Functional Electrical Stimulation as a Neuroprosthesis for Sitting Balance: Measuring Respiratory Function and Seated Postural Control in Able-bodied Individuals and Individuals with Spinal Cord Injury

by

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The level and completeness of spinal cord injury (SCI) above the first lumbar vertebra determine the degree of multi-system impairments including altered respiratory function and decreased capacity to maintain upright posture and seated postural stability in humans. Both systems were studied in able-bodied (AB) subjects and individuals with tetraplegia to explore the potential of functional electrical stimulation (FES) as a neuroprosthesis for seated postural control without compromising respiratory function. Data for AB participants (n=10) indicated higher tidal volumes, greater trunk extensor muscle activity, and different values of seated postural stability in upright sitting compared to slouch sitting. In three case studies of individuals with tetraplegia, surface FES was applied to trunk muscles. Changes in tidal volume, respiratory rate, and seated postural stability were case specific. These studies inform the development of a strategy for non-invasive FES as a neuroprosthesis for sitting balance while preserving respiratory function in individuals with SCI.
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Dedication

To my friend Katie McAlindon, whose memory I often visited during the writing of this thesis.
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Glossary

Anatomical

Anterior Superior Iliac Spine (ASIS, L or R denote Left or Right)

highest bony prominence on the anterior margin of the iliac crest of the pelvis

Central Nervous System (CNS)

organs (i.e. the brain and spinal cord) responsible for processing information related to
the control of all bodily functions

Erector Spinae Muscle (ES)

posterior trunk muscle group that acts to extend the spine along its entire length

Latissimus Dorsi Muscle (LD)

posterolateral trunk muscle that contributes to extension and lateral flexion of the lumbar
spine; LD primarily acts on the shoulder joint

Lower Motor Neuron

nerve fibre that connects the CNS to muscle fibres; originates in the anterior horn of the
spinal cord; transmits nerve impulses from the upper motor neuron to the muscle

Obliquus Externus Muscle (OE)

anterolateral abdominal trunk muscle that acts to compress the abdomen and retract the
chest downwards; OE contributes to trunk flexion and rotation

Posterior Superior Iliac Spine (PSIS, L or R denote Left or Right)

highest bony prominence on the posterior margin of the iliac crest of the pelvis

Rectus Abdominis Muscle (RA)

paired anterior abdominal trunk muscle that acts to flex the spine and compress the
abdomen

Spinal Cord Injury (SCI)

a traumatic injury that inflicts damage on the nervous tissue within the bony vertebrae of
the spine

Upper Motor Neuron

nerve fibre that originates in the motor cortex or brainstem, extending down the spinal
cord; at appropriate level of the spinal cord, it synapses with the lower motor neuron
Respiratory Function

Spirometry Testing
measures breathing characteristics including the amount (volume) and speed (flow) of air that can be inhaled or exhaled during a standard breathing manoeuvre

Forced Expiratory Volume in One Second (FEV₁)
volume of air exhaled in the first second of a forced expiration manoeuvre, measured in litres (L) or percent of predicted value (% Pred)

Forced Vital Capacity (FVC)
determination of vital capacity from a forced expiration manoeuvre, measured in litres (L) or percent of predicted value (% Pred)

Respiratory Rate (RR)
frequency of breathing, measured in breaths per minute (min⁻¹)

Tidal Volume (Vₜ)
volume of air exchanged in a person’s lungs in one breath, measured in litres (L)

Minute Ventilation (Vₐ)
volume of air exchanged in a person’s lungs in one minute, measured in litres per minute (L/min)

Postural Control

Postural Control System
sensorimotor feedback system involved in generating a frame of reference that allows maintenance of stability within the external environment under various circumstances

Postural Stability
the ability to control the centre of mass in relationship to the base of support

Centre of Mass (COM)
center point of total body mass calculated as the weighted mean of the COM of each body segment

Base of Support (BOS)
geometric surface area under the buttocks, thighs, and feet in a sitting position, measured in millimetres (mm)

Centre of Pressure (COP)
position of the vertical reaction vector on the surface of a force platform on which a person sits

Seated Postural Stability
capacity of the postural control system to maintain balance in sitting postures during performance of various movements by optimizing the position of the spine and the associated muscle activity
Sitting Posture
alignment of the axial skeleton in a seated position

Limits of Stability (LOS)
area within which a person can control his or her balance

Vertical Reaction Forces (Fz)
vertical (Z axis) component of the applied force vector detected over the surface of a force plate, measured in Newton (N)

Anteroposterior (AP)
describes movement of the COP in the Y-Z plane of the force plate; the arithmetic mean of the AP time series data is the y-coordinate of the mean COP (yCOP)

Mediolateral (ML)
describes movement of the COP in the X-Z plane of the force plate; the arithmetic mean of the ML time series data is the x-coordinate of the mean COP (xCOP)

Resultant Distance Time Series
vector distance from the mean COP to each pairs of points in the AP and ML time series, measured in millimetres (mm)

Mean COP Distance (MDIST)
mean of the resultant distance time series; represents the average distance of the COP trajectory from the mean COP, measured in millimetres (mm)

Total COP Excursion (TOTEX)
total length of the COP trajectory; approximately the sum of the distances between consecutive points on the COP path, measured in millimetres (mm)

Mean COP Velocity (MVELO)
average velocity of the COP trajectory; represents total excursion normalized to the analysis interval, measured in millimetres (mm)

Mean COP Frequency (MFREQ)
rotational frequency of the COP trajectory if it had traveled the total COP excursion around a circle with a radius equal to the mean COP distance, measured in Hertz (Hz)

Technical

Data Acquisition (DAQ)
computer (including hardware and software) used to collect data

Electromyography (EMG)
method of detecting and recording electrical activity of skeletal muscle

Functional Electrical Stimulation (FES)
method of activating muscle tissue by administering extrinsic electric impulses
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Chapter 1

1 Introduction

This chapter begins with a description of spinal cord injury (SCI) and the current status of SCI in Canada and the US, and the specific neuroanatomical considerations relevant to the case studies that were investigated in the thesis. The concepts of postural control and functional electrical stimulation are then introduced with a discussion of the salient points of both topics in the context of SCI. The chapter concludes with a description of the rationale for the study, an overview of the study and an outline of the thesis structure.

1.1 Spinal cord injury overview

Spinal cord injury is a neurological condition characterized by damage to the spinal cord of the central nervous system that can temporarily or permanently affect sensation, motor control, and/or autonomic functions of the body [1]. Two types of SCI are distinguished by the nature of the damage to the spinal cord: traumatic SCI, in which an acute, external physical impact causes damage to the nerves of the spinal cord, and non-traumatic SCI caused by disease, infection or the effects of a tumour on the spinal cord [2]. The level of physical impairment following SCI classifies the consequences as either tetraplegia or paraplegia; the two types are differentiated by the functional impairment of the arms in tetraplegia that is not present in paraplegia, in addition to impairments of the trunk, legs and pelvic organs [1]. Often, the neurological level of the SCI is described by the level of the spinal cord that demonstrates normal bilateral sensation and motor function [1]. The degree of impairment of an SCI is classified by the Impairment Scale defined by the American Spinal Injury Association. Letter grades A, B, C, D and E classify the completeness of the injury: ‘A’ designates a ‘complete’ injury, or an injury with no preservation of sensory or motor function below the level of the lesion; ‘B’ designates a motor complete injury, with some degree of sensation preserved below the level of the lesion; grades ‘C’ and ‘D’ have preserved sensory and motor function with progressively greater strength in key muscles below the level of injury; ‘E’ describes a normal level of both sensory and motor function [3]. Both the neurological level and the degree of injury thus determine the extent of impairment in the motor and sensory systems following SCI.

Following SCI, damage to the nerves of the spinal cord will result in some degree of temporary or permanent paralysis of the muscles and organs receiving inputs from the nerves that exit the
spinal cord below the level of injury. An injury occurring at any level of the vertebral column may affect the musculoskeletal and/or nervous systems; injuries occurring above the first lumbar vertebra (L1) may also affect the respiratory, digestive, and urinary systems due to paresis or paralysis of voluntary and involuntary components of the trunk musculature and dysfunction of abdominal organs innervated by nerves above L1. People with spinal cord injuries sustained at a level above L1 may experience simultaneous dysfunctions in respiration and trunk stability due to weakness in a relatively few number of key trunk muscles, which may include the rectus abdominis (RA), erector spinae, (ES) obliquus externus (OE), and latissimus dorsi (LD), among other muscles of the trunk. Injuries sustained at a high cervical level may also impart dysfunction on the diaphragm due to full or partial paralysis; weakness of this primary muscle of respiration combined with inadequate support for the respiratory organs results in significant respiratory deficits following SCI. The innervations of the RA, ES, OE, LD and diaphragm and other muscles relevant to respiratory function and postural control will be described in Chapter 2.

In 2010, the Rick Hansen Institute (RHI) released a publication co-authored by Angela Farry of the RHI and David Baxter of Urban Features Institute, entitled “The Incidence and Prevalence of Spinal Cord Injury in Canada: Overview and estimates based on current evidence” [2]. The report provides a glimpse at the incidence and prevalence of SCI in the Canadian context. Based on a set of stringent assumptions, Farry and Baxter estimated that in 2010, a total of 4,259 new cases of SCI occurred in Canada, and that 85,556 Canadians were living with an SCI.

The United States National Spinal Cord Injury Statistical Center reports that, since 2005, the most common category of neurological dysfunction occurring in the SCI population is incomplete tetraplegia (40.8 %), followed by complete paraplegia (21.6 %), incomplete paraplegia (21.4 %), and complete tetraplegia (15.8 %). An injury occurring above the first lumbar vertebra (L1) usually results in some degree of trunk muscle paralysis, which can have detrimental effects on both the postural control system and the respiratory system. Considering only the incidence and prevalence of cervical level injuries – those occurring within the first eight segments of the spinal cord, and resulting in tetraplegia – the number of individuals with

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According to the NSCISC, less than one percent of people who sustain an SCI experience complete recovery of neurologic function by the time they are discharged from hospital.
diminished postural control and significant respiratory compromise may include almost one half of the total SCI population, or nearly 40,000 individuals.

The absence of a standard and validated methodology for capturing and representing aetiological information to describe the myriad sequelae following SCI highlights what is likely another insufficiency: a comprehensive repository of pathological data on the SCI population – including the physical, physiological, functional, psychological, etc issues that people with SCI experience – that would enable the development of cross-disciplinary research paradigms that address multi-systemic dysfunctions that result from SCI. For example, a brief glance at the data presented above suggests that almost 40,000 people with a SCI may have both respiratory and trunk motor control deficits, since trunk impairments – i.e. dysfunctions relating to the musculature of the trunk, including muscles responsible for breathing and trunk stability – can occur in both tetraplegia and paraplegia. It is the intersection of the pathophysiology of the respiratory function and postural control systems following SCI that formed the basis of this research project.

1.2 Neuroanatomical considerations of cervical level SCI

Without sensitive physiological testing or neuroimaging measures, it is impossible to determine the contributions of various tracts of the sensory or motor pathways within the damaged spinal cord. At this time, the ASIA Impairment Scale is the accepted, standardized measure for assessing neurological function following SCI, which is capable of describing injury characteristics in terms of completeness, but not in discriminating pathology at the level of the cord. Nevertheless, any injury at the cervical level means that the trunk muscles will be impaired.

There are major differences for those injuries that are between neurological level C1 to C4 and those injuries at C5 and below. Individuals with complete injuries between C1-C4 experience extensive impairments that often lead to full dependency for performing daily tasks and in some cases (i.e. injuries between C1-C3) dependence on a mechanical ventilator for breathing due to the loss of diaphragmatic innervation [4]. For individuals with cervical level SCI, the degree of impairment in muscle function below the neurological level is of great importance as it determines the extent of the functional deficit in both postural control and respiratory function. It is important to note that in this study, only individuals with activation of the diaphragm were
studied (i.e. individuals requiring diaphragmatic pacing or mechanical ventilation were not included).

1.3 Postural control

The human body is capable of performing myriad tasks that place unique demands on the postural control system. Consider for a moment the different goals of a mother picking up her child, a tennis player rallying his opponent’s serve, a dancer performing a pirouette, or a soldier standing stock-still while guarding his post. In each of these conditions, the ultimate goal is to maintain stability for the duration of a given task, whether dynamic or static in nature. The postural control system is responsible for interpreting a frame of reference of the external environment that permits an individual to maintain stability in resting positions or while executing a movement by balancing all of the forces that act on the body [5] while concurrently integrating sensory inputs that contribute to postural control. As humans who perform most functional tasks in a vertical orientation relative to the external environment, we rely on multiple sensory references to establish our upright orientation: the vestibular, somatosensory, and visual systems provide information regarding our perception of gravity, of our bodies in relation to a support surface, and of our physical relationship to the external environment and the objects within it [6].

Shumway-Cook and Woollacott [6] define postural stability (synonymous with balance) as “the ability to control the centre of mass (COM) in relationship to the base of support.” The postural control system is said to ultimately control the COM – the center point of total body mass calculated as the weighted mean of the COM of each body segment. Research has demonstrated that perturbed individuals will regain the original COM position, instead of the original biomechanical orientation of the body, which suggests that the postural control system primarily acts on the COM [7]. The base of support (BOS) is the geometric surface area in contact with the body on a support surface (i.e. the buttocks, thighs and feet in a seated position). Finally, the centre of pressure (COP) – the position of the vertical reaction vector on the surface of a force platform – is an easily measured variable that can be used to describe the activity of the postural control system, as COM is maintained within the BOS by the movement of the COP around the COM [8].
The components of the postural control system are multiple, varied and even competing, and the coordination of information from the various systems in order to perform a particular task is complex. Figure 1 depicts the exceedingly complex interaction of the components of postural control. The musculoskeletal components include muscle properties (contractility, conductivity, excitability, and tonicity), joint range of motion, biomechanical relationship between different body segments, and spinal flexibility. The neural components of postural control include the intrinsic properties of the neural maps that connect sensation to a motor action, as well as motor processes, sensory processes, and cognitive processes [6]. The motor processes involved in postural control refer to the neuromuscular synergies, or the actions of groups of synergistic muscles that are activated together in order to efficiently and effectively perform a particular motor function. Sensory integration of the triad of sensory inputs integral to postural control (visual, vestibular and somatosensory, as described above) encompass the sensory processes influencing postural control. Through cognitive processes, the postural control system demonstrates the capacity for motor learning both adaptive and anticipatory mechanisms that permit a person to rapidly respond to unexpected postural perturbations and to prepare for expected postural perturbations. Attention, intent, and motivation are three additional cognitive processes that contribute to postural control [9]. In SCI, the major pathology affects neuromuscular status with the effect of requiring attention, intent and motivation to be maximized at a conscious level.
Detailed understanding of the anatomical correlates and the neurophysiological circuitry of human postural control is still in its infancy and much of our knowledge has been derived from behavioural studies. The description of postural control and its components come largely from studies of stance [8, 11-19]. The relatively small body of research that addresses seated postural control in able-bodied (AB) individuals has focussed on evaluating the kinematics of quiet (or static) sitting posture [20-24], and evaluating muscle synergies and/or seated postural stability components during dynamic sitting experiments (i.e. sitting perturbation paradigms) [25-30] and,
to a lesser extent, during static sitting experiments [31]. In the context of SCI, various components of seated postural control have been examined including kinematics of static sitting posture [23, 32-34], as well as muscle activity and sitting stability in multiple perturbed sitting paradigms [35-45], intervention studies [38, 46-49], as well as studies focussed on clinical measures of sitting balance [50-52]. Currently, there are two research groups investigating seated postural control in SCI, and the first comprehensive description of postural stability in quiet sitting in individuals with SCI was published recently [53], which provided insight into postural control through evaluation of the role of upper limb support in maintaining seated postural stability in static positions.

1.3.1 Implications of diminished postural control in SCI

The risk of developing pressure ulcers is elevated when lack of sensation and poor postural control contribute to extended periods of time spent sitting in compromised positions: the resultant changes in vertebral alignment, particularly lumbar hypolordosis, redistribute pressure posteriorly across the sitting surface, shifting weight support from the thighs to the buttocks, and increasing the load on the ischial tuberosities and the coccyx [54]. Individuals with a history of pressure ulcer development over these bony prominences show reduced trunk movement in the forward and backward reaching directions [41], highlighting the association between vertebral alignment and seated postural stability.

Functional tasks using the upper extremities that are performed during sitting necessitate adequate postural control of the trunk. Individuals with SCI affecting the trunk tend to develop alternative strategies for maintaining seated postural stability, which include greater recruitment of non-postural muscles, or muscles that are not typically active in AB individuals during the same tasks [35, 37, 42]. Studies of individuals with thoracic SCI performing dynamic sitting tasks, such as forward reaching, have identified the latissimus dorsi, in addition to the trapezius muscle, the pectoralis major, and the serratus anterior, as muscles contributing to stabilization of the body during movement [35, 36, 42, 43]. These patterns were especially prominent in individuals with high thoracic injuries who did not demonstrate a large degree of restoration of ES function at the lumbar level [42]. In addition, the obliquus abdominis muscle showed continual activation during reaching in individuals with injuries at all thoracic levels, preventing posterior pelvic tilt [37, 42].
1.3.2 Current postural control strategies in SCI

In many cases of complete SCI, passive seating adaptations are used to achieve a more comfortable and aesthetic posture, while attempting to increase function. In four case studies of individuals with complete cervical level SCI (C5-C6), wheelchair configuration and seating adaptations including adjustment of seat dimensions and reclining angles, use of cushions, backrests, supportive trunk braces, and foot and leg stabilisation, were successfully implemented to decrease posterior pelvic tilt, thoracic kyphosis and cervical lordosis; increase the height of the torso and achieve a more erect posture [32]. However, balance, as measured by a standard clinical test modified for people with SCI, was improved in only one case; notably, in all cases respiratory function, measured as vital capacity assessed by spirometry, was unchanged or negatively affected by the change in sitting position and posture. Granted, the abdominal binder is one physical intervention that confers positive change on respiratory function in individuals with SCI by reducing compliance of the abdominal wall [55-62], but the functional restriction it imparts on its users (and potential subsequent complications) cannot be discounted.

Despite muscular compensations and a measurable gain of compensatory or passive sitting balance, individuals with SCI still demonstrate markedly reduced limits of stability (LOS) – the area within which they can actively control their centre of pressure (COP) without losing balance [36, 37, 43]. In the rehabilitation setting, postural control is often regained by active, task-specific training and can be successful even in individuals with chronic SCI. Balance control for the purpose of functional task completion or recovery of balance from perturbation has been trained using various protocols. Extended training programs encompassing reaching and leaning tasks, kayak ergometry, and video games controlled by the user’s centre of pressure have conferred increases in maximal balance range – a measure of one’s limits of stability in the forward and backward leaning directions [51]; trunk responses to dynamic translational perturbations [38, 47]; and independent short sitting balance and completion of clinical assessments of balance on unstable sitting surfaces [46]. However, it remains to be investigated how respiratory function is affected by changes in postural control in individuals with SCI.

In terms of postural control, functional electrical stimulation has demonstrated the potential to alter posture and sitting stability. Several studies from one laboratory have explored the use of implanted stimulating electrodes as a functional intervention for postural control. These studies have shown that artificial muscle activation by implanted stimulating electrodes targeting
specific muscles of the trunk and hips can: shift the centre of pressure forward, suggesting anterior rotation of the pelvis with trunk extension [48]; alter spinal alignment and pelvic orientation, while at the same time increasing functional reach and workspace [63]; and simultaneously alter spinal alignment and pelvic orientation, increase resistance to postural perturbation, and increase respiratory function to a greater magnitude than abdominal binders [49]. While implanted electrodes impart no functional barrier, and seem to confer all of the targeted increases in postural control and respiratory function, the surgical procedure required for implantation is invasive and can pose its own health risks, such as infection, complications during or after surgery, etc.

There is a need for a multimodal intervention in the SCI population to target the postural control system and respiratory system in a functional, non-invasive way. Transcutaneous functional electrical stimulation, which is delivered by stimulating electrodes placed over the skin surface (herein referred to as surface FES), is an alternative strategy to assist recovery of motor control following SCI.

1.3.3 Using surface FES to augment respiratory function in SCI

Functional electrical stimulation is a potential solution for an intervention for postural control that preserves or even enhances respiratory function. Stimulating electrodes have been used in both the postural control context and the respiratory function context, but very little has been reported about the intersection of these two functional systems. In the respiratory function context, positive results have been established using two different methods of electrical stimulation: a priori abdominal muscle training [64, 65], involving extended periods of stimulation-based respiratory muscle training prior to respiratory testing; and direct-effect testing [66], involving no prior respiratory muscle training and participants who were naïve to abdominal muscle stimulation. Each study employed surface electrodes with similar stimulation

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ii The training received by participants in Cheng et al. [64] and Lee et al. [65] lasted four weeks, with 5-7 days per week of training; both studies participants’ received transcutaneous electrical stimulation of the abdominal muscles in a 4 s on/off paradigm (pulse width 300 μs) that elicited muscle contractions of the abdominal muscles with a stimulation frequency of 30 Hz or 50 Hz (respectively) and a pulse amplitude of 100 mA.
parameters used during respiratory function testing, with the exception of one study that did not use a stimulation protocol during testing: Cheng et al. [64] tested only the outcome of the extended training protocol, not the effect of abdominal muscle stimulation during respiratory function testing. Among these studies, improvements in respiratory function included increases in forced expiratory measures such as forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) [64-66]. Few studies have investigated the effect of continuous electrical stimulation on respiratory function; two groups of investigators that did use a continuous stimulation signal reported gains in thoracic and abdominal volume (correlated to tidal volume [V_T]), and measurable gains in V_T [67, 68]. It should be noted that participants in the latter two studies were tested in the supine position, which may have already conferred a mechanical advantage on the diaphragm for improving respiration (see section 2.1 for detailed explanation of effects of posture on respiratory function in SCI); the measured respiratory gains should therefore be interpreted with caution. Tables 1 and 2 present a summary of the studies reviewed in this section, including research design, participant characteristics (Table 1), methodology for using stimulation, and major findings (Table 2).

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**iii** In the studies reviewed for this paper, stimulation parameters ranged as follows: stimulation train: 4-8 s; stimulation frequency: 30-50 Hz; pulse width: 200-350 μs; maximum pulse amplitude: 100-120 mA, with the exception of the study by Lee et al. [65] that tested the single participant at a stimulation intensity of 225 mA.

