the degree of abdominal compression, the greater the improvement in cardiorespiratory function.

3) Abdominal binding significantly increased the distance covered during a maximal 4-min push in the field, attenuated the rise in blood lactate accumulation during repeated 4-min pushes, and reduced the time taken to complete an acceleration/deceleration test in highly-trained athletes with cervical SCI.

4) Abdominal binding improved peak oxygen uptake and reduced the metabolic acidosis during maximal wheelchair propulsion on a treadmill in highly-trained athletes with cervical SCI. Abdominal binding also provided beneficial mechanical alterations to the respiratory system by reducing operating lung volumes and the degree of ventilatory constraint.

REFERENCES

Aaron, E.A., Seow, K.C., Johnson, B.D. and Dempsey, J.A. (1992). Oxygen cost of exercise hyperpnea: implications for performance. *J Appl Physiol,* 72(5):1818-1825.

Aaron, S.D., Dales, R.E. and Cardinal, P. (1999). How accurate is spirometry at predicting restrictive pulmonary impairment? *Chest,* 115(3):869-873.

Abel, T., Peters, C. and Platen, P. (2003). Performance profile and health assessment of elite quad rugby players. *Eur J Sport Sci,* 3(2):1-7.

Acton, P.A., Farley, T., Freni, L.W., Ilegbodu, V.A., Sniezek, J.E. and Wohlleb, J.C. (1993). Traumatic spinal cord injury in Arkansas, 1980 to 1989. *Arch Phys Med Rehabil,* 74(10):1035-1040.

Alexander, M.S., Biering-Sorensen, F., Bodner, D., Brackett, N.L., Cardenas, D., Charlifue, S., Creasey, G., Dietz, V., Ditunno, J., Donovan, W., Elliott, S.L., Estores, I., Graves, D.E., Green, B., Gousse, A., Jackson, A.B., Kennelly, M., Karlsson, A.K., Krassioukov, A., Krogh, K., Linsenmeyer, T., Marino, R., Mathias, C.J., Perkash, I., Sheel, A.W., Schilero, G., Schurch, B., Sonksen, J., Stiens, S., Wecht, J., Wuermser, L.A. and Wyndaele, J.J. (2009). International standards to document remaining autonomic function after spinal cord injury. *Spinal Cord,* 47(1):36-43.

Alison, J.A., Regnis, J.A., Donnelly, P.M., Adams, R.D., Sullivan, C.E. and Bye, P.T. (1998). End-expiratory lung volume during arm and leg exercise in normal subjects and patients with cystic fibrosis. *Am J Respir Crit Care Med,* 158(5 Pt 1):1450-1458.

Aliverti, A., Bovio, D., Fullin, I., Dellaca, R.L., Lo Mauro, A., Pedotti, A. and Macklem, P.T. (2009). The abdominal circulatory pump. *PLoS One,* 4(5):e5550.

Aliverti, A., Cala, S.J., Duranti, R., Ferrigno, G., Kenyon, C.M., Pedotti, A., Scano, G., Sliwinski, P., Macklem, P.T. and Yan, S. (1997). Human respiratory muscle actions and control during exercise. *J Appl Physiol,* 83(4):1256-1269.

Amann, M., Eldridge, M.W., Lovering, A.T., Stickland, M.K., Pegelow, D.F. and Dempsey, J.A. (2006). Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *J Physiol,* 575(Pt 3):937-952.

Amodie-Storey, C., Nash, M.S., Roussell, P.M., Knox, A.W. and Crane, L.D. (1996). Head position and its effect on pulmonary function in tetraplegic patients. *Spinal Cord,* 34(10):602-607.

Anke, A., Aksnes, A.K., Stanghelle, J.K. and Hjeltnes, N. (1993). Lung volumes in tetraplegic patients according to cervical spinal cord injury level. *Scand J Rehabil Med,* 25(2):73-77.

ASIA. (2003). *Reference Manual of the International Standards for Neurological Classification of Spinal Cord Injury*. Chicago, IL. American Spinal Injuries Association.

Astrand, P.O., Cuddy, T.E., Saltin, B. and Stenberg, J. (1964). Cardiac Output during Submaximal and Maximal Work. *J Appl Physiol,* 19:268-274.

ATS/ACCP. (2003). ATS/ACCP statement on cardiopulmonary exercise testing. *Am J Respir Crit Care Med,* 167(2):211-277.

Barstow, T.J., Scremin, A.M., Mutton, D.L., Kunkel, C.F., Cagle, T.G. and Whipp, B.J. (1996). Changes in gas exchange kinetics with training in patients with spinal cord injury. *Med Sci Sports Exerc,* 28(10):1221-1228.

Bassett, D.R., Jr and Howley, E.T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Med Sci Sports Exerc,* 32(1):70-84.

Bassuk, S.S. and Manson, J.E. (2005). Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. *J Appl Physiol,* 99(3):1193-1204.

Baydur, A., Adkins, R.H. and Milic-Emili, J. (2001). Lung mechanics in individuals with spinal cord injury: effects of injury level and posture. *J Appl Physiol,* 90(2):405-411.

Baydur, A., Behrakis, P.K., Zin, W.A., Jaeger, M. and Milic-Emili, J. (1982). A simple method for assessing the validity of the esophageal balloon technique. *Am Rev Respir Dis,* 126(5):788-791.

Beck, K.C., Offord, K.P. and Scanlon, P.D. (1994). Bronchoconstriction occurring during exercise in asthmatic subjects. *Am J Respir Crit Care Med,* 149(2 Pt 1):352-357.

Bellemare, F. and Bigland-Ritchie, B. (1984). Assessment of human diaphragm strength and activation using phrenic nerve stimulation. *Respir Physiol,* 58(3):263-277.

Bellemare, F. and Grassino, A. (1982). Effect of pressure and timing of contraction on human diaphragm fatigue. *J Appl Physiol,* 53(5):1190-1195.

Benditt, J.O. (2005). Esophageal and gastric pressure measurements. *Respir Care,* 50(1):68-75.

Bergofsky, E.H. (1964). Mechanism for Respiratory Insufficiency After Cervical Cord Injury; a Source of Alveolar Hypoventilation. *Ann Intern Med,* 61:435-447.

Bhambhani, Y., Mactavish, J., Warren, S., Thompson, W.R., Webborn, A., Bressan, E., De Mello, M.T., Tweedy, S., Malone, L., Frojd, K., Van De Vliet, P. and Vanlandewijck, Y. (2010). Boosting in athletes with high-level spinal cord injury: knowledge, incidence and attitudes of athletes in paralympic sport. *Disability and Rehabilitation,* 32(26):2172-2190.

Bhambhani, Y.N., Burnham, R.S., Wheeler, G.D., Eriksson, P., Holland, L.J. and Steadward, R.D. (1995). Ventilatory threshold during wheelchair exercise in untrained and endurance-trained subjects with. *Adap Phys Act Quart,* 12(4):333-343.

Bjerkefors, A., Carpenter, M.G., Cresswell, A.G. and Thorstensson, A. (2009). Trunk muscle activation in a person with clinically complete thoracic spinal cord injury. *J Rehabil Med,* 41(5):390-392.

Boaventura, C.M., Gastaldi, A.C., Silveira, J.M., Santos, P.R., Guimaaes, R.C. and Lima, L.C.D. (2003). Effect of an abdominal binder on the efficacy of respiratory muscles in seated and supine tetraplegic patients. *Physiotherapy,* 89(5):290-295.

Bodin, P., Fagevik Olsen, M., Bake, B. and Kreuter, M. (2005). Effects of abdominal binding on breathing patterns during breathing exercises in persons with tetraplegia. *Spinal Cord,* 43(2):117-122.

Bogdanis, G.C., Nevill, M.E., Boobis, L.H., Lakomy, H.K. and Nevill, A.M. (1995). Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *J Physiol,* 482 (Pt 2):467-480.

Borg, G. (1998). *Borg's Perceived Exertion and Pain Scales*. Champaign, IL. Human Kinetics.

Bradley, C.A. and Anthonisen, N.R. (1980). Rib cage and abdominal restrictions have different effects on lung mechanics. *J Appl Physiol,* 49(6):946-952.

Brouwer, E. (1957). On simple formulae for calculating the heat expenditure and the quantities of carbohydrate and fat oxidized in metabolism of men and animals, from gaseous exchange (Oxygen intake and carbonic acid output) and urine-N. *Acta Physiol Pharmacol Neerl,* 6:795-802.

Brown, C.M. and Hainsworth, R. (2000). Forearm vascular responses during orthostatic stress in control subjects and patients with posturally related syncope. *Clin Auton Res,* 10(2):57-61.

Brown, R., DiMarco, A.F., Hoit, J.D. and Garshick, E. (2006). Respiratory dysfunction and management in spinal cord injury. *Respir Care,* 51(8):853-868.

Bruschi, C., Cerveri, I., Zoia, M.C., Fanfulla, F., Fiorentini, M., Casali, L., Grassi, M. and Grassi, C. (1992). Reference values of maximal respiratory mouth pressures: a population-based study. *Am Rev Respir Dis,* 146(3):790-793.

Buess, C., Pietsch, P., Guggenbuhl, W. and Koller, E.A. (1986). A pulsed diagonal-beam ultrasonic airflow meter. *J Appl Physiol,* 61(3):1195-1199.

Burnham, R., Wheeler, G. and Bhambhani, Y. (1994). Intentional induction of autonomic dysreflexia among quadriplegic athletes for performance enhancement: efficacy, safety, and mechanism of action. *Clin J Sport Med,*(4):1-10.

Burnley, M., Doust, J.H. and Vanhatalo, A. (2006). A 3-min all-out test to determine peak oxygen uptake and the maximal steady state. *Med Sci Sports Exerc,* 38(11):1995-2003.

Bush, V.E., Wight, V.L., Brown, C.M. and Hainsworth, R. (2000). Vascular responses to orthostatic stress in patients with postural tachycardia syndrome (POTS), in patients with low orthostatic tolerance, and in asymptomatic controls. *Clin Auton Res,* 10(5):279-284.

Campbell, E.J.M. (1958). *The Respiratory Muscles and the Mechanics of Breathing*. London. Hazell Watson and Viney.

Casiglia, E., Pizziol, A., Piacentini, F., Biasin, R., Onesto, C., Tikhonoff, V., Prati, R., Palatini, P. and Pessina, A.C. (1999). 24-hour leg and forearm haemodynamics in transected spinal cord subjects. *Cardiovascular Research,* 41(1):312-316.