**iv** Both studies reported the same stimulation parameters: 1s stimulation train; 50 Hz stimulation frequency; 350 μs pulse width. Neither group reported the precise intensity of stimulation; Sorli et al. [67] stated that “the level of electrical stimulation was adjusted individually to the level that noticeable contractions were observed without causing discomfort to the subjects”, while Stanic et al. [68] stated that the minima and maxima were approximately 20-100 mA.
Table 1. Summary of studies using FES to augment respiratory function in individuals with SCI: experimental paradigm and participant characteristics

<table>
<thead>
<tr>
<th>Study authors</th>
<th>Method</th>
<th>Sample size</th>
<th>Groups</th>
<th>Level of injury</th>
<th>ASIA score</th>
<th>Time post injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cheng et al. [64]</td>
<td>RT with control group</td>
<td>n=26</td>
<td>FES group: n=13</td>
<td>C4-C7</td>
<td>A-B</td>
<td>0.25y</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>control group: n=13</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lee et al. [65]</td>
<td>Single case study</td>
<td>n=1</td>
<td>FES intervention</td>
<td>C4</td>
<td>C</td>
<td>0.68y</td>
</tr>
<tr>
<td>Langbein et al.[66]</td>
<td>Quasi-experimental*</td>
<td>n=10</td>
<td>FES intervention</td>
<td>C5-T7</td>
<td>A-D</td>
<td>1-24y</td>
</tr>
<tr>
<td>Sorli et al. [67]</td>
<td>Single case study</td>
<td>n=1</td>
<td>FES intervention</td>
<td>C6</td>
<td>A</td>
<td>0.75y</td>
</tr>
<tr>
<td>Stanic et al. [68]</td>
<td>Quasi-experimental*</td>
<td>n=5</td>
<td>FES intervention</td>
<td>C4-C7</td>
<td>B and D</td>
<td>5-6y</td>
</tr>
</tbody>
</table>

Quasi-experimental* - a single group intervention paradigm where each participant serves as their own control.
Table 2. Summary of studies using FES to augment respiratory function in individuals with SCI: methodology and results

<table>
<thead>
<tr>
<th>Study authors</th>
<th>FES training</th>
<th>Testing protocol</th>
<th>FES parameters</th>
<th>Electrode placement (no. of electrodes)</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cheng et al. [64]</td>
<td>0.5 hr/d, 5 d/wk x 4 wk</td>
<td>Supine spirometry without FES</td>
<td>Training only: 4 s, 300 μs, 30 Hz, 100 mA</td>
<td>Abdominal wall, 3 cm from umbilicus; clavicular portion of pectoralis muscle (NR)</td>
<td>FES vs. control group: +0.44 L FVC (p=0.003) +0.26 L/s FEV1 (p=0.011)</td>
</tr>
<tr>
<td>Lee et al. [65]</td>
<td>0.5 hr/d x 4 wk</td>
<td>Seated spirometry with and without FES</td>
<td>Training: 4 s, 50 Hz, 100 mA Testing: 1 s, 200 μs, 50 Hz, 225 mA</td>
<td>Training: anterior abdomen (4) Testing: posterolateral abdomen</td>
<td>† Baseline vs. post-training: without FES: +0.4 L FVC, +0.3 L/s FEV1 with FES: +0.7 FVC, +0.7 L/s FEV1</td>
</tr>
<tr>
<td>Langbein et al. [66]</td>
<td>None</td>
<td>Seated spirometry with FES by manual trigger</td>
<td>8 s, 250 μs, 50 Hz, 100 mA</td>
<td>Upper/lower abdomen (8)</td>
<td>FES vs. no FES: +0.41 L FVC (p=0.006) +0.26 L/s FEV1 (p=0.019)</td>
</tr>
<tr>
<td>Sorli et al. [67]</td>
<td>None</td>
<td>Quiet breathing in supine</td>
<td>1 s, 350 μs, 50 Hz, NR</td>
<td>Along midline 6 cm above 5 cm below umbilicus (2)</td>
<td>† FES vs. no FES: +0.45 L volume of thorax*</td>
</tr>
<tr>
<td>Stanić et al. [68]</td>
<td>None</td>
<td>Quiet breathing in supine</td>
<td>1 s, 350 μs, 50 Hz, NR</td>
<td>Along midline below costal margin, above symphysis bone (2)</td>
<td>FES vs. no FES: +0.22 L VT (p&lt;0.05), +4.50 L/min VE (p&lt;0.05), +1.5 min⁻¹ RR (not significant)</td>
</tr>
</tbody>
</table>

FES parameters correspond to: stimulation train (s), pulse width (μs), frequency (Hz), pulse amplitude (mA); NR – not reported; * - authors inferred VT from kinematic analysis of optical marker coordinates on the torso; † - statistical tests were not performed in single-case studies
1.4 Functional electrical stimulation (FES)

Baker et al. [69] have described in detail neuromuscular electrical stimulation for the purposes of many therapeutic applications. In this research, the electrical stimulation is delivered via transcutaneous stimulating electrodes in a method known as surface FES. The objective of FES is to elicit a contraction of muscle fibres via an extrinsic electrical stimulus to achieve a targeted functional outcome [70], which is described as a neuroprosthesis when used as a therapeutic approach [70-74].

Muscle tissue, with its innate electrical properties, is an ideal medium for receiving FES. Artificial activation of nerve and muscle cells is possible due to the relative electronegativity of the tissue versus the extracellular environment, separated by the cell membrane. Application of an electrical stimulus to an area of the cell membrane causes localized depolarization, which then spreads along a motor nerve axon as an action potential, ultimately causing contraction of the muscle fibres that it innervates.

In surface FES, two stimulating electrodes (one anode, one cathode) are placed on the surface of the skin. When stimulation is initiated, electrical current flows from positive electrode (anode) to negative electrode (cathode), the latter of which is the site of initial depolarization for all motor nerve axons in the proximity of the electrodes. Propagation of an action potential (if the threshold for depolarization has been reached) occurs in both directions along the motor nerve axon. Activation of a motor nerve axon (and thus contraction of the motor units supplied by the axon) depends on its proximity to the stimulating electrodes and the amount of current that reaches the axon. There are multiple relevant factors important for achieving a functional muscle contraction that are incorporated in a neuroprosthetic approach to delivering FES.

1.4.1 Methodological considerations for surface FES

There are several factors that precede an optimal muscle contraction via surface FES. Success of neural excitation by an extrinsic, transcutaneous electrical current is dependent on the impedance of the tissues at the site of stimulation; the precise placement and size of electrodes; as well as the delivery settings of the electrical stimulus.

Surface FES presents a unique challenge, since the electrical current must pass through a number of layers of tissue before activating the muscle. Electrical current will always flow through the
path of least resistance: tissues with low impedance will permit current to flow more easily than tissues with high impedance. A tissue will have better conductivity if it has lower impedance, which is approximately equivalent to the inverse of its water content; for example, the epidermis has a low water content translating to one of the highest relative impedances of all bodily tissues, and is thus a relatively poor conductor [75]. Adipose tissue has similar water content to skin and bony structures with relatively high impedance and is likewise a poor conductor. On the other hand, muscle tissue has high water content and is a good conductor with low impedance. The same principle applies to current flowing through high and low resistance axons. Axon size is inversely related to its relative impedance, and as such, more of the stimulating current will flow through axons with the largest diameters. Again, skin, fat, bone, and other tissues located between the electrode site and the target motor nerve axon will affect the amount of current available for neural excitation.

The first consideration for FES electrode placement concerns the orientation of the target muscle fibres. Conductivity of a muscle is altered by the orientation of stimulating electrodes: signal integrity is degraded when the flow of current is directed perpendicular to the target muscle fibres, rather than along the length of the muscle fibre [76, 77]. Thus, it is important that electrode pairs are aligned in tandem parallel to the direction of the muscle fibres.

Since relative impedance plays a role in the amount of current flowing through different tissues between electrodes, current density is not quantified as the total amount of current between electrodes, but rather as the amount of current flow per unit area. Neural excitation will only occur when a sufficient amount of current is available at the depolarization site of the motor nerve axon. Since nerves are relatively deep tissue, the current must be available to the tissues below the surface (skin and fat). The placement of electrodes in a close proximity results in a pathway of least resistance restricted to surface tissues; a pathway of least resistance that passes through deeper tissues necessitates that electrodes are placed further apart.

Current density is further dependent on the size of electrode: under constant current stimulation settings, the total current remains the same regardless of size of electrode. Thus, a smaller electrode will concentrate current density over a smaller surface area, which has certain implications for application. Specificity of the stimulation target can be increased by placing a small electrode in as close proximity to the target nerve or motor point as possible. At the same time, high current density concentrated on the skin surface in contact with the electrode is likely
to be perceived as a painful stimulus, reducing stimulation tolerance for the individual. Furthermore, a muscle with multiple innervations at a number of motor points will not be efficiently stimulated using this method; this becomes especially important in muscles such as erector spinae, which is segmentally innervated along the length of its fibres. Therefore, the size of a given muscle, its neuromuscular physiology, and individual variation must be considered when determining appropriate electrode size.

The best stimulation paradigm is one that optimizes the desired motor function and is comfortable for the individual, an outcome that can be achieved by carefully balancing the deliver settings for the electrical stimulus. The following is a list of parameters that are important in producing the optimal paradigm: current amplitude, phase and/or pulse duration, pulse frequency, stimulation waveform, duration of the stimulus train and time between stimulus trains, ramp times on either side of the stimulus train, number of stimulation channels, channel synchronization, and triggering modes (see Baker et al. [69] for detailed guidelines for each of these parameters in designing a stimulation paradigm).

When determining the current amplitude required for generating a muscle contraction using surface FES, the pain threshold will constitute a limiting factor. In individuals with intact sensory function, the motor threshold is often reached before the pain threshold; in this case, the current amplitude is increased to maximize the functional contraction of the target muscle, without exceeding the pain threshold. There are two important caveats to consider when determining current amplitudes for individuals with SCI. The first is the possibility that the spinothalamic tract has been damaged by the SCI; in this case, the pain threshold may be altered, and individuals may tolerate higher (increased pain threshold) or lower (decreased pain threshold) current amplitudes or not perceive pain at all in the case of a complete loss of pain sensation. The second is the risk of developing autonomic dysreflexia (AD)\(^\text{v}\) during application of surface FES.

\(^{v}\) AD is classified as collection of signs and/or symptoms exhibited by an individual with an SCI usually at and above the sixth thoracic vertebra (T6) in response to stimuli that may or may not be noxious in nature; an episode of AD is defined as an increase of more than 20mmHg in systolic blood pressure, which may be accompanied by headache, flushing, piloerection, nasal congestion, perspiration above the level of injury, vasoconstriction below the level of injury, and dysrhythmias [78].
There have been a few published accounts of instances of AD occurring with the application of surface FES [79, 80] making assessment of risk of AD required on an individual basis.

1.4.2 Feasibility of using surface FES in SCI

Following SCI, the integrity of the neuromuscular junction is compromised when the pathway connecting the motor cortex of the brain to the motor effector (muscle) is damaged. At the level of the SCI, the spinal motor neuron is damaged, while below the level of injury the fibres of the LMN tend to remain intact. At and below the level of injury, the LMN no longer receives inputs (neuronal impulses) from the motor cortex and consequently generates either no output (nerve impulses) or generates continuous or intermittent output along the nerve tissue that innervates the target muscle. In cases of SCI where the target muscle has been denervated by damage to the LMN or when significant decreases in muscle volume occur from lack of use (disuse atrophy), it may be challenging to use surface FES. In most cases though, it is possible to artificially activate muscles that cannot be voluntarily contracted due to paralysis following SCI.

1.5 Rationale for this study

The need is paramount for a functional, non-invasive multimodal intervention that targets the postural control system after SCI, while maintaining at least the same level of function of the respiratory system. Using a sitting balance neuroprosthesis that applies surface FES technology to select trunk muscles, it will be possible to investigate the relationship between sitting balance and breathing.

By investigating multiple single-case studies, it will be possible to individualise surface FES paradigms for each participant according to the level of physiological function of impaired muscle groups. The purpose of this study is to investigate the feasibility of using surface FES intervention to alter sitting position and posture, and to assess concurrent change in respiratory function.

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\[ vi \] In incomplete injuries, impulses may be received and generated but at reduced capacity due to abnormal conductance.
1.5.1 Rationale for surface FES paradigm

In order to construct an informative surface FES paradigm, several factors had to be considered including: 1) the biomechanical function of individual muscles; 2) the role of individual muscles in both respiratory function and postural control, as evidenced by the literature; 3) and, the feasibility of stimulating a given muscle.

An underlying objective of this research was to alter sitting posture by means of increasing trunk extension, measurable by a change in sagittal spinal alignment. Integrity of the spine during upright sitting has been reported to be achieved by activation of antagonistic flexor-extensor trunk muscles, a mechanism for spine stabilization via intra-abdominal pressure [81-83]. Thus, selection of muscles was considered based on the criterion that pairs of muscles should have opposing biomechanical functions, specifically antagonistic trunk flexor-extensor actions.

Individual muscles were assessed based on evidence in the literature for their role in respiratory function and/or postural control in SCI. Triolo and colleagues [48, 49, 63] have used an implanted FES neuroprosthesis targeting the trunk extensors (ES at the lumbar level; gluteus maximus and quadratus lumborum were also stimulated) of individuals with SCI to demonstrate increased forced expiratory lung function, improved sagittal spinal alignment, and increases in functional workspace. In the respiratory function context, several studies have used surface FES targeting either RA (or OE in the case of the protocol of Lee et al.[65]) to increase measures of forced expiratory lung function and quiet breathing respiratory function [64-68]. A number of studies using electromyography (EMG) to investigate altered muscle synergies following SCI have indicated that LD is increasingly recruited during posturally demanding tasks [35, 37, 42, 43] and that LD plays a role in expiratory function in individuals with cervical level SCI [84]. While other muscles have been studied in this context, many of the accessory muscles of respiration are not otherwise involved in postural control at the level of the trunk (i.e. trapezius) and would not be suitable to include.

The final consideration for construction of the surface FES paradigm was the feasibility of stimulating selected muscles using transcutaneous electrodes. Each of the four aforementioned muscles – RA, OE, LD and ES – is anatomically located at the superficial aspect of the anterior, lateral or posterior surface of the trunk. Furthermore, the location of all intended stimulation sites on the trunk permitted a modest stimulation protocol, ensuring maximum comfort for participants.
According to the criteria outlined above, RA and ES were the ideal pair of antagonistic muscles to test. For comparison, an alternative muscle pair was chosen with similar biomechanical functions to RA and ES using OE and LD. Thus, the surface FES paradigm was designed to test two alternative pairs of muscles: 1) RA and ES on the anteroposterior surface of the trunk (herein referred to as the AP-FES condition), and 2) OE and LD on the lateral surface of the trunk (herein referred to as the ML-FES condition) – see Figure 2.

Figure 2. Diagram of superficial trunk musculature targeted by surface FES. Muscles that are labelled are latissimus dorsi (LD), obliquus externus (OE); thoracolumbar fascia, the membrane overlying erector spinae; and rectus sheath, the membrane overlying rectus abdominis. FES conditions AP-FES and ML-FES describe simultaneous surface FES of RA and ES, and OE and LD, respectively. Image adapted from 20th online edition of Gray (1918) Anatomy of the Human Body [85].
1.6 Study overview

This study was part of a larger research programme investigating the feasibility of using a surface neuroprosthesis to maintain sitting balance in individuals with compromised trunk control due to SCI and the potential effects of trunk stimulation on respiratory function.

The objective of this study was two-fold, given the different populations for investigation: 1) in AB individuals, determine whether quantifiable changes occurred in postural control and respiratory function between two postures: upright and slouch sitting; and 2) in individuals with SCI, determine whether surface FES induces quantifiable changes in postural control and respiratory function between two conditions: unsupported sitting and sitting with FES.

1.7 Thesis outline

This thesis will focus on the consequences of SCI on the postural control and respiratory systems in Chapter 2. Chapter 3 states the objectives and hypotheses of this study. A concise description of the experimental paradigm used in this study will follow, with emphasis on the modifications for participants with SCI in Chapter 4. Chapter 5 presents the results of the AB group, followed by the results of each SCI case study. The discussion is divided in the same manner as the previous section in Chapter 6. Chapter 7 summarizes the findings of this study, draws what I hope to be profound and enlightening conclusions about this research, and provides recommendations for progressive work.
Chapter 2

2  Literature review

The purpose of this chapter is to introduce the topics of respiratory mechanics and postural control, highlighting the dysfunctions in these systems that occur in SCI. This chapter focuses on the breathing, stability, and postural challenges that individuals with SCI experience related to upright sitting.

2.1  Respiratory function following SCI

Beyond acute injury survival, respiratory complications represent a major health threat to people with SCI, causing significant morbidity and mortality in the SCI population [86, 87]. Acute respiratory failure, pneumonia and other pulmonary infections, and atelectasis are the most common respiratory issues following SCI, which disproportionately affect people with tetraplegia [88]. Compared to the general population, the rate of death due to such secondary medical complications occurs at significantly higher rates in individuals with SCI: incidences as high as 125 times greater have been reported for individuals with SCI under age 40 when compared to a normal population sample [86]. Together, respiratory complications contribute significantly to morbidity, mortality, and increasing health care costs in the SCI population [87-90], indicating a healthcare and economic priority.

Many respiratory complications are related to the changes in respiratory mechanics that follow an SCI. The degree of functional impairment of the respiratory system depends on the spinal level of the neurological deficit; the time since injury; and the residual function of spared (or partially-affected) respiratory muscles [91]. Respiratory deficits tend to be more severe as the level of injury occurs more cephalad: individuals with tetraplegia experience greater breathing impairments than those with lower level injuries [92-94]. Some recovery of respiratory function following SCI typically occurs within one year of injury: once an individual is medically stable, some spontaneous recovery of respiratory function can occur [95-97], which may be facilitated by the resolution of spinal shock [98]. Few studies provide evidence of further improvements in respiratory function, as measured by spirometry, during rehabilitation: while intensive resistance training of either inspiratory or expiratory muscles does significantly increase respiratory function, including FVC and FEV₁ over time, the increases are not different than those achieved
with conventional rehabilitation therapy [99, 100]. Different combinations of partially functional primary and/or secondary respiratory muscles impart a range of dysfunctional breathing patterns on individuals with SCI [94, 97, 101-106]. Ultimately, the degree of paresis or paralysis of the muscles of the trunk – including the primary respiratory muscle, the diaphragm – is the final determinant in the extent of respiratory deficit experienced post-SCI.

In the SCI population, respiratory dysfunction is susceptible to exacerbation by an effect of posture. Individuals with cervical cord injuries already experiencing the greatest deficits in respiration are vulnerable to a positional phenomenon by which their respiratory function is reduced in the upright (sitting or standing) versus the supine position [55, 56]. Compared to AB individuals, individuals with injuries as low as the tenth thoracic vertebrae (T10) demonstrate reduced respiratory function, as measured by FVC, FEV\textsubscript{1} and other lung volumes, when measured in either sitting or supine position [94, 107]. This phenomenon occurs due to paralysis of the abdominal muscles: the abdomen distends due to increased compliance of the abdominal wall and the force of gravity acting on the contents of the abdominal cavity, resulting in the loss of the leverage point for contraction of the diaphragm [88].

Able-bodied individuals who assume a slouched posture (with significant posterior pelvic tilt and kyphotic spine) while sitting can significantly reduce respiratory function compared with normal (trunk upright, lumbar-supported) sitting in terms of both forced expiratory capacity [108] and measures of quiet breathing [109] – $V_T$, respiratory rate (RR), and minute ventilation ($V_E$). The values of clinical standardized spirometry reported for AB individuals during slump sitting lie within the range of values reported for individuals with SCI while sitting (Table 3). The implication that fully innervated, uncompromised trunk musculature is insufficient to ameliorate respiratory deficits during sitting provides further evidence for the postural dependence of respiratory function.
Table 3. Summary of spirometry values for AB and SCI populations

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Sample size</th>
<th>Condition</th>
<th>FVC mean ±1 SD</th>
<th>FEV₁ mean ±1 SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lin et al.[108]</td>
<td>Able-bodied</td>
<td>n=40</td>
<td>Normal sitting</td>
<td>4.13 ±1.01 L</td>
<td>3.31 ±0.90 L</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Slump sitting</td>
<td>3.96 ±1.03 L</td>
<td>3.18 ±0.94 L</td>
</tr>
<tr>
<td>Baydur et al. [94]</td>
<td>Tetraplegia</td>
<td>n=31</td>
<td>Seated</td>
<td>2.70 ±0.91 L</td>
<td>2.36 ±0.74 L</td>
</tr>
<tr>
<td></td>
<td>Paraplegia</td>
<td>n=43</td>
<td>Seated</td>
<td>4.09 ±0.96 L</td>
<td>3.40 ±0.75 L</td>
</tr>
<tr>
<td>Linn et al.[93]</td>
<td>Tetraplegia, complete</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>High (C2-C5)</td>
<td>n=26</td>
<td>Seated</td>
<td>49 ±13 % Pred</td>
<td>53 ±14 % Pred</td>
</tr>
<tr>
<td></td>
<td>Low (C6-C8)</td>
<td>n=9</td>
<td>Seated</td>
<td>62 ±10 % Pred</td>
<td>69 ±10 % Pred</td>
</tr>
<tr>
<td></td>
<td>Paraplegia, complete</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>High (T1-T7)</td>
<td>n=28</td>
<td>Seated</td>
<td>81 ±13 % Pred</td>
<td>85 ±15 % Pred</td>
</tr>
<tr>
<td></td>
<td>Low (T8-L5)</td>
<td>n=13</td>
<td>Seated</td>
<td>95 ±11 % Pred</td>
<td>97 ±8 % Pred</td>
</tr>
</tbody>
</table>

FVC – forced vital capacity; FEV₁ – forced expiratory volume in one second; 1 SD – one standard deviation; L – litres; % Pred – percentage of predicted values; all spirometric reference values calculated using equations from Hankinson et al.[110]

2.1.1 Respiratory mechanics

At rest, the process of quiet breathing consists of two phases: active inspiration and passive expiration. During inspiration, lung expansion occurs when the pressure in the thoracic cavity drops; negative pressure in the thorax is created by contraction of the diaphragm against the abdominal contents, and by in-phase activity of the external intercostal muscles, and to a lesser degree the scalene muscles, which act to elevate the rib cage and effectively increase the volume of the thoracic cavity. During the expiratory phase of quiet breathing, the diaphragm and accessory respiratory muscles relax; elastic recoil causes the lungs to deflate passively. Although not actively recruited until the work of respiration increases, the abdominal muscles have an important mechanical role in quiet breathing: proper diaphragmatic function is dependent on the abdominal contents acting as a leverage point to assist in rib cage expansion during inspiration [111, 112], and during expiration, intra-abdominal pressure (IAP) facilitates emptying of the lung by reversing the pressure inside the thoracic cavity [113]. The activities of RA and OE, which contribute to the stiffness of the abdominal wall and the control of IAP [114, 115], play a passive but important role in performance of quiet breathing respiratory function.
As a cycle, inspiration and exhalation determine the volume of air entering the lungs and the ventilatory component of oxygen exchange. RR – the number of breaths taken per minute, and tidal $V_T$ – the volume of air moved during a single cycle of respiration (one inspiration and one expiration), are the components of $V_E$. Minute ventilation is a rate describing the volume of gas displaced by the lungs in one minute; it is inversely related to the level of carbon dioxide in the blood. Minute ventilation is optimized in the body to maintain physiologic homeostasis; thus, a change in either RR or $V_T$ will produce subsequent changes in the other that facilitate a return to normal $V_E$ [116]. In AB individuals, $V_E$ compensation has been achieved by increasing $V_T$, not RR, when respiratory function is adversely affected by a slouch sitting posture [109].

Any injury to the cervical spinal cord has tremendous potential to compromise breathing. At the cervical level, the function of any of the primary inspiratory muscles and all of the expiratory muscles would be affected (see Table 4); post-SCI, the recruitment of accessory respiratory and non-respiratory muscles might be necessary to perform quiet breathing. Examination of recordings by electromyography (EMG) in individuals with various levels of tetraplegia have shown reduced or absent muscle activity, or irregular spasticity, in the intercostales, scalenus, RA, and OE [105, 117], and recruitment of Sternocleidomastoid [102, 118], trapezius [102, 105, 118], and pectoralis major [84, 105] during quiet breathing, which contribute to paradoxical breathing patterns. The resultant breathing patterns are characterized by rapid, shallow breathing – i.e. increased RR and decreased $V_T$, relative to AB controls – that persist in individuals with SCI during the first year following injury and throughout the chronic stages of injury [96, 104].

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$vii$ Respiratory rate, the number of breaths taken per minute, and tidal volume, the volume of air moved during a single cycle of respiration (one inspiration, one expiration) are the components of minute ventilation – a rate describing the volume of gas displaced by the lungs in one minute.
### Table 4. Muscles of respiration and their innervations

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Function</th>
<th>Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaphragm</td>
<td>Primary inspiratory</td>
<td>C3-C5</td>
</tr>
<tr>
<td>Scalenes (all parts)</td>
<td></td>
<td>C3-C8</td>
</tr>
<tr>
<td>Intercostales (external)</td>
<td></td>
<td>T1-T11</td>
</tr>
<tr>
<td>Sternocleidomastoid</td>
<td>Accessory inspiratory</td>
<td>CN-XI</td>
</tr>
<tr>
<td>Trapezius</td>
<td></td>
<td>CN-XI</td>
</tr>
<tr>
<td>Pectoralis major</td>
<td></td>
<td>C5-T1</td>
</tr>
<tr>
<td>Intercostales (internal)</td>
<td>Expiratory</td>
<td>T1-T12</td>
</tr>
<tr>
<td>Obliquus abdominis externus</td>
<td></td>
<td>T6-L1</td>
</tr>
<tr>
<td>Rectus abdominis</td>
<td></td>
<td>T7-T12</td>
</tr>
</tbody>
</table>

C – cervical, T – thoracic, CN – cranial nerve

### 2.2 Postural control following SCI

Following an SCI above the first lumbar vertebra (L1), the musculature of the trunk that provides support to the spine is compromised by some degree of paralysis and atrophy. The subsequent functional deficits of the trunk result in poor postural control during sitting. The inability to stabilise the trunk while sitting is a significant problem for individuals with a cervical or thoracic-level injury who are wheelchair-bound and consequently perform most daily activities from a seated position [4]. Varying degrees of impairment, dependent on the level and severity of SCI, of the postural control system is evident in the outcome measures that are diminished relative to AB individuals: seated postural stability, trunk muscle activity, sitting posture. Individuals with SCI may attempt to compensate for impaired postural control by recruiting non-postural muscles to substitute for compromised muscular function. This section discusses postural control in AB individuals and diminished postural control in individuals with SCI; below, Table 5 presents a summary of the relevant trunk musculature that is discussed in this chapter.
### Table 5. Select postural muscles of the trunk and their innervations

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Function</th>
<th>Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectus abdominis</td>
<td>Abdominal wall stiffness</td>
<td>T7-T12</td>
</tr>
<tr>
<td>Obliquus externus</td>
<td></td>
<td>T6-L1</td>
</tr>
<tr>
<td>Trapezius</td>
<td></td>
<td>CN-XI</td>
</tr>
<tr>
<td>Serratus anterior</td>
<td>Upper limb and/or scapular manipulation</td>
<td>C5-C7</td>
</tr>
<tr>
<td>Pectoralis major</td>
<td></td>
<td>C5-T1</td>
</tr>
<tr>
<td>Latissimus dorsi *</td>
<td></td>
<td>C6-C8</td>
</tr>
<tr>
<td>Erector spinae</td>
<td>Trunk extension</td>
<td>Segmental innervation by posterior branch of spinal nerve</td>
</tr>
<tr>
<td>Multifidus</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

C – cervical, T – thoracic, CN – cranial nerve; * - trunk extension and lateral flexion

#### 2.2.1 Seated postural stability

Seated postural stability is the capacity of the postural control system to maintain upright sitting postures, during performance of other movements by optimizing the position of the spine and the associated muscle activity. Prieto et al. [12] provide a comprehensive, but not exhaustive, list of centre of pressure (COP)-based measures to evaluate postural steadiness in the time and frequency domains, such as mean COP distance (MDIST) and mean COP velocity (MVELO). Some of these measures have been linked to particular aspects of postural control: for a given level of stability (MDIST) there is an associated level of neurological regulatory activity (MVELO) from the postural control system [14, 119]. The relationship between MDIST and MVELO is quantifiable using a hybrid measure, mean COP frequency (MFREQ), that relates the area and the total distance of the COP trajectory [12].

The methodology of using COP-based measures derived from forces to quantify postural steadiness in standing has been adopted for the seated postural stability context. In SCI, seated postural stability has been evaluated using different paradigms and with various COP-based measures. Seelen et al. [42] used a reaching task to assess changes in seated postural stability over the rehabilitation phase. Individuals in two groups, high level injuries (T2-T8) and low level injuries (T9-T12), both showed increases in maximum COP displacement over the course of the rehabilitation period [42]. At a single time point, Seelen et al. [36] contrasted AB versus SCI individuals’ seated postural stability: individuals with SCI had less stability than AB individuals,
as demonstrated by a reduced maximum COP displacement, with higher levels of injury corresponding to increasing levels of stability impairment. Representative traces of COP displacement at a given reaching distance showed that the trajectory of the COP displacement path was much smoother (less variation in the plane of the leaning direction) for an AB participant within and across trials, while an individual with T4 SCI showed the largest magnitude of variation in the COP trajectory within single trials and for all trials at a given reaching distance [36]. The limits of stability (LOS) in sitting have been evaluated in both the AB and SCI populations using multi-directional leaning or reaching paradigms.[26, 120] In comparison, individuals with SCI (T12 and above) achieve lower LOS in all reaching directions, indicated by lower COP displacements than those achieved by AB individuals [120].

In quiet sitting, Grigorenko et al. used COP-based measures to evaluate seated postural stability in individuals with SCI (T2-T11) at baseline and post-kayak training compared to a control group of AB individuals [47]. At baseline, standard deviation (SD) of the frontal plane COP displacement was greater in SCI individuals and decreased with kayak training [47]. Compared to the control group, MVELO and mean frequency of COP acceleration in both frontal (mediolateral, ML) and sagittal (anteroposterior, AP) planes were reduced in individuals with SCI at baseline; kayak training did not ameliorate the differences in COP-based measures between controls and SCI individuals [47]. Grangeon et al. showed that individuals with SCI (C3-L1) had poorer seated postural stability compared to AB controls in quiet sitting positions with and without support from the upper limbs [53]. Seated postural stability deficits in the SCI group were indicated by increased MDIST, MVELO and sway area in the time domain, and reduced values of COP-based measures in the frequency domain compared to the AB group; within the SCI group, time and frequency domain measures increased in unsupported sitting positions. Separate AP and ML directional components of each measure were less consistently different than the composite measures [53].

As the examination of seated postural stability in SCI increases, the measures by which investigators quantify sitting balance must be carefully constructed and defined such that they are appropriate for this population. COP-based measures are computed as directionless composite values from the anteroposterior (AP) and mediolateral (ML) components of the planar trajectory of the vertical reaction force over the sitting surface; the AP and ML components of COP displacement (and many other measures in the time and frequency domain) can be assessed
independently. Assessment of AP and ML components separately, which describe only the true directional components, is only appropriate when the orientation of the body segment in contact with the sitting surface is constrained to the axis of the force platform [12]. Patients with SCI have compromised control of the limbs and trunk and may not be able to constrain the placement of the hips and thighs along the axes of the force platform; thus, the composite COP measures may more accurately reflect the dynamics of seated postural stability in the natural sitting position of an individual with SCI.

2.2.2 Trunk muscle activity

In neurologically intact individuals, lumbar spine stability is achieved by the normal function of the core trunk muscles. A neutral static sitting position – having an upright posture without slouching or slumping – is associated with greater EMG activity in the lumbar extensor muscles and the internal oblique muscles, with no difference in EMG activity of the thoracic extensor muscles nor the medial or lateral superficial abdominal muscles when compared to slouch sitting; this position also allows relaxation of the thoracic extensor muscles compared to upright sitting with extension of the thoracic spine [22], while still engaging the lumbar extensor muscles [22, 121]. Activity of the thoracic extensor and abdominal muscles has been quantified by measuring the EMG activity of the trunk flexor-extensor muscles, including ES and multifidus spinae, and the muscles of the medial and lateral abdominal wall, including RA and obliquus abdominis, during static sitting,[27] during voluntary trunk manoeuvres [81, 82], and sudden expected or unexpected trunk perturbations [25, 27, 30, 122]. Further, the co-activation of these muscles provides the necessary support for the spine during wheeling: at both low and high speeds, RA, obliquus abdominis, and the trunk extensors longissimus thoracis, iliocostalis and multifidus were actively recruited during the push cycle in AB individuals [123].

The challenge of maintaining dynamic sitting balance following SCI is often achieved by evolving a novel pattern of activations of muscles where innervations and functions were spared above the level of injury. In reaching tasks, individuals with SCI tend to have lower EMG activity of lumbar ES, serratus anterior, and RA than AB individuals [37, 43, 124], while individuals with progressively higher levels of injury tend to recruit LD, pectoralis major, and trapezius to a greater degree than their non-injured counterparts and individuals with lower level injuries [37, 42, 43]. Even in static sitting, individuals with SCI recruit muscles of the upper body such as pectoralis major to maintain sitting balance in different seating configurations
This pattern of non-postural muscle recruitment for maintaining sitting balance during static or dynamic sitting is problematic: firstly, in that the muscles being used are ill-suited to providing adequate postural control. Secondly, if non-postural muscles are used to substitute for postural compensation, the concurrent normal function and biomechanical action of the non-postural muscles is likely to be compromised.

2.2.3 Sitting posture

Optimal ergonomic sitting posture is often debated, yet a number of studies have substantiated claims that maintaining lumbar lordosis relays benefits through the entire length of the spine. A sitting posture that fosters lumbar lordosis will reduce intradiscal pressure [20, 126], and disc degeneration [127], reduce the risk of lower back pain [128], and prevent injury [81]. Compared to slouching, an upright sitting posture with neutral lumbar lordosis and a relaxed thoracic spine permits the greatest anterior rotation of the pelvis [22, 121], [21].

Compared to AB individuals, individuals with SCI above L1 tend to assume a suboptimal posture in various sitting positions. Individuals with SCI sitting with legs extended and without trunk support have been reported to demonstrate hyperkyphosis of the thoracic spine, reduced lumbar kyphosis, and posterior pelvic tilt [34]; even sitting in a body positioning chair, individuals with SCI showed a significant 15° decrease in pelvic angle, indicating posterior tilt, compared to AB people sitting in the same chair [23]. Loss of voluntary postural control thus leads to a sitting posture – with its characteristic “C”-shape achieved by hyperkyphosis of the thoracic spine, flattening of the lumbar spine and posterior tilting of the pelvis – that is problematic in terms of optimal respiratory function, adequate functional workspace, aesthetic posture, and risk of developing pressure ulcers.

2.3 Model of trunk muscle function in SCI

The objectives of this project specifically investigate trunk function in the context of SCI above the first lumbar vertebra (L1). A model of the neuroanatomy of the trunk musculature demonstrates and distinguishes the specific groups of muscles that would be affected at various levels of a SCI (Figure 3). It is important to note that ES is a segmentally innervated muscle that runs the length of the spine from the base of the skull to the pelvis, and that injury at any level above L1 that affects the motor system will impart motor deficits on the muscle fibres of ES that
are innervated below the level of injury (refer back to Tables 4 and 5 for a description of the innervations of muscles important for respiratory function and postural control, respectively).

Figure 3. Neuroanatomy of the postural and respiratory muscles of the trunk. Image adapted from: Myotomes (Spine to Body Reference) by Tami Jo Urban 2008 [129].
At levels of injury above L1, postural control and respiratory function will be affected differently by the combination of muscles that are partially or fully paralyzed, as discussed in the previous sections of Chapter 2. For example, an injury at the sixth thoracic vertebra (T6) will result in compromised ES in the lower trunk, as well as the lower half of both RA and OE. However, the upper half of RA and OE would remain intact, as well as the upper trunk muscles serratus anterior, pectoralis major, and LD, and the diaphragm. In this case, many of the key muscles involved in breathing remain functional, but stability of the lower spine is likely to be compromised.

An injury sustained at the first thoracic vertebra (T1), ES would be affected just below the shoulders. The muscles of the abdominal wall would also be affected, which may compromise diaphragmatic function as well as spine stability below T1. Respiratory function in this case would depend on the function of the diaphragm without abdominal wall stiffness and the capacity of the accessory respiratory muscles.

At C5, an injury would compromise the function of the LD and other muscles of the upper trunk; the diaphragm and the muscles of the neck and shoulders would be primarily responsible for respiratory function, while most trunk extensors and pairs of spine stabilizing muscles would be compromised depending on completeness of injury. Injuries sustained above the fifth cervical vertebra will invariably affect the diaphragm to some extent; above C3, an individual would require mechanical ventilation to sustain breathing.
Chapter 3

3 Objective and hypothesis

3.1 Objective

In this thesis, two studies were conducted to address the research objectives. The objective of the first study was to examine respiratory function and postural control in AB individuals in two sitting postures: upright and slouch. The objective in the second study was to explore the effects of surface FES activation of trunk muscles on respiratory function and seated postural stability in individuals with SCI.

3.2 Hypothesis

Able-bodied individuals

Respiratory function

H1: $V_T$ will decrease in slouch sitting compared to upright sitting.
H2: $RR$ will increase in slouch sitting compared to upright sitting.
H3: $V_E$ will not change in slouch compared to upright sitting.

Seated postural stability

$H_0$: Seated postural stability, as measured by MDIST, MVELO, and MFREQ, will not change in slouch sitting compared to upright sitting.

Muscle activity

H1: EMG activity of the trunk extensor muscles will decrease in slouch sitting compared to upright sitting.

Individuals with SCI

The second study was comprised of a series of case studies of individuals with SCI where the level of injury affected their trunk musculature, with the consequence of having altered sitting postures and seated postural stability. Functional electrical stimulation was applied to the trunk muscles of these individuals to explore the effects of involuntary muscle activation on respiratory function and seated postural stability. Previous literature demonstrates that implanted FES to trunk musculature facilitates upright posture and respiration [48, 49, 63] and surface FES
to trunk musculatures augments respiratory function [67, 68]. Several configurations of stimulating electrodes were explored in this study, according to the tolerance of each participant, as described in section 1.5.1 (Rationale for surface FES paradigms): AP-FES, stimulating muscles on the anterior and posterior surface of the body (RA and ES); ML-FES, stimulating muscles on the lateral aspects of the body (LD and OE); and FES-ALL, stimulating muscles around the whole trunk (ES, RA, LD, and OE). The hypotheses for the second study were as follows:

H1: Surface FES of trunk muscles will alter respiratory function and postural stability concurrently.

H2: There will be measurable influence of surface FES of the trunk muscles on respiratory function, as evidenced by changes in $V_T$, RR, and/or $V_E$.

H3: There will be measurable effect of surface FES of the trunk muscles on postural stability, as evidenced by changes in MDIST, MVELO, and MFREQ.
Chapter 4

4 Methods

This section begins with a description of the methodology for measuring each experimental variable, including a description of the equipment required for each measurement (a detailed list of apparatus used in the experiments is included in Appendix A), the method used for data collection, and the calibration tests required. A detailed account of the experimental protocol follows the setup description.

4.1 Overview

All participants took part in experimental sessions during which baseline spirometry testing was performed by a trained Respiratory Therapist to determine baseline respiratory function according to a standardized and accepted methodology disseminated by the American Thoracic Society [130]. For both AB individuals and individuals with SCI, each experimental trial consisted of multiple trials, 60s in duration, of quiet sitting in alternating sitting postures; during each trial, simultaneous recordings of postural kinematics, centre of pressure, and respiratory function variables were made. In addition, bilateral trunk muscle activity was recorded from select muscle groups during each trial for AB individuals, while FES was applied to the same group of muscles during each trial with SCI participants. Preliminary recordings of bilateral muscle activity were also done with SCI participants.

4.2 Equipment

4.2.1 Respiratory function measurements

For the purposes of this thesis, two systems were used to assess respiratory function. First, a digital spirometer (SpiroPro® CT, VIASYS Healthcare, San Diego, CA) was used to measure spirometry for each participant under the supervision of a trained respiratory therapist. The digital spirometer captured the standardized outcomes of a single respiratory manoeuvre performed under standard instructions, which allowed for an initial assessment of each individual’s respiratory capacity compared to accepted standards. All participants were asked to do a forced expiratory manoeuvre by the following instruction: “Take a deep breath in, form a tight seal with your lips around the mouthpiece and blow out as long and as hard as you can
through your mouth.” Forced expiratory manoeuvres were performed until three efforts achieved FVC and FEV1 values within a range of 0.15 L (i.e. participants had to reproduce three values of FVC within 0.15 L of one another) for AB participants as per the ATS guidelines for standard spirometry testing as described by Miller et al. [130]. The allowable range for SCI participants was set at 0.2 L; according to Kelley et al. [131], between 77 % and 90 % of individuals with SCI who violate at least one criterion for an acceptable forced expiratory manoeuvre still achieve reproducible values within 0.2 L.

The system used in this thesis to record quiet breathing respiratory function during the experimental sessions was a stand-alone clinical metabolic testing system or CPET (cardiopulmonary exercise testing) system (MetaMax® 3X, Cortex Biophysik GmbH, Germany). This CPET system allowed for the continuous recording of the respiratory cycle (inspiration and expiration) over an extended period of time; there was no standardized methodology for comparison or interpretation of the values obtained by the CPET system, so the results could only be interpreted within the context of this study. A low dead-space full-face mask with a head strap was worn by each participant for the duration of the experimental session to measure $V_T$, RR and $V_E$. The face mask was connected to the external component of the CPET system via the sample line and the volume transducer data cable. Participants were instructed to breathe normally at all times while wearing the face mask.

4.2.2 Seated postural stability measurements

The dual force plate set-up (two split force plates (AccuswayPlus®, Advanced Mechanical Technology, Inc., Watertown, MA) in-built to a custom-made, height-adjustable sitting apparatus [conversion of hydraulic table base (Akron, UK) by REL, Toronto, ON]), allowed the simultaneous capture of the forces and moments in the AP, ML, and vertical planes over the surface of the seat and feet (each with a surface area of 50 cm x 50 cm and a height of 4.4 cm), with participants seated on one plate, the surface area covered by a section of yoga mat, and feet resting on the second plate. These values were converted offline to AP and ML COP components (yCOP and xCOP, respectively) for the seat and feet.

It is known that the use of a footrest by AB individuals increases COP during reaching movements [40]; however, evidence from a pilot experiment conducted by the researcher with an individual with a C5-C6 motor complete injury suggested that persons with SCI at the cervical and high thoracic levels may not be able to remain seated on a platform without foot support.
When analysing dynamic stability during sitting, especially in individuals with compromised trunk muscle activity, it may be necessary to consider a global COP, inclusive of the force and moment contributions of the seat and feet. Therefore, seated postural stability was evaluated using a dual force plate set-up in this study.

4.2.3 Muscle activity measurements

EMG electrode application was identical for AB participants (during the experimental session) and SCI participants (during the consultation session). One EMG system (Bortec AMT-8 EMG system, Bortec Biomedical, Calgary, AB) with a total of eight channels was used to collect surface EMG measurements using bipolar silver-silver chloride disposable electrodes (BiPole electrodes, Bortec Biomedical, Calgary, AB). Skin preparation was performed prior to electrode application: the electrode site was first marked, and, if necessary, the skin area was shaved; the skin was then abraded with 3M™ Skin Prep Abrader Tape and swabbed with an alcohol pad. Once the skin was dry, eight electrodes were placed bilaterally, 18 mm apart, over the following muscles: 1) RA, 3 cm lateral to the umbilicus, aligned vertically; 2) OE, 15 cm lateral to the umbilicus, aligned 45° to the vertical; 3) ES at the lumbar level, 3 cm lateral to the L3 spinous process, aligned vertical; and, 4) latissimus dorsi (LD), 15 cm lateral to T9 spinous process, aligned 45° to the vertical (Figure 4), for a total of 8 EMG channels. A reference electrode (Kendall Medi-Trace™, Covidien, Mansfield, MA) was placed over the clavicle.
4.2.4 Sitting posture measurements

Sitting posture kinematics were captured by the detection of digital marker signals by two position sensors, components of the digital 3D motion capture system (Optotrak® 3020, Northern Digital Inc., Waterloo, ON). Ten infrared digital markers (Northern Digital Inc., Waterloo, ON) were adhered with double-sided tape to landmarks on the skin surface: a single marker each indicated the occipital bone, and vertebral levels C7 and T9; two groups of three markers, arranged on two custom rigid bodies created using NDI 6D Architect, indicated the left and right posterior superior iliac spines (PSIS$_L$ and PSIS$_R$, respectively). The tenth marker was adhered to the seating platform as a reference.