Cerny, F.J. and Ucer, C. (2004). Arm work interferes with normal ventilation. *Applied Ergonomics,* 35(5):411-415.

Clanton, T., Calverly, P.M. and Celli, B.R. (2002). Tests of respiratory muscle endurance: ATS/ERS statement on respiratory muscle testing. *Am J Respir Crit Care Med,* 166(4):518-624.

Claydon, V.E., Steeves, J.D. and Krassioukov, A. (2006). Orthostatic hypotension following spinal cord injury: understanding clinical pathophysiology. *Spinal Cord,* 44(6):341-351.

Coutts, K.D., Rhodes, E.C. and McKenzie, D.C. (1983). Maximal exercise responses of tetraplegics and paraplegics. *J Appl Physiol,* 55(2):479-482.

Cunningham, D.J., Guttmann, L., Whitteridge, D. and Wyndham, C.H. (1953). Cardiovascular responses to bladder distension in paraplegic patients. *J Physiol,* 121(3):581-592.

Dahlberg, A., Kotila, M., Leppanen, P., Kautiainen, H. and Alaranta, H. (2005). Prevalence of spinal cord injury in Helsinki. *Spinal Cord,* 43(1):47-50.

Danon, J., Druz, W.S., Goldberg, N.B. and Sharp, J.T. (1979). Function of the isolated paced diaphragm and the cervical accessory muscles in C1 quadriplegics. *Am Rev Respir Dis,* 119(6):909-919.

Davis, G., M. (1993). Exercise capacity of individuals with paraplegia. *Med Sci Sports Exerc,* 25(4):423-432.

Davis, G.M., Servedio, F.J., Glaser, R.M., Gupta, S.C. and Suryaprasad, A.G. (1990). Cardiovascular responses to arm cranking and FNS-induced leg exercise in paraplegics. *J Appl Physiol,* 69(2):671-677.

de Groot, P.C., van Dijk, A., Dijk, E. and Hopman, M.T. (2006). Preserved cardiac function after chronic spinal cord injury. *Arch Phys Med Rehabil,* 87(9):1195-1200.

De Troyer, A. (1997). Effect of hyperinflation on the diaphragm. *Eur Respir J,* 10(3):708-713.

De Troyer, A. (2005). Actions of the respiratory muscles. In: *Physiological Basis of Respiratory Disease* (eds Hamid, Q., Shannon, J. and Martin, J.). pp. 263-275. Hamilton, Ontario, Canada. BC Decker Inc.

De Troyer, A. and Estenne, M. (1984). Coordination between rib cage muscles and diaphragm during quiet breathing in humans. *J Appl Physiol,* 57(3):899-906.

De Troyer, A., Estenne, M. and Heilporn, A. (1986a). Mechanism of Active Expiration in Tetraplegic Subjects. *New England Journal of Medicine,* 314(12):740-744.

De Troyer, A., Estenne, M. and Vincken, W. (1986b). Rib cage motion and muscle use in high tetraplegics. *Am Rev Respir Dis,* 133(6):1115-1119.

De Troyer, A., Ninane, V., Gilmartin, J.J., Lemerre, C. and Estenne, M. (1987). Triangularis sterni muscle use in supine humans. *J Appl Physiol,* 62(3):919-925.

De Troyer, A., Sampson, M., Sigrist, S. and Macklem, P.T. (1982). Action of costal and crural parts of the diaphragm on the rib cage in dog. *J Appl Physiol,* 53(1):30-39.

Decramer, M. (1997). Hyperinflation and respiratory muscle interaction. *Eur Respir J,* 10(4):934-941.

Dela, F., Mohr, T., Jensen, C.M., Haahr, H.L., Secher, N.H., Biering-Sorensen, F. and Kjaer, M. (2003). Cardiovascular control during exercise: insights from spinal cord-injured humans. *Circulation,* 107(16):2127-2133.

Dempsey, J.A., Adams, L., Ainsworth, D.M., Fregosi, R.F., Gallagher, C.G. and Guz, A. (1996). Airway, lung, and respiratory muscle function during exercise. In: *Handbook of Physiology. Regulation and Integration of Multiple Systems*. pp. 448-514. Bethesda, MD. Am. Physiol. Soc.

Dempsey, J.A., Amann, M., Romer, L.M. and Miller, J.D. (2008a). Respiratory system determinants of peripheral fatigue and endurance performance. *Med Sci Sports Exerc,* 40(3):457-461.

Dempsey, J.A., McKenzie, D.C., Haverkamp, H.C. and Eldridge, M.W. (2008b). Update in the understanding of respiratory limitations to exercise performance in fit, active adults. *Chest,* 134(3):613-622.

Dempsey, J.A. and Wagner, P.D. (1999). Exercise-induced arterial hypoxemia. *J Appl Physiol,* 87(6):1997-2006.

Derchak, P.A., Sheel, A.W., Morgan, B.J. and Dempsey, J.A. (2002). Effects of expiratory muscle work on muscle sympathetic nerve activity. *J Appl Physiol,* 92(4):1539-1552.

Devereux, R.B., Alonso, D.R., Lutas, E.M., Gottlieb, G.J., Campo, E., Sachs, I. and Reichek, N. (1986). Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Am J Cardiol,* 57(6):450-458.

DeVivo, M.J., Fine, P.R., Maetz, H.M. and Stover, S.L. (1980). Prevalence of spinal cord injury: a reestimation employing life table techniques. *Arch Neurol,* 37(11):707-708.

DeVivo, M.J., Go, B.K. and Jackson, A.B. (2002). Overview of the national spinal cord injury statistical center database. *J Spinal Cord Med,* 25(4):335-338.

DeVivo, M.J., Krause, J.S. and Lammertse, D.P. (1999). Recent trends in mortality and causes of death among persons with spinal cord injury. *Arch Phys Med Rehabil,* 80(11):1411-1419.

DeVivo, M.J. and Stover, S.L. (1995). Long-term survival and causes of death. In: Spinal Cord Injury: Clinical Outcome from the Model System. Stover SL, DeLisa JA, Whiteneck GG (eds). pp. 289-316. Gaithersburg, MD. Aspen Publishers.

Diaz, O., Villafranca, C., Ghezzo, H., Borzone, G., Leiva, A., Milic-Emil, J. and Lisboa, C. (2000). Role of inspiratory capacity on exercise tolerance in COPD patients with and without tidal expiratory flow limitation at rest. *Eur Respir J,* 16(2):269-275.

DiMarco, A.F., Kelsen, S.G., Cherniack, N.S., Hough, W.H. and Gothe, B. (1981). Effects on breathing of selective restriction of movement of the rib cage and abdomen. *J Appl Physiol,* 50(2):412-420.

Ditunno, J.F., Little, J.W., Tessler, A. and Burns, A.S. (2004). Spinal shock revisited: a four-phase model. *Spinal Cord,* 42(7):383-395.

Dorfman, T.A., Rosen, B.D., Perhonen, M.A., Tillery, T., McColl, R., Peshock, R.M. and Levine, B.D. (2008). Diastolic suction is impaired by bed rest: MRI tagging studies of diastolic untwisting. *J Appl Physiol,* 104(4):1037-1044.

Douglas, C. (1911). A method for determining the total respiratory exchange in man. *J Physiol,* 42:XVII-XVIII.

Dryden, D.M., Saunders, L.D., Rowe, B.H., May, L.A., Yiannakoulias, N., Svenson, L.W., Schopflocher, D.P. and Voaklander, D.C. (2003). The epidemiology of traumatic spinal cord injury in Alberta, Canada. *Can J Neurol Sci,* 30(2):113-121.

Duffield, D. and Hart, A. (2008). *International Wheelchair Rugby Federation Classification Manual (3rd ed)*. pp. 1-25. IWRF.

Ellis, L., Gastin, P., Lawrence, S., Savage, B., Buckeridge, A., Stapff, A., Tumility, D., Quinn, A., Woolford, S. and Young, W. (2000). *Protocols for the physiological assessment of team sport players. In: Physiological Tests for Elite Athletes*. pp. 128-145. Champaign, IL. Human Kinetics.

Eriksson, P., Lofstrom, L. and Ekblom, B. (1988). Aerobic power during maximal exercise in untrained and well-trained persons with quadriplegia and paraplegia. *Scand J Rehabil Med,* 20(4):141-147.

Estenne, M. and De Troyer, A. (1986). The effects of tetraplegia on chest wall statics. *Am Rev Respir Dis,* 134(1):121-124.

Estenne, M. and De Troyer, A. (1987). Mechanism of the postural dependence of vital capacity in tetraplegic subjects. *Am Rev Respir Dis,* 135(2):367-371.

Estenne, M. and De Troyer, A. (1990). Cough in tetraplegic subjects: an active process. *Ann Intern Med,* 112(1):22-28.

Estenne, M., Gevenois, P.A., Kinnear, W., Soudon, P., Heilporn, A. and De Troyer, A. (1993). Lung volume restriction in patients with chronic respiratory muscle weakness: the role of microatelectasis. *Thorax,* 48(7):698-701.

Estenne, M. and Gorini, M. (1992). Action of the diaphragm during cough in tetraplegic subjects. *J Appl Physiol,* 72(3):1074-1080.

Estenne, M., Knoop, C., Vanvaerenbergh, J., Heilporn, A. and De Troyer, A. (1989). The effect of pectoralis muscle training in tetraplegic subjects. *Am Rev Respir Dis,* 139(5):1218-1222.

Estenne, M., Pinet, C. and De Troyer, A. (2000). Abdominal muscle strength in patients with tetraplegia. *Am J Respir Crit Care Med,* 161(3 Pt 1):707-712.

Estenne, M., Van Muylem, A., Gorini, M., Kinnear, W., Heilporn, A. and De Troyer, A. (1994). Evidence of dynamic airway compression during cough in tetraplegic patients. *Am J Respir Crit Care Med,* 150(4):1081-1085.

Estenne, M., Van Muylem, A., Gorini, M., Kinnear, W., Heilporn, A. and De Troyer, A. (1998). Effects of abdominal strapping on forced expiration in tetraplegic patients. *Am J Respir Crit Care Med,* 157(1):95-98.

Eysmann, S.B., Douglas, P.S., Katz, S.E., Sarkarati, M. and Wei, J.Y. (1995). Left ventricular mass and diastolic filling patterns in quadriplegia and implications for effects of normal aging on the heart. *Am J Cardiol,* 75(2):201-203.