4.2.5 Surface FES application for SCI participants

Two, 4-channel electric stimulators (Compex Motion, Compex, Switzerland) were used to deliver electrical currents with symmetric, biphasic waveforms, to four pairs of muscles with a frequency of 40Hz and pulse duration of 350 μs. During trials with surface FES, stimulation was
delivered concurrently to all electrodes in order to activate antagonistic muscles for the duration of each trial (60 s). Pairs of surface FES electrodes were arranged transversely across the muscle fibres to stimulate the largest segment of the muscle; surface FES electrodes were prepared with conductive gel before being applied to the skin of SCI participants. Self-adhesive surface FES electrodes (5 cm x 5 cm Stimtrode, Cefar Compex Medical, Sweden; 5 cm x 9 cm Stim Trode, Axelgaard Manufacturing Co., Denmark) were arranged bilaterally over the following muscles: 1) RA: four 5 cm x 9 cm electrodes, two per side arranged 3 cm laterally to the umbilicus, one in parallel with umbilicus, one arranged inferiorly, aligned horizontally; 2) OE: four 5 cm x 9 cm electrodes, two per side arranged 15 cm lateral to the umbilicus, aligned 45° to the vertical; and, 3) LD: four 5 cm x 9 cm electrodes, two per side arranged 15 cm lateral to T9 spinous process, aligned 45° to the vertical; 4) ES at the lumbar level: four 5 cm x 5 cm electrodes were placed, two per side, 3 cm lateral to the L3 spinous process (one in parallel with L3, one arranged inferiorly), aligned horizontally. In total, sixteen self-adhesive surface FES electrodes were distributed among eight FES channels (Figure 5).

Figure 5: Anterior (left) and posterior (right) view of surface FES electrode placements for SCI participants
4.2.6 Data acquisition

Data collection for all measurement variables was time-locked at the onset of each experimental trial: recording of EMG signals and COP forces and moments took place via the Central data acquisition (DAQ) system, and motion capture signals captured via the Secondary DAQ were time-locked via an external voltage-based synchronization signal. The nature of the CPET system necessitated continuous data collection over the duration of the experimental session via a Tertiary DAQ; data markers inputted manually allowed indication of the onset of each experimental trial.

Both sets of equipment for respiratory assessment had self-contained data collection and analysis hardware and software components; all respiratory values were independently acquired and analyzed. The digital spirometer acquired single-breath output data; collected values included a measure of respiratory volume (forced vital capacity, FVC), a measure of respiratory flow (forced expiratory volume in 1 second, FEV₁), and the respective percentage of predicted value for each. During the experimental session, tidal volume (VT), breathing frequency (RR), and minute ventilation (VE) were sampled at ten-second intervals (collection frequency 0.16 Hz) on the Tertiary DAQ.

The Central DAQ, responsible for the collection of surface EMG signals and COP forces and moments, was electronically synched to the motion capture system Secondary DAQ via an external trigger (single channel output amplitude 5 V), which was recorded alongside the EMG signals and COP components in LabVIEW. The trigger, EMG signals, and COP components were recorded at a sampling frequency of 2,000 Hz; EMG signals were pre-amplified with a gain of 5,000. Postural kinematic data was collected using First Principles motion capture software at a sampling frequency of 120 Hz.

4.2.7 Calibration

Prior to conducting experimental sessions, calibration was performed on the digital spirometer, CPET system, EMG system, force plates, and motion capture system. Calibration of the stimulators constituted determining an individual’s pain threshold for surface FES; the final surface FES current for experimental trials was set at 1 mA below the pain threshold.

The digital spirometer, equipped with in-built software, automatically calibrates individual results to population-standardized values using anthropometric inputs including age, height,
weight, and gender. Full calibration of the CPET system, including gas and volume standards, was performed using the manufacturer’s calibration kit (Cortex Calibration Kit) including a syringe with known volume (Hans Rudolph 3.0 L) and a standard gas canister (5% CO₂, 15% O₂). Pressure calibration was performed using barometric pressure reported online by Environment Canada. An automatic room-air calibration was also performed just prior to the start of data collection.

A voltmeter was used to determine the impedance of surface EMG electrodes. Where high values (>10 kΩ) or exaggerated asymmetry in impedance across bilateral muscle pairs was indicated, re-preparation of the skin, replacement of the electrode, and lead wire replacement were done as necessary to reduce the value or achieve greater symmetry.

Each force plate was zeroed prior to each experimental session. Once participants were seated, the height of the sitting apparatus was adjusted for each participant to achieve 90° angles at the hip, knee, and ankle; then, the corners of the seat and feet force plates were aligned using weighted drop lines. Orientation of the coordinate system for the force plates is shown in Figure 6. Formal calibration testing was conducted for the force plates in an early study. The force plates were calibrated for spatial resolution and force versus weight and drift measurements. Sin [29] found that the spatial resolution error (0.4 cm), weight calibration SD (0.784 N to 0.882 N), and vertical force drift (0.18 N to 2.7 N) of the force plates fell within the manufacturer’s acceptable standards.

Motion capture position sensor registration and alignment was performed with a 16-point calibration cube (Northern Digital Inc., Waterloo, ON); this was aligned such that one plane was parallel to the ground and the planar axes aligned with the axes of the force plates and the reference point of the coordinate system was located at the right, rear corner of the dual force platform set up (see Figure 6). Calibration of the motion capture system indicated a root mean square measurement error of ~ 0.2 mm. Calibration of the digitizing probe was done by performing a pivot test with the probe tip, of known diameter, placed stationary on a surface and pivoting the probe in three planes around the tip.
4.3 Experiments

4.3.1 Participants

All persons who participated in the experiments described in this document were eligible volunteers who provided informed consent after reading an information sheet (Appendix B), as per the study conditions approved by the Research Ethics Board of Toronto Rehabilitation Institute (TRI REB#05-017 Sub-Study) and the Health Science Ethics Review Office of the University of Toronto (Protocol Reference #25789).

Able-bodied participants

Individuals were invited to volunteer for this study if they were young (25-45 years of age), healthy individuals, willing and able to participate in an experimental session for two hours. Individuals were not permitted to volunteer if he/she was not able to comprehend instructions; had a musculoskeletal or neurological condition; had a respiratory disease or infection, such as asthma, cystic fibrosis, COPD, emphysema, pneumonia, or bronchitis; was a heavy smoker (>10 pack years)\(^{\text{viii}}\); or had a body mass index (BMI) that exceeded 24.9. The medical and lifestyle restrictions were placed on AB participants such that baseline information was not potentially contaminated by underlying deficits or dysfunctions of the relevant systems.

SCI participants

The recruitment guidelines for individuals with SCI were initially set to match the anthropometric characteristics defined for AB participants; however, inclusion criteria were later amended to include individuals whose BMI exceeded 25.0. Individuals were invited to participate if they had an SCI from C4-T12, based on the assumption that the diaphragm would be intact at these injury levels; individuals could participate if they had a traumatic or non-traumatic injury. Participants had to be able to perform a manual transfer with some assistance and be able to sit unsupported for 60s. Individuals had to be willing and able to participate in an experimental session for 1.5h, with the provision of multiple breaks for rest or weight relief.

\(^{\text{viii}}\) One pack year is equivalent to the number of cigarettes smoked per day divided by 20 (20 cigarettes per one pack) and multiplied by the number of years an individual has smoked. At least one study investigating the prevalence of common lung diseases worldwide suggested that individuals with a lifetime pack-year history of 0-10 had a similar rate of disease incidence as individuals who had never smoked [132].
Individuals were not permitted to volunteer if he/she was not able to comprehend instructions; had a musculoskeletal or neurological condition; had a respiratory disease or infection, such as asthma, cystic fibrosis, chronic obstructive pulmonary disorder (COPD), emphysema, pneumonia, or bronchitis; was a heavy smoker (>10 pack years). Similarly for SCI individuals, medical and lifestyle restrictions were placed on participants such that baseline information was not potentially contaminated by underlying deficits or dysfunctions of the relevant systems, other than that imparted by the SCI.

4.3.2 Experimental set-up

The equipment required in this experiment was arranged on a 3.5 m x 3.5 m platform, behind a large privacy screen (Figure 6, privacy screen not shown). The custom sitting apparatus was placed off-centre on the platform; this allowed the sitting force plate, which was secured to the apparatus, and thus the seated participant, to be centred on the platform. The second force plate was located directly in front of the sitting apparatus, on the platform, in line with the sitting force plate. The CPET system was placed to the left of the seated participant, on a raised shelf. The motion capture system position sensors were placed at a distance of 4m from the centre of the sitting force plate; this distance permitted the optimal capture range for the sensors. The remaining components of the experimental setup (including computers equipped with acquisition software) were placed outside the perimeter of the platform.
4.3.3 Protocol

Able-bodied participants

Able-bodied participants participated in a single experimental session. The session began with spirometry testing, supervised by the Respiratory Therapist. The participant was then asked to adjust his or her shirt to allow access to the skin surface and muscles of the trunk. Surface EMG electrodes were applied, and impedance was measured using a voltmeter and recorded (Appendix C).

The participant was asked to sit down on the sitting apparatus, remove their shoes, and place their feet flat on the feet force plate. With the participant comfortably seated, the height of the sitting apparatus was hydraulically adjusted by the experimenter with a foot pedal, such that the participant’s hips, knees, and ankles were all aligned at 90°; alignment of the front corners of the seat force plate and the rear corners of the feet force plate was then performed using weighted drop lines. Preamplifier cables were connected to the EMG electrodes and secured to the skin.

Figure 6. Layout of experimental dual force plate set-up. Dual force plate set-up shown in reference to other measurement components (a) and origin of force plate axes (b).
with medical tape to reduce signal artefacts from movement. Signal integrity was tested by having the participant contract each muscle of interest against external resistance provided by the experimenter.

Next, infrared markers and rigid bodies were adhered to the participants’ body with double-sided tape to C7, T9, PSIS_L and PSIS_R; a digitizing probe was used to virtually landmark the left and right anterior superior iliac spines (ASIS_L and ASIS_R, respectively) in reference to each ipsilateral PSIS rigid body.

Following the automatic room-air calibration by the CPET system, the face mask was applied to the participant and secured with the head strap, and the occipital infrared marker was clipped into place with a hair clip, completing AB participant preparation (Figure 7).

The participant was then given a minimum of 60s to acclimatise to breathing through the face mask before beginning the experimental trials. During this time, the experimenter explained the different postures that the participant would alternately adopt during the experimental session, and reminded the participant to continue to breathe normally. For AB participants, the experimental session consisted of six, discrete 60s trials conducted in one of two postures: 1) upright sitting, or 2) slouch sitting. The participant was given the following instructions: for the upright sitting condition, “Keeping your feet flat on the floor, sit upright, in a comfortable position with your hands on your lap”; for the slouch sitting condition, “Keeping your feet flat on the floor, slouch down into your body, letting yourself relax completely”.

Once breathing stabilized, the experimenter explained that the trials would commence, beginning with an upright sitting trial. Data collection of quiet breathing respiratory function, muscle activity, sitting forces, and postural kinematics for a given trial commenced at least 60s after the participant assumed the appropriate posture, to allow time for the breathing to stabilise; following 60s of recording, the experimenter gave the instruction to assume the alternate posture.

Data collection continued until three 60s trials had been collected for each posture. Upon completion of the experimental session, the face mask was removed, followed by the infrared digital markers and EMG electrodes.
SCI participants

SCI participants participated in this study in two parts: a preliminary session and an experimental session. During the preliminary session, all assessments were done with participants seated in their own wheelchair. First, participants performed spirometry testing, supervised by the Respiratory Therapist. The participant was then asked to remove his shirt to allow access to the skin surface and muscles of the trunk. Surface EMG electrodes were then applied, and impedance was measured using a voltmeter and recorded (Appendix C). The participant was asked to assume a typical sitting position, and 60s of EMG activity was collected; the participant was then asked to assume a sitting position with the body shifted away from the backrest, where possible, and another 60 s of EMG activity was collected.
At the start of the experimental session, the participant remained in his own wheelchair while the experimenter applied surface FES electrodes with conductive gel to the RA, OE, LD, and ES at the lumbar level. The participant was then asked to transfer from his wheelchair to the sitting apparatus. If required, the experimenter provided assistance with the transfer; a safety harness was also provided that attached to ceiling-mounted hardware to prevent falls, and to assist with rest periods between trials. During participant preparation, the safety harness was used to provide trunk stability to the participant when necessary. Once seated comfortably, the experimenter assisted the participant to remove his shoes and place his feet flat on the feet force plate. The sitting apparatus was then height-adjusted as for AB participants, and the force plates were aligned.

The next step was to establish stimulation intensity in each of the two surface FES configurations (AP-FES, ML-FES). The goal of administering FES is to generate a motor activation of the muscle within the limits of the pain threshold; however, this threshold may be altered or absent in individuals with SCI. In the case where the participants’ intact sensation allowed them to perceive the stimulation (SCI-1), the pain threshold was considered in the determination of the appropriate level of stimulation and the stimulation intensity was set at 1mA below the pain threshold. When participants could not perceive stimulation (SCI-2, SCI-3), stimulation intensity was determined by increasing intensity until muscle contraction was detected by palpation, without causing discomfort to participants. Stimulation intensity for each participant is listed in Appendix C.

Next, infrared digital markers were applied, and ASIS\textsubscript{L} and ASIS\textsubscript{R} were digitized, as for AB participants. Following the automatic room-air calibration by the CPET system, the face mask was applied to the participant and secured with the head strap, and the occipital marker was clipped into place with a hair clip, completing SCI participant preparation (Figure 8). The participant was then given a minimum of 60 s to acclimatise to breathing through the face mask before beginning the experimental trials. During this time, the researcher explained the different conditions that would take place during the experimental trials, and reminded participants to continue to breathe normally. When the researcher finished all explanations, a continuous music playlist was initiated as an auditory stimulus to prevent sleepiness during the experimental session. For SCI participants, the experimental session consisted of four to six 60 s trials, depending on the comfort of each participant, conducted in one of three conditions: 1)
unsupported sitting, where no external support of the trunk was provided to the participant for the duration of the trial (Unsupported), 2a) anteroposterior surface FES-assisted sitting (AP-FES), activating ES at the lumbar level and RA, and 2b) mediolateral surface FES-assisted sitting (ML-FES), activating OE and LD. In one case, the participant was able to tolerate application of surface FES using all channels simultaneously (ALL-FES); one trial of ALL-FES was therefore completed with participant SCI-3. Participants were given the following instructions: for unsupported sitting, “Keeping your feet flat on the floor, try to sit upright while maintaining your balance in a comfortable position with your hands on your lap”; for AP- and ML-FES-assisted sitting, “Keeping your feet flat on the floor, relax your body while maintaining your balance in a comfortable position with your hands on your lap”.

Once the participant’s breathing stabilized, the experimenter explained that the trials would commence, beginning with an unsupported sitting trial. Data collection for a given trial commenced as soon after the participant achieved stability in the given condition as possible, so as to limit the duration of time a participant spent in a static position; following 60 s of recording, the participant had the opportunity to rest or to perform pressure relief, as necessary. Data collection continued until two to three trials had been collected for each condition. Upon completion of the experimental session, the face mask was removed, followed by the infrared markers and surface FES electrodes. The participant then transferred back to his own wheelchair and answered a brief questionnaire regarding his experience with surface FES-assisted sitting (Appendix D).
4.4 Analysis

Time-locked data sets for each variable from each experimental trial were analyzed. Force plate signals and EMG signals were analyzed offline using custom code written in the MATLAB programming environment (The Mathworks Inc., Natick, MA). Respiratory data were analyzed separately using in-built software (SpiroPro) or the standard CPET software Metasoft3.

For each section of the analysis (quiet breathing respiratory function, seated postural stability, and muscle activity), outcome measures from each of three trials within a single condition were averaged to yield a mean value per experimental condition for each AB participant (i.e. ten individual means were yielded for each variable for both the upright and slouch sitting conditions). Statistical analyses compared the group mean (n=10) and SD of each variable in upright versus slouch sitting. For SCI participants, data were examined for trends between each trial across the entire experimental session.

Figure 8. Posterior view of experimental set-up for participants with SCI. Origin (right-hand corner of force plate) indicates axis of motion capture system reference frame.
All data sets were subject to a test of normality. Where the data deviated from a normal distribution (Shapiro-Wilk test, $\alpha \geq 0.05$), a Wilcoxon signed rank test (WSR) was performed in place of a paired t-test to determine statistical significance between variables.

4.4.1 Respiratory function

Spirometry values were determined using in-built clinical diagnostic software with each participant’s age, height, and weight as predictors. The ATS has recommended the use of lower limits of normal for setting the threshold that defines abnormal test results [133]. In the United States, the Centers for Disease Control and Prevention recommends the methodology developed by Hankinson et al. [110] be used for predicting spirometric reference values; thus, individual results were compared to the lower limit of normal predicted by these equations. Absolute and percent of predicted values for FVC and FEV$_1$ were reported orally to the experimenter by the respiratory technician and recorded. The highest spirometry values were used for comparison, as per the ATS spirometry testing guidelines [133]. Real-time values of quiet breathing respiratory function ($V_T$, RR, and $V_E$) were recorded at the end of each experimental session. Individual trial means and SDs were recorded for all participants; for AB participants, individual trial means were converted to three-trial condition means and SDs for comparison.

A paired t-test was performed on the mean quiet breathing respiratory function values across all participants to test for a difference between upright and slouch sitting postures. Since directional changes were expected for all quiet breathing respiratory function variables, two-tail t-tests were performed. Statistical significance indicating a conditional difference was found when $p<0.05$.

4.4.2 Seated postural stability

For each sitting condition, custom code written and applied in the MATLAB program was used to convert raw voltage signals to COP and forces [29]. COP signals were then filtered using a 4th-order, zero-phase lag, low-pass Butterworth filter with a cut-off frequency of 5 Hz. For the analyses of the COP data, COP-based measures were computed using the methodology of Prieto et al. [12] and modified by Sin [29]. Mean vertical forces (Fz) were thus computed directly from raw voltage signals across the surface of each force plate. Mean COP distance was calculated from the COP time series data. Total length of the COP displacement in the x- and y- directions was calculated; MVELO was estimated by taking the first derivative with respect to time of the
COP displacement data. Mean COP frequency was then calculated as the modified ratio of MVELO to MDIST (MVELO:2πMDIST).

Mean vertical forces (Fz) were reported for each force plate and as a proportion of the composite total Fz. For SCI participants, Fz minima and maxima for all sitting trials were reported; the proportion of the composite total Fz was determined using the mean total Fz from all sitting trials. For each force plate, mean distance, velocity, and frequency of the COP were computed as overall values (MDIST, MVELO, MFREQ respectively) and as directional components (anteroposterior: apMDIST, apMVELO, apMFREQ; mediolateral: mlMDIST, mlMVELO, mlMFREQ). These measures will collectively be referred to as COP parameters.

For AB participants’ data, COP-based measures will be reported as both overall values and as directional components, since the orientation of the base of support (BOS) was constrained to the axes of the force plate. In the individual case studies of participants with SCI, only the overall values of COP-based measures will be reported, since the orientation of the BOS (specifically, the thighs and feet) could not be constrained due to the minimal or absent muscle tone of the lower limbs, in some cases allowing the thighs and feet to splay outward, adopting an orientation closer to 45° along the X-Y axis of the force plates. This is considered a more accurate way of characterizing the COP path when the BOS is not constrained [12].

A paired t-test was performed on the mean seated postural stability values across all participants for each sitting condition to test for a difference between the upright and slouch postures. Since no difference was predicted in seated postural stability between upright and slouch sitting, two-tail t-tests were performed. Statistical significance indicating a conditional difference was found when p<0.05.

4.4.3 Muscle activity

For each sitting condition, raw EMG signals were filtered using a 4th-order, zero-phase lag, low-pass Butterworth filter with a cut-off frequency of 5 Hz with additional in-code notch filters at 60 Hz, 120 Hz, and 180 Hz. Next, filtered signals were rectified and used to determine the mean EMG amplitude (mV) for each sitting condition. For each individual trial, the mean amplitude of the EMG signal over 60 s and the power spectral density (generated using a Welch’s method FFT analysis) were plotted for visual inspection.
A paired t-test was performed on the mean EMG amplitude across all participants for each muscle bilaterally to test for: a) an increase from the slouch to the upright sitting posture (a one-tail t-test was performed); and, b) symmetry between the left and right side of each muscle within a given sitting condition (a two-tail t-test was performed). Statistical significance indicating a conditional difference or asymmetry was found when \( p<0.05 \) (e.g. EMG amplitude in the upright posture being different from the slouch posture, or right-sided muscle EMG amplitude being different from left-sided, respectively).

Analysis of the EMG signals collected for SCI participants was beyond the scope of this thesis; data will be presented only as an appendix (Appendix E).

4.4.4 Sitting posture

There were no analyses performed on the kinematics dataset as this component was beyond the scope of this thesis; upright sitting posture will be reported elsewhere.
Chapter 5

5 Results

This section presents the results of the analysis outlined in Chapter 4. In the text, values in parentheses indicate the range of individual means, followed by the p-value generated by the associated t-test or Wilcoxon signed rank test, unless stated otherwise; where data presented in tabular or graphical format, values are the group mean and one SD. The results are presented in two sections: 1) a summary of the results of experiments with AB participants; and 2) three case studies describing each experiment with a participant with SCI. Since the number of trials conducted in the SCI case studies was not sufficient to submit to statistical testing, meaningful differences will be considered only relative to the outcomes of the AB participants.