Faghri, P.D. and Yount, J. (2002). Electrically induced and voluntary activation of physiologic muscle pump: a comparison between spinal cord-injured and able-bodied individuals. *Clin Rehabil,* 16(8):878-885.

Fagraeus, L. and Linnarsson, D. (1976). Autonomic origin of heart rate fluctuations at the onset of muscular exercise. *J Appl Physiol,* 40(5):679-682.

Ferguson, G.T. (2006). Why Does the Lung Hyperinflate? *Proc Am Thorac Soc,* 3(2):176-179.

Figoni, S.F. (1993). Exercise responses and quadriplegia. *Med Sci Sports Exerc,* 25(4):433-441.

Figoni, S.F., Rodgers, M.M., Glaser, R.M., Hooker, S.P., Feghri, P.D., Ezenwa, B.N., Mathews, T., Suryaprasad, A.G. and Gupta, S.C. (1990). Physiologic responses of paraplegics and quadriplegics to passive and active leg cycle ergometry. *J Am Paraplegia Soc,* 13(3):33-39.

Forner, J.V. (1980). Lung volumes and mechanics of breathing in tetraplegics. *Paraplegia,* 18(4):258-266.

Franklin, B.A., Swantek, K.I., Grais, S.L., Johnstone, K.S., Gordon, S. and Timmis, G.C. (1990). Field test estimation of maximal oxygen consumption in wheelchair users. *Arch Phys Med Rehabil,* 71(8):574-578.

Fredberg, J.J., Inouye, D., Miller, B., Nathan, M., Jafari, S., Raboudi, S.H., Butler, J.P. and Shore, S.A. (1997). Airway smooth muscle, tidal stretches, and dynamically determined contractile states. *Am J Respir Crit Care Med,* 156(6):1752-1759.

Frisbie, J.H. (2004). Postural hypotension, hyponatremia, and salt and water intake: case reports. *J Spinal Cord Med,* 27(2):133-137.

Frisbie, J.H. and Brown, R. (1994). Waist and neck enlargement after quadriplegia. *J Am Paraplegia Soc,* 17(4):177-178.

Fugl-Meyer, A.R. and Grimby, G. (1971a). Rib-cage and abdominal volume ventilation partitioning in tetraplegic patients. *Scand.J.Rehabil.Med.,* 3(4):161-167.

Fugl-Meyer, A.R. and Grimby, G. (1971b). Ventilatory function in tetraplegic patients. *Scand J Rehabil Med,* 3(4):151-160.

Fujiwara, T., Hara, Y. and Chino, N. (1999). Expiratory function in complete tetraplegics - Study of spirometry, maximal expiratory pressure, and muscle activity of pectoralis major and latissimus dorsi muscles. *Am J Phys Med Rehabil,* 78(5):464-469.

Gaitanos, G.C., Williams, C., Boobis, L.H. and Brooks, S. (1993). Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol,* 75(2):712-719.

Gandevia, S.C., Butler, J.E., Hodges, P.W. and Taylor, J.L. (2002). Balancing acts: respiratory sensations, motor control and human posture. *Clin Exp Pharmacol Physiol,* 29(1-2):118-121.

Garshick, E., Kelley, A., Cohen, S.A., Garrison, A., Tun, C.G., Gagnon, D. and Brown, R. (2005). A prospective assessment of mortality in chronic spinal cord injury. *Spinal Cord,* 43(7):408-416.

Gass, E.M., Gass, G.C. and Gwinn, T.H. (1992). Sweat rate and rectal temperatures in teteraplegic men during exercise. *Sports Med Training Rehabil,* 3:243-249.

Gass, G.C. and Camp, E.M. (1979). Physiological characteristics of trained Australian paraplegic and tetraplegic subjects. *Med Sci Sports Exerc,* 11(3):256-259.

Gass, G.C., Camp, E.M., Nadel, E.R., Gwinn, T.H. and Engel, P. (1988). Rectal vs. esophageal temperatures in paraplegic men during prolonged exercise. *J Appl Physiol,* 64(6):2265-2271.

Gass, G.C., Watson, J., Camp, E.M., Court, H.J., McPherson, L.M. and Redhead, P. (1980). The effects of physical training on high level spinal lesion patients. *Scand J Rehabil Med,* 12(2):61-65.

Gehrig, R. and Michaelis, L.S. (1968). Statistics of acute paraplegia and tetraplegia on a national scale. Switzerland 1960-67. *Paraplegia,* 6(2):93-95.

Gillis, D.J., Wouda, M. and Hjeltnes, N. (2008). Non-pharmacological management of orthostatic hypotension after spinal cord injury: a critical review of the literature. *Spinal Cord,* 46(10):652-659.

Glaister, M., Howatson, G., Pattison, J.R. and McInnes, G. (2008). The reliability and validity of fatigue measures during multiple-sprint work: an issue revisited. *J Strength Cond Res,* 22(5):1597-1601.

Glaser, R., Rattan, M.S.N., Davies, G.M., Servedio, F.J., Gupta, S.C. and Suryaprasad, A.G. 1987. Central hemodynamic responses to lower-limb FNS. In *Proceedings of the 9th Annual Conference on Engineering and Medical Biological Society*.615-617.

Glaser, R.M. (1985). Exercise and locomotion for the spinal cord injured. *Exerc Sport Sci Rev,* 13:263-303.

Goldman, J.M., Rose, L.S., Morgan, M.D. and Denison, D.M. (1986a). Measurement of abdominal wall compliance in normal subjects and tetraplegic patients. *Thorax,* 41(7):513-518.

Goldman, J.M., Rose, L.S., Williams, S.J., Silver, J.R. and Denison, D.M. (1986b). Effect of abdominal binders on breathing in tetraplegic patients. *Thorax,* 41(12):940-945.

Goldman, J.M., Williams, S.J. and Denison, D.M. (1988). The rib cage and abdominal components of respiratory system compliance in tetraplegic patients. *Eur Respir J,* 1(3):242-247.

Goosey-Tolfrey, V. (2005). Physiological Profiles of Elite Wheelchair Basketball Players in Preparation for the 2000 Paralympic Games. *Adap Phys Act Quart,* 22:57-66.

Goosey-Tolfrey, V., Castle, P. and Webborn, N. (2006). Aerobic capacity and peak power output of elite quadriplegic games players. *Br J Sports Med,* 40:684-687.

Goosey-Tolfrey, V., Swainson, M., Boyd, C., Atkinson, G. and Tolfrey, K. (2008). The effectiveness of hand cooling at reducing exercise-induced hyperthermia and improving distance-race performance in wheelchair and able-bodied athletes. *Journal of Applied Physiology,* 105(1):37-43.

Goshgarian, H.G. (2010). *Anatomy and function of the spinal cord. In: Spinal Cord Medicine Principles and Practice*. 2nd edition. pp. 15. New York. Demos Medical Publishing.

Gounden, P. (1997). Static respiratory pressures in patients with post-traumatic tetraplegia. *Spinal Cord,* 35(1):43-47.

Gray, H. (2008). *Gray's Anatomy*. London. Arcturus.

Gray, S., Shaver, J.A., Kroetz, F.W. and Leonard, J.J. (1969). Acute and prolonged effects of G suit inflation on cardiovascular dynamics. *Aerosp Med,* 40(1):40-43.

Green, M., Road, J., Sieck, G.C. and Similowski, T. (2002). Tests of Respiratory Muscle Strength. In. ATS/ERS Statement on respiratory muscle testing. *Am J Respir Crit Care Med,* 166(4):518-624.

Grigorean, V.T., Sandu, A.M., Popescu, M., Iacobini, M.A., Stoian, R., Neascu, C., Strambu, V. and Popa, F. (2009). Cardiac dysfunctions following spinal cord injury. *J Med Life,* 2(2):133-145.

Grimby, G., Bunn, J. and Mead, J. (1968). Relative contribution of rib cage and abdomen to ventilation during exercise. *J Appl Physiol,* 24(2):159-166.

Grimby, G. and Sóderholm, B. (1963). Spirometric Studies in Normal Subjects. *Acta Medica Scandinavica,* 173(2):199-206.

Gross, D., Ladd, H.W., Riley, E.J., Macklem, P.T. and Grassino, A. (1980). Effect of Training on Strength and Endurance of the Diaphragm in Quadriplegia. *Am J Med,* 68(1):27-35.

Grossman, W. (1990). Diastolic dysfunction and congestive heart failure. *Circulation,* 81(2 Suppl):1-7.

Gruner, J.A., Glaser, R.M., Feinberg, S.D., Collins, S.R. and Nussbaum, N.S. (1983). A system for evaluation and exercise-conditioning of paralyzed leg muscles. *J Rehabil Res Dev,* 20(1):21-30.

Guenette, J.A. and Sheel, A.W. (2007). Physiological consequences of a high work of breathing during heavy exercise in humans. *J Sci Med Sport,* 10(6):341-350.

Guenette, J.A., Witt, J.D., McKenzie, D.C., Road, J.D. and Sheel, A.W. (2007). Respiratory mechanics during exercise in endurance-trained men and women. *J Physiol,* 581(Pt 3):1309-1322.

Guttman, L. (1973). *Spinal Cord Injuries: Comprehensive Management and Research*. pp. 51. Oxford. Blackwell Scientific.

Guttmann, L., Silver, J. and Wyndham, C.H. (1958). Thermoregulation in spinal man. *J Physiol,* 142(3):406-419.

Hadley, M. (2002). Blood pressure management after acute spinal cord injury. *Neurosurgery,* 50(3 Suppl):S58-62.

Hagobian, T.A., Jacobs, K.A., Kiratli, B.J. and Friedlander, A.L. (2004). Foot cooling reduces exercise-induced hyperthermia in men with spinal cord injury. *Med Sci Sports Exerc,* 36(3):411-417.

Haisma, J.A., van der Woude, L.H., Stam, H.J., Bergen, M.P., Sluis, T.A. and Bussmann, J.B. (2006). Physical capacity in wheelchair-dependent persons with a spinal cord injury: a critical review of the literature. *Spinal Cord,* 44(11):642-652.

Harms, C.A., Babcock, M.A., McClaran, S.R., Pegelow, D.F., Nickele, G.A., Nelson, W.B. and Dempsey, J.A. (1997). Respiratory muscle work compromises leg blood flow during maximal exercise. *J Appl Physiol,* 82(5):1573-1583.

Harms, C.A., Wetter, T.J., St Croix, C.M., Pegelow, D.F. and Dempsey, J.A. (2000). Effects of respiratory muscle work on exercise performance. *J Appl Physiol,* 89(1):131-138.

Harridge, S.D., Andersen, J.L., Hartkopp, A., Zhou, S., Biering-Sorensen, F., Sandri, C. and Kjaer, M. (2002). Training by low-frequency stimulation of tibialis anterior in spinal cord-injured men. *Muscle & Nerve,* 25(5):685-694.

Hart, C. and Williams, E. (1994). Epidemiology of spinal cord injuries: a reflection of changes in South African society. *Paraplegia,* 32(11):709-714.

Hart, N., Laffont, I., de la Sota, A.P., Lejaille, M., Macadou, G., Polkey, M.I., Denys, P. and Lofaso, F. (2005). Respiratory effects of combined truncal and abdominal support in patients with spinal cord injury. *Arch Phys Med Rehabil,* 86(7):1447-1451.

Hartkopp, A., Bronnum-Hansen, H., Seidenschnur, A.M. and Biering-Sorensen, F. (1997). Survival and cause of death after traumatic spinal cord injury. A long-term epidemiological survey from Denmark. *Spinal Cord,* 35(2):76-85.

Hartung, G.H., Lally, D.A. and Blancq, R.J. (1993). Comparison of treadmill exercise testing protocols for wheelchair users. *Eur J Appl Physiol Occup Physiol,* 66(4):362-365.

Haverkamp, H.C., Dempsey, J.A., Miller, J.D., Romer, L.M., Pegelow, D.F., Rodman, J.R. and Eldridge, M.W. (2005). Gas exchange during exercise in habitually active asthmatic subjects. *J Appl Physiol,* 99(5):1938-1950.

Haverkamp, H.C., Dempsey, J.A., Pegelow, D.F., Miller, J.D., Romer, L.M., Santana, M. and Eldridge, M.W. (2007). Treatment of airway inflammation improves exercise pulmonary gas exchange and performance in asthmatic subjects. *J Allergy Clin Immunol,* 120(1):39-47.

Hettinga, D.M. and Andrews, B.J. (2008). Oxygen consumption during functional electrical stimulation-assisted exercise in persons with spinal cord injury: implications for fitness and health. *Sports Med,* 38(10):825-838.

Hicks, A.L., Martin, K.A., Ditor, D.S., Latimer, A.E., Craven, C., Bugaresti, J. and McCartney, N. (2003). Long-term exercise training in persons with spinal cord injury: effects on strength, arm ergometry performance and psychological well-being. *Spinal Cord,* 41(1):34-43.

Higginbotham, M.B., Morris, K.G., Williams, R.S., McHale, P.A., Coleman, R.E. and Cobb, F.R. (1986). Regulation of stroke volume during submaximal and maximal upright exercise in normal man. *Circ Res,* 58(2):281-291.

Himelman, R.B., Cassidy, M.M., Landzberg, J.S. and Schiller, N.B. (1988). Reproducibility of quantitative two-dimensional echocardiography. *Am Heart J,* 115(2):425-431.

Hjeltnes, N., Aksnes, A.K., Birkeland, K.I., Johansen, J., Lannem, A. and Wallberg-Henriksson, H. (1997). Improved body composition after 8 wk of electrically stimulated leg cycling in tetraplegic patients. *Am J Physiol,* 273(3):R1072-R1079.

Hooker, S.P., Figoni, S.F., Rodgers, M.M., Glaser, R.M., Mathews, T., Suryaprasad, A.G. and Gupta, S.C. (1992). Physiologic effects of electrical stimulation leg cycle exercise training in spinal cord injured persons. *Arch Phys Med Rehabil,* 73(5):470-476.

Hopman, M.T., Dueck, C., Monroe, M., Philips, W.T. and Skinner, J.S. (1998a). Limits to maximal performance in individuals with spinal cord injury. *Int J Sports Med,* 19(2):98-103.

Hopman, M.T., Houtman, S., Groothuis, J.T. and Folgering, H.T. (2004). The effect of varied fractional inspired oxygen on arm exercise performance in spinal cord injury and able-bodied persons. *Arch Phys Med Rehabil,* 85(2):319-323.

Hopman, M.T., Monroe, M., Dueck, C., Phillips, W.T. and Skinner, J.S. (1998b). Blood redistribution and circulatory responses to submaximal arm exercise in persons with spinal cord injury. *Scand J Rehabil Med,* 30(3):167-174.

Hopman, M.T., Oeseburg, B. and Binkhorst, R.A. (1992). The effect of an anti-G suit on cardiovascular responses to exercise in persons with paraplegia. *Med Sci Sports Exerc,* 24(9):984-990.

Hopman, M.T., van der Woude, L.H., Dallmeijer, A.J., Snoek, G. and Folgering, H. (1997). Respiratory muscle strength and endurance in individuals with tetraplegia. *Spinal Cord,* 35(2):104-108.

Hostettler, S., Leuthold, L., Brechbuhl, J., Mueller, G., Illi, S.K. and Spengler, C.M. 2010. Cardiorespiratory responses to maximal wheelchair and arm crank exercise in persons with tetraplegia. In *European Respiratory Society* Barcelona.869.

Houtman, S., Oeseburg, B. and Hopman, M.T. (2000). Blood volume and hemoglobin after spinal cord injury. *Am J Phys Med Rehabil,* 79(3):260-265.

Huang, C.T., Kuhlemeier, K.V., Ratanaubol, U., McEachran, A.B., DeVivo, M.J. and Fine, P.R. (1983). Cardiopulmonary response in spinal cord injury patients: effect of pneumatic compressive devices. *Arch Phys Med Rehabil,* 64(3):101-106.

Huldtgren, A.C., Fugl-Meyer, A.R., Jonasson, E. and Bake, B. (1980). Ventilatory dysfunction and respiratory rehabilitation in post-traumatic quadriplegia. *Eur J Respir Dis,* 61(6):347-356.

Hussain, S.N., Rabinovitch, B., Macklem, P.T. and Pardy, R.L. (1985). Effects of separate rib cage and abdominal restriction on exercise performance in normal humans. *J Appl Physiol,* 58(6):2020-2026.

IPC. (2000). International Paralympic Committe handbook. Part I. 2nd ed.

Jain, N.B., Brown, R., Tun, C.G., Gagnon, D. and Garshick, E. (2006). Determinants of forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC), and FEV1/FVC in chronic spinal cord injury. *Arch Phys Med Rehabil,* 87(10):1327-1333.

Jalinous, R. (2001). *Guide to Magnetic Nerve Stimulation*.

Janssen, T.W., Dallmeijer, A.J., Veeger, D.J. and van der Woude, L.H. (2002). Normative values and determinants of physical capacity in individuals with spinal cord injury. *J Rehabil Res Dev,* 39(1):29-39.

Jensen, J.I., Lyager, S. and Pedersen, O.F. (1980). The relationship between maximal ventilation, breathing pattern and mechanical limitation of ventilation. *J Physiol,* 309:521-532.

Johnson, B.D., Babcock, M.A., Suman, O.E. and Dempsey, J.A. (1993). Exercise-induced diaphragmatic fatigue in healthy humans. *J Physiol,* 460:385-405.

Johnson, B.D., Beck, K.C., Zeballos, R.J. and Weisman, I.M. (1999a). Advances in pulmonary laboratory testing. *Chest,* 116(5):1377-1387.

Johnson, B.D., Reddan, W.G., Pegelow, D.F., Seow, K.C. and Dempsey, J.A. (1991). Flow limitation and regulation of functional residual capacity during exercise in a physically active aging population. *Am Rev Respir Dis,* 143(5 Pt 1):960-967.

Johnson, B.D., Saupe, K.W. and Dempsey, J.A. (1992). Mechanical constraints on exercise hyperpnea in endurance athletes. *J Appl Physiol,* 73(3):874-886.

Johnson, B.D., Weisman, I.M., Zeballos, R.J. and Beck, K.C. (1999b). Emerging concepts in the evaluation of ventilatory limitation during exercise: the exercise tidal flow-volume loop. *Chest,* 116(2):488-503.

Kantrowitz, A. (1960). Electronic Physiological Aids. *Report on the Maimonides Hospital Brooklyn*:4-5.

Kelley, A., Garshick, E., Gross, E.R., Lieberman, S.L., Tun, C.G. and Brown, R. (2003). Spirometry testing standards in spinal cord injury. *Chest,* 123(3):725-730.

Kemi, O.J., Haram, P.M., Loennechen, J.P., Osnes, J., Skomedal, T., Wisloff, U. and Ellingsen, O. (2005). Moderate vs. high exercise intensity: Differential effects on aerobic fitness, cardiomyocyte contractility, and endothelial function. *Cardiovascular Research,* 67(1):161-172.

Kerk, J.K., Clifford, P.S., Snyder, A.C., Prieto, T.E., O'Hagan, K.P., Schot, P.K., Myklebust, J.B. and Myklebust, B.M. (1995). Effect of an abdominal binder during wheelchair exercise. *Med Sci Sports Exerc,* 27(6):913-919.

Kessler, K.M., Pina, I., Green, B., Burnett, B., Laighold, M., Bilsker, M., Palomo, A.R. and Myerburg, R.J. (1986). Cardiovascular findings in quadriplegic and paraplegic patients and in normal subjects. *Am J Cardiol,* 58(6):525-530.

Kitano, Y., Takata, M., Sasaki, N., Zhang, Q., Yamamoto, S. and Miyasaka, K. (1999). Influence of increased abdominal pressure on steady-state cardiac performance. *J Appl Physiol,* 86(5):1651-1656.

Klefbeck, B., Mattsson, E., Weinberg, J. and Svanborg, E. (1998). Oxygen desaturations during exercise and sleep in fit tetraplegic patients. *Arch Phys Med Rehabil,* 79(7):800-804.

Kohl, H.W. (2001). Physical activity and cardiovascular disease: evidence for a dose response. *Med Sci Sports Exerc,* 33(6):S472-S483.

Koulouris, N., Mulvey, D.A., Laroche, C.M., Goldstone, J., Moxham, J. and Green, M. (1989). The effect of posture and abdominal binding on respiratory pressures. *Eur Respir J,* 2(10):961-965.

Koulouris, N.G., Dimopoulou, I., Valta, P., Finkelstein, R., Cosio, M.G. and Milic-Emili, J. (1997). Detection of expiratory flow limitation during exercise in COPD patients. *J Appl Physiol,* 82(3):723-731.

Koulouris, N.G., Valta, P., Lavoie, A., Corbeil, C., Chasse, M., Braidy, J. and Milic-Emili, J. (1995). A simple method to detect expiratory flow limitation during spontaneous breathing. *Eur Respir J,* 8(2):306-313.