5.1 Able-bodied participants

Ten healthy, AB participants (n=6 male, 4 female; for all ten participants (mean ±1SD): age 31 ±5.9 y; height 174.4 ±9.5 cm; weight 68.5 ±13.2 kg; BMI 22.3 ±2.0) volunteered to participate in this study (see Appendix F for detailed participant characteristics). None had a history of chronic respiratory disease, nor had an acute respiratory episode at the time of testing. All participants had FVC within the normal range according to the predicted spirometry values calculated using the recommended equations from Hankinson et al. [110], and all but two had FEV₁ within the normal range: AB1 and AB5 had 76 % predicted FEV₁, which was 6 % below the lower limit of normal FEV₁ (see Appendix F for detailed spirometry for AB participants). Seven participants were never smokers; for one current and two former smokers, mean pack-year history was 6.3 ±3.7 pack-years. A summary of AB group average outcomes for all variables can be found in Appendix G.

5.1.1 Respiratory function

The total number of trials included in the analysis of quiet breathing respiratory function included 29 upright sitting trials and 28 slouch sitting trials (Participant AB-1 had two upright sitting trials and one slouch sitting trial). The entire dataset was determined to be normally distributed (Shapiro-Wilk test, p>0.10 for all quiet breathing respiratory function variables). The group average quiet breathing respiratory function values were computed for all AB participants by averaging the ten individual means for each sitting condition (Table 6 and Figure 9).
Comparison between sitting postures showed that $V_T$ and $V_E$ in the upright condition were higher than in the slouch condition; the differences were statistically significant for both a 13% increase in $V_T$ during upright versus slouch sitting (upright: 0.4-1.2 L, slouch: 0.4-1.0 L, $p=0.009$) and an 8% increase in $V_E$ during upright versus slouch sitting (upright: 5.6-11.8 L/min, slouch: 4.7-10.6 L/min, $p=0.01$). A paired t-test comparing upright and slouch condition RR revealed no statistical significance of the 4% difference between the postures (upright 9.6-19.1 min$^{-1}$, slouch 9.8-19.2 min$^{-1}$, $p=0.18$), although RR was higher in the slouch than the upright condition.

Table 6. Quiet breathing respiratory function for AB participants (n=10) during upright and slouch sitting

<table>
<thead>
<tr>
<th>Quiet breathing respiratory function (mean ±1SD)</th>
<th>Upright</th>
<th>Slouch</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_T$ (L)</td>
<td>0.7 ±0.3</td>
<td>0.6 ±0.2</td>
<td>0.009</td>
</tr>
<tr>
<td>RR (min$^{-1}$)</td>
<td>14.2 ±1.4</td>
<td>14.7 ±1.0</td>
<td>0.01</td>
</tr>
<tr>
<td>$V_E$ (L/min)</td>
<td>9.3 ±1.1</td>
<td>8.6 ±0.8</td>
<td>0.18</td>
</tr>
</tbody>
</table>
Figure 9. Quiet breathing respiratory function for AB participants (n=10) during upright and slouch sitting. Group average (bars, plus 1 SD) $V_T$ and $V_E$ were higher in the upright versus the slouch posture, whereas RR showed no significant difference between sitting conditions (*p<0.05, **p<0.01). Upright sitting is represented by solid bars, hatched bars indicate slouch sitting; dark grey, light grey, and black bars indicate $V_T$, RR, and $V_E$, respectively.

5.1.2 Seated postural stability

The analysis of seated postural stability included a total of 28 upright sitting trials and 28 slouch sitting trials; missing data due to technical recording error included one upright sitting trial and two slouch sitting trials from Participant AB-1, and one upright sitting trial from participant AB-9. Several variables within the seated postural stability dataset were determined to be non-normally distributed (Shapiro-Wilk test, p<0.10); WSR indicates those trials where nonparametric statistics were performed.

Comparisons of vertical forces between conditions revealed little change among the AB participants; group average mean Fz detected by each force plate during upright and slouch sitting are summarized below (Table 7). In comparisons of Fz in upright versus slouch sitting postures, there was no significant difference in the absolute vertical forces detected by either the seat force plate (upright: 407.63-712.96 N, slouch: 404.83-709.55 N, p=0.15) not the feet force plate (upright: 109.76-216.21 N, slouch: 99.32-204.75 N, p=0.22) between sitting conditions, nor
was there a significant change in the proportion of total vertical forces distributed between the seat and feet force plates (upright: 71.1-84.8 N, slouch: 72.6-84.6 N, p=0.22).

<table>
<thead>
<tr>
<th>Absolute Fz, mean ±1 SD</th>
<th>Vertical force (N)</th>
<th>Upright</th>
<th>Slouch</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seat</td>
<td>564.90 ±104.39</td>
<td>557.62 ±100.15</td>
<td>0.15</td>
<td></td>
</tr>
<tr>
<td>Feet</td>
<td>146.99 ±33.72</td>
<td>153.16 ±35.85</td>
<td>0.22</td>
<td></td>
</tr>
<tr>
<td>Proportion total Fz</td>
<td>Seat</td>
<td>0.79</td>
<td>0.78</td>
<td>0.22</td>
</tr>
<tr>
<td></td>
<td>Feet</td>
<td>0.21</td>
<td>0.22</td>
<td>0.22</td>
</tr>
</tbody>
</table>

In comparisons of MDIST among the AB participants, group average MDIST followed no common pattern between conditions among directional components (Table 8, Figure 10). Overall MDIST was greater for each participant and significantly greater on average during upright versus slouch sitting for the seat force plate (upright: 0.73-2.78 mm, slouch: 0.32-2.46 mm, p<0.001) and for the feet force plate (upright: 1.12-3.47 mm, slouch: 0.30-1.87 mm, p<0.001). In the AP direction, apMDIST was greater on average in the slouch condition for the seat and feet (during slouch sitting, 7 and 5 participants had higher apMDIST measured on the seat and feet force plates, respectively), but the differences were not significant for the seat force plate (upright: 0.48-1.28 mm, slouch: 0.52-3.41 mm, p=0.16) nor for the feet force plate (upright: 0.25-2.25 mm, slouch: 0.92-2.89 mm, p=0.17). Although mlMDIST was greater on average in the upright compared to the slouch sitting posture for both the seat and the feet, the differences were not significant for the seat force plate (upright: 0.22-3.07 mm, slouch: 0.23-0.94 mm, p=0.27) nor for the feet force plate (upright: 0.18-1.53 mm, slouch: 0.11-1.60 mm, p=0.75); among participants, both force plates had 3 instances (individual means) of mlMDIST during slouch exceeding mlMDIST during upright sitting.
Table 8. Mean COP distance for AB participants (n=10) during upright and slouch sitting

<table>
<thead>
<tr>
<th></th>
<th>Mean COP distance (mm)</th>
<th>Upright</th>
<th>Slouch</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MDIST</td>
<td>Seat</td>
<td>1.52 ±0.63</td>
<td>1.09 ±0.62</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Feet</td>
<td>2.23 ±0.75</td>
<td>1.07 ±0.56</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>apMDIST</td>
<td>Seat</td>
<td>0.70 ±0.26</td>
<td>1.05 ±0.86</td>
<td>0.16</td>
</tr>
<tr>
<td></td>
<td>Feet</td>
<td>1.16 ±0.71</td>
<td>1.59 ±0.59</td>
<td>0.17</td>
</tr>
<tr>
<td>mlMDIST</td>
<td>Seat</td>
<td>0.70 ±0.87</td>
<td>0.43 ±0.21</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>Feet</td>
<td>0.66 ±0.48</td>
<td>0.61 ±0.56</td>
<td>0.75</td>
</tr>
</tbody>
</table>

Figure 10. Mean COP distance for AB participants (n=10) during upright and slouch sitting. For both force plates, overall MDIST was significantly greater in upright vs. slouch sitting (p<0.001). Bars represent group average plus 1 SD; solid bars represent upright sitting, while hatched bars represent slouch sitting; dark grey represents the seat, light grey represents the feet.
All group average MVELO were higher in the upright posture for both the seat and feet force plates when compared with the slouch posture (Table 9, Figure 11); this observation was also true for individual participants, with the exception of AB-4 whose slouch sitting MVELO values exceeded the upright sitting values in all COP velocity components for both force plates (see Appendix G). With the exception of apMVELO for the seat force plate, all MVELO comparisons between sitting conditions were significantly higher in the upright versus the slouch posture (for the seat force plate: MVELO upright: 1.67-4.86 mm/s, slouch: 1.32-4.74 mm/s, p= 0.02 [WSR], mlMVELO upright: 1.13-3.95 mm/s, slouch: 0.82-2.85 mm/s, p=0.006 [WSR]; for the feet force plate: MVELO upright: 2.18-12.77 mm/s, slouch: 1.62-10.74 mm/s, p=0.01 [WSR], apMVELO upright: 1.29-4.10 mm/s, slouch: 1.13-3.29 mm/s, p=0.036, mlMVELO upright: 1.36-12.04 mm/s, slouch: 0.93-10.20 mm/s, p=0.03 [WSR]); apMVELO of the seat appears to trend toward significance (upright: 0.92-2.87 mm/s, slouch: 0.77-3.69 mm/s, p=0.07 [WSR]).

Table 9. Mean COP velocity for AB participants (n=10) during upright and slouch sitting

<table>
<thead>
<tr>
<th></th>
<th>Mean COP velocity (mm/s)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Upright</td>
<td>Slouch</td>
</tr>
<tr>
<td>MVELO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>3.04±1.01</td>
<td>2.15±1.12</td>
</tr>
<tr>
<td>Feet</td>
<td>4.96±3.08</td>
<td>3.61±2.71</td>
</tr>
<tr>
<td>apMVELO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>1.65±0.62</td>
<td>1.32±0.89</td>
</tr>
<tr>
<td>Feet</td>
<td>2.65±0.90</td>
<td>1.97±0.70</td>
</tr>
<tr>
<td>mlMVELO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>2.16±0.85</td>
<td>1.40±0.68</td>
</tr>
<tr>
<td>Feet</td>
<td>3.51±3.18</td>
<td>2.53±2.79</td>
</tr>
</tbody>
</table>

* – Wilcoxon signed rank test result
Figure 11. Mean COP velocity for AB participants (n=10) during upright and slouch sitting. All COP velocity components on the feet force plate were significantly greater in upright vs. slouch sitting; on the seat force plate, only VELO and mVELO were significantly greater in upright sitting (*p<0.05, **p<0.01). Bars represent group average plus 1 SD; solid bars represent upright sitting, hatched bars represent slouch sitting; dark grey represents the seat, light grey represents the feet.

All group average values of MFREQ were higher in the slouch sitting posture than in upright sitting (Table 10, Figure 12). Only one component of MFREQ was significantly different between sitting conditions: apMFREQ for the seat was significantly higher in the slouch posture (upright: 0.24-0.63 Hz, slouch: 0.25-0.78 Hz, p=0.016). Changes in the overall MDIST and MVELO of the seat between upright and slouch conditions were proportional, such that the resultant overall MFREQ in both conditions were very similar (MFREQ upright: 0.24-0.55 Hz, slouch: 0.25-0.56 Hz, p=0.30; mlMFREQ upright: 0.38-0.85 Hz, slouch: 0.40-1.13 Hz, p=0.09); this was true to some degree of the overall MFREQ of the feet as well (MFREQ upright: 0.24-0.65 Hz, slouch: 0.24-0.85 Hz, p=0.71; apMFREQ upright: 0.28-1.04 Hz, slouch: 0.31-1.36 Hz, p=0.14; mlMFREQ upright: 0.26-1.01 Hz, slouch: 0.27-1.96 Hz, p=0.09).
Table 10. Mean COP frequency AB participants (n=10) during upright and slouch sitting

<table>
<thead>
<tr>
<th></th>
<th>Upright</th>
<th>Slouch</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MFREQ</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.36 ±0.09</td>
<td>0.39 ±0.11</td>
<td>0.30</td>
</tr>
<tr>
<td>Feet</td>
<td>0.34 ±0.12</td>
<td>0.35 ±0.18</td>
<td>0.71</td>
</tr>
<tr>
<td><strong>apMFREQ</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.37 ±0.12</td>
<td>0.53 ±0.17</td>
<td>0.16</td>
</tr>
<tr>
<td>Feet</td>
<td>0.59 ±0.25</td>
<td>0.79 ±0.38</td>
<td>0.14</td>
</tr>
<tr>
<td><strong>mlMFREQ</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.60 ±0.16</td>
<td>0.66 ±0.24</td>
<td>0.09</td>
</tr>
<tr>
<td>Feet</td>
<td>0.66 ±0.26</td>
<td>0.98 ±0.50</td>
<td>0.09</td>
</tr>
</tbody>
</table>

Figure 12. Mean COP frequency for AB participants (n=10) during upright and slouch sitting. Only apMFREQ of the seat and mlMFREQ of the feet were significantly different between postures (*p<0.05). Solid bars represent upright sitting; hatched bars represent slouch sitting; dark grey represents the seat; light grey represents the feet.
Interpretation of COP parameters entails consideration of the following caveat: the analysis of seated postural stability in this study included those trials with sudden and/or extreme COP excursions (deviations). By visual inspection, 24 of the 56 trials used in the analysis (42% of all trials) showed at least one incidence of deviation in both planes of the force plate (AP and ML) that was visible on both force plates during a given 60 s trial. Several participants reported feeling altered states of alertness during the course of the experimental session: participant AB6 reported feeling very relaxed and in a “meditative state” – this participant had the second highest incidence of COP deviations. Participant AB-4, who had the highest incidence of sudden or extreme COP deviations, reported feeling “sleepy” during all trials of the experimental session. Interestingly, AB-2, who had no trials with COP deviations, reported feeling “more attentive” in the upright posture. An example of COP deviations in both upright and slouch sitting is shown during 30 s of data collection for participant AB-10 (Figure 13). These deviations were more common and more frequent in trials of the upright sitting posture (n=16 trials with 34 incidences) than in the slouch sitting posture (n=8 trials with 16 incidences). These unique, time-limited events (generally lasting 2 s-3 s, but as long as 4 s) appear to reflect a momentary loss of stability that may be a common phenomenon during periods of uninterrupted quiet sitting even in AB individuals.
5.1.3 Muscle activity

Data recorded from the left-side OE of participant AB-7 (paired trials 1 and 3) were excluded from the EMG analysis due to physiological interference with the recording. Participants AB-1 and AB-9 had missing data from both sitting conditions (paired trials 2 and 3, and paired trial 2, respectively) due to technical recording error. In total, data from 27 paired trials (27 upright trials and 27 slouch trials) were analyzed for the right-side OE, and RA, LD, and ES muscles bilaterally; for the left-side OE, 25 paired trials were analyzed. Several variables within the muscle activity dataset were determined to be non-normally distributed (Shapiro-Wilk test, p<0.10); WSR indicates those tests where nonparametric statistics were performed.
Average bilateral muscle activity, as measured by mean EMG amplitude, for each sitting condition is shown (Table 11, Figure 14). Asymmetry in trunk muscle activity was detected in RA and OE in both the upright and slouch sitting postures: in RA, right side muscle activity exceeded left side muscle activity in the upright and slouch postures in seven participants; left OE muscle activity exceeded right OE muscle activity in both postures in six participants. There is insufficient evidence from the impedance measurement results to suggest that line noise may have contributed to this difference. Paired t-tests for both muscles indicated statistically significant sidedness in EMG amplitude within each sitting condition (RA upright left: 2.19-3.76 mV, right: 1.22-7.72 mV, p=0.01 [WSR]; RA slouch left: 2.16-3.52 mV, right: 1.18-7.66 mV, p=0.01 [WSR]; OE upright left: 2.48-6.73 mV, right: 2.32-3.36 mV, p=0.002; OE slouch left: 2.47-7.91 mV, right: 2.25-2.83 mV, p=0.005 for slouch posture). No asymmetry was found in LD or ES in either sitting condition (LD upright left: 2.40-3.31 mV, right: 2.37-3.15 mV, p=0.30; LD slouch left: 2.37-2.88 mV, right: 2.30-2.87 mV, p=0.48; ES upright left: 2.94-8.18 mV, right: 2.49-8.78 mV, p=0.11; slouch left: 2.35-6.13 mV, right: 2.33-8.60 mV, p=0.28).

Table 11. Average muscle activity for AB participants (n=10) during upright and slouch sitting

<table>
<thead>
<tr>
<th></th>
<th>Mean EMG amplitude (mV)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean ±1 SD</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Upright</td>
<td>Slouch</td>
</tr>
<tr>
<td>RA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>2.93 ±0.47</td>
<td>2.91 ±0.44</td>
</tr>
<tr>
<td>R</td>
<td>5.30 ±2.34†</td>
<td>5.26 ±2.33†</td>
</tr>
<tr>
<td>OE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>4.38 ±1.43</td>
<td>4.56 ±1.89</td>
</tr>
<tr>
<td>R</td>
<td>2.70 ±0.38†</td>
<td>2.50 ±0.20†</td>
</tr>
<tr>
<td>LD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>2.85 ±0.28</td>
<td>2.64 ±0.19</td>
</tr>
<tr>
<td>R</td>
<td>2.75 ±0.26</td>
<td>2.57 ±0.20</td>
</tr>
<tr>
<td>ES</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>5.51 ±1.92</td>
<td>3.27 ±1.20</td>
</tr>
<tr>
<td>R</td>
<td>4.37 ±1.84</td>
<td>3.18 ±1.93</td>
</tr>
</tbody>
</table>

† - significant difference in left versus right EMG amplitude within condition (RA: p=0.01*, slouch p=0.28); OE: p=0.002, slouch p=0.005); * - Wilcoxon signed rank test result
Figure 14. Average muscle activity for AB participants during upright and slouch sitting. Mean EMG amplitude for AB subjects showed sidedness in the RA and OE in both conditions; the OE (right-side only), and bilateral LD and ES showed larger EMG amplitudes in the upright versus the slouch posture (p<0.05*, p<0.01**). Solid bars indicate upright sitting, hatched bars indicate slouch sitting; dark grey is left-sided muscles, light grey is right-sided muscles.

The comparison between upright versus slouch sitting EMG amplitude revealed significant posture-related differences in bilateral LD and ES, and a unilateral posture-related difference in OE. In each of the three muscle groups, the upright sitting posture had higher average muscle activity than the slouch sitting posture: nine participants had higher OE muscle activity during upright versus slouch sitting (right-side OE upright: 1.22-7.72 mV, slouch: 2.25-2.83 mV, p=0.03); for the left and right side LD, nine and ten participants respectively had higher LD muscle activity during upright sitting compared to slouch sitting; overall, the differences were significant on the left (upright: 2.40-3.31 mV, slouch: 2.37-2.88 mV, p=0.004) and right (upright: 2.37-3.15 mV, slouch: 2.30-2.87 mV, p=0.01). Similarly for ES, which showed the greatest difference in average muscle activity between upright and slouch postures, all ten participants had higher mean EMG amplitudes during upright compared to slouch sitting on both the right and left sides, resulting in overall statistical significance in average muscle activity between postures on the left (upright: 2.94-8.18 mV, slouch: 2.35-6.13 mV, p=0.004 [WSR]) and
the right (upright: 2.49-8.78 mV, slouch: 2.33-8.60 mV, p=0.002 [WSR]). No significant difference in muscle activity was found between postures for left-side OE (upright: 2.48-6.73 mV, slouch: 2.47-7.91 mV, p=0.27) or bilateral RA (left upright: 2.19-3.76 mV, slouch: 2.16-3.52 mV, p=0.21; right upright: 1.22-7.72 mV, slouch: 1.18-7.66 mV, p=0.49).

There are two important caveats to consider when interpreting the muscle activity results from this study. The first is that the EMG signal recorded from both ventral trunk muscles, RA and OE, appeared to be contaminated. Specifically, in eight participants’ right-side RA (six were bilateral) and seven participants’ left-side OE multi-trial recordings, there was a pulsing signal throughout the EMG trace. Representative EMG amplitude traces from participant AB-3 shows bilateral recordings from each muscle group during upright and slouch sitting (Figure 15); a pulsing signal can be seen in right-side RA and left-side OE. It seems likely that the signal embedded in the left-side OE EMG recording is a radiating arterial pulse. A signal with a similar profile being detected on the right RA may represent pulse detection of the external iliac artery or one of its abdominal branches (either of the inferior epigastric or deep circumflex arteries).

The second caveat is that the absolute values of the EMG amplitudes that were recorded during this study were quite small – on the scale of single digit millivolts (mV). For two of the muscles that showed significant difference between the upright and slouch postures (right side OE, and bilateral LD), the absolute difference between conditions was on the minute scale of tenths of millivolts (or a tenth of a thousandth [$10^{-4}$] of a volt). Given the relatively low and constant impedances of the EMG recording signals (Appendix G), the significance of this result is likely due to the very small variance within each condition, thus representing a mathematical significance rather than a physiologically meaningful difference.
Figure 15. Representative 10s epoch EMG amplitude traces from participant AB-3. For each muscle, a 30s epoch of bilateral muscle activity is shown. Traces from both upright and slouch sitting conditions are superimposed; solid lines represent slouch sitting, dotted lines represent upright sitting. Note the high-amplitude pulses in right-side RA and left-side OE EMG traces.
5.2 SCI participants

In this subsection, results are discussed as individual case studies: trends within each set of experimental trials for a given variable were discussed in the same approach used for AB participants, followed by a tabular summary for each variable. A summary of individual SCI case study outcomes for all variables can be found in Appendix H. The first trial of each session was always an ‘unsupported sitting’ trial; this trial and the quiet breathing respiratory function outcome measures will be referred to as the ‘baseline.’ Since no statistical analysis of this data was feasible, the outcomes reported for AB participants served as a basis for comparison. For example, for the interpretation of SCI participants’ outcomes, a relative change in quiet breathing respiratory function of less than 5% will not be considered meaningful.

Three individuals with SCI volunteered to participate in this study. None had a history of chronic respiratory disease, nor had an acute respiratory episode at the time of testing; two participants were current smokers. A summary of the anthropometric characteristics of each of the three SCI participants is presented first (Table 12).