Krassioukov, A. (2004). Autonomic dysreflexia in acute spinal cord injury: incidence, mechanisms, and management. *SCI Nurs,* 21(4):215-216.

Krassioukov, A. (2009). Autonomic function following cervical spinal cord injury. *Respir Physiol Neurobiol,* 169(2):157-164.

Krassioukov, A., Claydon, V.E., Lynne, C.W. and Canio, P. (2006). The clinical problems in cardiovascular control following spinal cord injury: an overview. In: *Progress in Brain Research*. pp. 223-229. Elsevier.

Krassioukov, A., Eng, J.J., Warburton, D.E. and Teasell, R. (2009). A systematic review of the management of orthostatic hypotension after spinal cord injury. *Arch Phys Med Rehabil,* 90(5):876-885.

Krum, H., Louis, W.J., Brown, D.J. and Howes, L.G. (1992). Pressor dose responses and baroreflex sensitivity in quadriplegic spinal cord injury patients. *J Hypertens,* 10(3):245-250.

Lakomy, H.K., Campbell, I. and Williams, C. (1987). Treadmill performance and selected physiological characteristics of wheelchair athletes. *Br J Sports Med,* 21(3):130-133.

Lan, C., Lai, J.S., Chang, K.H., Jean, Y.C. and Lien, I.N. (1993). Traumatic spinal cord injuries in the rural region of Taiwan: an epidemiological study in Hualien county, 1986-1990. *Paraplegia,* 31(6):398-403.

Lang, R.M., Bierig, M., Devereux, R.B., Flachskampf, F.A., Foster, E., Pellikka, P.A., Picard, M.H., Roman, M.J., Seward, J., Shanewise, J.S., Solomon, S.D., Spencer, K.T., Sutton, M.S. and Stewart, W.J. (2005). Recommendations for chamber quantification. *J Am Soc Echocardiogr,* 18(12):1440-1463.

Lasko-McCarthey, P. and Davis, J. (1991). Effect of work rate increment on peak oxygen uptake during wheelchair ergometry in men with quadriplegia. *Eur J Appl Physiol Occup Physiol,* 63(5):349-353.

Ledsome, J.R. and Sharp, J.M. (1981). Pulmonary function in acute cervical cord injury. *Am Rev Respir Dis,* 124(1):41-44.

Lehmann, K.G., Lane, J.G., Piepmeier, J.M. and Batsford, W.P. (1987). Cardiovascular abnormalities accompanying acute spinal cord injury in humans: incidence, time course and severity. *J Am Coll Cardiol,* 10(1):46-52.

Lenton, J. 2008. Mechanical efficiency of push strategies during manual hand-rim wheelchair propulsion. Manchester: Manchester Metropolitan University. pp. 184.

Lewis, J.E., Nash, M.S., Hamm, L.F., Martins, S.C. and Groah, S.L. (2007). The relationship between perceived exertion and physiologic indicators of stress during graded arm exercise in persons with spinal cord injuries. *Arch Phys Med Rehabil,* 88(9):1205-1211.

Lidal, I.B., Snekkevik, H., Aamodt, G., Hjeltnes, N., Biering-Sorensen, F. and Stanghelle, J.K. (2007). Mortality after spinal cord injury in Norway. *J Rehabil Med,* 39(2):145-151.

Lin, F., Parthasarathy, S., Taylor, S.J., Pucci, D., Hendrix, R.W. and Makhsous, M. (2006). Effect of different sitting postures on lung capacity, expiratory flow, and lumbar lordosis. *Arch Phys Med Rehabil,* 87(4):504-509.

Lin, H.T., Su, F.C., Wu, H.W. and An, K.N. (2004). Muscle forces analysis in the shoulder mechanism during wheelchair propulsion. *Proc Inst Mech Eng H,* 218(4):213-221.

Lin, K.H., Lai, Y.L., Wu, H.D., Wang, T.Q. and Wang, Y.H. (1998). Effects of an abdominal binder and electrical stimulation on cough in patients with spinal cord injury. *J Formos Med Assoc,* 97(4):292-295.

Linn, W.S., Spungen, A.M., Gong, H., Adkins, R.H., Bauman, W.A. and Waters, R.L. (2001). Forced vital capacity in two large outpatient populations with chronic spinal cord injury. *Spinal Cord,* 39(5):263-268.

Lord, P.W. and Edwards, J.M. (1978). Variation in airways resistance when defined over different ranges of airflows. *Thorax,* 33(3):401-405.

Loring, S.H. and Mead, J. (1982). Action of the diaphragm on the rib cage inferred from a force-balance analysis. *J Appl Physiol,* 53(3):756-760.

Lougheed, M.D., Flannery, J., Webb, K.A. and O'Donnell, D.E. (2002). Respiratory sensation and ventilatory mechanics during induced bronchoconstriction in spontaneously breathing low cervical quadriplegia. *Am J Respir Crit Care Med,* 166(3):370-376.

Mador, M.J., Khan, S. and Kufel, T.J. (2002). Bilateral anterolateral magnetic stimulation of the phrenic nerves can detect diaphragmatic fatigue. *Chest,* 121(2):452-458.

Mador, M.J., Magalang, U.J., Rodis, A. and Kufel, T.J. (1993). Diaphragmatic fatigue after exercise in healthy human subjects. *Am Rev Respir Dis,* 148(6 Pt 1):1571-1575.

Maloney, F.P. (1979). Pulmonary function in quadriplegia: effects of a corset. *Arch Phys Med Rehabil,* 60(6):261-265.

Martini, F.R. (2001). The heart. In: *Fundamentals of Anatomy and Physiology* 5th edition. pp. 684-686. New Jersey. Prentice-Hall.

Mateus, S.R., Beraldo, P.S. and Horan, T.A. (2006). Cholinergic bronchomotor tone and airway caliber in tetraplegic patients. *Spinal Cord,* 44(5):269-274.

Mateus, S.R., Beraldo, P.S. and Horan, T.A. (2007). Maximal static mouth respiratory pressure in spinal cord injured patients: correlation with motor level. *Spinal Cord,* 45(8):569-575.

Mathias, C.J., Christensen, N.J., Corbett, J.L., Frankel, H.L., Goodwin, T.J. and Peart, W.S. (1975). Plasma catecholamines, plasma renin activity and plasma aldosterone in tetraplegic man, horizontal and tilted. *Clin Sci Mol Med,* 49(4):291-299.

Mathias, C.J. and Frankel, H.L. (1983). Clinical manifestations of malfunctioning sympathetic mechanisms in tetraplegia. *J Aut Nerv Sys,* 7(3-4):303-312.

Maynard, F.M., Bracken, M.B., Creasey, G., Ditunno, J.F., Donovan, W.H., Ducker, T.B., Garber, S.L., Marino, R.J., Stover, S.L., Tator, C.H., Waters, R.L., Wilberger, J.E. and Young, W. (1997). International standards for neurological and functional classification of spinal cord injury. American Spinal Injury Association. *Spinal Cord,* 35(5):266-274.

McClaran, S.R., Wetter, T.J., Pegelow, D.F. and Dempsey, J.A. (1999). Role of expiratory flow limitation in determining lung volumes and ventilation during exercise. *J Appl Physiol,* 86(4):1357-1366.

McCool, F.D. (2006). Global physiology and pathophysiology of cough - ACCP evidence-based clinical practice guidelines. *Chest,* 129(1):48s-53s.

McCool, F.D., Pichurko, B.M., Slutsky, A.S., Sarkarati, M., Rossier, A. and Brown, R. (1986). Changes in lung volume and rib cage configuration with abdominal binding in quadriplegia. *J Appl Physiol,* 60(4):1198-1202.

Mead, J. (1979). Functional significance of the area of apposition of diaphragm to rib cage [proceedings]. *Am Rev Respir Dis,* 119(2 Pt 2):31-32.

Miller, J.D., Pegelow, D.F., Jacques, A.J. and Dempsey, J.A. (2005a). Effects of augmented respiratory muscle pressure production on locomotor limb venous return during calf contraction exercise. *J Appl Physiol,* 99(5):1802-1815.

Miller, M.R., Hankinson, J., Brusasco, V., Burgo, F., Casaburi, R., Coates, A., Crapo, R., Enright, P., van der Grinten, C.P., Gustafsson, P., Jensen, R., Johnson, D.C., MacIntyre, N., McKay, R., Navajas, D., Pedersen, O.F., Pellegrino, R., Viegi, G. and Wanger, J. (2005b). Standardisation of spirometry. *Eur Respir J,* 26:319-338.

Mills, G.H., Kyroussis, D., Hamnegard, C.H., Polkey, M.I., Green, M. and Moxham, J. (1996). Bilateral magnetic stimulation of the phrenic nerves from an anterolateral approach. *Am J Respir Crit Care Med,* 154(4 Pt 1):1099-1105.

Morgan, M.D., Gourlay, A.R., Silver, J.R., Williams, S.J. and Denison, D.M. (1985). Contribution of the rib cage to breathing in tetraplegia. *Thorax,* 40(8):613-617.

Mortola, J.P. and Sant'Ambrogio, G. (1978). Motion of the rib cage and the abdomen in tetraplegic patients. *Clin Sci Mol Med,* 54(1):25-32.

Mota, S., Casan, P., Drobnic, F., Giner, J., Ruiz, O., Sanchis, J. and Milic-Emili, J. (1999). Expiratory flow limitation during exercise in competition cyclists. *J Appl Physiol,* 86(2):611-616.

Moulton, A. and Silver, J.R. (1970). Chest movements in patients with traumatic injuries of the cervical cord. *Clin Sci,* 39(3):407-422.

Mueller, G., de Groot, S., van der Woude, L. and Hopman, M.T. (2008). Time-courses of lung function and respiratory muscle pressure generating capacity after spinal cord injury: a prospective cohort study. *J Rehabil Med,* 40(4):269-276.

Munakata, M., Kameyama, J., Nunokawa, T., Ito, N. and Yoshinaga, K. (2001). Altered Mayer wave and baroreflex profiles in high spinal cord injury. *Am J Hypertens,* 14(2):141-148.

Mutton, D.L., Scremin, A.M., Barstow, T.J., Scott, M.D., Kunkel, C.F. and Cagle, T.G. (1997). Physiologic responses during functional electrical stimulation leg cycling and hybrid exercise in spinal cord injured subjects. *Arch Phys Med Rehabil,* 78(7):712-718.