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age (y)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>BMI</th>
<th>Neurological level of injury (completeness)</th>
<th>Post injury (mo)</th>
<th>Sensation</th>
<th>Pack years</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI-1</td>
<td>25</td>
<td>180.3</td>
<td>61.2</td>
<td>18.8</td>
<td>C5 (I)</td>
<td>31</td>
<td>Yes</td>
<td>8</td>
</tr>
<tr>
<td>SCI-2</td>
<td>25</td>
<td>170.2</td>
<td>72.0</td>
<td>24.9</td>
<td>C5 (MC)</td>
<td>36</td>
<td>SOME</td>
<td>NS</td>
</tr>
<tr>
<td>SCI-3</td>
<td>33</td>
<td>175.3</td>
<td>79.4</td>
<td>25.9</td>
<td>C5-6 (MC)</td>
<td>192</td>
<td>Limited</td>
<td>2.25</td>
</tr>
</tbody>
</table>

BMI – body mass index; I – incomplete; MC – motor complete; NS – never smoker
Case study #1

Participant SCI-1 was a 25 year old male, with a C5 incomplete injury sustained in 2009 – some motor function and sensation below the level of the lesion were present at the time of participation. For example, SCI-1 was capable of transferring independently and could easily achieve sitting balance in a self-chosen posture. He had no history of respiratory disease or illness; he was a current smoker with a pack-year history of 8. His self reported height and weight translated to a BMI at the low end of the normal range \(^{ix}\) (18.8). SCI-1 completed eight experimental trials in the following sequence: unsupported sitting, AP-FES, ML-FES, unsupported sitting, FES-ALL, unsupported sitting, AP-FES, and ML-FES.

Case study #2

Participant SCI-2 was a 25 year old male, who sustained a C5 motor complete injury (light touch sensation was preserved) in 2008. SCI-2 required assistance to transfer to and from the sitting apparatus; he required initial external support in order to achieve sitting balance in a self-chosen posture and occasional externally-provided support (i.e. leaning against a back support). He had no history of respiratory disease or illness and was a non-smoker. His self-reported height and weight translated to a BMI at the top end of the normal range (24.9). SCI-2 completed seven experimental trials in the following sequence: unsupported sitting, AP-FES, ML-FES, unsupported sitting, unsupported sitting, AP-FES, and ML-FES.

Case study #3

Participant SCI-3 was a 33 year old male, with a C5-C6 motor complete injury (minimal sensation preserved) sustained in 1995. SCI-3 required assistance to transfer to and from the sitting apparatus; he required initial external support in order to achieve sitting balance in a self-chosen posture and was able to do pressure relief using his upper limbs. He had no history of respiratory dysfunction; he had a pack-year history of 2.25. His self-reported height and weight translated to a BMI of 25.9. SCI-3 completed six experimental trials \(^x\) in the following sequence: unsupported sitting, AP-FES, AP-FES, unsupported sitting, ML-FES, unsupported sitting.

\(^{ix}\) According to the World Health Organization, normal body mass index falls between 18.5 to 24.9 kg/m\(^2\).

\(^x\) Although SCI-3 completed six experimental trials, COP data for only four trials was captured. The first AP-FES and second unsupported sitting trials (trials #2 and #4) do not have accompanying COP data.
5.2.1 Respiratory function

The results of spirometry testing for the SCI case studies revealed that two of the three participants had values of FVC and FEV\(_1\) that fell below the predicted lower limit of normal (SCI-1 and SCI-2), while all three participants’ FEV\(_1\):FVC ratios were elevated (Table 13).

<table>
<thead>
<tr>
<th>Table 13. Spirometry results for SCI participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spirometry</td>
</tr>
<tr>
<td>FVC (L)</td>
</tr>
<tr>
<td>FEV(_1) (L)</td>
</tr>
<tr>
<td>FEV(_1)/FVC</td>
</tr>
<tr>
<td>SCI-1</td>
</tr>
<tr>
<td>FVC (L)</td>
</tr>
<tr>
<td>FEV(_1) (L)</td>
</tr>
<tr>
<td>FEV(_1)/FVC</td>
</tr>
<tr>
<td>SCI-2</td>
</tr>
<tr>
<td>FVC (L)</td>
</tr>
<tr>
<td>FEV(_1) (L)</td>
</tr>
<tr>
<td>FEV(_1)/FVC</td>
</tr>
</tbody>
</table>

Actual – measured by spirometry; % Pred – percentage of predicted values; all spirometric reference values calculated using equations from Hankinson et al.[110]

In the case of participant SCI-1, the baseline unsupported sitting trial indicated that his respiratory strategy was different from the average AB participant, with a reduced V\(_T\) and an elevated RR. The respiratory components combined to reveal a V\(_E\) that was similar to the AB group average. The effects of surface FES on the quiet breathing respiratory function of SCI-1 appear to be most positive in the latter half of the experimental trials, where V\(_T\) remains stable during trials with and without application of surface FES, and the initial negative effect of surface FES on RR is dampened (Table 14). During the final three trials (unsupported sitting, AP-FES, ML-FES), V\(_T\) and V\(_E\) were consistently greater than all preceding trials; during these trials, RR was consistently higher than the baseline unsupported sitting trial. For participant SCI-1, surface FES had the greatest effect on RR, with the ML-FES configuration demonstrating the only meaningful (albeit negative) change (a 6 % increase on average compared to unsupported sitting); however, the resultant overall change was diminished by a small decrease in V\(_T\) (2 %),
such that the maximum difference in $V_E$ across all conditions relative to the average of unsupported sitting trials (an increase of 3% achieved during ML-FES) was not considered meaningful.

Table 14. Quiet breathing respiratory function per trial for SCI-1 during sitting with and without surface FES

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>US</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AP-FES</td>
<td>0.54</td>
<td>0.45</td>
<td>0.45</td>
<td>0.45</td>
<td>0.54</td>
<td>0.56</td>
<td>0.55</td>
<td>0.55</td>
</tr>
<tr>
<td>V_T (L)</td>
<td>9.82</td>
<td>8.30</td>
<td>9.55</td>
<td>8.40</td>
<td>9.48</td>
<td>10.77</td>
<td>11.15</td>
<td>10.30</td>
</tr>
</tbody>
</table>

For participant SCI-2, although his initial $V_E$ measured below the AB group average, the contributing $V_T$ was very close to, and the RR actually lower than, the average AB participant. The application of surface FES on participant SCI-2 in either configuration produced alterations in quiet breathing respiratory function. Following the baseline unsupported sitting trial, the first surface FES trial (AP-FES) produced decreases in both $V_T$ (24%) and $V_E$ (6%) with a concomitant increase in RR (22%). Over the remainder of the experimental trials, these initial effects were ameliorated and reversed in unsupported and surface FES sitting conditions (Table 15). The final four trials of the experimental session (in sequential order: two unsupported sitting, AP-FES, ML-FES) all had absolute values of RR that were lower, and values of $V_T$ that were higher, than each of the three preceding trials, including the baseline unsupported sitting trial. The absolute values of $V_E$ during the final three trials (unsupported sitting, AP-FES, ML-FES) were also higher than each of the preceding trials. Notably, the highest values of $V_T$ and $V_E$, and the lowest values of RR were achieved in the final two trials – AP-FES and ML-FES.
Participant SCI-3 had an initial $V_E$ that exceeded the AB group average, achieved by an elevated $V_T$ coupled with a reduced RR compared to the average AB participant. Participant SCI-3 was the least tolerant of the sitting apparatus and the surface FES protocol due to difficulty in achieving adequate weight relief resulting in increasing pressure over the ischial tuberosities and increasing sensitivity to the stimulation resulting in autonomic dysreflexia-like symptoms. As such, a limited number of trials were completed, including only one ML-FES trial. While the first AP-FES trial with SCI-3 was accompanied by directional changes similar to those in the cases of SCI-1 and SCI-2 – the initial 12 % drop in $V_T$ and 21 % increase in RR – there was no amelioration of these changes (Table 16). After a second AP-FES trial during which little change was measured, RR continued to increase until the last trial (unsupported sitting), when it dropped to its lowest value for the entire experimental session. $V_T$ increased during the last two trials, but never regained the baseline value; this included the 28 % increase in $V_T$ with the application of ML-FES compared to the unsupported sitting trial immediately preceding. The ML-FES trial (second from last) was also characterized by the highest RR measured during the experimental session (a 5 % increase in RR compared to the preceding unsupported sitting trial), while the resultant 30 % increase in $V_E$. Both changes in RR and $V_E$ were within the range to be considered meaningful.

---

Table 15. Quiet breathing respiratory function per trial for SCI-2 during sitting with and without surface FES

<table>
<thead>
<tr>
<th>Trial</th>
<th>Quiet breathing respiratory function</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>US</td>
</tr>
<tr>
<td>$V_T$ (L)</td>
<td>0.72</td>
</tr>
<tr>
<td>RR (min$^{-1}$)</td>
<td>11.68</td>
</tr>
<tr>
<td>$V_E$ (L/min)</td>
<td>8.18</td>
</tr>
</tbody>
</table>

US - unsupported

---

$\text{xi}$ As in all cases, the participant was guiding the continuation of the experimental session based on their own perceptions of well-being.
5.2.2 Seated postural stability

No individual participant had an absolute minimum to maximum (min-max) range that exceeded 5% of the mean total Fz for either force plate (Table 17). The min-max range for each the seat and feet force plates for both SCI-1 (2 % and 1 %, respectively) and SCI-3 (1 % for both force plates) was very close to the average difference between conditions for AB participants (1 % of the mean total Fz; see Table 7), which was not significantly different. Participant SCI-2 had a larger min-max range for both the seat force plate (4 % mean total Fz) and the feet force plate (5 % mean total Fz), which represented a range 2.5 times greater than the maximum proportion of mean total Fz reported for AB participants; no consistent pattern of force distribution was observed among the sitting conditions (e.g. higher vertical forces generated through either force plate was not restricted to a particular sitting condition).
Table 17. Minimum and maximum vertical reaction forces for SCI participants (n=3) for all trials

<table>
<thead>
<tr>
<th>Vertical force (N)</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI-1</td>
<td>Seat</td>
<td>495.85</td>
</tr>
<tr>
<td></td>
<td>Feet</td>
<td>125.43</td>
</tr>
<tr>
<td>SCI-2</td>
<td>Seat</td>
<td>598.81</td>
</tr>
<tr>
<td></td>
<td>Feet</td>
<td>129.11</td>
</tr>
<tr>
<td>SCI-3</td>
<td>Seat</td>
<td>618.04</td>
</tr>
<tr>
<td></td>
<td>Feet</td>
<td>170.08</td>
</tr>
</tbody>
</table>

For SCI-1, the baseline unsupported sitting trial revealed a very different level of sitting stability compared to AB participants sitting in similar postures (Table 18). Compared to AB participants in both upright and slouch sitting, SCI-1’s MDIST was more than three times greater. Coupled with an MVELO similar to the AB group average, the resultant MFREQ was considerably lower than for the average AB participant. Application of surface FES in either configuration increased the MVELO in all but one trial (trial 3 – ML-FES), at the same time decreasing the mean COP displacement over the 60s trial. Comparing AP-FES, ML-FES, and FES-ALL, only AP-FES achieved a ratio of MDIST: MVELO comparable to the average AB participant, indicated by similar values of MFREQ.
Table 18. Seated postural stability for each trial (n=8) for SCI-1 during sitting with and without surface FES

<table>
<thead>
<tr>
<th>Seated postural stability</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDIST (mm)</td>
<td>4.93</td>
<td>1.80</td>
<td>1.91</td>
<td>1.08</td>
<td>2.70</td>
<td>2.97</td>
<td>2.06</td>
<td>2.49</td>
</tr>
<tr>
<td>MVELO (mm/s)</td>
<td>3.23</td>
<td>4.25</td>
<td>2.88</td>
<td>1.96</td>
<td>4.90</td>
<td>4.24</td>
<td>4.72</td>
<td>3.28</td>
</tr>
<tr>
<td>MFREQ (Hz)</td>
<td>0.10</td>
<td>0.38</td>
<td>0.24</td>
<td>0.29</td>
<td>0.29</td>
<td>0.23</td>
<td>0.37</td>
<td>0.21</td>
</tr>
<tr>
<td>US - unsupported</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

By visual inspection of the COP excursion for SCI-1 (Figure 16), unsupported sitting shows the largest magnitude deviations, except for trial 4 (top row, middle plot); AP-FES (trials 2, 7) appears to reduce the magnitude and frequency of sudden/extreme deviations, but in trial 7 some phasic movement (e.g., a phenomenon resembling breathing frequency) is emphasized. In ML-FES (trials 3, 8), some deviations do occur, but the magnitude is ameliorated to some degree. The effect of all-around stimulation in FES-ALL has a similar effect as ML-FES, with deviations occurring at a lower magnitude than unsupported sitting.
Figure 16. COP excursion plots for SCI-1 during all sitting trials. Plots numbered in order of trial sequence: 1-unsupported, 2-AP-FES, 3-ML-FES, 4-unsupported, 5-FES-ALL, 6-unsupported, 7-AP-FES, and 8-ML-FES. Scale of y-axis is -50 mm to 50 mm; x-axis is 60 s. Displacement in the AP direction indicated by solid line, and in the ML direction by the dotted line.

The COP excursion plots for SCI-2 revealed an even greater magnitude of COP deviations during unsupported sitting (Figure 17). In the baseline trial, MDIST for SCI-2 was more than four times greater than the AB group average, and MVELO was similarly elevated, resulting in an MFREQ of less than half that measured among AB participants (Table 19). Application of surface FES following the initial trial decreased both MDIST and MVELO, although only AP-FES achieved an MFREQ most similar to the AB group average. Mean COP distance reached a maximum during two back-to-back unsupported sitting trials with very large COP deviations (Figure 17, plots 4, 5); instability was then mitigated in both surface FES configurations, indicated by reduced values of MDIST and MVELO relative to the two preceding trials, although the values suggest that greater stability was achieved during AP-FES. Furthermore, the second trials of AP-FES and ML-FES (Figure 17, plots 6, 7) both showed the phasic movement
emphasis as in SCI-1. Overall, AP-FES achieved the lowest values of MDIST and MVELO, as well as the value of MFREQ most similar to the AB group average. Although AP-FES did not achieve the lowest values of MDIST or MVELO (trial 4 – unsupported sitting), both applications of AP-FES achieved values of these COP components translating to an MFREQ most similar to the AB group average.

Figure 17. COP excursion plots for SCI-2 during all sitting trials. Plots numbered in order of trial sequence: 1-Unsupported, 2-AP-FES, 3-ML-FES, 4-unsupported sitting, 5-unsupported sitting, 6-AP-FES, and 7-ML-FES. Scale of y-axis is -50 mm to 50 mm; x-axis is 60 s. Displacement in the AP direction indicated by dashed lines; dotted lines correspond to displacement in the ML direction.
Table 19. Seated postural stability for each trial (n=7) for SCI-2 during sitting with and without surface FES

<table>
<thead>
<tr>
<th>Seated postural stability</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDIST (mm)</td>
<td>6.42</td>
<td>1.04</td>
<td>3.34</td>
<td>11.53</td>
<td>5.45</td>
<td>2.78</td>
<td>1.81</td>
</tr>
<tr>
<td>MVELO (mm/s)</td>
<td>6.94</td>
<td>1.46</td>
<td>2.58</td>
<td>5.70</td>
<td>3.68</td>
<td>3.17</td>
<td>1.79</td>
</tr>
<tr>
<td>MFREQ (Hz)</td>
<td>0.17</td>
<td>0.22</td>
<td>0.12</td>
<td>0.08</td>
<td>0.11</td>
<td>0.18</td>
<td>0.16</td>
</tr>
<tr>
<td>US - unsupported</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The lack of sudden or extreme COP deviations on the COP excursion plots is somewhat misleading about the seated postural stability of SCI-3 (Figure 18). MDIST was greater in all conditions than the AB group average; this was also true for MVELO, but to a lesser degree in unsupported sitting trials (Table 20). In both unsupported sitting trials, MFREQ was below the AB group average. Due to missing data for trials 2 and 4 (AP-FES and unsupported sitting, respectively), only the results from remaining trials 3, 5, and 6 will be described. Compared to the baseline trial, application of AP-FES almost doubled MVELO, at the same time decreasing MDIST by a lesser degree; the result was an increased MFREQ, although the magnitude of the increase elevated the value of MFREQ above the AB group average. The remaining trials, ML-FES followed by unsupported sitting, had very similar MFREQ values, although the application of ML-FES increased COP displacement to such an extent that MFREQ dropped even further. Of the four trials, the AP-FES condition achieved MDIST and MVELO values corresponding to the MFREQ most similar to the AB group average, though it was still elevated.
Figure 18. COP excursion plots for SCI-3 during sitting with and without surface FES. Plots are numbered in order of sequence in the experimental session; 1-unsupported sitting, 2-AP-FES (not shown), 3-AP-FES, 4-unsupported sitting (not shown), 5-ML-FES, 6-unsupported sitting. Scale of y-axis is -50 mm to 50 mm; x-axis is 60 s. Displacement in the AP direction indicated by dashed lines; dotted lines correspond to displacement in the ML direction.

Table 20. Seated postural stability for each trial (n=6*) for SCI-3 during sitting with and without surface FES

<table>
<thead>
<tr>
<th>Seated postural stability</th>
<th>1</th>
<th>2*</th>
<th>3</th>
<th>4*</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDIST (mm)</td>
<td>2.84</td>
<td>--</td>
<td>1.96</td>
<td>--</td>
<td>6.62</td>
<td>3.95</td>
</tr>
<tr>
<td>MVELO (mm/s)</td>
<td>3.14</td>
<td>--</td>
<td>6.05</td>
<td>--</td>
<td>7.12</td>
<td>3.86</td>
</tr>
<tr>
<td>MFREQ (Hz)</td>
<td>0.18</td>
<td>--</td>
<td>0.49</td>
<td>--</td>
<td>0.17</td>
<td>0.16</td>
</tr>
</tbody>
</table>

US - Unsupported; * - two trials without COP data
5.2.3 Questionnaire responses

Responses to the questionnaire and other comments from the SCI participants are summarized in Appendix F. In general, SCI-1 expressed a preference for the trials with stimulation turned on; he felt that stimulation improved his sitting posture, but did not describe any difference in ease of breathing. Participant SCI-2 did not respond to the questions due to lack of time. Participant SCI-3 mainly reported on feeling spasticity and increasing symptoms of autonomic dysreflexia as the experimental session progressed.
Chapter 6

6 Discussion

In this section, an overview of the main results will be discussed for AB participants and each of the three SCI case studies. A discussion of the implications of the findings, limitations of these studies, and future work will follow.

6.1 Able-bodied participants

The outcomes of the experiments with AB participants confirm that sitting in an upright posture, compared to a slouch posture, is beneficial in terms of optimizing $V_E$ by increasing $V_T$ without changing RR. The changes in breathing volume substantiate the findings of Lee et al. [134] who found significant changes in the shape of the chest wall between an upright sitting posture and a slouch sitting posture, which specifically included a decrease in the diameter of the thorax at the abdominal level; such a decrease in the diameter of the thorax found by Lee et al. suggests that abdominal wall stiffness, and therefore IAP, is increased in upright sitting versus slouch sitting, and improves the function of the diaphragm. Lee et al. did not distinguish if this difference was biomechanical or caused by a change in muscle activation.

In AB individuals, one would expect to find differences in EMG activity between upright and slouch sitting postures to be similar bilaterally. As predicted, there was an increase in EMG activity in LD and ES bilaterally in upright sitting; no difference was detected between postures in RA bilaterally, which may be attributed to the fact that RA does not explicitly participate in the maintenance of upright posture. The difference in OE where there was increased activity in the left-side only during upright sitting may be attributed to measurement error due to signal contamination by arterial pulses. These findings substantiate the characterisation of muscle activation patterns between upright and slouch sitting by O’Sullivan et al. [121], who found an increase in lumbar multifidus activity without changes in abdominal muscle activity, which was accompanied by increases in lumbar lordosis and forward rotation of the pelvis in an upright compared to a slouch sitting posture. Analysis of the kinematic data recorded in this thesis might further support the findings of O’Sullivan and colleagues.

In the present study, upright sitting was associated with higher overall values of MDIST and MVELO compared to slouch sitting. Increased MDIST or MVELO does not necessarily indicate
a lack of seated postural stability in upright sitting, but merely reflects the resultant dynamics of inherent biomechanical differences between the two postures. It may be that upright sitting raises the centre of mass and consequently changes the resultant dynamics. Vette et al. [31] described the dynamic differences between sitting and standing: as the centre of mass increases in vertical height in standing, the trunk assumes a greater moment of inertia, and subsequently affects the displacement of the COP, increasing MDIST and MVELO.

Mean COP frequency, which reflects the relationship between MDIST and MVELO, did not change significantly between upright and slouch sitting postures. Interestingly, the values of MFREQ found by Prieto et al. [12] for AB young adults in two standing conditions were similar to the values reported in this study (from Prieto et al.: MFREQ=0.374±0.092 and 0.400±0.120, for n=20 young adults standing with their eyes open and eyes closed, respectively). The similarities in the MFREQ among four different postural conditions allude to a potential optimization of the intact postural control system in young, AB individuals, at least in semi-static conditions. One might be interested to explore this more thoroughly in other conditions in order to determine if there is validity to this suggested optimization.

### 6.2 SCI participants

Interpretation of the results from the three SCI case studies is more complex, owing to the heterogeneous characteristics of the participants. Common among all three participants was an elevated FEV₁ to FVC ratio. The measured values were between 5-18% greater than the predicted values calculated using the equations of Hankinson et al.[110] An elevated FEV₁ to FVC ratio (>85-90%) is common among individuals with neuromuscular disease and weakened respiratory musculature [92]. In contrast, only SCI-1 had a notably different respiratory strategy (decreased VT and increased RR) compared to AB participants at baseline. It may be that both SCI-2 and SCI-3, who both had motor complete injuries, have gained increased quiet breathing respiratory function through compensatory strategies necessitated by the dysfunction of key respiratory muscles.

In the baseline unsupported trials, all SCI participants displayed more postural instability compared to AB participants. Overall, Grangeon et al. [53] found increased MDIST and MVELO in upright, unsupported sitting in SCI participants; these results were pooled from fourteen individuals with SCI with wide-ranging injuries (i.e. levels of injury ranging from C3 to
L1 with varying degrees of sensory and motor completeness). Our findings were similar for MDIST, but not for MVELO, which was only greater for SCI-2. In addition, the MFREQ for all three SCI participants was reduced, which may be an important measure to characterize seated postural stability in this population.

Each of the three SCI participants in this study had similar levels of injury, ranging between C5 and C6, but with different degrees of neurological sensorimotor dysfunction, and each individual received surface FES to activate the same set of trunk muscles. However, there were highly variable responses to stimulation among each of the case studies in terms of both experimental outcomes and autonomic function, which may be accounted for by the differences in completeness of injury and ultimate neurological sensorimotor integrity of the trunk and respiratory muscles, as well as the differences in duration of injury of each individual which may lead to different behavioural adaptations to optimize postural stability and respiratory function. Over the course of the experimental sessions for all three participants, conflicting alterations in stability and breathing were recorded during FES trials.

**Case study #1**

In the experimental session with SCI-1, two of the eight trials completed (trials 2, 7), which were both AP-FES trials, appeared to have minimized COP deviations compared to unsupported sitting. Compared to the baseline trial, both AP-FES trials showed a simultaneous decrease in MDIST and increase in MVELO; by visual inspection, the excursion of the COP during these two trials was at a minimum compared to trials in other conditions. During trials 2 and 7, MFREQ reached a value comparable to the AB group average. However, the outcomes for quiet breathing respiratory function during AP-FES were not positive or consistent: in trial 2, \(V_T\) fell markedly, but no change in respiratory rate occurred; in trial 7, there was essentially no change in \(V_T\) and a moderate increase in RR. For SCI-1, the AP-FES condition achieved the greatest seated postural stability, but the functional detriment to breathing is problematic; thus, it would be important to explore how altering the AP-FES parameters might reduce the negative impact on quiet breathing respiratory function in individuals like SCI-1 with incomplete injuries.

**Case study #2**

For participant SCI-2, application of FES improved seated postural stability: MDIST and MVELO decreased in both FES conditions (AP-FES, ML-FES) compared to all trials of unsupported sitting. This was evident by visual inspection as COP excursion was reduced;
however, MFREQ changed minimally between conditions. Overall, FES also increased tidal volume and minute ventilation, without large increases in respiratory rate; AP-FES and ML-FES had opposite effects on $V_T$ and RR in the first versus second trials of each condition, with the most positive effects on quiet breathing respiratory function realized in the second set of FES trials. Thus, participant SCI-2 benefited mostly from FES in all cases, with no significant loss of quiet breathing respiratory function; a period of acclimatization to the sensation of FES may benefit individuals who initially experience breathing deficits due to FES.

Case study #3
The results from the experimental session with participant SCI-3 are highly conflicting in terms of seated postural stability and breathing – visual inspection of the COP excursion plots is not informative. AP-FES conferred increased stability at the cost of tidal volume, while ML-FES increased tidal volume at the cost of stability; in fact, it seems that the least impairments in both systems were realized in the baseline unsupported sitting trial. The current FES paradigms were problematic in this case study; this may be attributed to the high levels of stimulation that were required to achieve changes in muscle activation, and potentially contributing to the autonomic dysreflexia-like symptoms reported by SCI-3. A new FES paradigm including stimulation of ES and OE simultaneously might be considered to explore whether better seated postural stability can be achieved without the negative effects on quiet breathing respiratory function; however, the potential intolerance of FES by some individuals with SCI who have significant loss of sensation also demands consideration.

With such a small sample size of SCI participants and the heterogeneous nature of their individual anthropometrics and injury characteristics, it is very challenging to generalize the results. What has been demonstrated is that surface FES has the potential to alter seated postural stability and quiet breathing respiratory function in individuals with SCI, while the current FES stimulating paradigm has limitations in augmenting both systems simultaneously. Using FES at levels tolerable by individual participants, it is possible to alter the amount of postural adjustments as measured by mean COP velocity, the associated level of stability as measured by mean COP distance, and the breathing pattern as measured by tidal volume and respiratory rate – how to control the consequent changes is the next challenge. In future studies, another important development of the experimental protocol will be to determine the criteria for what direction and magnitude of change in the measured variables will be considered clinically important.
6.3 Implications and future directions

It is well established that, with an intact postural control system, a person is capable of achieving stability in different body positions including standing and sitting, and that stability is compromised among individuals with SCI. As described in Chapter 2, the maintenance of a physiologically sound posture requires active recruitment of the musculature of the trunk, which is weakened among individuals with SCI; the LOS have also been quantified and contrasted among AB and SCI individuals in previous studies, using reaching tasks to challenge the postural control system, and the consequent muscle activation patterns for realizing the LOS have been characterized, at least for some directions. However, the consequences of compensatory seated postural stability on the respiratory system in SCI have largely been overlooked, despite evidence that COP displacement is closely linked to breathing. Hodges et al. [135] describe that in a semi-static condition, movement of the COP in phase with respiratory rate is complicated by the changing shape of the thorax during the respiratory cycle, inherent trunk stiffness (or lack thereof), and the basic posture of the person. Furthermore, in dynamic conditions, the activity of the diaphragm is not isolated to a respiratory function, but participates in trunk stabilization prior to perturbation [136] and during movement of the upper limb [137]. In the intact person, a highly redundant musculature permits the recruitment of inherently non-postural muscles to perform multiple functions. In the individual with SCI, recruitment of non-postural muscles for the purpose of maintaining seated postural stability could result in poor performance of the secondary function and loss of intended function (e.g. breathing, in the case of the diaphragm). Performance of a functional task requiring sitting balance and use of the upper limbs will be limited if the individual must hold their breath!

This research project was undertaken in order to provide insight into the physiological consequences of trunk muscle stimulation in people with SCI: the development and implementation of a neuroprosthesis for sitting balance should aim to impart the highest level of functional improvement on the target system – postural control – at the same time minimizing disturbances to other physiological processes such as breathing. In this thesis, the use of FES with individuals with SCI was not free from disruptions to either seated postural stability or quiet breathing respiratory function. Considerations of the FES paradigm used in this project may be warranted.
A closed-loop control system could provide a sophisticated stimulation paradigm to more closely reflect the natural characteristics of muscle activation that provide postural stability and support quiet breathing respiratory function. The stimulation signal used in the current experimental sessions was continuous throughout a given FES trial, which might be considered rudimentary in comparison to signal characteristics in use by other research groups. It is possible to develop a feedback control system for FES for specific motor functions. For instance, Gollee et al.[138, 139] developed a control system for FES-assisted breathing in individuals with tetraplegia: the control system was able to recognize different respiratory patterns, including quiet breathing and coughing, as well as discriminate non-regular respiratory patterns, such as breathing while talking, and to trigger the onset of the appropriate stimulation signal. In three of four individuals with complete injuries at the cervical level, $V_T$ increased during quiet breathing assisted by FES. Gollee et al.[138] also reported the ongoing need to adjust stimulation intensity due to muscle fatigue. Other FES interventions have suggested this type of feedback control system – or closed-loop controller – to optimize motor training in SCI [140, 141], and in our laboratory Masani et al.[27] have developed a model of postural control during sitting that could inform the development of such a controller. It may be possible to use an approach similar to that of Gollee and colleagues to develop a multi-function control system for FES-assisted postural control paired with quiet breathing respiratory function. This would be a more sophisticated method for applying FES that would be more sensitive to the subtle dynamic changes in both respiratory and postural control systems.

6.4 Limitations of this study

There are limitations of these studies that are relevant to both the participants and the experimental paradigm itself.

The first consideration is of the sudden or extreme COP deviations experienced by AB participants during the experimental sessions. This researcher suspects that these events may be due in part to postural adjustments precipitated by discomfort from prolonged sitting or due to a sudden increase in wakefulness (hypnic jerk) [142]. Some AB participants did report altered states of alertness during the experiments. To avoid this effect in the study with SCI participants, music was played in the background to provide an attention stimulus. The sudden or extreme COP deviations during trials of this nature warrant further investigation.
The time requirement for participation in this study was a limitation for individuals with SCI. All participants were seated on the surface of a force plate, which was covered by a yoga mat. (Several different foam cushions were considered for this purpose, but the contour and thickness were problematic in obtaining noise-free COP data.) At the conclusion of the session with SCI-3, the mat was noted to have two indentations underneath the buttocks of the participant. The indentations occurred despite multiple weight relief manoeuvres. This was of major concern to the researcher and academic supervisor, who followed-up with the participant daily until the participant felt sure that the risk of developing a pressure sore had passed. Participant safety must remain a priority at all times; in future studies, adequate cushioning of the sitting apparatus must be of paramount importance in conducting experimental trials with individuals with SCI.

Some aspects of this study might have been improved. The analysis of EMG signals could have been improved in two ways: firstly, by normalizing the signal detected during experimental trials to the participants’ maximum voluntary contraction (MVC) against external resistance; MVC normalization would provide a more standardized value for comparison. However, this approach is highly problematic for individuals with SCI: firstly, because MVC normalization of the trunk requires completion of an extended set of manoeuvres [143] that are not always possible with this population, and secondly, because the characteristics of paralysis are highly variable and individually specific among this population. Secondly, there are some methodologies that could be used to better filter EMG signals: specifically, methods to filter heart rate and arterial pulses from trunk EMG that could be used in future experiments. For SCI participants, the initial effects of FES on breathing (i.e. sudden decrease in V\text{T} and increase in RR) that were not seen in subsequent trials suggest that an acclimatization period to the sensation of FES might have reduced the possibility of an autonomic reaction to the stimulation. However, this may be important for further investigation to determine whether this is a ubiquitous effect of FES in the SCI population.

Finally, in examining the effects of FES on respiratory function during quiet breathing, it would be pertinent to utilize a methodology that would provide precise determination of the function of the diaphragm and the trunk musculature for each individual case. This step is paramount in determining the precise factors within an experimental protocol that are responsible for altering respiratory function during quiet breathing in such a heterogeneous population of individuals with SCI. The standard approaches to neurological assessment for SCI do not capture functional
aspects of the trunk. For example, it is possible to test the intactness of dermatomes across the trunk to determine sensation, however these are not relevant to the function of the diaphragm and currently there is not a robust motor assessment for trunk musculature, making the clinical assessment of trunk impairment at best uninformative.

The methodological considerations for future studies should include detailed neuroimaging of the cord at the level of the injury; systematic electrophysiological assessment of the trunk musculature, and kinematics of chest wall movement. Precise assessment of diaphragmatic function via a standardized methodology such as fluoroscopic imaging [144], ultrasound imaging [145], or measurement of sniff transdiaphragmatic pressure with an esophageal (and gastric) balloon-catheter system [146] or magnetic phrenic nerve stimulation [147] could be used.

In future studies, it would be important to consider inclusion of a subset of these methodologies either for screening or during experimental protocols (where permitted) in order to gain a detailed presentation of how the SCI pathology manifests itself in the interplay between postural control and respiratory function.
Chapter 7

7  Conclusions

“We live in a time when the words impossible and unsolvable are no longer part of the scientific community's vocabulary. Each day we move closer to trials that will not just minimize the symptoms of disease and injury but eliminate them.”

– Christopher Reeve

In this thesis, the changes in quiet breathing respiratory function, muscle activity, and seated postural stability have been described between upright and slouch sitting postures. Increases in $V_T$, trunk extensor muscle activity, and COP displacement were recorded in AB individuals sitting in an upright posture compared to the slouch posture. Application of surface FES in individuals with SCI indicated that involuntary activation of trunk muscles could alter both quiet breathing respiratory function – $V_T$ and RR – and seated postural stability, although control of the direction and magnitude of the changes was inconsistent. There was a trend within the AP-FES condition to increase the efficiency of the postural control system without imparting a disturbance to respiratory function among individuals. However, a more thorough and detailed assessment of the neurophysiological integrity of the sensorimotor function of the trunk as well as the existing functional respiratory status of each individual participant are required to prescribe a more discriminating surface FES paradigm. This would be a necessary set of studies before investigating a more sophisticated, closed-loop FES control system.

People living with disability due to SCI face challenges in achieving independence. Indeed, becoming competent in the control of body movement precedes learning new skills and strategies that may be required to perform self-care, mobility, and participation in society. Many people with SCI have been able to regain some degree of independence by increasing control over bodily functions, facilitating movement and task performance of the upper and lower limbs, enhancing respiratory function, and improving posture and stability using FES interventions for therapy and restoration of function. In the context of SCI, individualized stimulation paradigms are significant owing to the highly heterogeneous injury characteristics within the SCI population. Future studies could be performed to test more sophisticated and individualized surface FES paradigms for improving posture, quiet breathing respiratory function, and seated postural stability.
References


Appendix A

List of equipment

The experimental apparatus consisted of:

- A digital spirometer (SpiroPro® CT, VIASYS Healthcare, San Diego, CA) with disposable mouthpiece, to measure, record, and analyse values of respiratory capacity during a PFT.

- A portable cardiopulmonary exercise testing (CPET) system (MetaMax® 3X, Cortex Biophysik GmbH, Germany) with full face mask and head strap, to perform mixing-chamber analysis.

- A dual force platform (AccuswayPlus®, Advanced Mechanical Technology, Inc., Watertown, MA), in-built to a custom-made, height-adjustable sitting apparatus (conversion of hydraulic table base (Akron, UK) by REL, Toronto, ON), to capture COP of the seat; a second force platform arranged on the floor in front of the sitting apparatus to capture COP of the feet; the two force platforms together (also force plates) are the dual force plate setup.

- Eight 10mm silver-silver chloride disposable surface electromyography electrodes (BiPole electrodes, Bortec Biomedical, Calgary, AB), and EMG amplifier (Bortec AMT-8 EMG system, Bortec Biomedical, Calgary, AB) to capture bilateral muscle activity of select trunk muscles; ECG electrode for grounding EMG signals (Kendall Medi-Trace™, Covidien, Mansfield, MA).

- A digital 3D motion capture system (Optotrak® 3020, Northern Digital Inc., Waterloo, ON) to track the position of body segments using two position sensors, multiple custom-made rigid bodies and independently arranged infrared digital markers, and a six-marker digitizing probe (Northern Digital Inc., Waterloo, ON) to virtually mark and capture anatomical landmarks with respect to a rigid body within the global reference frame.

- A programmable 8-channel neuromuscular electrical stimulator (Compex Motion, Compex SA, Switzerland) and self-adhesive FES electrodes (5cm x 5cm Stimtrode, Cefar
Compex Medical, Sweden; 5cm x 9cm Stim Trode, Axelgaard Manufacturing Co., Denmark) to deliver surface FES to select trunk muscles (SCI individuals only).

- Data acquisition systems.
- Multiple computers (primary, secondary and tertiary DAQ).
- Miscellaneous: abrasive tape, medical tape, double-sided tape, conductive gel, hair clips, alcohol swabs, drop-line weights for aligning force plates, yoga mat (6mm in thickness), volt meter, safety harness, privacy screen.
- MetaSoft 3.9 software (Cortex Biophysik GmbH, Germany).
- LabVIEW 6.1 software (National Instrument, Austin, TX).
- NDI First Principles and 6D Architect (Northern Digital Inc., Waterloo, ON).
- MATLAB 7.0.4 software (The Mathworks Inc., Natick, MA).
Appendix B

Participant Information Sheet

Rehabilitation Engineering Laboratory

Toronto Rehabilitation Institute and University of Toronto

Full Study Title: Neuroprosthesis for Sitting Function in Spinal Cord Injury
Sub-Study: Measuring Postural Control and Respiratory Capacity
Principal Investigator: Professor Molly Verrier
Co-investigators: Meredith Kuipers (MSc Candidate), Dr. Milos Popovic

You are invited to take part in a research study. Before you agree to participate, it is important that you read the information below about the study. It describes the purpose of the study, the risks and benefits to yourself and your right to withdraw at any time. Make sure that all of your questions have been answered before you consent to participate.

1. What is the purpose of this study?

Functional electrical stimulation (FES) uses electricity to make muscles contract. FES can be used to get the muscles to do useful work. This is done by applying the right amount of electricity in the right places at the right time. Some people with limited or no control of muscle function have used FES for exercising their muscles.

Currently, the Rehabilitation Engineering Lab at the Lyndhurst Centre is developing an FES system for sitting balance. This system will allow patients with spinal cord injury to achieve sitting balance using a non-invasive stimulating technology. The purpose of this study is to see the effect of such stimulation on respiratory capacity and wellbeing.

2. How does this study work?

There are two parts to this study: Part 1 is a preliminary consultation, lasting no more than 30 minutes, and Part 2 is the experimental session lasting no more than 2 hours. During Part 1, we will measure your voluntary muscle activity using electromyography, a non-invasive procedure for detecting muscle contractions. Sticky electrodes will be put on the front and back of your torso (over your trunk muscles: abdominals and back muscles). Next, we will ask you to do some movements to activate certain muscles – this will allow us to determine what level of stimulation to apply during the experimental session. This should take about fifteen minutes. We will also ask you to perform three standard breathing tests; this is done by breathing voluntarily into a spirometer. This should take about ten minutes.

Part 2 is the experimental session, which may take place on a different day if you choose. At the start of each session, you will transfer to a padded seat equipped with a force plate, backrest, and side supports, and sit down. Your feet will be placed on a footrest. You will also wear a harness secured to an overhead beam to prevent falling during the entire session. If you need to, you can change position during the breaks to relieve any pressure points. Sticky electrodes will be put on the front and back of your torso (over your trunk muscles: abdominals and back muscles). Next, the researcher will test the electrodes to make sure that they are working properly. This will take less than 10 minutes. The electrodes will be used to stimulate your trunk muscles with FES. This will make your trunk muscles contract, and, as a result, alter your sitting posture.
Prior to the beginning of each trial, the researcher will adjust the intensity of the stimulation to your tolerance levels. If you can control your trunk muscles without FES, you will be suggested to perform the trials unassisted. During the experimental session, you will be asked to perform alternating trials of quiet sitting, reaching, and an eye-tracking task with and without FES of your trunk muscles. You will also be asked to wear a face mask attached to a cardiopulmonary exercise system (a device that measures respiratory rates and volumes, and gas exchange in the lungs) during the experiment. At the end of the session, the electrodes and the face mask will be removed.

Before, during, and after the experimental session we will evaluate your postural alignment, sitting stability, and respiratory capacity. For that, we will use an independent motion-capture system, a force plate that you will be sitting on, and a cardiopulmonary exercise testing system that the face mask is attached to; we will record these measurements while you sit unsupported and with FES-assisted sitting. After the experimental session we will request that you respond to a questionnaire to evaluate your experience using the FES system during sitting.

3. How long is this study?

You will participate in two sessions at the Lyndhurst Center of Toronto Rehab: the first session will not exceed thirty minutes in duration; the second session will not exceed two hours in duration.

4. What are the risks involved in this study?

There is no known risk of using a CPET system to measure breathing capacity. If you become uncomfortable while wearing the mask, you will be allowed to remove it and rest between experimental trials, if required.

There is a very small risk of skin irritation when using FES. However, we have never had this happen in our lab.

Subjects with a spinal cord injury could potentially experience autonomic dysreflexia (AD) due to FES during the training session, though the chances of FES induced AD occurring are extremely low. AD can happen when your blood pressure gets too high. Usually, AD is caused by your bladder being too full. AD can also be caused by other things like a blocked catheter or a urinary tract infection. If you start to get a bad headache during a session, please tell the researcher right away. This can be a symptom of AD. There is a doctor on call at the Lyndhurst Centre to help you if you get AD during an experimental session. AD can not happen due to FES after the training session. However, if you will experience signs of AD (sudden increase in your blood pressure, severe headache, flushed skin above the level of the injury, “goose bumps” below the level of the injury, sweating, anxiety, and nasal congestion) please seek urgent medical attention.

There is a very small risk of falling while attempting to maintain an upright posture during the tests. To prevent a fall during the standing the subject will wear a harness for the duration of the session and be closely monitored by at least two assistants for the duration of the trials.

5. What are the drawbacks of this study?

You will have to commit to two sessions at the Lyndhurst Centre, the first lasting no more than thirty minutes, the second lasting no more than two hours. You will have to make your own arrangements for traveling to and from the Lyndhurst Centre. You won’t receive any kind of compensation for participating in this study.
6. What are the potential benefits of this study?

Your trunk muscles will get some exercise from the electrical stimulation, which is healthy for your heart, and help to decrease a risk of such complications as pressure sores.

You may be helping the researchers in the Rehabilitation Engineering Lab develop a new and improved FES neuroprosthesis for sitting balance and to advance knowledge of the relationship between postural control and respiratory capacity in the SCI population.

7. What will happen to incidental findings?

During the course of this research, there is a possibility that the results of spirometry tests may suggest an undiagnosed respiratory problem greater than would be expected for a person with SCI alone. In this case, your physician at Lyndhurst Center will be notified for follow-up to ensure that you are assessed for a suspected respiratory problem.

8. How will personal information be protected in this study?

Your personal information will be stored in a locked filing cabinet or a password-protected computer file. This includes your name, date of birth, and phone number.

• Only the people in charge of the study will ever see this information.

• A special code will be used instead of your name on any documents or computer files that could be seen by other people. This code will not have anything to do with your name or date of birth. For example, John Smith might be the code Subject WXYZ.

• Only the people in charge of the study will know your special code.

• The researchers can include only your age, sex, or type of injury in a presentation or a report. For example, a researcher could say “a 35 year old male with a complete SCI at level T6 participated in this study”. No other personal information will be included without your written consent.

• Video tapes of your sessions will be stored in a locked cabinet. They will only be viewed by the researchers. If the researchers would like to show the videotapes to anyone else, they have to get written consent from you first.

• The Data Protection Policy of the Rehabilitation Engineering Laboratory lists all the rules that the researchers have to follow with respect to your personal information. If you would like a copy of this policy, please ask the researcher. He or she will be happy to give you a copy and answer any questions you may have.

9. If I participate in this study, what are my rights?

You will be given a copy of this information sheet and consent form. You can have as much time and privacy as you need to read this information before you choose whether or not to participate in this study.

You can stop participating in this study at any time. Stopping this study won’t have any effect on other treatments and services that you get at the Lyndhurst Centre. You can continue your other treatments and services even if you stop this study.
Your participation in this study can be stopped at any time by the researcher. However, you can continue your other treatments and services at the Lyndhurst Centre even if the researcher stops your participation in this study.

If you choose to participate, sign the consent form and return it to the designated person.

If you want to learn more about the final results of this study, the researcher will provide you with copies of the published report(s) pertaining to this study.

10. Who is in charge of this study?

We encourage you to ask questions and give feedback at any point in the project. If you have any questions or comments, please contact any of the individuals listed below.

Professor Molly Verrier  
Senior Scientist, SCI Mobility Lab  
Department of Physical Therapy  
160-500 University Ave, Rm 184  
Toronto, ON  
M5G 1V7  
Phone: 416-978-5935  
E-mail: m.verrier@utoronto.ca

Meredith Kuipers  
Master’s candidate, University of Toronto  
Rehabilitation Engineering Laboratory  
Lyndhurst Centre, Toronto Rehab 520 Sutherland Dr.  
Toronto, Ontario, M4G 3V9  
Phone: 416-597-3422x6199/Fax:416-425-9923  
E-mail: m.kuipers@utoronto.ca

Dr. Milos R. Popovic  
Senior Scientist, Rehabilitation Engineering Laboratory  
IBBME, Galbraith Building  
319C-35 St. George Street  
Toronto, ON  
M5S 1A4  
Phone: 416-978-6676  
E-mail: milos.popovic@utoronto.ca

If you have questions about your rights as a research participant, or about any ethical issues relating to this study, you can contact someone who is independent of the research team. Please call the Research Ethics Board Office at (416) 597-3422 x 3081.
Participant Consent Form

Rehabilitation Engineering Laboratory

Toronto Rehabilitation Institute and University of Toronto

Full Study Title: Neuroprosthesis for Sitting Function in Spinal Cord Injury
Sub-Study: Measuring Postural Control and Respiratory Capacity
Principal Investigator: Professor Molly Verrier
Co-investigators: Meredith Kuipers (MSc Candidate), Dr. Milos Popovic

I acknowledge that I have read the attached information sheet and understand its contents including the purpose, the potential benefits, the drawbacks, the method and the role I will play associated with participation in this study. By signing this consent form, I am indicating that I choose to participate in the “Neuroprosthesis for sitting balance for people with spinal cord injury: Measuring postural control and respiratory capacity” study.

________________________________________  ___________________________  ___________________________
Participant’s Printed Name                  Participant’s Signature               Date

________________________________________
Name of Person Obtaining Consent

________________________________________  ___________________________
Signature of Person Obtaining Consent       Date
## Appendix C

### Table 1. Summary of EMG signal impedances for all participants

<table>
<thead>
<tr>
<th></th>
<th>Impedance (kΩ)</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AB1</td>
<td>AB2</td>
<td>AB3</td>
<td>AB4</td>
<td>AB5</td>
<td>AB6</td>
<td>AB7</td>
<td>AB8</td>
<td>AB9</td>
<td>AB10</td>
<td>SCI-1</td>
</tr>
<tr>
<td>RA</td>
<td>L</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>2.5</td>
<td>20.1</td>
<td>19.2</td>
<td>6.8</td>
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<tr>
<td></td>
<td>R</td>
<td>--</td>
<td>--</td>
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<td>--</td>
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<td>2.5</td>
<td>18.9</td>
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<td>5.3</td>
</tr>
<tr>
<td>OE</td>
<td>L</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>6.3</td>
<td>8.4</td>
<td>13.7</td>
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<td></td>
<td>R</td>
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<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>5.4</td>
<td>10.3</td>
<td>7.3</td>
<td>9.8</td>
</tr>
<tr>
<td>LD</td>
<td>L</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>11.3</td>
<td>34.5</td>
<td>12.8</td>
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<tr>
<td></td>
<td>R</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>11.1</td>
<td>26.3</td>
<td>16.2</td>
<td>53.5</td>
</tr>
<tr>
<td>ES</td>
<td>L</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>1.2</td>
<td>23</td>
<td>5.7</td>
<td>4.7</td>
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<tr>
<td></td>
<td>R</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>1.3</td>
<td>29.4</td>
<td>7.5</td>
<td>2.5</td>
</tr>
</tbody>
</table>

### Table 2. Summary of FES signal parameters for SCI participants

<table>
<thead>
<tr>
<th>Stimulation intensity (mA)</th>
<th>SCI-1</th>
<th>SCI-2</th>
<th>SCI-3</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA</td>
<td>15</td>
<td>8</td>
<td>50</td>
</tr>
<tr>
<td>OE</td>
<td>16</td>
<td>13</td>
<td>46</td>
</tr>
<tr>
<td>LD</td>
<td>15</td>
<td>14</td>
<td>50</td>
</tr>
<tr>
<td>ES</td>
<td>21</td>
<td>12</td>
<td>50</td>
</tr>
</tbody>
</table>

Note: Stimulation was applied equally bilaterally; mA – milli-amperes
## Appendix D

**Questionnaire responses: SCI participants only**

Did the use of the surface FES in either configuration - AP-FES or ML-FES - make it easier to sit quietly?

<table>
<thead>
<tr>
<th>Participant</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI-1</td>
<td>- Yes - stimulation helped participant to sit up straight; normally, participants’ back “brings me down”</td>
</tr>
<tr>
<td>SCI-2</td>
<td>No response</td>
</tr>
<tr>
<td>SCI-3</td>
<td>- when stimulation was ON, “it created more spasticity in my left side”; participant felt some Autonomic Dysreflexia reaction occurring as trials progressed: sweating, increased heart rate, higher blood pressure (felt diastolic peak over 100mmHg)</td>
</tr>
</tbody>
</table>

Did you think that it was easier to breathe during stimulation trials?

<table>
<thead>
<tr>
<th>Participant</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI-1</td>
<td>- No – it was harder to breathe while sitting on force plate; no change in ease of breathing with or without stimulation</td>
</tr>
<tr>
<td>SCI-2</td>
<td>No response</td>
</tr>
<tr>
<td>SCI-3</td>
<td>- with ML-FES, it was harder to breathe: more spasticity in this condition; participant was flexing trapezius muscle; struggled to maintain balance</td>
</tr>
</tbody>
</table>

Were you able to breathe more deeply during stimulation?

<table>
<thead>
<tr>
<th>Participant</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI-1</td>
<td>No response</td>
</tr>
<tr>
<td>SCI-2</td>
<td>No response</td>
</tr>
<tr>
<td>SCI-3</td>
<td>- when AP-FES was ON, participant felt “big forward flexion” from abdominal muscle (RA) stimulation</td>
</tr>
</tbody>
</table>
Were you less fatigued following trials with stimulation?

<table>
<thead>
<tr>
<th>Participant</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI-1</td>
<td>- felt relief when stimulation was turned off</td>
</tr>
<tr>
<td>SCI-2</td>
<td>No response</td>
</tr>
<tr>
<td>SCI-3</td>
<td>No response</td>
</tr>
</tbody>
</table>

Do you have any other comments about your experience?

<table>
<thead>
<tr>
<th>Participant</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI-1</td>
<td>- preference was for trials with stimulation</td>
</tr>
<tr>
<td>SCI-2</td>
<td>No response</td>
</tr>
<tr>
<td>SCI-3</td>
<td>No response</td>
</tr>
</tbody>
</table>
### Table 1. Summary of muscle activity for SCI participants

<table>
<thead>
<tr>
<th>Muscle activity for SCI participants</th>
<th>SCI-1</th>
<th>SCI-2</th>
<th>SCI-3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Engaged</td>
<td>Relaxed</td>
<td>Engaged</td>
</tr>
<tr>
<td><strong>EMG amplitude (mV) over 30s</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RA L</td>
<td>1.05</td>
<td>0.74</td>
<td>0.40</td>
</tr>
<tr>
<td>RA R</td>
<td>2.85</td>
<td>1.58</td>
<td>0.74</td>
</tr>
<tr>
<td>OE L</td>
<td>2.23</td>
<td>1.02</td>
<td>0.60</td>
</tr>
<tr>
<td>OE R</td>
<td>1.37</td>
<td>0.56</td>
<td>0.13</td>
</tr>
<tr>
<td>LD L</td>
<td>0.69</td>
<td>0.51</td>
<td>0.40</td>
</tr>
<tr>
<td>LD R</td>
<td>0.83</td>
<td>0.49</td>
<td>0.43</td>
</tr>
<tr>
<td>ES L</td>
<td>0.55</td>
<td>0.56</td>
<td>0.31</td>
</tr>
<tr>
<td>ES R</td>
<td>0.52</td>
<td>0.48</td>
<td>0.28</td>
</tr>
<tr>
<td><strong>Proportion of AB group mean EMG amplitude</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RA L</td>
<td>0.36</td>
<td>0.25</td>
<td>0.14</td>
</tr>
<tr>
<td>RA R</td>
<td>0.54</td>
<td>0.30</td>
<td>0.14</td>
</tr>
<tr>
<td>OE L</td>
<td>0.51</td>
<td>0.22</td>
<td>0.14</td>
</tr>
<tr>
<td>OE R</td>
<td>0.51</td>
<td>0.22</td>
<td>0.05</td>
</tr>
<tr>
<td>LD L</td>
<td>0.24</td>
<td>0.19</td>
<td>0.14</td>
</tr>
<tr>
<td>LD R</td>
<td>0.30</td>
<td>0.19</td>
<td>0.16</td>
</tr>
<tr>
<td>ES L</td>
<td>0.10</td>
<td>0.17</td>
<td>0.06</td>
</tr>
<tr>
<td>ES R</td>
<td>0.12</td>
<td>0.15</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Engaged - unsupported, sitting away from backrest; Relaxed - typical posture sitting in wheelchair
### Appendix F

**Table 1. Summary of anthropometric data for AB participants (n=10)**

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age (y)</th>
<th>Height (m)</th>
<th>Weight (kg)</th>
<th>BMI</th>
<th>Waist circumference (cm)</th>
<th>Pack years</th>
</tr>
</thead>
<tbody>
<tr>
<td>AB1*</td>
<td>27</td>
<td>165.1</td>
<td>54.4</td>
<td>19.8</td>
<td>68.2</td>
<td>NS</td>
</tr>
<tr>
<td>AB2</td>
<td>27</td>
<td>180.0</td>
<td>76.0</td>
<td>23.5</td>
<td>83.4</td>
<td>NS</td>
</tr>
<tr>
<td>AB3</td>
<td>25</td>
<td>188.0</td>
<td>88.5</td>
<td>24.9</td>
<td>85.9</td>
<td>NS</td>
</tr>
<tr>
<td>AB4</td>
<td>38</td>
<td>180.0</td>
<td>74.0</td>
<td>22.8</td>
<td>82.5</td>
<td>NS</td>
</tr>
<tr>
<td>AB5*</td>
<td>29</td>
<td>162.6</td>
<td>53.1</td>
<td>20.1</td>
<td>68.0</td>
<td>NS</td>
</tr>
<tr>
<td>AB6</td>
<td>41</td>
<td>182.9</td>
<td>83.0</td>
<td>24.8</td>
<td>92.5</td>
<td>8.8</td>
</tr>
<tr>
<td>AB7*</td>
<td>38</td>
<td>165.1</td>
<td>61.2</td>
<td>22.7</td>
<td>77.5</td>
<td>2.0</td>
</tr>
<tr>
<td>AB8*</td>
<td>31</td>
<td>162.6</td>
<td>49.9</td>
<td>19.0</td>
<td>67.6</td>
<td>NS</td>
</tr>
<tr>
<td>AB9</td>
<td>29</td>
<td>177.8</td>
<td>70.3</td>
<td>22.2</td>
<td>79.0</td>
<td>NS</td>
</tr>
<tr>
<td>AB10</td>
<td>25</td>
<td>180.0</td>
<td>75.0</td>
<td>23.1</td>
<td>82.6</td>
<td>8.0</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>31 (5.9)</td>
<td>174.4 (9.5)</td>
<td>68.5 (13.2)</td>
<td>22.3 (2.0)</td>
<td>78.7 (8.5)</td>
<td>6.3 (3.7)†</td>
</tr>
</tbody>
</table>

* - female participant; NS - never smoker; † - mean pack year calculated for three ever smokers

**Table 2. Summary of spirometry for AB participants (n=10).**

Data presented is highest of three PFT efforts measuring within ±0.15L.

<table>
<thead>
<tr>
<th>Participant</th>
<th>FVC (L)</th>
<th>FEV₁ (L)</th>
<th>FEV₁: FVC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Actual</td>
<td>% Pred</td>
<td>Actual</td>
</tr>
<tr>
<td>AB1</td>
<td>3.19</td>
<td>82</td>
<td>2.54</td>
</tr>
<tr>
<td>AB2</td>
<td>5.55</td>
<td>98</td>
<td>4.95</td>
</tr>
<tr>
<td>AB3</td>
<td>7.25</td>
<td>117</td>
<td>6.18</td>
</tr>
<tr>
<td>AB4</td>
<td>5.46</td>
<td>100</td>
<td>4.66</td>
</tr>
<tr>
<td>AB5</td>
<td>3.25</td>
<td>86</td>
<td>2.44</td>
</tr>
<tr>
<td>AB6</td>
<td>5.83</td>
<td>104</td>
<td>4.96</td>
</tr>
<tr>
<td>AB7</td>
<td>4.68</td>
<td>122</td>
<td>3.80</td>
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<tr>
<td>AB8</td>
<td>3.70</td>
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<td>3.22</td>
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<tr>
<td>AB9</td>
<td>4.98</td>
<td>87</td>
<td>4.15</td>
</tr>
<tr>
<td>AB10</td>
<td>6.11</td>
<td>104</td>
<td>4.15</td>
</tr>
</tbody>
</table>

Actual – measured by spirometer; % Pred – percentage of predicted values; all spirometric reference values calculated using equations from Hankinson et al. [110].
## Appendix G

### Table 1. Summary of experimental results for able-boded participants

<table>
<thead>
<tr>
<th>Able bodied participants (n=10)</th>
<th>Upright (mean ± 1 SD)</th>
<th>Slouch (mean ± 1 SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Quiet breathing respiratory function</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VT (L)</td>
<td>0.70 ±0.27</td>
<td>0.61 ±0.19</td>
</tr>
<tr>
<td>RR (min⁻¹)</td>
<td>14.2 ±1.4</td>
<td>14.7 ±1.0</td>
</tr>
<tr>
<td>VE (L/min)</td>
<td>9.3 ±1.1</td>
<td>8.6 ±0.8</td>
</tr>
<tr>
<td><strong>Vertical forces</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absolute Fz (N)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>564.90 ±104.39</td>
<td>557.62 ±100.15</td>
</tr>
<tr>
<td>Feet</td>
<td>146.99 ±33.72</td>
<td>153.16 ±35.85</td>
</tr>
<tr>
<td>Proportion total Fz</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.79</td>
<td>0.78</td>
</tr>
<tr>
<td>Feet</td>
<td>0.21</td>
<td>0.22</td>
</tr>
<tr>
<td><strong>Mean COP distance (mm)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MDIST</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>1.52 ±0.63</td>
<td>1.09 ±0.62</td>
</tr>
<tr>
<td>Feet</td>
<td>2.23 ±0.75</td>
<td>1.07 ±0.56</td>
</tr>
<tr>
<td>apMDIST</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.70 ±0.26</td>
<td>1.05 ±0.86</td>
</tr>
<tr>
<td>Feet</td>
<td>1.16 ±0.71</td>
<td>1.59 ±0.59</td>
</tr>
<tr>
<td>mlMDIST</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.70 ±0.87</td>
<td>0.43 ±0.21</td>
</tr>
<tr>
<td>Feet</td>
<td>0.66 ±0.48</td>
<td>0.61 ±0.56</td>
</tr>
<tr>
<td><strong>Mean COP velocity (mm/s)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MVELO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>3.04 ±1.01</td>
<td>2.15 ±1.12</td>
</tr>
<tr>
<td>Feet</td>
<td>4.96 ±3.08</td>
<td>3.61 ±2.71</td>
</tr>
<tr>
<td>apMVELO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>1.65 ±0.62</td>
<td>1.32 ±0.89</td>
</tr>
<tr>
<td>Feet</td>
<td>2.65 ±0.90</td>
<td>1.97 ±0.70</td>
</tr>
<tr>
<td>mlMVELO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>2.16 ±0.85</td>
<td>1.40 ±0.68</td>
</tr>
<tr>
<td>Feet</td>
<td>3.51 ±3.18</td>
<td>2.53 ±2.79</td>
</tr>
<tr>
<td><strong>Mean COP frequency (Hz)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MFREQ</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.36 ±0.09</td>
<td>0.39 ±0.11</td>
</tr>
<tr>
<td>Feet</td>
<td>0.34 ±0.12</td>
<td>0.35 ±0.18</td>
</tr>
<tr>
<td>apMFREQ</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.37 ±0.12</td>
<td>0.53 ±0.17</td>
</tr>
<tr>
<td>Feet</td>
<td>0.59 ±0.25</td>
<td>0.79 ±0.38</td>
</tr>
<tr>
<td>mlMFREQ</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seat</td>
<td>0.60 ±0.16</td>
<td>0.66 ±0.24</td>
</tr>
<tr>
<td>Feet</td>
<td>0.66 ±0.26</td>
<td>0.98 ±0.50</td>
</tr>
<tr>
<td><strong>Muscle activity (mV)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RA EMG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>2.93 ±0.47</td>
<td>2.91 ±0.44</td>
</tr>
<tr>
<td>R</td>
<td>5.30 ±2.34</td>
<td>5.26 ±2.33</td>
</tr>
<tr>
<td>OE EMG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>4.38 ±1.43</td>
<td>4.56 ±1.89</td>
</tr>
<tr>
<td>R</td>
<td>2.70 ±0.38</td>
<td>2.50 ±0.20</td>
</tr>
<tr>
<td>LD EMG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>2.85 ±0.28</td>
<td>2.64 ±0.19</td>
</tr>
<tr>
<td>R</td>
<td>2.75 ±0.26</td>
<td>2.57 ±0.20</td>
</tr>
<tr>
<td>ES EMG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L</td>
<td>5.51 ±1.92</td>
<td>3.27 ±1.20</td>
</tr>
<tr>
<td>R</td>
<td>4.37 ±1.84</td>
<td>3.18 ±1.93</td>
</tr>
</tbody>
</table>
## Appendix H

### Table 1. Summary of experimental results for SCI-1

<table>
<thead>
<tr>
<th>Participant SCI-1</th>
<th>results for all sitting trials (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT (L)</td>
<td>0.54 0.45 0.45 0.45 0.54 0.56 0.55 0.55</td>
</tr>
<tr>
<td>RR (min⁻¹)</td>
<td>18.3 18.5 21.2 18.9 17.6 19.3 20.4 18.6</td>
</tr>
<tr>
<td>VE (L/min)</td>
<td>9.82 8.30 9.55 8.40 9.48 10.77 11.15 10.30</td>
</tr>
<tr>
<td>Absolute Fz (N)</td>
<td>503.24 499.82 505.23 495.85 501.16 502.58 504.53 499.56</td>
</tr>
<tr>
<td>Feet</td>
<td>127.21 130.35 125.43 135.73 131.06 130.11 129.13 134.15</td>
</tr>
<tr>
<td>Proportion total Fz</td>
<td>0.80 0.79 0.80 0.79 0.79 0.79 0.80 0.79</td>
</tr>
<tr>
<td>Feet</td>
<td>0.20 0.21 0.20 0.21 0.21 0.21 0.20 0.21</td>
</tr>
<tr>
<td>MDIST (mm)</td>
<td>4.93 1.80 1.91 1.08 2.70 2.97 2.06 2.49</td>
</tr>
<tr>
<td>MVELO (mm/s)</td>
<td>3.23 4.25 2.88 1.96 4.90 4.24 4.72 3.28</td>
</tr>
<tr>
<td>MFREQ (Hz)</td>
<td>0.10 0.38 0.24 0.29 0.29 0.23 0.37 0.21</td>
</tr>
</tbody>
</table>

Sequence of sitting trials: 1-Unsupported, 2-AP-FES, 3-ML-FES, 4-Unsupported, 5-ALL FES, 6-Unsupported, 7-AP-FES, 8-ML-FES

### Table 2. Summary of experimental results for SCI-2

<table>
<thead>
<tr>
<th>Participant SCI-2</th>
<th>results for all sitting trials (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT (L)</td>
<td>0.72 0.55 0.79 0.84 0.88 1.23 1.14</td>
</tr>
<tr>
<td>RR (min⁻¹)</td>
<td>11.7 14.3 12.1 10.6 11.5 10.1 9.9</td>
</tr>
<tr>
<td>VE (L/min)</td>
<td>8.18 7.68 9.40 8.70 10.03 12.45 11.15</td>
</tr>
<tr>
<td>Absolute Fz (N)</td>
<td>618.60 629.20 623.59 609.03 616.72 598.81 605.54</td>
</tr>
<tr>
<td>Feet</td>
<td>139.49 129.11 135.43 150.57 144.85 165.99 159.58</td>
</tr>
<tr>
<td>Proportion total Fz</td>
<td>0.82 0.83 0.82 0.80 0.81 0.78 0.79</td>
</tr>
<tr>
<td>Feet</td>
<td>0.18 0.17 0.18 0.20 0.19 0.22 0.21</td>
</tr>
<tr>
<td>MDIST (mm)</td>
<td>6.42 1.04 3.34 11.53 5.45 2.78 1.81</td>
</tr>
<tr>
<td>MVELO (mm/s)</td>
<td>6.94 1.46 2.58 5.70 3.68 3.17 1.79</td>
</tr>
<tr>
<td>MFREQ (Hz)</td>
<td>0.17 0.22 0.12 0.08 0.11 0.18 0.16</td>
</tr>
</tbody>
</table>

Sequence of sitting trials: 1-Unsupported, 2-AP-FES, 3-ML-FES, 4-Unsupported, 5-Unsupported, 6-AP-FES, 7-ML-FES
Table 3. Summary of experimental results for SCI-3

<table>
<thead>
<tr>
<th>Participant SCI-3</th>
<th>Quiet breathing respiratory function</th>
<th>Vertical forces</th>
<th>Seated postural stability</th>
</tr>
</thead>
<tbody>
<tr>
<td>results for all sitting trials (n=6)</td>
<td>VT (L)</td>
<td>RR (min⁻¹)</td>
<td>VE (L/min)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>0.91</td>
<td>0.81</td>
<td>0.81</td>
</tr>
<tr>
<td></td>
<td>12.1</td>
<td>14.7</td>
<td>14.4</td>
</tr>
<tr>
<td></td>
<td>10.55</td>
<td>11.13</td>
<td>11.17</td>
</tr>
<tr>
<td></td>
<td>6.62</td>
<td>3.95</td>
<td>1.96</td>
</tr>
<tr>
<td></td>
<td>624.06</td>
<td>170.08</td>
<td>6.62</td>
</tr>
<tr>
<td></td>
<td>618.04</td>
<td>176.31</td>
<td>3.95</td>
</tr>
</tbody>
</table>

Sequence of sitting trials: 1-Unsupported, 2-AP-FES, 3-AP-FES, 4-unsupported, 5-ML-FES, 6-Unsupported