Nash, M.S., Bilsker, S., Marcillo, A.E., Isaac, S.M., Botelho, L.A., Klose, K.J., Green, B.A., Rountree, M.T. and Shea, J.D. (1991). Reversal of adaptive left ventricular atrophy following electrically-stimulated exercise training in human tetraplegics. *Paraplegia,* 29(9):590-599.

Nash, M.S., Montalvo, B.M. and Applegate, B. (1996). Lower extremity blood flow and responses to occlusion ischemia differ in exercise-trained and sedentary tetraplegic persons. *Arch Phys Med Rehabil,* 77(12):1260-1265.

Nava, S., Rubini, F., Zanotti, E. and Caldiroli, D. (1996). The tension-time index of the diaphragm revisited in quadriplegic patients with diaphragm pacing. *Am J Respir Crit Care Med,* 153(4):1322-1327.

Nevill, A.M. and Atkinson, G. (1997). Assessing agreement between measurements recorded on a ratio scale in sports medicine and sports science. *Br J Sports Med,* 31(4):314-318.

Ninane, V., Baer, R.E. and De Troyer, A. (1989). Mechanism of triangularis sterni shortening during expiration in dogs. *J Appl Physiol,* 66(5):2287-2292.

Ning, G.Z., Yu, T.Q., Feng, S.Q., Zhou, X.H., Ban, D.X., Liu, Y. and Jiao, X.X. (2011). Epidemiology of traumatic spinal cord injury in Tianjin, China. *Spinal Cord,* 49(3):386-390.

O'Connor, P.J. (2005a). Forecasting of spinal cord injury annual case numbers in Australia. *Arch Phys Med Rehabil,* 86(1):48-51.

O'Connor, P.J. (2005b). Prevalence of spinal cord injury in Australia. *Spinal Cord,* 43(1):42-46.

O'Connor, P.J. (2006). Trends in spinal cord injury. *Accid Anal Prev,* 38(1):71-77.

O'Donnell, D.E., Bertley, J.C., Chau, L.K. and Webb, K.A. (1997). Qualitative aspects of exertional breathlessness in chronic airflow limitation: pathophysiologic mechanisms. *Am J Respir Crit Care Med,* 155(1):109-115.

O'Donnell, D.E., Hamilton, A.L. and Webb, K.A. (2006). Sensory-mechanical relationships during high-intensity, constant-work-rate exercise in COPD. *J Appl Physiol,* 101(4):1025-1035.

O'Donnell, D.E., Hong, H.H. and Webb, K.A. (2000). Respiratory sensation during chest wall restriction and dead space loading in exercising men. *J Appl Physiol,* 88(5):1859-1869.

O'Donnell, D.E. and Laveneziana, P. (2007). Dyspnea and activity limitation in COPD: mechanical factors. *Copd,* 4(3):225-236.

O'Donnell, D.E., Revill, S.M. and Webb, K.A. (2001). Dynamic hyperinflation and exercise intolerance in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med,* 164(5):770-777.

Olafsson, S. and Hyatt, R.E. (1969). Ventilatory mechanics and expiratory flow limitation during exercise in normal subjects. *J Clin Invest,* 48(3):564-573.

Oo, T., Watt, J.W., Soni, B.M. and Sett, P.K. (1999). Delayed diaphragm recovery in 12 patients after high cervical spinal cord injury. A retrospective review of the diaphragm status of 107 patients ventilated after acute spinal cord injury. *Spinal Cord,* 37(2):117-122.

Peckham, P.H., Mortimer, J.T. and Van Der Meulen, J.P. (1973). Physiologic and metabolic changes in white muscle of cat following induced exercise. *Brain Res,* 50(2):424-429.

Pellegrino, R., Viegi, G., Brusasco, V., Crapo, R.O., Burgos, F., Casaburi, R., Coates, A., van der Grinten, C.P., Gustafsson, P., Hankinson, J., Jensen, R., Johnson, D.C., MacIntyre, N., McKay, R., Miller, M.R., Navajas, D., Pedersen, O.F. and Wanger, J. (2005). Interpretative strategies for lung function tests. *Eur Respir J,* 26(5):948-968.

Pendergast, D.R. (1989). Cardiovascular, respiratory, and metabolic responses to upper body exercise. *Med Sci Sports Exerc,* 21(5 Suppl):S121-125.

Perhonen, M.A., Zuckerman, J.H. and Levine, B.D. (2001). Deterioration of left ventricular chamber performance after bed rest : "Cardiovascular deconditioning" or hypovolemia? *Circulation,* 103(14):1851-1857.

Petrofsky, J.S. (1992). Thermoregulatory stress during rest and exercise in heat in patients with a spinal cord injury. *Eur J Appl Physiol Occup Physiol,* 64(6):503-507.

Pickering, M. and Jones, J.F. (2002). The diaphragm: two physiological muscles in one. *J Anat,* 201(4):305-312.

Pickett, G.E., Campos-Benitez, M., Keller, J.L. and Duggal, N. (2006). Epidemiology of traumatic spinal cord injury in Canada. *Spine,* 31(7):799-805.

Piepmeier, J.M., Lehmann, K.B. and Lane, J.G. (1985). Cardiovascular instability following acute cervical spinal cord trauma. *Cent Nerv Syst Trauma,* 2(3):153-160.

Pitetti, K.H., Barrett, P.J., Campbell, K.D. and Malzahn, D.E. (1994). The effect of lower body positive pressure on the exercise capacity of individuals with spinal cord injury. *Med Sci Sports Exerc,* 26(4):463-468.

Polkey, M.I., Luo, Y., Guleria, R., Hamnegard, C.H., Green, M. and Moxham, J. (1999). Functional magnetic stimulation of the abdominal muscles in humans. *Am J Respir Crit Care Med,* 160(2):513-522.

Pollack, S.F., Axen, K., Spielholz, N., Levin, N., Haas, F. and Ragnarsson, K.T. (1989). Aerobic training effects of electrically induced lower extremity exercises in spinal cord injured people. *Arch Phys Med Rehabil,* 70(3):214-219.

Popovic, M.R., Curt, A., Keller, T. and Dietz, V. (2001). Functional electrical stimulation for grasping and walking: indications and limitations. *Spinal Cord,* 39(8):403-412.

Powers, S.K., Dodd, S., Lawler, J., Landry, G., Kirtley, M., McKnight, T. and Grinton, S. (1988). Incidence of exercise induced hypoxemia in elite endurance athletes at sea level. *Eur J Appl Physiol Occup Physiol,* 58(3):298-302.

Prefaut, C., Durand, F., Mucci, P. and Caillaud, C. (2000). Exercise-induced arterial hypoxaemia in athletes: a review. *Sports Med,* 30(1):47-61.

Press, V., Freestone, I. and George, C.F. (2003). Physical activity: the evidence of benefit in the prevention of coronary heart disease. *QJM,* 96(4):245-251.

Price, C., Makintubee, S., Herndon, W. and Istre, G.R. (1994). Epidemiology of traumatic spinal cord injury and acute hospitalization and rehabilitation charges for spinal cord injuries in Oklahoma, 1988-1990. *Am J Epidemiol,* 139(1):37-47.

Price, M.J. (2006). Thermoregulation during exercise in individuals with spinal cord injuries. *Sports Med,* 36(10):863-879.

Price, M.J. and Campbell, I.G. (1997). Thermoregulatory responses of paraplegic and able-bodied athletes at rest and during prolonged upper body exercise and passive recovery. *Eur J Appl Physiol Occup Physiol,* 76(6):552-560.

Price, M.J. and Campbell, I.G. (2003). Effects of spinal cord lesion level upon thermoregulation during exercise in the heat. *Med Sci Sports Exerc,* 35(7):1100-1107.

Quanjer, P. (1983). Standardized lung function testing. *Bull Eur Physiopathol Respir,* 19(Suppl. 5):33-38.

Quanjer, P.H., Tammeling, G.J., Cotes, J.E., Pedersen, O.F., Peslin, R. and Yernault, J.C. (1993). Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. *Eur Respir J Suppl,* 16:5-40.

Radulovic, M., Schilero, G.J., Wecht, J.M., Weir, J.P., Spungen, A.M., Bauman, W.A. and Lesser, M. (2008). Airflow Obstruction and Reversibility in Spinal Cord Injury: Evidence for Functional Sympathetic Innervation. *Arch Phys Med Rehabil,* 89(12):2349-2353.

Raper, A.J., Thompson, W.T., Shapiro, W. and Patterson, J.L. (1966). Scalene and sternomastoid muscle function. *J Appl Physiol,* 21(2):497-502.

Ready, A.E. (1994). Response of quadriplegic athletes to maximal and submaximal exercise. *Physiother Can,* 36:124-128.

Robergs, R.A., Dwyer, D. and Astorino, T. (2010). Recommendations for improved data processing from expired gas analysis indirect calorimetry. *Sports Med,* 40(2):95-111.

Romer, L.M., Harms, C.A. and Sheel, A.W. (in press). The respiratory system. In: *ACSM's advanced exercise physiology* (eds Farrell, P., Joyner, M. and Caiozzo, V.). Baltimore, MD. Lippincott, Williams and Wilkins.

Romer, L.M., Haverkamp, H.C., Lovering, A.T., Pegelow, D.F. and Dempsey, J.A. (2006a). Effect of exercise-induced arterial hypoxemia on quadriceps muscle fatigue in healthy humans. *Am J Physiol Regul Integr Comp Physiol,* 290(2):R365-375.

Romer, L.M., Lovering, A.T., Haverkamp, H.C., Pegelow, D.F. and Dempsey, J.A. (2006b). Effect of inspiratory muscle work on peripheral fatigue of locomotor muscles in healthy humans. *J Physiol,* 571(Pt 2):425-439.

Romer, L.M. and Polkey, M.I. (2008). Exercise-induced respiratory muscle fatigue: implications for performance. *J Appl Physiol,* 104(3):879-888.

Roth, E.J., Lu, A., Primack, S., Oken, J., Nusshaum, S., Berkowitz, M. and Powley, S. (1997). Ventilatory function in cervical and high thoracic spinal cord injury. Relationship to level of injury and tone. *Am.J.Phys.Med.Rehabil.,* 76(4):262-267.

Rowell, L.B. (1973). Regulation of splanchnic blood flow in man. *Physiologist,* 16(2):127-142.

Rowell, L.B., Detry, J.M., Blackmon, J.R. and Wyss, C. (1972). Importance of the splanchnic vascular bed in human blood pressure regulation. *J Appl Physiol,* 32(2):213-220.

Rowell, L.B., Masoro, E.J. and Spencer, M.J. (1965). Splanchnic metabolism in exercising man. *J Appl Physiol,* 20(5):1032-1037.

Scanlon, P.D., Loring, S.H., Pichurko, B.M., McCool, F.D., Slutsky, A.S., Sarkarati, M. and Brown, R. (1989). Respiratory mechanics in acute quadriplegia. Lung and chest wall compliance and dimensional changes during respiratory maneuvers. *Am Rev Respir Dis,* 139(3):615-620.

Schilero, G.J., Grimm, D.R., Bauman, W.A., Lenner, R. and Lesser, M. (2005). Assessment of airway caliber and bronchodilator responsiveness in subjects with spinal cord injury. *Chest,* 127(1):149-155.

Schilero, G.J., Spungen, A.M., Bauman, W.A., Radulovic, M. and Lesser, M. (2009). Pulmonary function and spinal cord injury. *Respir Physiol Neurobiol,* 166(3):129-141.

Schmid, A., Huonker, M., Barturen, J.M., Stahl, F., Schmidt-Trucksass, A., Konig, D., Grathwohl, D., Lehmann, M. and Keul, J. (1998). Catecholamines, heart rate, and oxygen uptake during exercise in persons with spinal cord injury. *J Appl Physiol,* 85(2):635-641.

Scroop, G.C. and Shipp, N.J. (2010). Exercise-Induced Hypoxemia: Fact or Fallacy? *Med Sci Sports Exerc,* 42(1):120-126.

Seaworth, J.F., Jennings, T.J., Howell, L.L., Frazier, J.W., Goodyear, C.D. and Grassman, E.D. (1985). Hemodynamic effects of anti-G suit inflation in a 1-G environment. *J Appl Physiol,* 59(4):1145-1151.

Sheel, A.W., Reid, W.D., Townson, A.F. and Ayas, N. (2008a). Respiratory Management Following Spinal Cord Injury. In. Eng JJ, Teasall RW, Miller WC, Wolfe DL, Twonson AF, Hsieh JTC, Konnyu KF, Connolly SJ, Foulon BL, Aubut JL, editors, Spinal Cord Injury Rehabilitation Evidence, Volume 2.0. Vancouver. p. 8.1-8.40.

Sheel, A.W., Reid, W.D., Townson, A.F., Ayas, N.T. and Konnyu, K.J. (2008b). Effects of exercise training and inspiratory muscle training in spinal cord injury: a systematic review. *J Spinal Cord Med,* 31(5):500-508.

Shingu, H., Ikata, T., Katoh, S. and Akatsu, T. (1994). Spinal cord injuries in Japan: a nationwide epidemiological survey in 1990. *Paraplegia,* 32(1):3-8.

Short, D.J., Silver, J.R. and Lehr, R.P. (1991). Electromyographic study of sternocleidomastoid and scalene muscles in tetraplegic subjects during respiration. *Int Disabil Stud,* 13(2):46-49.

Siebens, A.A., Kirby, N.A. and Poulos, D.A. (1964). Cough following transection of spinal cord at C-6. *Arch Phys Med Rehabil,* 45:1-8.

Silva, A.C., Neder, J.A., Chiurciu, M.V., Pasqualin, D.C., da Silva, R.C., Fernandez, A.C., Lauro, F.A., de Mello, M.T. and Tufik, S. (1998). Effect of aerobic training on ventilatory muscle endurance of spinal cord injured men. *Spinal Cord,* 36(4):240-245.

Silver, J.R. and Lehr, R.P. (1981). Electromyographic investigation of the diaphragm and intercostal muscles in tetraplegics. *J Neurol Neurosurg Psychiatry,* 44(9):837-841.

Similowski, T., Fleury, B., Launois, S., Cathala, H.P., Bouche, P. and Derenne, J.P. (1989). Cervical magnetic stimulation: a new painless method for bilateral phrenic nerve stimulation in conscious humans. *J Appl Physiol,* 67(4):1311-1318.

Sinderby, C., Ingvarsson, P., Sullivan, L., Wickstrom, I. and Lindstrom, L. (1992). The role of the diaphragm in trunk extension in tetraplegia. *Paraplegia,* 30(6):389-395.

Sinderby, C., Weinberg, J., Sullivan, L., Borg, J., Lindstrom, L. and Grassino, A. (1996a). Diaphragm function in patients with cervical cord injury or prior poliomyelitis infection. *Spinal Cord,* 34(4):204-213.

Sinderby, C., Weinberg, J., Sullivan, L., Lindstrom, L. and Grassino, A. (1996b). Electromyographical evidence for exercise-induced diaphragm fatigue in patients with chronic cervical cord injury or prior poliomyelitis infection. *Spinal Cord,* 34:594-601.

Singas, E., Lesser, M., Spungen, A.M., Bauman, W.A. and Almenoff, P.L. (1996). Airway hyperresponsiveness to methacholine in subjects with spinal cord injury. *Chest,* 110(4):911-915.

Smit, A.A., Wieling, W., Fujimura, J., Denq, J.C., Opfer-Gehrking, T.L., Akarriou, M., Karemaker, J.M. and Low, P.A. (2004). Use of lower abdominal compression to combat orthostatic hypotension in patients with autonomic dysfunction. *Clin Auton Res,* 14(3):167-175.

Smith, J.C. and Hill, D.W. (1991). Contribution of energy systems during a Wingate power test. *Br J Sports Med,* 25(4):196-199.

Soden, R.J., Walsh, J., Middleton, J.W., Craven, M.L., Rutkowski, S.B. and Yeo, J.D. (2000). Causes of death after spinal cord injury. *Spinal Cord,* 38(10):604-610.

Spirito, P., Pelliccia, A., Proschan, M.A., Granata, M., Spataro, A., Bellone, P., Caselli, G., Biffi, A., Vecchio, C. and Maron, B.J. (1994). Morphology of the "athlete's heart" assessed by echocardiography in 947 elite athletes representing 27 sports. *Am J Cardiol,* 74(8):802-806.

St Croix, C.M., Morgan, B.J., Wetter, T.J. and Dempsey, J.A. (2000). Fatiguing inspiratory muscle work causes reflex sympathetic activation in humans. *J Physiol,* 529 (Pt 2):493-504.

Stepp, E.L., Brown, R., Tun, C.G., Gagnon, D.R., Jain, N.B. and Garshick, E. (2008). Determinants of lung volumes in chronic spinal cord injury. *Arch Phys Med Rehabil,* 89(8):1499-1506.

Stover, S.L., DeVivo, M.J. and Go, B.K. (1999). History, implementation, and current status of the National Spinal Cord Injury Database. *Arch Phys Med Rehabil,* 80(11):1365-1371.

Stubbing, D.G., Pengelly, L.D., Morse, J.L. and Jones, N.L. (1980). Pulmonary mechanics during exercise in normal males. *J Appl Physiol,* 49(3):506-510.

Supinski, G.S., Fitting, J.W. and Bellemare, F. (2002). ATS/ERS Statement on respiratory muscle testing. *Am J Respir Crit Care Med,* 166(4):518-624.

Sutbeyaz, S.T., Koseoglu, B.F. and Gokkaya, N.K. (2005). The combined effects of controlled breathing techniques and ventilatory and upper extremity muscle exercise on cardiopulmonary responses in patients with spinal cord injury. *Int J Rehabil Res,* 28(3):273-276.

Swain, D.P. and Franklin, B.A. (2006). Comparison of cardioprotective benefits of vigorous versus moderate intensity aerobic exercise. *Am J Cardiol,* 97(1):141-147.

Takahashi, M., Matsukawa, K., Nakamoto, T., Tsuchimochi, H., Sakaguchi, A., Kawaguchi, K. and Onari, K. (2007). Control of heart rate variability by cardiac parasympathetic nerve activity during voluntary static exercise in humans with tetraplegia. *J Appl Physiol,* 103(5):1669-1677.

Takano, N. (1993). Ventilatory responses during arm and leg exercise at varying speeds and forces in untrained female humans. *J Physiol,* 468:413-424.

Taylor, A. (1960). The contribution of the intercostal muscles to the effort of respiration in man. *J Physiol,* 151:390-402.

Taylor, B.J., West, C.R. and Romer, L.M. (2010). No effect of arm-crank exercise on diaphragmatic fatigue or ventilatory constraint in Paralympic athletes with cervical spinal cord injury. *J Appl Physiol,* 109(2):358-366.

Teasell, R.W., Arnold, J.M.O., Krassioukov, A. and Delaney, G.A. (2000). Cardiovascular consequences of loss of supraspinal control of the sympathetic nervous system after spinal cord injury. *Arch Phys Med Rehabil,* 81(4):506-516.

Teichholz, L.E., Kreulen, T., Herman, M.V. and Gorlin, R. (1976). Problems in echocardiographic volume determinations: echocardiographic-angiographic correlations in the presence or absence of asynergy. *Am J Cardiol,* 37(1):7-11.

The Consensus Committee of the American Autonomic Society and the American Academy of Neurology. (1996). Consensus statement on the definition of orthostatic hypotension, pure autonomic failure, and multiple system atrophy. *Neurology,* 46(5):1470.

Theisen, D., Francaux, M., Fay, A. and Sturbois, X. (1996). A new procedure to determine external power output during handrim wheelchair propulsion on a roller ergometer: A reliability study. *Int J Sports Med,* 17(08):564,571.

Thijssen, D.H., Heesterbeek, P., van Kuppevelt, D.J., Duysens, J. and Hopman, M.T. (2005). Local vascular adaptations after hybrid training in spinal cord-injured subjects. *Med Sci Sports Exerc,* 37(7):1112-1118.

Thijssen, D.H., Steendijk, S. and Hopman, M.T. (2009). Blood redistribution during exercise in subjects with spinal cord injury and controls. *Med Sci Sports Exerc,* 41(6):1249-1254.

Tweedy, S. and Diaper, N. (2010). Introduction to wheelchair sport. In: *Wheelchair Sport: A complete guide for athletes, coaches and teachers* (ed Goosey-Tolfrey, V.). pp. 3-29. Champaign, IL. Human Kinetics.

Urmey, W., Loring, S., Mead, J., Slutsky, A.S., Sarkarati, M., Rossier, A. and Brown, R. (1986). Upper and lower rib cage deformation during breathing in quadriplegics. *J Appl Physiol,* 60(2):618-622.

Vallbona, C., Spencer, W.A., Cardus, D. and Dale, J.W. (1963). Control of orthostatic hypotension of quadriplegic patients with a pressure suite. *Arch Phys Med Rehabil,* 44:7-18.

Valta, P., Corbeil, C., Lavoie, A., Campodonico, R., Koulouris, N., Chasse, M., Braidy, J. and Milic-Emili, J. (1994). Detection of expiratory flow limitation during mechanical ventilation. *Am J Respir Crit Care Med,* 150(5 Pt 1):1311-1317.

van Asbeck, F.W., Post, M.W. and Pangalila, R.F. (2000). An epidemiological description of spinal cord injuries in The Netherlands in 1994. *Spinal Cord,* 38(7):420-424.

van der Woude, L.H., de Groot, G., Hollander, A.P., van Ingen Schenau, G.J. and Rozendal, R.H. (1986). Wheelchair ergonomics and physiological testing of prototypes. *Ergonomics,* 29(12):1561-1573.

van der Woude, L.H.V., Veeger, H.E.J., Dallmeijer, A.J., Janssen, T.W.J. and Rozendaal, L.A. (2001). Biomechanics and physiology in active manual wheelchair propulsion. *Medical Engineering & Physics,* 23(10):713-733.

Van Loan, M.D., McCluer, S., Loftin, J.M. and Boileau, R.A. (1987). Comparison of physiological responses to maximal arm exercise among able-bodied, paraplegics and quadriplegics. *Paraplegia,* 25(5):397-405.

Vanlandewijck, Y., Theisen, D. and Daly, D. (2001). Wheelchair propulsion biomechanics: implications for wheelchair sports. *Sports Med,* 31(5):339-367.

Vaziri, N.D. (2003). Nitric oxide in microgravity-induced orthostatic intolerance: relevance to spinal cord injury. *J Spinal Cord Med,* 26(1):5-11.

Verellen, J., Vanlandewijck, Y., Andrews, B. and Wheeler, G.D. (2007). Cardiorespiratory responses during arm ergometry, functional electrical stimulation cycling, and two hybrid exercise conditions in spinal cord injured. *Disabil Rehabil Assist Technol,* 2(2):127-132.

Vivier, E., Metton, O., Piriou, V., Lhuillier, F., Cottet-Emard, J.M., Branche, P., Duperret, S. and Viale, J.P. (2006). Effects of increased intra-abdominal pressure on central circulation. *Br J Anaesth,* 96(6):701-707.

Vogiatzis, I., Nanas, S., Kastanakis, E., Georgiadou, O., Papazahou, O. and Roussos, C. (2004). Dynamic hyperinflation and tolerance to interval exercise in patients with advanced COPD. *Eur Respir J,* 24(3):385-390.

Wade, O.L. (1954). Movements of the thoracic cage and diaphragm in respiration. *J Physiol,* 124(2):193-212.

Wadsworth, B.M., Haines, T.P., Cornwell, P.L. and Paratz, J.D. (2009). Abdominal binder use in people with spinal cord injuries: a systematic review and meta-analysis. *Spinal Cord,* 47(4):274-285.

Wahr, D.W., Wang, Y.S. and Schiller, N.B. (1983). Left ventricular volumes determined by two-dimensional echocardiography in a normal adult population. *J Am Coll Cardiol,* 1(3):863-868.

Wallin, B.G. and Stjernberg, L. (1984). Sympathetic activity in man after spinal cord injury. *Brain,* 107(1):183-198.

Wanger, J., Clausen, J.L., Coates, A., Pedersen, O.F., Brusasco, V., Burgos, F., Casaburi, R., Crapo, R., Enright, P., van der Grinten, C.P., Gustafsson, P., Hankinson, J., Jensen, R., Johnson, D., Macintyre, N., McKay, R., Miller, M.R., Navajas, D., Pellegrino, R. and Viegi, G. (2005). Standardisation of the measurement of lung volumes. *Eur Respir J,* 26(3):511-522.

Wasserman, K., Hansen, J.E., Darryl, S.Y., Stringer, W.W. and Whipp, B.J. (2005). Clinical applications of cardiopulmonary exercise testing. In: *Principles of exercise testing and interpretation*. pp. 209. Philadelphia. Lippincott Williams and Wilkins.

Webborn, N., Price, M.J., Castle, P. and Goosey-Tolfrey, V.L. (2008). Cooling strategies improve intermittent sprint performance in the heat of athletes with tetraplegia. *Br J Sports Med,* 44(6):455-460.

Webborn, N., Price, M.J., Castle, P.C. and Goosey-Tolfrey, V.L. (2005). Effects of two cooling strategies on thermoregulatory responses of tetraplegic athletes during repeated intermittent exercise in the heat. *J Appl Physiol,* 98(6):2101-2107.

Whipp, B.J. and Wasserman, K. (1969). Efficiency of muscular work. *J Appl Physiol,* 26(5):644-648.

Wicks, J.R., Oldridge, N.B., Cameron, B.J. and Jones, N.L. (1983). Arm cranking and wheelchair ergometry in elite spinal cord-injured athletes. *Med Sci Sports Exerc,* 15(3):224-231.

Wilson, T.A., Legrand, A., Gevenois, P.A. and De Troyer, A. (2001). Respiratory effects of the external and internal intercostal muscles in humans. *J Physiol,* 530(Pt 2):319-330.

Winslow, C. and Rozovsky, J. (2003). Effect of spinal cord injury on the respiratory system. *Am J Phys Med Rehabil,* 82(10):803-814.

Winslow, E.B., Lesch, M., Talano, J.V. and Meyer, P.R. (1986). Spinal cord injuries associated with cardiopulmonary complications. *Spine,* 11(8):809-812.

Woodruff, B.A. and Baron, R.C. (1994). A description of nonfatal spinal cord injury using a hospital-based registry. *Am J Prev Med,* 10(1):10-14.

Wragg, S., Aquilina, R., Moran, J., Ridding, M., Hamnegard, C., Fearn, T., Green, M. and Moxham, J. (1994). Comparison of cervical magnetic stimulation and bilateral percutaneous electrical stimulation of the phrenic nerves in normal subjects. *Eur Respir J,* 7(10):1788-1792.

Yekutiel, M., Brooks, M.E., Ohry, A., Yarom, J. and Carel, R. (1989). The prevalence of hypertension, ischaemic heart disease and diabetes in traumatic spinal cord injured patients and amputees. *Paraplegia,* 27(1):58-62.

Yeo, J.D., Walsh, J., Rutkowski, S., Soden, R., Craven, M. and Middleton, J. (1998). Mortality following spinal cord injury. *Spinal Cord,* 36(5):329-336.

APPENDIX A-1: ETHICAL APPROVAL LETTERS

Head of School of Sport & Education
Professor Susan Capel

Brunel UNIVERSITY WEST LONDON

Heinz Wolff Building,
Brunel University, Uxbridge,
Middlesex, UB8 3PH, UK
Telephone +44 (0)1895 266494
Fax +44 (0)1895 269769
Web www.brunel.ac.uk

Mr Christopher West
Research (PhD Sport Science) Student
c/o School of Sport and Education
Brunel University

25th March 2009

Dear Chris

RE22-08 - Respiratory and cardiovascular responses to abdominal binding in athletes with spinal cord injury

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 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

Head of School of Sport & Education
Professor Susan Capel

Brunel UNIVERSITY WEST LONDON

Heinz Wolff Building,
Brunel University, Uxbridge,
Middlesex, UB8 3PH, UK
Telephone +44 (0)1895 266494
Fax +44 (0)1895 269769
Web www.brunel.ac.uk

Christopher West
PhD (Sport Science) Student
School of Sport and Education
Brunel University

25th January 2010

Dear Chris

RE10-09: Effect of abdominal binding on exercise performance in athletes with chronic cervical spinal cord injury

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 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

Head of School of Sport & Education
Professor Susan Capel

Brunel UNIVERSITY WEST LONDON

Heinz Wolff Building,
Brunel University, Uxbridge,
Middlesex, UB8 3PH, UK
Telephone +44 (0)1895 266494
Fax +44 (0)1895 269769
Web www.brunel.ac.uk

Mr Christopher West
PhD (Sport Science) Student
School of Sport and Education
Brunel University

30th March 2010

Dear Chris

RE24-09: Effect of abdominal binding on breathing mechanics during maximal incremental exercise in wheelchair athletes with cervical spinal cord injury

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

APPENDIX A-2: DISABLITITY SPECIFIC QUESTIONNAIRE

Brunel University
Sport Sciences

Participant Questionnaire

This questionnaire is designed to aid the interpretation of data collected during your testing sessions. Please complete as honestly and accurately as possible. All information will be kept in the strictest of confidence and used only be the researchers.

General details

Name:

Age:

Date of Birth:

Contact Details

Home address:

Tel no.:

Mobile no.:

Email:

If known

Height:

Weight:

Injury/disability details

What is your disability?

What lesion level?

Is it complete or incomplete?

How long have you been a wheelchair user?

Please state any medication you are currently taking, including dose (e.g. antispasticity drugs or bronchodilators):

How long have you been taking the medication?

General Health

Do you smoke?

If no, have you ever smoked?

If yes, for how many years?

Do you suffer from epilepsy at all?

Have you ever/do you suffer from any health or respiratory complications?

Have you ever undertaken any sort of respiratory muscle training before?

If so how long did you do it for, what did it involve and when did you stop?

Did you notice any benefits?

Sport Participation

What sport do you play?

How many years have you played?

Do you play any other wheelchair based or other sports?

Where do you train?

How many times per week do you train?

On average, how long do your training sessions last?

What is the nature of these training sessions? i.e. 1 x strength training, 1 x game situation training

What was your sporting background prior to your injury?

APPENDIX A-3: PRE-EXERCISE HEALTH QUESTIONNAIRE

The participant should complete the whole of this sheet him/herself

	Please tick the appropriate box	
	YES	NO
Have you read the Research Participant Information Sheet?	☐	☐
Have you had an opportunity to ask questions and discuss this study?	☐	☐
Have you received satisfactory answers to all your questions?	☐	☐
Who have you spoken to?		
Do you understand that you will not be referred to by name in any report concerning the study?	☐	☐
Do you understand that you are free to withdraw from the study:		
- **At any time**	☐	☐
- **Without having to give a reason for withdrawing?**	☐	☐
- **Without affecting your future care?**	☐	☐
Do you agree to take part in this study?	☐	☐

Signature of Research Participant:

Date:

Name in capitals:

<u>Witness statement</u>

I am satisfied that the above-named has given informed consent.

Witnessed by:

Date:

Name in capitals: