ASSESSMENT AND MANAGEMENT OF SPASTICITY AFTER SCI

ASSESSMENT AND ACTIVITY-BASED MANAGEMENT OF SPASTICITY AFTER SPINAL CORD INJURY

By

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A Thesis

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ABSTRACT

Sixty-five to 78% of sample populations of individuals with chronic spinal cord injury (SCI) have symptoms of spasticity, but current assessment tools and management strategies are inadequate. Therefore, the purpose of this thesis was to: 1) conduct a review of the definition, pathophysiology, and management of symptoms of spasticity in individuals with SCI, 2) develop and assess a new self-report scale designed to measure the impact of spasticity on daily life in people with SCI and 3) examine the effects of body-weight supported treadmill training (BWSTT) and tilt-table standing (TTS) on spasticity outcomes. 1) Spasticity was found to be a relevant concern for the SCI population and possibly responsive to activity-based intervention. A need was identified, however, for an assessment tool that allows individuals with SCI to report the impact of their spasticity. 2) In total, 89 individuals with chronic SCI participated in three phases of scale design: development, pilot testing, and evaluation of test-retest reliability and construct validity. The Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET) was developed as a 7-day recall self-report questionnaire that takes into account both the problematic and useful effects of spasticity. The internal consistency (α) and intraclass correlation coefficient of the SCI-SET were 0.90 and 0.91, respectively. Construct validity was supported by correlations ($r=-0.48$ to 0.68; $p<0.01$) between SCI-SET scores and theoretically meaningful constructs (spasticity impact and severity, spasm rating, quality of life, and functional mobility). 3) Seven individuals with chronic SCI performed thrice-weekly BWSTT for 4 wks and thrice-weekly TTS for 4 wks in a random cross-over design and were assessed for changes in symptoms of spasticity immediately following the first session and 24-48 hrs following the twelfth session of activity. Compared to a single session of TTS, moderate and strong effect sizes (ES) supported the tendency of BWSTT to have greater beneficial effects on muscle tone (ES=0.69), flexor spasms (ES=0.57), and motor neuron excitability (ES=0.50). A single session of TTS appeared to reduce extensor spasms (ES=0.68). Flexor spasms (ES=0.79), clonus (ES=0.66), and self-reported mobility (ES=1.27) tended to benefit more from 4 wks of BWSTT than of TTS, whereas extensor spasms had greater reductions following TTS (ES=1.32). Participation in BWSTT appeared to have a favourable effect on quality of life (ES=0.50). In conclusion, the SCI-SET fills a need for a reliable and valid self-report measure of the impact of spasticity on daily life in people with SCI. Individuals with SCI and spasticity may benefit from participation in weight-bearing activity, but the effects of intervention may differ depending on the spasticity outcome and the timeline.

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LIST OF ABBREVIATIONS

ADL: activities of daily living
ASIA: American Spinal Injury Association
BWS: body-weight support
BWSTT: body-weight supported treadmill training
CNS: central nervous system
DOMS: delayed-onset muscle soreness
EMG: electromyographic
ES: effect size
FES: functional electrical stimulation
GABA: gamma-aminobutyric acid
H/M ratio: maximum H-reflex to maximum M-wave ratio
ICC: intraclass correlation coefficient
MAS: Modified Ashworth Scale
QLI: Quality of Life Index
QOL: quality of life
SCATS: Spinal Cord Assessment Tool for Spinal reflexes
SCI: spinal cord injury
SCI-SET: Spinal Cord Injury Spasticity Evaluation Tool
SD: standard deviation
TENS: transcutaneous electrical nerve stimulation
THC: tetrahydrocannabinol
TTS: tilt-table standing

PREFACE

The manuscripts prepared as a result of this thesis work (Chapters 2 to 4) have been published or submitted for publication under multiple authorship. As a doctoral candidate, I was responsible for all stages of the presented research, including study design and implementation, data collection and analysis, and result presentation / manuscript preparation.

CHAPTER 1

BACKGROUND INFORMATION

1.0 BACKGROUND INFORMATION

1.1 SPINAL CORD INJURY AND SPASTICITY

1.1.1 Introduction to Spinal Cord Injury

As the conduit of communication between the brain and all other parts of the body, the spinal cord is critical to the proper functioning of the nervous system. Therefore, damage to the spinal cord, whether through traumatic or non-traumatic etiology, can result in variable losses in motor and/or sensory function. The American Spinal Injury Association (ASIA) Impairment Scale (Table 1.1) is widely used to classify the degree of impairment following spinal cord injury (SCI). According to the International Standards for Neurological and Functional Classification of Spinal Cord Injury, segments of the body with normal motor and/or sensory function are determined. The neurological level of an individual with SCI is identified as the most caudal segment of the spinal cord with normal sensory and motor function on both sides of the body. An injury is classified as complete (ASIA A) if there is no motor or sensory function in the lowest sacral segment of the spinal cord. If there is partial preservation of motor and/or sensory function below the neurological level and in the lowest sacral segment, the injury is classified as incomplete (ASIA B-D). Injuries affecting the cervical spinal cord result in impairment of the upper and lower limbs as well as the pelvic organs and the trunk (tetraplegia). If the thoracic, lumbar, or sacral area of the spinal cord is injured, the trunk, legs and pelvic organs may incur some loss (paraplegia).[1]

Table 1.1 ASIA Impairment Scale [1]

ASIA Class	Complete or Incomplete	Description
A	Complete	No sensory or motor function preserved in the lowest sacral segments (S4-S5).
B	Sensory Incomplete	Sensory but no motor function preserved below the neurological level including the sacral segments S4-S5.
C	Motor Incomplete	Motor function is preserved below the neurological level, and more than half of the key muscles below the neurological level have a muscle grade less than 3. There must be some sparing of sensory and/or motor function in the segments S4/5.
D	Motor Incomplete	Motor function is preserved below the neurological level, and more than half of the key muscles below the neurological level have a muscle grade greater than or equal to 3. There must be some sparing of sensory and/or motor function in the segments S4/5.
E	"Normal"	Sensory and motor function are normal.

It is estimated that over 30,000 Canadians currently are living with a SCI (~ 35 per million population), with approximately 900 new injuries occurring each year.[2] The majority of the injuries in Canada are due to car collisions (35%) and falls (16.5%).[2] Of those living with a SCI, the majority were injured between the ages of 15 and 34 (78%)

and are male (80%).[2] With advancements in medicine over the last three decades in North America, there has been a 40% decline in mortality during the critical 2-year period post-SCI.[3] Therefore, it is very important to recognize and manage the many potential secondary complications of SCI that can impact long-term health and quality of life (Figure 1.1).

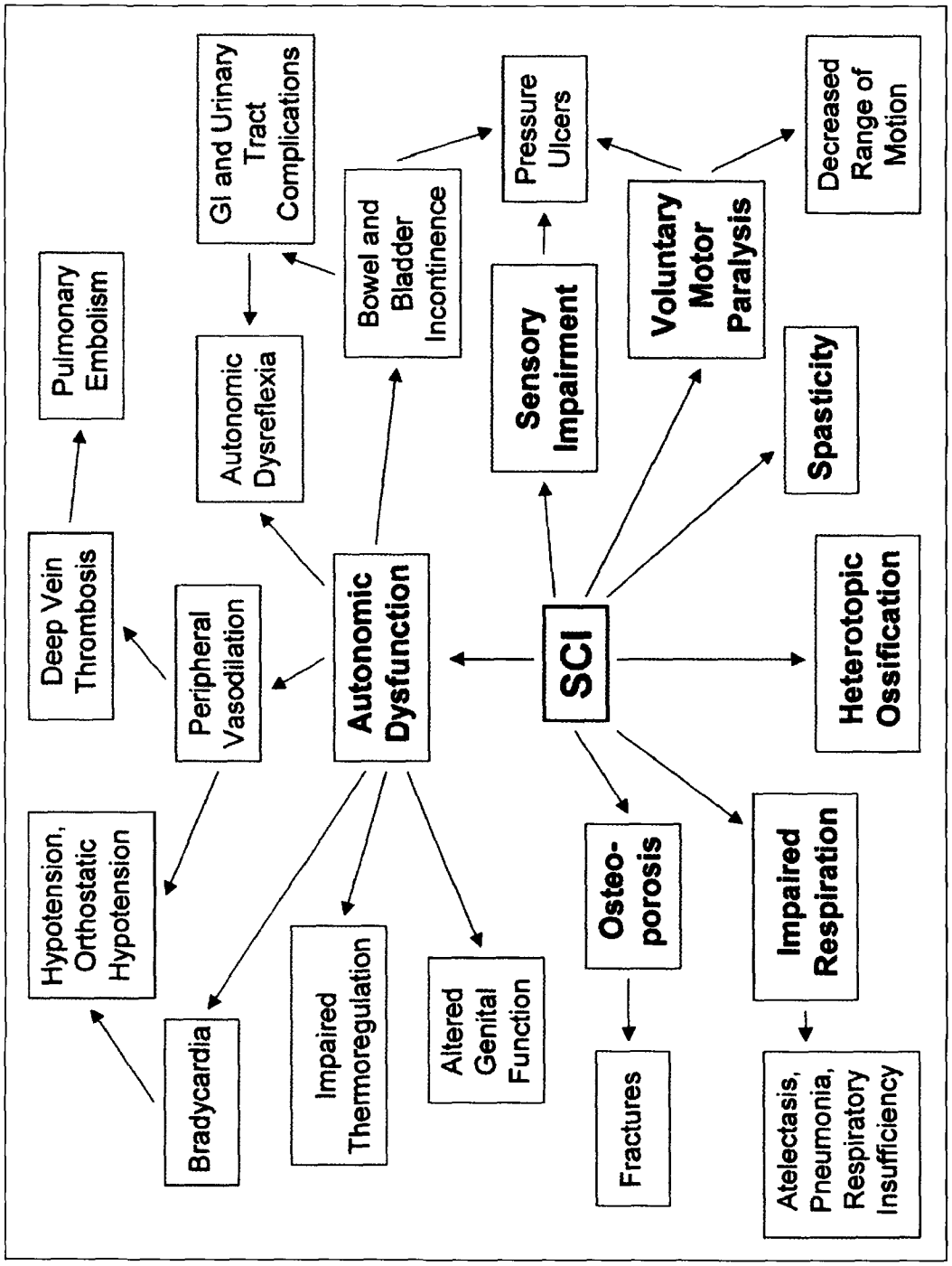

Figure 1.1 Schematic representation of the physical complications of spinal cord injury.[4]

1.1.2 Complications of Spinal Cord Injury: Introduction to Spasticity

Depending on the level and severity, SCI can contribute to the development of a wide spectrum of medical complications affecting organ systems above and below the level of the injury (Figure 1.1).[4,5] Such complications can have a considerable impact on the health, social participation, and quality of life of the individual.[6,7] Spasticity has been defined as "disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary contraction of muscles".[8] This "neurological consequence" is considered to be due to the injury itself, as a result of interruption and decentralization of the nervous system.[6] In particular, hypertonicity, spasms, and clonus (the "symptoms of spasticity") are believed to result in part from hyperexcitability of spinal reflexes.[9]

Symptoms of spasticity have been reported in 65% to 78% of sample populations of individuals with chronic SCI.[10,11] Spasticity can be problematic due to its effects on walking, self-care, pain, fatigue, sleep, safety, and self-image.[9,11] Alternatively, some individuals with SCI may also find spasticity to be helpful due to an increase in sitting or standing stability and the facilitation of the performance of some activities of daily living and transfers.[9,11] Therefore, to be of most benefit, spasticity management decisions should be made with the goal of achieving balance between the useful and problematic effects of these symptoms.[12,13]

The assessment of spasticity can be performed by using various methods, including clinical, biomechanical, and electrophysiological techniques.[14-18] Depending on the reason for assessment and the availabilities of equipment and expertise, certain methodologies may be more suitable than others. Widely used, the Ashworth scale[19] (or Modified Ashworth Scale[20]) allows an examiner to numerically rate muscle hypertonicity. Recently, a clinical scale for the numerical rating of spasms and clonus has also become available (the Spinal Cord Assessment Tool for Spinal reflexes – SCATS).[21] Pendulum testing and biomechanical gait analysis are used to quantify and characterize limb movement.[14,22] Electrophysiological assessment can help to identify underlying physiological abnormalities in spasticity and can include measurement of the H-reflex and the F-wave.[18] As no single outcome measure is considered adequate when assessing spasticity, it has been suggested that a variety of different approaches should be combined.[7,22-26] Also becoming more evident, is the importance of including assessments performed by the *individual*,[14,27] as examiner-based physical examination does not necessarily elicit spasticity in individuals who report the symptom[11] and individuals with SCI may experience spasticity in body segments that are not tested by an examiner[28]. It is also interesting to note that not all individuals with SCI who report *having* spasticity indicate that spasticity has an *impact* on their daily life.[28] Some research studies in the SCI population have begun to include self-ratings among the outcome measures of spasticity, in which reporting of both spasticity severity (e.g., using visual analogue scales[29] or single item ratings[30]) and spasticity impact (on daily pain and function[23], or daily life[28]) has been performed. Unfortunately, many of these self-report techniques lack appropriate validation and are limited in their abilities to capture both the detrimental and potentially beneficial impacts of spasticity on daily life in individuals with SCI.

Many approaches are also available for the management of spasticity symptoms, ranging from non-invasive physical rehabilitation modalities to pharmacologic treatment and surgical procedures (Figure 1.2). A combination of these strategies may be required to adequately manage spasticity in an individual.[31] While surgical strategies are typically reserved for cases of severe focal spasticity, pharmacologic management is quite common in the SCI population. For example, baclofen, whether administered orally or intrathecally, inhibits spinal reflexes due to decreased neurotransmitter release in excitatory spinal pathways.[9] Unfortunately, pharmacologic agents may also impart problematic side-effects, such as drowsiness, fatigue, nausea, muscle weakness, and depression.[12,32] In contrast, physical rehabilitation modalities are conservative strategies which cause few (if any) adverse side-effects, while also offering further possible advantages. There exists evidence for the benefits of cold/heat application, stretching, weight-bearing, and electrical stimulation, among others.[9] Further research is needed to clearly elucidate the role of physical management strategies on spasticity, however, as mixed results and poorly designed studies prevent firm conclusions from being drawn.[33]

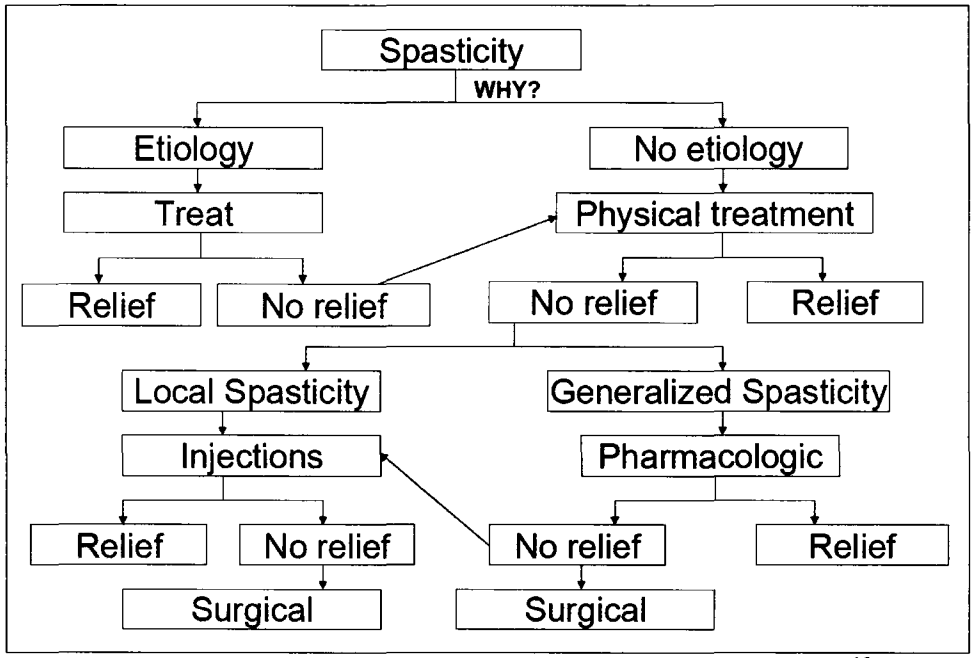

Figure 1.2 Spasticity management (adapted from Parziale and colleagues[13])

1.2 Activity-Based Therapy Following Spinal Cord Injury

1.2.1 Introduction to Activity-Based Therapy

Within the context of this thesis, the term "activity-based therapy" is used to discuss physical "rehabilitation" strategies that would be similar to physical activity-related participation in the able-bodied population. In individuals with SCI, such activity-based therapies (or "activities") may be either passive or active depending on the strategy

and the amount of preserved motor function by the individual. They may be performed within a rehabilitation setting, but performance within home or community environments is also possible. Examples of activity-based therapies include assisted-walking, -standing, and -cycling, as well as movements through a range of motion and stretching.

1.2.2 Assisted-Standing

In individuals who use a wheelchair as a primary mode of mobility, the "chronic seated posture" may contribute to the development of various secondary complications.[34] Compared to a seated position, standing places load on the lower limbs and imposes stretch on the skeletal muscles that cross the hip, knee, and ankle joints. The effects of assisted-standing have been studied for at least four decades[(e.g.,35)] using such devices as standing frames, standing wheelchairs, long leg braces and tilt tables to allow the individual to perform prolonged "standing" in a safe environment. Individuals with SCI who regularly participate in assisted-standing have been shown to have better well-being, quality of life, digestion, sleep, bladder and bowel regularity, and skin integrity, as well as less pain and fatigue.[36-38]

1.2.3 Body-Weight Supported Treadmill Training: Assisted-Walking

Pioneering research by Sherrington in 1910 and Brown in 1914 revealed that cats with severed spinal cords retain some ability to perform stepping motions with their hind limbs.[39,40] Years later, it was proposed that the cat spinal cord contains a "rhythm generator" for locomotion that is independent from the brain and sensory cues, often called a "central pattern generator".[40,41] With part of their body-weight supported by their tail, "spinalized" cats were found to have improved walking ability following a period of training on a treadmill.[42-44]

This "body-weight supported treadmill training" (BWSTT) has since been made available for use by humans who have deficits in independent walking ability.[45,46] A motor-driven treadmill with an overhead pulley system capable of supporting a percentage of the participant's body-weight is typically used during BWSTT. Therapists are also positioned at each of the legs of the participant to assist with lower limb movement when necessary. Although the presence of a central pattern generator in humans remains to be confirmed,[39,47-50] BWSTT has been shown to be beneficial both during the assisted-walking bout and as a means to improve walking ability in individuals with SCI.[51-58] Furthermore, the benefits of BWSTT have been found to extend beyond walking ability, as muscle morphology, glucose regulation, cardiovascular regulation, and psychological well-being have been shown to improve with training.[51,59-61]

1.2.4 Possible Effects of BWSTT and Tilt-Table Standing on Spasticity after SCI

There is evidence to support the beneficial effects of several activity-based therapies on various spasticity outcomes in individuals with SCI; these benefits have been noted after single[29,62-66] and multiple sessions[29,34,37,38,67-71] of activity. The immediate

effects of a single session of activity are often reported as being short-lasting (i.e., less than 24 hrs, typically 1 to 8 hrs).[24,62] Observed benefits of multiple sessions of activity may be due to maintained "chronic" changes, but the perception of the individual that repeated short-term effects contribute to overall daily management must also be considered. The mechanisms explaining the benefits of activity on spasticity remain elusive, but suggestions have included mechanical changes at the muscle, tendon, and soft-tissue level[72] and plastic changes within the central nervous system (e.g., reduction of motor unit activity/excitability[62,73]).

Research examining the effects of BWSTT on spasticity in individuals with SCI is limited, but there is some evidence that motor neuron excitability is reduced following a single session.[74] The characteristics of BWSTT certainly make it a likely activity to have beneficial effects on spasticity management, as the weight-bearing, muscle stretch, limb movement, and electromyographic activity[58,75,76] that occur during BWSTT provide a unique combination of potential stimuli. The costly equipment and personnel requirements of BWSTT, however, necessitate the assessment of similar, but less costly alternatives, such as tilt-table standing (TTS). Although not as comprehensive as BWSTT, TTS provides weight-bearing and stretch and has been shown to help with spasticity management in individuals with SCI.[34,37,38,62]

1.3 Development of Thesis

The Centre for Health Promotion and Rehabilitation in the Department of Kinesiology at McMaster University houses the MacWheelers program, an exercise and wellness program for individuals with SCI. This program, along with on-going research in the Department, allows individuals with SCI the opportunity to discuss problematic complications of their injury and perceived benefits of participation in physical activity and exercise. Spasticity was identified by our program participants as a problematic complication that tended to improve with regular participation in the MacWheelers program. Therefore, the first stage of my doctoral thesis was to conduct a review of the literature to develop an understanding of spasticity and its management in the SCI population (Chapter 2). This review allowed me to determine that, in fact, there was evidence in the scientific literature to support the anecdotal reports by our program participants: as an activity-based management strategy, the weight-bearing, stretch, and muscle activity that occurs during BWSTT may help to alleviate problematic effects of spasticity in individuals with SCI. It also became evident that the self-reporting of spasticity by the individual was recognized as an important component of research in clinical settings. No valid and reliable measure existed, however, to conduct self-reporting of the problematic and beneficial effects of spasticity on daily life by individuals with SCI. The second stage of my doctoral thesis, therefore, was to develop and evaluate the Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET; Chapter 3).

With a sound understanding of spasticity and the availability of appropriate outcome measures, it became possible to conduct investigations of the effects of BWSTT on spasticity in individuals with SCI. As BWSTT is unique in its provision of weight-bearing, stretch, *and* muscle activity, it was decided to compare the effects of BWSTT to

those of TTS, a less-comprehensive, but more widely-available form of activity. The literature was found to include two distinct bodies of information related to the effects of activity-based management of spasticity: a) the short-term or immediate effects of a single session of activity and b) the longer-term or prolonged effects of a series of sessions of activity. Therefore, stages 3 and 4 of my thesis included the investigation of the effects of single sessions of BWSTT and TTS (Chapter 4) and multiple sessions of BWSTT and TTS (Chapter 5) on various clinical, electrophysiological, and self-report measures of spasticity and theoretically-related constructs. As a whole, this thesis fulfilled three general purposes that are relevant to the quality of life of individuals with SCI:

1. To make readily available a comprehensive overview of the definition, pathophysiology, and management of symptoms of spasticity in individuals with SCI.
2. To describe the development and preliminary evaluation of the SCI-SET, a self-report measure of the impact of spasticity on daily life in people with SCI.
3. To investigate the effects of single-sessions and multiple-sessions of BWSTT and TTS on clinically-assessed and self-reported spasticity and motor neuron excitability in individuals with chronic SCI.

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CHAPTER 2

SPASTICITY AFTER SPINAL CORD INJURY (A REVIEW)

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2.0 SPASTICITY AFTER SPINAL CORD INJURY

2.1 ABSTRACT

Symptoms of spasticity are often experienced by individuals with spinal cord injury (SCI) following a period of spinal shock and, in many cases, these symptoms negatively affect quality of life. Despite its prevalence, spasticity as a syndrome in the SCI population is not always managed effectively. This is likely due to the fact that the syndrome can have various presentations, each with their own specific etiology. This overview summarizes the symptoms and pathophysiology of the various presentations of spasticity in the SCI population and discusses the currently accepted management techniques. There is a need for a better understanding of the syndrome of spasticity as well as the development of a valid and reliable assessment tool.

2.2 INTRODUCTION

Unequivocally, 'spasticity' is understood to be among the symptoms resulting from injury to the upper motor neurons within the central nervous system (CNS) and is a common, but not an inevitable sequelae of spinal cord injury (SCI).[1-3] The most commonly cited definition for spasticity is that published by Lance in 1980[4]: "Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch-reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motoneuron syndrome". There remains, however, discrepancy in the literature about the definition of spasticity; whereas some authors include symptoms such as clonus, hyperactive tendon reflexes, and spasms within the umbrella term 'spasticity',[1,5-7] others discuss these same symptoms as related to, but separate from spasticity, which is defined by these authors as increased muscle tone.[3,8-11] Decq[2] recently has suggested the use of a modified definition, whereby spasticity, in general, is defined as a symptom of the upper motor neuron syndrome characterized by an exaggeration of the stretch reflex secondary to hyperexcitability of spinal reflexes. He follows by separating the various components of spasticity into sub-definitions: *1) intrinsic tonic spasticity*: exaggeration of the tonic component of the stretch reflex (manifesting as increased tone), *2) intrinsic phasic spasticity*: exaggeration of the phasic component of the stretch reflex (manifesting as tendon hyperreflexia and clonus), and *3) extrinsic spasticity*: exaggeration of extrinsic flexion or extension spinal reflexes. Throughout the discussion to follow, the modified definition of spasticity suggested by Decq[2] will be utilized in order to clearly differentiate between the various spasticity-related symptoms that are experienced by individuals with SCI.

The literature has shown that 65%-78% of sample populations of individuals with chronic SCI (≥ 1 year post injury) have symptoms of spasticity.[6,12] Although unclear, it has been suggested that American Spinal Injury Association (ASIA) classification of SCI (severity) and level of injury may predict the likelihood of developing spasticity; for example, in individuals with a cervical SCI, 93% of those diagnosed as ASIA A and 78% of those diagnosed as ASIA B-D reported having symptoms of spasticity, whereas in individuals with thoracic SCI, 72% of those diagnosed as ASIA A and 73% of those diagnosed as ASIA B-D reported symptoms of spasticity.[6] The greater incidence of lower motor neuron injury associated with lower-level injuries results in a reduced tendency for spasticity development in these individuals.[6,12] Whereas the resolution of spinal shock may coincide with an increase in spasticity symptoms,[12] there is no clear relationship between the presence of spasticity symptoms and time since injury beyond the spinal shock period.[6]

Spasticity has the potential to negatively influence quality of life (QOL) through restricting activities of daily living (ADL), inhibiting effective walking and self-care, causing pain and fatigue, disturbing sleep, compromising safety, contributing to the development of contractures, pressure ulcers, infections, negative self-image, complicating the role of the caretaker, and impeding rehabilitation efforts.[3,6,7,13-17] Reports of problematic spasticity 1, 3, and 5 years following SCI occurred in 35%, ~31%, and ~27% of a sample of a population-based cohort of SCI survivors reported to the

Colorado Spinal Cord Injury Early Notification System.[18] Similarly, of those individuals reporting spasticity in the Stockholm Spinal Cord Injury Study, 40% reported their spasticity to be problematic, in that ADL were restricted and/or the spasticity caused pain.[19] In a study by Sköld and colleagues[6], 20% and 4% of their total sample perceived their spasticity to restrict ADL and cause pain, respectively. Although Krawetz and Nance [20] have identified that severity of spasticity is among the factors that can reduce the degree to which walking is effective in functional ambulators after SCI, Norman and colleagues [21] have emphasized that, despite the common association between spasticity and clinical signs of abnormal gait, the nature of this relationship remains unclear. Furthermore, it must be noted that, although spasticity can have a negative impact on QOL, it has been suggested that symptoms of spasticity may increase stability in sitting and standing, facilitate the performance of some ADL and transfers, increase muscle bulk and strength of spastic muscles (thereby helping prevent osteopenia), and increase venous return (possibly diminishing the incidence of deep vein thrombosis).[10,14-16] This potential for a beneficial effect of spasticity on QOL has a large impact upon decisions regarding its management.[7,16]

The use of varied definitions of spasticity complicate its valid and reliable assessment.[14,22] While the Ashworth and modified Ashworth scales[14,15,23] are commonly used to assess the severity of spasticity, there is some question about their validity in the lower limbs of persons with SCI.[24] As spasticity outcomes vary between clinical patient groups and depend on a variety of factors within each individual, a battery of assessment tools is recommended, incorporating clinical, electrophysiologic, neurophysiological[25] and/or biomechanical techniques.[14,26,27] It is important to note that there are generally poor correlations among clinical scales and, further, reductions in spasticity are not necessarily correlated with improvement in function.[27-29] The lack of agreed-upon measures of spasticity as a whole, or of the various 'components of spasticity' limit the quantification of physical status and the study of effectiveness of management strategies.[27] The ideal scale should not only quantify the degree and nature of the spasticity, but patient satisfaction, global function, and technological assessment should be considered.[29]

2.3 PATHOPHYSIOLOGY OF SPASTICITY AFTER SCI

In general, spasticity is classified as a symptom of the upper motor neuron syndrome, characterized by an exaggeration of the stretch reflex secondary to hyperexcitability of spinal reflexes [2]. Upper motor neurons originate in the brain and brain stem and project to lower motor neurons within the brain stem and spinal cord.[11] The lower motor neurons are of two types, both of which originate in the ventral horn of the spinal cord: 1) alpha motor neurons project to extrafusal skeletal fibres and 2) gamma motor neurons project to intrafusal muscle fibres within the muscle spindle.[11] With a lesion of the CNS comes interruption of the signals sent via the upper motor neurons to the lower motor neurons or related interneurons. Immediately following SCI, a period exists whereby the individual presents with flaccid muscle paralysis and loss of tendon reflexes below the level of the lesion.[5] This period was first described in 1750, with the

term 'spinal shock' introduced by Marshall Hall in 1850.[30] Spinal shock has been reported to end from 1-3 days[31] to a few weeks post-injury, with the gradual development of exaggerated tendon reflexes, increased muscle tone, and involuntary muscle spasms[5]: the symptoms of spasticity. Recent animal research suggested that a recovery of relatively normal motor neuron excitability and plateau potential behaviour (sustained depolarizations), in the absence of normal inhibitory control to turn off plateaus and associated sustained firing, may be implicated in the recovery of spinal shock following SCI.[32]

2.3.1 Intrinsic Tonic Spasticity

Decq[2] has differentiated intrinsic *tonic* spasticity (increased muscle tone) as that component of spasticity resulting from an exaggeration of the *tonic* component of the stretch reflex. Briefly, the stretch reflex is a monosynaptic reflex pathway that originates in the muscle spindles embedded parallel to the muscle fibres and travels via a Ia afferent to the spinal cord, where it synapses either first with interneurons, or directly with an alpha motor neuron innervating the muscle from which the stimulus originated.[11] The tonic component of the stretch reflex associated with increased muscle tone results from a maintained stretch of the central region of the muscle fibres and the reflex is polysynaptic.[11] Upon a sustained stretch, both type Ia and type II afferents (from secondary spindle endings) synapse with interneurons within the ventral horn of the spinal cord. Synapses of the interneurons with alpha motor neurons facilitate contraction in the muscle being stretched.[11]

It is the hyperexcitability of this tonic stretch reflex which is commonly thought to result in increased muscle tone in response to passive stretch following SCI.[3] This hypertonia is velocity-dependent, with faster stretching velocities being associated with greater amounts of reflex activity.[3] The development of tonic stretch reflex hyperexcitability could be due to a lower threshold, an increased gain of the stretch reflex, or a combination of the two.[22] The resultant increase in muscle tone is thought to be due to a combination of increased denervation hypersensitivity [2,3,5,9,33] and changed muscle properties.[11,13,22,34,35] Denervation leads to an initial down-regulation of neuronal membrane receptors, followed by an up-regulation, with enhanced sensitivity to neurotransmitters.[2] Gradual changes in muscle properties also occur following SCI, such as fibrosis, atrophy of muscle fibres, decrease in the elastic properties, decrease in the number of sarcomeres, accumulation of connective tissue, and alteration of contractile properties toward tonic muscle characteristics, which likely contribute to the increased passive tension.[11,13,22,34-36]

2.3.2 Intrinsic Phasic Spasticity

Intrinsic phasic spasticity encapsulates symptoms such as tendon hyperreflexia and clonus, and is due to exaggeration of the *phasic* component of the stretch reflex.[2] Tendon hyperreflexia is identified as an exaggerated muscle response to an externally applied tap of deep tendons.[7] Reduced presynaptic Ia inhibition is thought to play an

important role in this hyperreflexia, as the occurrence of reduced pre-synaptic inhibition of group Ia fibres appears to correlate with the excitability of tendon reflexes.[36]

Clonus has been defined as "involuntary rhythmic muscle contraction that can result in distal joint oscillation"[37] and most often occurs at the ankle. [2,7,9] Clonus is elicited by a sudden rapid stretch of a muscle.[38] The prevailing theory explaining the underlying mechanism responsible for clonus is that of recurrent activation of stretch reflexes.[11,37,38] According to this theory, dorsiflexing the ankle causes activation of the Ia muscle spindle afferents and induces a reflex of the triceps surae, resulting in plantar flexion of the ankle.[11,37,38] This reflex contraction is brief, essentially phasic, and ceases rapidly.[2] The muscle then relaxes, causing the ankle to be dorsiflexed once again, due either to gravity or to the sustaining of the stretch by an examiner.[2] The result is a new stretch reflex, etc.[2,37] Ultimately, it is the disinhibition of the stretch reflex due to interruption of descending influences with SCI which is thought to cause exaggeration of the phasic stretch reflex pathway, and hence, clonus.[3]

The second theory is that clonus is the result of activity of a central oscillator or generator within the spinal cord which rhythmically activates alpha motor neurons in response to peripheral events.[37,38] Beres-Jones and colleagues[37] outline observations that they feel support such a hypothesis: 1) reports of similar frequencies of clonus among ankle, knee, and wrist muscles, 2) observations that the clonus frequency is not entrained by the input frequency suggest that clonus cannot be solely stretch-mediated, 3) the finding that stimuli other than stretch evoke clonus, and 4) the observation of a refractory period following the clonic EMG burst where tendon tap, H-reflex stimulation, and vibration fail to elicit an efferent response. Therefore, whereas reduced pre-synaptic inhibition of group Ia fibres appears to be among the contributing factors to tendon hyperreflexia, the underlying mechanism of clonus has not been clearly elucidated.

2.3.3 Extrinsic Spasticity

In addition to the various intrinsic factors that contribute to symptoms of spasticity, involuntary muscle spasms can also occur in response to a perceived noxious stimulus originating extrinsic to the muscle: *extrinsic spasticity*.[2,3,7] Flexion spasms are the most common form of extrinsic spasticity, triggered by afferent input from skin, muscle, subcutaneous tissues, and joints (collectively referred to as 'flexor reflex afferents'). These flexor reflex afferents mediate the polysynaptic reflexes involved in the flexion withdrawal reflex.[3,35,39] SCI can interrupt the inhibition of these reflexes by supraspinal pathways, making them hyperexcitable.[2,3,40] In other words, whereas flexor withdrawal reflexes occur normally in individuals without SCI, upon disruption of normal descending influences, the threshold for the flexor withdrawal reflex may become lowered, the gain of the system may become raised, or both may occur together.[3] A recent study has provided evidence to implicate plateau potentials in the spinal interneuronal and motoneuronal circuitry in the hyperexcitability of flexion withdrawal reflexes in individuals with chronic SCI.[41] Intrasegmental polysynaptic connections cause the flexor reflex initiated by a localized stimulus to generate a widespread flexor spasm which can appear as a coordinated flexion of all joints of the leg.[35,39]

2.4 MANAGEMENT OF SPASTICITY FOLLOWING SCI

In contrast to the general lack of agreement within the literature about the definition and evaluation of spasticity, there appears to be widespread agreement that decisions regarding the management of spasticity must be based on the goal of achieving balance between the useful and detrimental effects of spasticity on an individual's QOL.[2,10,16,34] The management of spasticity may be desired for the reduction of 'passive problems', such as preventing contracture, reducing pain, facilitating splint wearing, easing positioning and hygiene, and preventing contractures, or of 'functional problems', including the individual's reduced ability to perform useful work with the motor system.[9]

In general, no one treatment option will successfully manage spasticity in all individuals; the most conservative tactics are utilized first, with a progression from physical rehabilitation modalities, pharmacologic interventions, injection techniques, intrathecal baclofen, and lastly, surgery.[15] In general, local treatments are used primarily by individuals with spasticity predominating in only certain muscle groups, such as occurs mainly in individuals with stroke or traumatic brain injury.[42] In the case of SCI, the distribution of spasticity tends to be more diffuse, making regional or systemic treatment preferable.[34] The decision of whether or not to treat spasticity and, if so, in what manner, is summarized nicely in a flow chart by Parziale and colleagues (figure 2.1[16]).

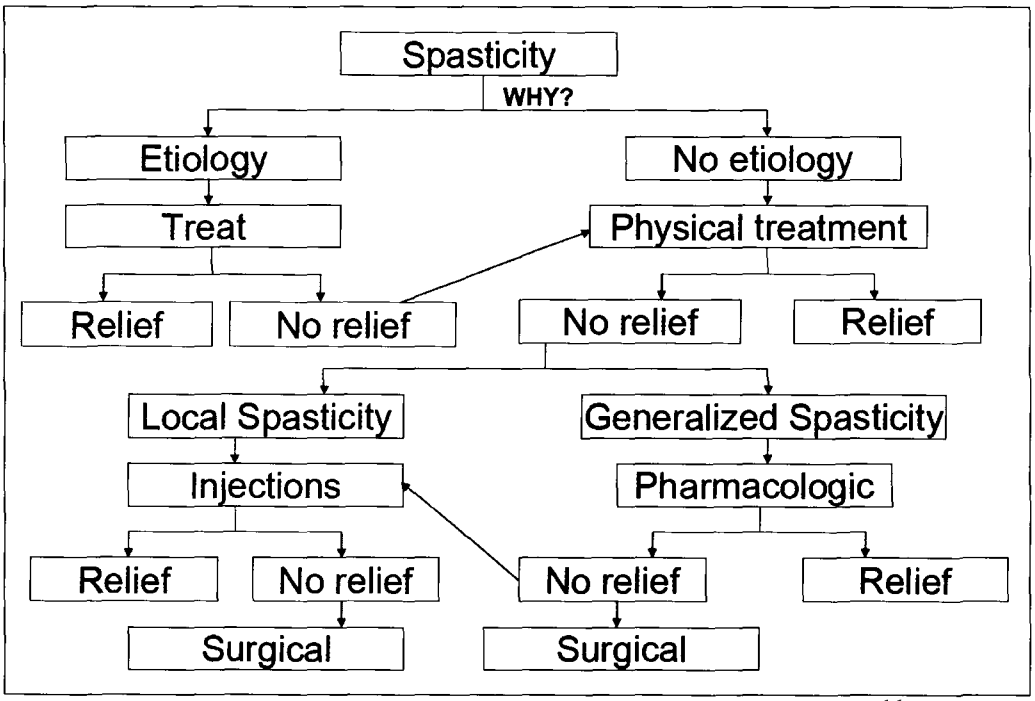

Figure 2.1 Spasticity management (adapted from Parziale and colleagues[16]).

2.4.1 Conservative/Physical Rehabilitation Management

It is generally agreed that physical therapy/rehabilitation is an essential component in the management of spasticity as a first line of defence, as well as in a long-term regimen during and after the implementation of pharmacological or surgical strategies.[10,17,36] The goal of physical therapy is to diminish spasticity in order to allow expression of voluntary mobility and movements and/or to improve the comfort and independence in tasks related to QOL, such as transfers, dressing, and using the washroom.[43] The literature on the conservative/physical treatment of spasticity is sparse, and some have questioned the effectiveness of these management strategies.[36] Table 2.1 summarizes the most common physical therapy approaches to spasticity management.

Table 2.1 Physical techniques in the management of spasticity.

Physical therapy technique description and comment on effectiveness	*Purpose/ suggested mechanisms*
Positioning[15,43] • In bed and during sitting • Reports of clinical effectiveness; impact remains to be proven scientifically[14]	• Important to the maintenance of muscle length[15,43]
Range of motion/stretching • Includes passive stretch and passive lengthening[14,43] • Benefits may carry over for several hours[15,16] • Effects remain to be quantified and the efficacy remains to be determined despite the clinical evidence for the benefits[14]	• Prevents contractures[42,43] • Causes temporary reduction in intensity of muscle contraction in reaction to muscle stretch[43] • May cause plastic changes within the central nervous system and/or mechanical changes at the muscle, tendon, and soft-tissue level[14]
Weight-bearing • Using a tilt table or standing frame • Benefits are greater than stretching alone and may persist into next day[8] • Effectiveness has been questioned[14]	• Prolonged stretch of ankle plantar flexor muscles[8,14,15] • Mechanism remains uncertain; suggested to include a modulating influence from cutaneous and joint receptor input to the spinal motor neurons, resulting in decreased excitability[8]
Muscle strengthening • Progressive addition of resistance to muscles with voluntary control[16,43]	• Emphasis of balance of agonist and antagonist groups of muscles with voluntary control[16,43]
Electrical stimulation • Various methods: stimulation to the antagonist muscle, application of tetanic contraction to the spastic muscle, functional electrical stimulation (FES), and transcutaneous electrical nerve stimulation (TENS)[14-16,43] • Reports of beneficial effects between only 10 minutes and 3 hours[14,15,43]	• Stimulation of the antagonist muscle: augmentation of reciprocal inhibition of the spastic muscle[14] • Repetitive tetanic stimulation of spastic muscle: fatigue of the muscle due to repetitive tetanic stimulation[14] • FES: change the mechanical properties of a spastic joint by strengthening the antagonists of the spastic muscle or might decrease the hyperactivity of spastic muscles through reciprocal inhibition[54] • TENS: may involve the stimulation of large diameter afferent fibres that travel from mechanoreceptors to the spinal cord[14]

Epidural spinal cord stimulation • For mild spasticity and incomplete lesions: stimulation below the level of the lesion found effective (spasms)[55] • For severe spasticity: stimulation of dorsal roots of the upper lumbar cord segment found effective (hypertonus and spasms)[56] • Shown to lack long-term effectiveness[57]	• May involve the activation of inhibitory networks within the spinal cord[56] • More strongly affected patients require stronger stimuli and/or higher frequencies[56]
Cold/heat application • Application of a cold pack or of a vapocoolant spray, or of superficial heat • Following cold application: tendon reflex excitability and clonus may be reduced for a short period of time (e.g., < 1 hr), allowing for intermittent improved motor function[15,16] • Following heat application: subsequent passive stretch is facilitated[16]	• Cold: may cause slowing of nerve conduction, decrease in sensitivity of cutaneous receptors, and alteration of CNS excitability[14-16] • Heat: facilitation of uptake of released neurotransmitters and return of calcium to the sarcoplasmic reticulum[16]
Splinting/orthoses • Helpful in the continuous application of muscle stretch • Use of splints is questioned[14]	• Enables long-term stretch[42,43] • Joint can be maintained in a position that does not elicit a spasm[15]

2.4.2 Pharmacologic Management

Systemic pharmacological treatments of spasticity symptoms are often prescribed in the SCI population.[34] Most antispasticity medications can be grouped roughly into three functional categories: 1) 'GABAergic' – drugs that act at interneurons that use the neurotransmitter gamma-aminobutyric acid (GABA) in the CNS (e.g., baclofen and diazepam), 2) alpha-2-adrenergic – those that act at alpha-2 receptors in the CNS (e.g., tizanidine and clonidine) and 3) peripheral acting – those that act at the neuromuscular level (e.g., dantrolene).[44] Numerous factors are taken into consideration when the physician and patient are deciding upon the role of pharmacologic management, including time since injury, onset of spasticity, severity, prognosis, available support system, cognitive status, concurrent medical problems, geographic location, symptom location, and financial issues.[9] Each type of medication has potentially serious side effects and no single medication has a beneficial effect in all individuals.[15,34]

2.4.2.1 Diazepam

GABA is an inhibitory neurotransmitter typically found in short interneurons.[45] There are two types of GABA receptors, with diazepam acting at the $GABA_A$ receptor, the more prominent of the two.[34] When GABA is released from a presynaptic membrane and binds to $GABA_A$ receptors on the postsynaptic membrane, chloride channels are opened, allowing chloride ions to flow into the postsynaptic membrane, hyperpolarizing the membrane.[34] As a result, GABA inhibits action potential transmission. Presynaptic inhibition occurs when a GABAergic interneuron connects with the terminal of a Ia

afferent and, therefore, decreases the excitability of the Ia afferent terminal and causes a decrease in the transmitter released from the Ia afferent to, ultimately, the motor neuron.[34]

Diazepam is the most commonly used agent among the benzodiazepines to treat spasticity, which, as a group, act by enhancing the efficiency of GABAergic transmission.[9,10,13,44] Diazepam does not directly mimic GABA.[15,34] Rather, it binds post-synaptically near $GABA_A$ receptors, facilitates GABA-mediated chloride conductance and, therefore, hyperpolarizes the membrane.[9,10,15,44] The result of diazepam administration is an increase in presynaptic inhibition of afferent neuronal terminals and, thus, a reduction of monosynaptic and polysynaptic reflexes.[9,13,34] Diazepam is often mentioned as being most effective in the treatment of hyperactive reflexes and painful spasms in individuals with SCI (compared to individuals with stroke or multiple sclerosis).[9,10,13,15] Functional measures, however, have been shown not to improve with diazepam treatment.[9] Clonazepam, another benzodiazepine, causes less sedation than diazepam and has slightly lower risk for dependence; it is typically used for the reduction of nighttime spasms.[15,34]

2.4.2.2 Baclofen

Unlike diazepam, baclofen is a structural anologue of GABA and an agonist of $GABA_B$ receptors.[9,10,13,15,44] When baclofen binds to $GABA_B$ receptors both presynaptically and postsynaptically, monosynaptic and polysynaptic spinal reflexes are inhibited.[9,10,13,15,44] Upon binding of baclofen presynaptically, influx of calcium into the presynaptic terminal is restricted and neurotransmitter release in excitatory spinal pathways is decreased, leading to a decrease in alpha motor neuron activity.[9,10,15,34,45] When baclofen binds to $GABA_B$ receptors on the postsynaptic membrane of a Ia afferent, potassium channels allow the flow of potassium out of the Ia afferent terminal, resulting in membrane hyperpolarization and, hence, interruption of action potential transmission.[9,10,34]

Baclofen is a commonly used drug for spasticity in the SCI population.[21] It has been reported to be particularly effective for reducing flexor spasms.[9,10,34] However, the literature also suggests that baclofen may have no positive effect on walking ability or the performance of ADL.[9,21,34,44] Similar to other muscle relaxants, baclofen may impair the ability of the patient to walk or stand.[13,15] Baclofen has been shown to be safe and effective for long-term use, with no evidence for tolerance.[15]

2.4.2.3 Clonidine

Clonidine is a centrally acting alpha-2 adrenergic agonist commonly used to treat hypertension.[9,34] Alpha-2 receptors are located, among other regions, on presynaptic nerve terminals in the CNS and are termed adrenergic because they are involved in release of norepinephrine and acetylcholine.[45] Generally speaking, an alpha-2-adrenergic agonist can bind to apha-2 receptors, thereby preventing the normal action of norepinephrine to act as a neurotransmitter.[45] Therefore, clonidine can act spinally to reduce spasticity by enhancing alpha-2-mediated presynaptic inhibition of sensory

afferents, thereby suppressing spinal polysynaptic reflexes.[9,15] Clonidine has been found to be associated with improved walking ability in individuals with incomplete SCI (e.g., longer cycles, increased treadmill speed, and more upright posture).[21,46]

2.4.2.4 Tizanidine

Tizanidine is an imidazole derivative and, like clonidine, is a centrally-acting (spinally and supraspinally) alpha-2-adrenergic agonist.[9,10,13,34,44] Therefore, it acts by inhibiting the release of excitatory amino acids from the presynaptic terminals of excitatory spinal neurons.[9,10] It may also facilitate the inhibitory neurotransmitter glycine.[9,10,13] Tizanidine has been shown to reduce muscle tone and frequency of muscle spasms in individuals with SCI, but no increase in functional measures has been noted.[9,10,13,15,34] Manual muscle testing while being treated with tizanidine has indicated that strength is not decreased,[10,15] although weakness has been reported.[34]

2.4.2.5 Dantrolene Sodium

Dantrolene sodium is the only oral medication that acts peripherally at the muscle tissue, rather than at the spinal cord level, to weaken muscles that are overexcited.[9,15,34] It is a hydantoin derivative that inhibits muscle action potential-induced release of calcium from the sarcoplasmic reticulum to the active myosin fibres during muscle contraction by increasing the binding of calcium to the sarcoplasmic reticulum.[9,10,13,15,34,44] The result is interference with excitation-contraction coupling that is necessary to produce muscle contraction.[10,44] It has also been suggested that dantrolene may alter muscle spindle sensitivity by acting on the gamma motor neurons.[9,34] Dantrolene appears to have a greater effect on phasic than on tonic stretch reflexes and on fast twitch rather than slow twitch muscle fibres, with the clinical significance of these discrepancies remaining unclear.[9,10,15,34] There is evidence that individuals with SCI respond well to dantrolene sodium, with possible reductions in muscle tone, tendon reflexes, and clonus, and increases in range of motion.[9,34] Improvement in performance of ADL is less evident.[34] Individuals with SCI are rarely treated with dantrolene, likely because its peripheral site of action results in its most common adverse effect: muscle weakness.[9,10,13]

2.4.2.6 Cyproheptadine

Although less commonly reported among the drugs used to treat spasticity, cyproheptadine has been associated with an improvement in walking pattern in individuals with SCI (e.g., reduced need for manual assistance, increased treadmill speed, and reduced ankle clonus).[21,47,48] Cyproheptadine is an histamine and a serotonin antagonist which is proposed to reduce spasticity via inhibition of motor neurons by "neutralizing the spinal and supraspinal serotoninergic excitatory inputs".[34]

2.4.2.7 Cannabis

Tetrahydrocannabinol (THC), available in the drug dronabinol, is the main active ingredient in cannabis.[15] Cannabinoids have been shown to have efficacy in treating spasticity and are currently being studied.[9] Anecdotal reports by individuals with SCI have also revealed a beneficial effect of marijuana on the management of spasticity.[34] Some literature supports the hypothesis that the relaxing effect of marijuana on muscles in patients with SCI-related spasticity is due to an antispastic effect, perhaps inhibition of polysynaptic reflexes, rather than to simply a general relaxation response.[34,44]

2.4.2.8 Pharmacologic-Induced Negative Side-Effects

Although pharmacologic agents have been shown to be effective in the reduction of spasticity symptoms, the overall QOL of the individual may become hampered by unwanted pharmacologic-induced side effects. Briefly, these side effects vary between pharmacologic agents and may include sedation, drowsiness, insomnia, fatigue, nausea, diarrhea, dry mouth, muscle weakness, ataxia, dizziness, hypotension, depression, impaired memory and attention, hallucinations, liver toxicity, and possible addiction.[9,10,13,15,34] Furthermore, abrupt cessation of certain agents, particularly diazepam and baclofen, may lead to anxiety, agitation, restlessness, irritability, tremor, muscle fasciculation and twitching, nausea, hypersensitivity to touch, taste, smell, light, and sound, hallucinations, insomnia, nightmares, and seizures.[10,13,16,34]

2.4.3 Intrathecal Administration of Baclofen

Intrathecal administration of baclofen combines the parmacologic administration of baclofen with a surgical technique.[15] In individuals who do not respond to oral administration of medications or to other techniques or who have had intolerable side effects from medications, intrathecal baclofen may be indicated and should be considered prior to surgical intervention.[10,15,34] Briefly, a pump with reservoir (~ 4 inches in diameter) is surgically implanted in the subcutaneous tissue of the abdominal wall, allowing the direct delivery of the drug to the cerebrospinal fluid.[10,15,34] A percutaneous puncture into the access port allows access to the pump reservoir.[15] The dose and flow rate of baclofen are individualized through external computer communication with a computer chip within the pump.[15]

Bypassing the blood-brain barrier allows as much as 4 times the concentration of baclofen to be delivered to the spinal cord with only 1% of the oral dose.[10,15] One of the main advantages is the reduction in negative systemic side effects compared to oral administration.[15] The effectiveness of intrathecal baclofen as an anti-spasticity management therapy has been shown in individuals with SCI, with little data on functional improvements or QOL being reported.[17,34,44,49] Recent reviews have described the effects of intrathecal baclofen on reducing hypertonus, spasm frequency, reflex intensity and/or spasticity-related discomfort, as well as improving QOL through

allowing the individual to eat, feel, and look better and facilitating transfers, nursing care, sleep, and, in some, walking ability.[17,50]

The long-term effects of treatment with intrathecal baclofen are not yet known.[17] Possible complications as a result of the surgical implantation of the pump include dislodgement, disconnection, migration, catheter kinking, blockage, pump failure, battery depletion, infection, and accidental overdose.[10,15] As with oral baclofen, drowsiness, dizziness, nausea, hypotension, headache, weakness, and withdrawal syndrome are possible side effects.[15]

2.4.4 Injection Techniques – Chemodenervation Agents

Injection for the purpose of local chemodenervation is one of the four possible routes of administration of a pharmacologic agent (with enteral, transdermal, and intrathecal administration being the other three).[9] The technique actually treats the upper motor neuron syndrome by simulating a lower motor neuron lesion.[9] Injection techniques are preferred for treatment of focal spasticity and when agonist muscles have the functional strength once freed from antagonist spasticity.[9,15] One of the benefits of injection is the minimization of systemic side effects.[9,15] The injections can be applied as nerve blocks or motor point blocks, which are temporary, or as chemical neurolysis, which permanently destroys a portion of the nerve; whether an injection results in temporary or permanent chemodenervation depends on the concentration of agent administered.[42] The chemodenervation agents used include phenol, ethanol, and, more recently, botulinum toxin.[9]

2.4.4.1 Phenol and Ethanol Injections

Local injections of phenol or ethanol are utilized less commonly in individuals with SCI; for a detailed review, see Gracies and colleagues.[42] Briefly, administration of phenol or ethanol to a nerve trunk causes short-term effects similar to a local anesthetic: blocking of sodium channels reduces nerve depolarization.[16] The mechanism of the longer-term nerve block involves denaturing of protein and fibrosis of neural tissue, causing disruption of nerve conduction and interruption of the reflex arc and, hence, muscle relaxation.[14-16,42] Recovery is variable between individuals, from a few days to months, as axons regenerate and reach motor endplates (Wallerian degeneration and regeneration).[14,16,42] A number of factors may influence the duration of the effects, including the concentration and volume used for injection, the site of the block, vascular complications, cutaneous side-effects, excessive motor weakness, sensory loss, wound infection, treatment variables after the block, and systemic side effects.[42] A progressive reduction in motor unit activity tends to occur with repeated injections, however, as there is some permanent denervation with every injection.[17]

There have been fewer reports of adverse effects of ethanol injection compared to phenol.[42] Among the complications of these are injection site pain (particularly when intramuscular), vascular complications (phlebitis), permanent nerve damage, skin irritation, acute systemic effects (tremor, convulsions, central nervous system depression,

and cardiovascular collapse), chronic dysesthesia, tissue necrosis, sensory dysaesthesia, post-block pain due to an incomplete block, and muscular weakness.[14-17,42] Most individuals who undergo a phenol or an ethanol injection have preservation of motor strength.[42]

2.4.4.2 Botulinum Toxin

Botulinum toxin is the most potent neurotoxin known to man and product of the anaerobic bacteria *clostridium botulinum.*[10,15,51] It was initially developed for clinical use in 1980 by an opthalmologist to treat involuntary contractions and spasms of the eyelid muscles and 'crossed-eyes', and first was examined formally for the treatment in spasticity in 1989.[15,51] Seven immunologically distinct toxins have been identified (type A through G),[10,15] with Type A and B being currently available for treatment.[9] Compared with ethanol and phenol, whose actions are mediated by their ability to denature protein at the nerve, the botulinum toxins manifest their effects at the neuromuscular junction where they inhibit the release of acetylcholine from presynaptic motor axons.[9,10,14,15]

More specifically, botulinum toxin is injected into a muscle at its end plate region and spreads throughout the muscle and fascia approximately 30 mm.[10,13] The mechanism of action of botulinum toxin then occurs in three stages, through coordinated action of the heavy and light chain components of the toxin: 1) *binding:* botulinum toxin binds to the presynpatic neuron at the neuromuscular junction via the heavy chain,[10,13,14] 2) *internalization:* the toxin is internalized into the cell by endocytosis, where the heavy chain forms a channel to allow the light chain to enter the cytosol and 3) *inhibition of acetylcholine release:* the release of acetylcholine from presynaptic vesicles is inhibited.[15] In step 3, the system involved in ACh exocytosis (the soluble N-ethlylmaleimide-sensitive fusion protein attachment protein receptor (SNARE) complex) is proteolytically cleaved in different critical sections by the different toxin types.[9,15,51] Type A has been described as activating zinc-dependant proteolysis of SNAP-25 (a synaptosome-associated protein), whereas toxin B is active on synaptobrevin-2, a protein attached to the acetylcholine vesicle (involved in docking and fusion of the synaptic vesicle to the presynaptic membrane).[9,14,15,51] With a disrupted SNARE complex, acetylcholine cannot be released from the presynaptic terminal, and muscle contraction is inhibited. Therefore, without affecting the synthesis of acetylcholine, botulinum toxin causes reversible chemical denervation atrophy, thereby weakening muscle.[10,13,15]

Because of the complex mechanism of action, chemical denervation subject to botulinum toxin injection develops slowly over the course of 24 to 72 hours, peaking at 2 to 6 weeks.[10,13,15] Collateral sprouting and slow reinnervation of chemically denervated nerve terminals allows for a gradual reversal of the clinical response.[14,15,51] The duration of the botulinum toxin response can depend on a number of factors, including muscle size, the dose of the toxin administered, activity of the muscle, and perhaps factors including physiotherapy and bracing.[14] In general, the duration has been reported to be between 2 and 6 months, with ~3 months being common.[10,13-15,51]

Botulinum toxin has recently been touted "the pharmacological treatment of first choice for focal spasticity"[17] because of the evidence for its effectiveness in reducing pain

and tone, and improving range of motion, function, brace tolerance, and walking ability.[14,17] Although botulinum toxin is not as commonly used in individuals with generalized spasticity (such as in SCI), improvements in pain, nursing care, hygiene, comfort, and functional activities can be induced by botulinum toxin injections into isolated muscle groups.[15] Botulinum toxin therapy can also be combined with other treatments to enhance rehabilitation and function.[15] In general, the literature indicates that botulinum toxin injections potentially can be useful in the treatment of spasticity secondary to SCI.[15,51,52]

There have been very few reports of severe adverse reactions due to the injection of botulinum toxin.[15] Any possible complications of botulinum toxin are often related to the blocking of acetylcholine release from parts of the autonomic nervous system by the toxin (dry mouth, reduced sweating), or to the spreading of the toxin beyond the desired area, resulting in excessive weakness (which is ultimately reversible).[10,13-15,51]

2.4.5 Surgical Management

As most surgeries performed on patients with spasticity take place at the muscle or the tendon, they are useful for treatment of focal spasticity with a purpose of improving function, correcting a deformity, or for cosmetic reasons.[14,53] Although there are and have been several possible surgical techniques to treat spasticity (for a review, see Chambers[53]), only those currently relevant to individuals with SCI will be discussed here. For example, selective rhizotomy (cutting of posterior roots to interrupt the peripheral reflex arc), though shown to be encouraging for children with cerebral palsy, is not frequently used in individuals with SCI.[13,15,53] Intrathecal baclofen administration, often discussed under the topic of surgical management of spasticity,[13,15] has been discussed above; currently, it is considered to be the most commonly used and successful of the surgical treatments for spasticity in individuals with SCI.[13]

Orthopedic surgical techniques (as opposed to neurosurgical) are reserved for only selected cases.[14] A tenotomy, the release of a tendon from a severely spastic muscle, might be performed in individuals with severe spasticity and without voluntary movement.[14] Tendon lengthening serves to reduce the pull on spastic muscles, thereby positioning the joints at a more natural and useful angle.[14] A tendon transfer, moving the tendon attachment to bone closer to the muscle, is performed in muscles which have at least partial voluntary function, with the goal of allowing these muscles to produce useful movements.[14] The mechanism of action of tendon lengthening and tendon transfers in terms of spasticity-reduction is via alteration of the tension in the intrafusal muscle spindle, resulting in a decreased stimulus for further contraction and, hence, in theory, reduced spasticity.[53] It has been reported, however, that the effect of tendon lengthening and tendon transfer on spasticity is variable and unpredictable.[53]

2.5 Conclusion

Symptoms of spasticity are experienced by the majority of individuals with SCI and are a possible contributor to reduced QOL.[3,6,7,13-17] The emerging understanding of

the different pathophysiologies of the various presentations of spasticity symptoms has led to a recent suggestion that distinct terminology be used for these symptoms.[2] By considering intrinsic tonic spasticity, intrinsic phasic spasticity, and extrinsic spasticity as having distinct etiologies, the identification and classification of troubling symptoms becomes more specific, thereby allowing for more effective application of management strategies. Our growing understanding of spasticity in the SCI population is serving to further enhance the QOL of those who find their spasticity symptoms to be problematic; the development of valid and reliable assessment tools is now an important consideration for continued improvement in spasticity classification and management decisions.

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CHAPTER 3

SPINAL CORD INJURY SPASTICITY EVALUATION TOOL (SCI-SET): DEVELOPMENT AND EVALUATION

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3.0 SPINAL CORD INJURY SPASTICITY EVALUATION TOOL (SCI-SET): DEVELOPMENT AND EVALUATION

3.1 ABSTRACT

Objective: Develop and assess the reliability and validity of a new scale designed to measure the impact of spasticity on daily life in people with spinal cord injury (SCI).
Design: Scale development and assessment.
Setting: General community.
Participants: Community-dwelling individuals with chronic SCI and spasticity participated in Study 1 (n=9), Study 2 (n=19) and Study 3 (n=61).
Interventions: Not applicable.
Main Outcome Measures: Study 1: participant definitions of spasticity and list of scale items. Study 2: scale refinement, face validity, and time to complete. Study 3: internal consistency, test-retest reliability, and construct validity.
Results: The Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET) is 7-day recall self-report questionnaire that takes into account both the problematic and useful effects of spasticity on daily life in people with SCI. The scale exhibited good face validity and required 6.8±2.6 min to complete. The internal consistency (α) and intraclass correlation coefficient of the SCI-SET were 0.90 and 0.91, respectively. Construct validity was supported by correlations ($r=-0.48$ to 0.68; $p<0.01$) between SCI-SET scores and theoretically meaningful constructs.
Conclusions: The SCI-SET fills a need for a reliable and valid self-report measure of the impact of spasticity on daily life in people with SCI, taking into account both the problematic and useful effects of spasticity.

3.2 INTRODUCTION

A spinal cord injury (SCI) is most commonly sustained by individuals who are relatively young in age and, over the last three decades, there has been a 40% decline in mortality during the critical 2-year period post-SCI.[1] Therefore, it has become increasingly important to focus on the long-term effective management of the secondary consequences of SCI that can impact quality of life. Spasticity has been reported in 65% to 78% of samples of individuals with chronic SCI.[2,3] The SPASM group[4, p.72] has recently suggested that spasticity be defined as: "disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary contraction of muscles". Therefore, the term 'spasticity' is no longer limited to only 'muscle hypertonicity' (as was initially suggested by Lance[5]) but, rather, is acknowledged to include numerous positive features of the upper motor neuron syndrome (i.e., clonus, spasms).

It is generally accepted that the effects of spasticity can be perceived as both problematic and beneficial by individuals with SCI.[6,7] Spasticity can contribute to pain, falls, fatigue, and poor self-esteem/body image, and can interfere with mobility, transfers, self-care, activities of daily living, social participation, caregiving, sleep, and sexual functioning.[8-14] However, it also has been reported that spasticity can have beneficial effects on sitting, standing, transfers, walking, and lower body dressing / performance of activities of daily living.[8-12,15,16] Accordingly, there is widespread agreement in the literature that decisions regarding the treatment of spasticity must be based on the goal of achieving balance between the useful and problematic effects of these symptoms on an individual's quality of life.[12,17-19]

In order to make decisions about spasticity management and to evaluate the effects of treatment in people with SCI, it is necessary to apply measurement instruments shown to be valid and reliable in this population. There are a number of measures of spasticity (clinical, biomechanical, electrophysiological) described within the literature that are performed/evaluated by the examiner.[20-24] Although some of these measures have limitations, careful selection of a combination of measures can provide an appropriate overall *examiner-based* assessment of spasticity.[25,26] There are many indications, however, that spasticity should also be assessed by the *individual*. For example, examiner-based physical examination does not necessarily elicit spasticity in individuals who report the symptom[3] and individuals with SCI may experience spasticity in body segments that are not tested by an examiner[7]. Research has also indicated that *examiner-based* assessments of one or more symptoms of spasticity do not correlate well with *self*-assessments of spasticity,[7,25] with improved function,[13,27] or with each other.[25] Self-assessments have also been found to be more sensitive to changes in aspects relevant to daily life following implementation of a spasticity management intervention.[15,20,28] These findings support the suggestion that the best judge of spasticity severity is the individual, who can assess the impact of spasticity on daily life.[11]

Routine clinical work often includes self-evaluation or self-descriptions of the extent and impact of spasticity.[7] Although limited, there are also examples of research studies that have begun to include self-ratings among the outcome measures of spasticity

in the SCI population. Examples of these self-ratings include measures of both spasticity severity (e.g., using visual analogue scales[15] or single item ratings[29]) or spasticity impact (on daily pain and function[25], or daily life[7]). It is interesting to note that not all individuals with SCI who report *having* spasticity indicate that spasticity has an *impact* on their daily life.[7]

It is encouraging that the importance of participant-rated spasticity is being recognized. None of the methods of measurement implemented to date, however, have been appropriately validated or are broad enough in scope to capture both the detrimental and potentially beneficial impact of spasticity on daily life in individuals with SCI. It is noteworthy that an 88-item self-report spasticity scale has been recently designed for the multiple sclerosis population.[30] This scale, however, is inadequate for the SCI population because it only measures the problematic effects of spasticity and it was specifically designed for the multiple sclerosis population. As a result, the scale includes items that would not be relevant to many individuals with SCI. Therefore, the purpose of the present paper is to describe the development and preliminary assessment of the Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET), a self-report measure of the impact of spasticity on daily life in people with SCI.

3.3 METHODS

3.3.1 Overview of Studies and Participants

The development and evaluation of the SCI-SET were performed in three studies. Study 1: A definition of spasticity was developed and a list of possible aspects of daily life that may be affected by spasticity was generated through a detailed search of the spasticity literature and interviews with individuals with SCI. Study 2: The SCI-SET underwent pilot testing and refinement based on participant suggestions. Study 3: Test-retest reliability and construct validity of the SCI-SET were assessed through administration of the scale to a sample of people with SCI. Community-dwelling individuals with SCI were recruited for these studies from three sources: physiatrist referral, a community exercise program for people with SCI, and at local events targeting the SCI population. Because the SCI population is very heterogeneous,[31] participants for this study represented a wide range of characteristics; individuals with neurological levels from C2 (C; cervical) to T12 (T; thoracic), American Spinal Injury Association (ASIA) impairment ratings[32] from A to D, and number of years post-injury from 1yr to 34yrs were included (Table 1). Participants also varied in terms of age, highest education level completed, and current employment status (Table 1). All individuals provided verbal informed consent before participating and the study was approved by the McMaster Research Ethics Board.

3.3.2 General Statistical Analyses

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS) for Windows, v. 10.0. Data are presented as mean±SD. For all analyses, the level of statistical significance was set at $p < 0.05$.

3.4 Methods and Results

3.4.1 Study 1: Development of the SCI-SET

3.4.1.1 Methods

Semi-structured individual interviews and a comprehensive search of the literature were conducted to generate: a) an operational definition of spasticity and b) a comprehensive list of aspects of daily life that can be affected (benefited or hindered) by spasticity. Additional participants were interviewed until no new themes emerged, resulting in a final sample of nine individuals with SCI and self-reported spasticity (see Table 3.1). Information collected during the interviews was used to develop scale items and design a preliminary questionnaire.

3.4.1.2 Results

The first version of the SCI-SET was a 38-item, 7-day recall questionnaire that took into account the potential problematic and helpful effects of spasticity on daily life in people with SCI. Responses could range from -3 (extremely problematic) to +3 (extremely helpful), with the option of choosing "0" if spasticity had no effect on that aspect of life. The instructions developed for the SCI-SET included a statement asking participants to recall the previous week: "*For each of the following, please choose the answer that best describes how your spasticity symptoms have affected that area of your life during the past 7 days.*". The following operational definition of spasticity was also developed and included in the instructions: "*When I talk about "spasticity symptoms", I mean: a) uncontrolled, involuntary muscle contraction or movement (slow or rapid; short or prolonged), b) involuntary, repetitive, quick muscle movement (up and down; side to side), c) muscle tightness, and d) what you might describe as "spasms".*"

Table 3.1 Study sample characteristics

Study	Para/ Tetra *N* (%)	Age M (SD)	YPI M (SD)	M.Inc/ M.Comp *N* (%)	Male/ Female *N* (%)	Primary Mode of Mobility: Power Chair *N* (%)	Primary Mode of Mobility: Manual Chair *N* (%)	Primary Mode of Mobility: Assisted Walking *N* (%)	Highest Education Completed: Uni/ Coll *N* (%)	Highest Education Completed: HS *N* (%)	Highest Education Completed: MS *N* (%)	Employment Status: Student *N* (%)	Employment Status: Emp/ Vol *N* (%)	Employment Status: Unemp/ Retired *N* (%)
1. Development (*N*=9)	3/6 (33/67)	37.6 (11.1)	13.6 (11.5)	5/4 (56/44)	8/1 (89/11)	4 (44)	4 (44)	1 (11)	6 (67)	3 (33)	-	-	5 (56)	4 (44)
2. Pilot Testing (*N*=19)	2/17 (11/89)	45.7 (13.9)	16.1 (9.7)	12/7 (63/37)	15/4 (79/21)	9 (47)	9 (47)	1 (5)	10 (53)	8 (42)	1 (5)	2 (11)	6 (32)	11 (58)
3. Evaluation of reliability and validity (*N*=61)	24/37 (39/61)	41.9 (12.6)	10.2 (8 6)	25/36 (41/59)	45/16 (74/26)	16 (26)	42 (69)	3 (5)	38 (62)	18 (30)	5 (8)	7 (11)	16 (26)	38 (62)

Continuous variables are presented as means with standard deviations in parentheses. Categorical variables are expressed as numbers with percentages in parentheses. Due to rounding, not all categories add up to 100%.

Para, paraplegia; Tetra, tetraplegia; YPI, years post-injury; M.Inc, motor-incomplete (ASIA C or D); M.Comp, motor-complete (ASIA A or B); Uni/Coll, university or college; HS, high school; MS, middle school; Emp/Vol, employed full-time or part-time or volunteer; Unemp/Retired, unemployed or retired.

3.4.2 Study 2: Pilot-Testing the SCI-SET

3.4.2.1 Methods

The preliminary version of the SCI-SET underwent two phases of pilot testing in 19 individuals with SCI and self-reported spasticity (see Table 1); minor changes were made based on comments of the first fourteen participants and the revised SCI-SET was then pilot tested in an additional five participants. The SCI-SET instructions (including the operational definition of spasticity) and all scale items were administered to participants by telephone or in person. Immediately upon completion of the questionnaire, participants were asked to provide feedback about the scale items and the questionnaire as a whole through four open-ended questions addressing: 1) clarity/ease of understanding, 2) ease of completion, 3) appropriateness of the items (redundancy/clarity/sense), and 4) whether the questionnaire captured how they felt about the impact of spasticity on daily life and, if not, what was missing. Participant feedback was used to refine the SCI-SET. Overall face validity of the SCI-SET was assessed on a 5-point scale (1 = very irrelevant; 5 = very suitable) and time to complete the questionnaire was recorded.

3.4.2.2 Results

Pilot testing of the preliminary version of the SCI-SET identified the need for a statement at the end of the instructions allowing participants to rate items as non-applicable when appropriate: "*Please let me know when a question is not applicable to you.*". The original 38 SCI-SET items were pared down to 35; of the original items, two were combined because of similarity (indicated by a high correlation between the items; r=0.89) and two were removed from the questionnaire because further scrutiny by the investigators identified them as being unsuitable. During final pilot testing of the SCI-SET, no items were considered redundant, unclear, or nonsensical, and no additional items were suggested. Rating of the relevance/suitability of the SCI-SET by the participants resulted in a mean score of 4.4±0.6 out of 5, indicating good face validity. The SCI-SET required 6.8±2.6 min to complete. The final version of the SCI-SET (see Appendix A) is scored by summing the responses from all applicable items and dividing the sum by the number of applicable items. Therefore, scores on the SCI-SET can range from -3.00 to +3.00.

3.4.3 Study 3: Assessing the test-retest reliability and construct validity of the SCI-SET

3.4.3.1 Methods

Sixty-one individuals with SCI and self-reported "stable" spasticity (i.e., consistent medication routine and absence of conditions known to affect spasticity intensity, frequency, or impact[15]) completed all of the study measures. Reliability of the

SCI-SET was assessed by administration of the SCI-SET in person or by telephone, three times, three weeks in a row, on the same day of the week. The same administration format was used across all three time-points for each participant.

Prior to the first scheduled questionnaire administration, participants received the SCI-SET instructions and rating scale (see Appendix A), and an example question. If participants could not be reached on a scheduled questionnaire administration date, efforts to reach the individuals were continued with the goal of minimizing alterations to the planned schedule. For 37 participants, the questionnaire administration proceeded as planned. For 24 participants, the second or third administration was greater than 7 days (8 to 14 days) following the first or second administration, respectively. At the time of his second scheduled telephone interview, one participant revealed the recent onset of a urinary tract infection. Because of previous experience with this complication, the participant assured us he would be well within one week and would be ready to perform his second interview two weeks from the scheduled interview date. This was the case and, therefore, the second and third interviews were performed 14 and 21 days following the first interview Removing this participant from the analyses did not alter the results; therefore, his data were included.

Self-assessments of spasticity have repeatedly shown to have poor correlations with examiner-based measures of spasticity[7,25] and there is no accepted "gold-standard" criterion measure of spasticity. Therefore, rather than criterion validity, the construct validity of the SCI-SET was assessed. Construct validity reflects the correlation between the SCI-SET and other theoretically related constructs for which current measures exist.[33] Since spasticity is thought to be related to functional performance and quality of life,[20] SCI-SET scores (third administration) were validated against: 1) self-assessment of spasticity severity, 2) self-assessment of spasticity impact, 3) the Penn Spasm Frequency Scale, 4) the FIM Motor subscale, and 5) the Quality of Life Index (QLI) SCI Version – III Health and Functioning subscale (satisfaction). Prior to the first administration of the SCI-SET, participants were asked to rate the overall severity of their spasticity on a 6-point scale (0 = no spasticity; 5 = extreme spasticity) and the overall impact of spasticity on their daily life on a 6-point scale (0 = no impact; 5 = extreme impact). The FIM Motor subscale, the QLI Health and Functioning subscale, and the Penn Spasm Frequency scale were administered in a random order immediately following the third administration of the SCI-SET.

The FIM is an 18-item subjective scale that assesses burden of care.[34] It has been suggested that some, although not all, of the FIM items are relevant for assessing the effects of spasticity intervention.[20] A self-assessment version of the FIM has been shown to be a reliable and valid measure of perceived functional independence in the SCI population.[35] Most likely to be relevant to spasticity, is the Motor subscale of the FIM (13 items assessing self-care, sphincter control, transfers, and locomotion).[34] Participants in the present study were asked to rate their levels of ability for the independent performance of the motor tasks during the previous 7 days on a 7-point Likert-type scale (1 = low ability; 7 = high ability). The individual item ratings were summed to produce a total score that could range from 13 to 91.

The QLI[36] SCI Version – III is a 35-item questionnaire designed to assess several components of quality of life relevant specifically to the SCI population.[37] The Health and Functioning subscale of the QLI was selected as most likely to be related to spasticity. This subscale has 15 items and has been shown to have adequate internal consistency ($\alpha > 0.80$).[36] Although the QLI was designed to assess importance-based weighted satisfaction, the weighting component of this scale was omitted for the present study because of the known limitations when scales use multiplicative composite scores.[33] Participants were asked to rate how satisfied they have been with each item over the past 7-days on a 6-point Likert-type scale (1 = very dissatisfied; 6 = very satisfied). Individual item ratings were summed to produce a total score that could range from 15 to 90.

The Penn Spasm Frequency Scale[38] is a measure of self-assessed spasm frequency commonly applied in studies involving the SCI population (e.g., [25]). Participants were asked to rate their spasms during the past 7 days on a 5-point scale (0 = no spasms; 4 = spasms occurring more than 10 times per hour). This scale has been used recently for examination of validity of a new clinical measure of spasms and clonus.[39]

Means and standard deviations (SD) and internal consistency (α) were calculated from the third administration of the SCI-SET. For the purposes of calculating α, non-applicable items were coded as "-4"; this was necessary, as a code of "N/A" would have falsely resulted in items having missing data. The intraclass correlation coefficient (ICC) was calculated as an index of test-retest reliability; two-way random effects models and an absolute agreement definition of reliability were used. The estimate for single-trial reliability was reported. Two-tailed, Pearson product-moment correlations (r) were computed between the average SCI-SET score (third administration) and self-assessed spasticity severity, self-assessed impact of spasticity, FIM Motor subscale, QLI SCI Version – III Health and Functioning subscale, and Penn Spasm Frequency Scale.

3.4.3.2 Results

Completion of the SCI-SET by 61 individuals with chronic SCI and 'stable' spasticity (n = 8 in-person; n = 53 by telephone) produced a range of scores from -2.35 to 0.00 (-0.65±0.56). From the sample, the group of individuals with paraplegia (n=24) had a score of -0.62±0.57 and the group of individuals with tetraplegia (n=37) had a mean score of -0.67±0.57. Males (n=45) and females (n=16) had mean scores of -0.60±0.55 and -0.80±0.57, respectively. Individual scores of 0.00 were due to tabulation of either all items rated as "0", or an equal positive and negative rating. Internal consistency (α = 0.90) and test-retest reliability (ICC = 0.91) were both adequate.

Although the correlation between the SCI-SET and the FIM Motor subscale was weak (r = 0.21; p =0.12), the SCI-SET demonstrated statistically significant ($p<0.001$) moderate to strong correlations with all other measures: 1) self-assessed spasticity severity (r = -0.48), 2) self-assessed spasticity impact (r = -0.61), 3) the QLI SCI Version III Health and Functioning subscale (r = 0.68), and 4) the Penn Spasm Frequency Scale (-0.66; Table 3.2).

Table 3.2 Pearson correlations between SCI-SET score and construct validity indices.

	SCI-SET	Spasticity Severity	Spasticity Impact	FIM – Motor	QLI – Health & Functioning	Penn Spasm Frequency
SCI-SET	1.00					
Spasticity Severity	-0.480*	1.00				
Spasticity Impact	-0.607*	0.566*	1.00			
FIM – Motor	0.206	-0.032	-0.146	1.00		
QLI – Health & Functioning	0.679*	-0.481*	-0.432*	0.469*	1.00	
Penn Spasm Frequency	-0.661*	0.575*	0.673*	-0.049	-0.464*	1.00

* $p<0.001$. SCI-SET, Spinal Cord Injury Spasticity Evaluation Tool; FIM – Motor, Functional Independence Measure Motor subscale; QLI – Health & Functioning, Quality of Life Index Spinal Cord Injury Version III – Health and Functioning subscale.

3.5 General Discussion

The overall purpose of these studies was to develop and conduct a preliminary psychometric evaluation of a new self-assessment measure of the impact of spasticity on daily life in individuals with chronic SCI. The resulting scale is a 35-item, 7-day recall questionnaire that targets aspects of daily life relevant to the SCI population and allows respondents to rate the impact of their spasticity from extremely problematic to extremely helpful. Our analyses provide support for the reliability and validity of the SCI-SET for this population.

As a self-report questionnaire, the SCI-SET addresses the need for a measure that considers the individual's perception of the impact of spasticity on his/her daily life.[11] Compared to many current measures that consider spasticity to be a physical impairment, the SCI-SET allows spasticity to be considered as it relates to activity and participation restrictions. Therefore, the SCI-SET offers a method to acknowledge the often-overlooked aspects of the International Classification of Functioning, Disability, and Health (ICF). The questionnaire was designed to be comprehensive, but easy to understand by respondents and potentially useful in both research and clinical settings. Self-ratings of spasticity at any given moment have been shown to correlate poorly with self-ratings of general spasticity[7] and spasticity presence can fluctuate hourly and with different days of the week (as was also noted in our interviews).[13,15] Therefore, a 7-day recall self-report format was chosen as a time period most likely to be representative of perceived impact of spasticity on daily life, despite hourly and daily fluctuations. The high internal consistency of the SCI-SET ($\alpha = 0.90$) was within the suggested range of 0.70[40] to 0.90,[33] supporting this combination of items as a cohesive measure. Participants also felt that the questionnaire was easy to complete. With repeated administration of the SCI-SET, the investigators developed an understanding of the importance, in some cases,

of reminding participants to consider the impact of spasticity (and not other SCI-related impairments) when responding to questionnaire items. Future users of the SCI-SET may want to implement a standardized statement to this effect.

The bidirectional response scale (-3 to +3) is new to spasticity assessment instruments. Numerous reports in the literature have emphasized the importance of considering both the problematic and the potentially beneficial effects of spasticity on aspects of daily life.[12,17-19] Some participants in the present study identified spasticity as being helpful (positive rating) on items such as "your ability to change positions in bed" (n = 5) and "your ability to stand/weight-bear" (n = 3). During the third administration of the SCI-SET (reliability assessment), 21 of the 35 items were given a positive rating by at least one study participant. The mean SCI-SET score was negative, however, indicating an overall problematic impact of spasticity on daily life in this population despite perceptions of some benefits. Items receiving the most ratings of "-3" (spasticity extremely problematic) were "your pain" (n = 7), "your hobbies/recreational activities" (n = 6), and "the quality of your sleep" (n = 6). Therefore, the SCI-SET should prove useful as an instrument to aid in decision-making regarding spasticity management and as a measure to assess the effectiveness of management strategies designed to minimize the detrimental impact of spasticity. In particular, the SCI-SET may help individuals with SCI to consider and communicate their thoughts about the impact of spasticity on their daily life, facilitating their involvement in spasticity-related management decisions.

With regard to test-retest reliability, the ICC for the SCI-SET (0.91) was above the recommended minimum reliability values of 0.70 and 0.90 for use of a scale in research and clinical settings, respectively.[40] This value is impressive given the variable nature of spasticity. Although the causes are not well-understood, spasticity severity and frequency are known to vary hourly and daily in some individuals.[13,15] Interviews during the first study revealed that even certain "uncontrollable" factors, such as the weather, can have an influence on spasticity. Because published self-reports of spasticity are lacking and because test-retest reliability has not been assessed for many clinical scales,[41] there are no related, accepted scales to consider as a basis of comparison with our findings. Nevertheless, it is clear that the ICC has good reliability.

Investigators have typically considered validity coefficients between 0.30 to 0.50 to be satisfactory.[42] Therefore, the validity of the SCI-SET as a self-report measure of the impact of spasticity on daily life in people with SCI was supported by the moderate to strong and statistically significant correlations between average SCI-SET scores and: 1) self-ratings of spasticity severity, 2) self-ratings of spasticity impact, 3) the QLI SCI Version III Health and Functioning subscale, and 4) the Penn Spasm Frequency Scale. Because the SCI-SET was designed to assess the *impact* of spasticity on daily life, it is particularly noteworthy that the correlation between the SCI-SET and self-ratings of spasticity impact (r = -0.61) was larger than the correlation between the SCI-SET and self-ratings of spasticity severity (r = -0.48). The negative correlations suggest that individuals who reported the most problematic spasticity (i.e., the most negative scores), also tended to report greater severity, impact, and frequency (Penn Spasm Frequency Scale; r = -0.66) of spasticity. Conversely, the positive correlation between the SCI-SET scores and QLI SCI Version III Health and Functioning subscale scores (r = 0.68)

suggests that individuals who reported the highest quality of life were those who experienced the smallest impact of spasticity (i.e., scores near or at "0" on the SCI-SET). These results are encouraging when compared to previous studies assessing correlations between measures of spasticity in the SCI population. In particular, self-assessed spasm frequency (single item; 5 possible responses) by 85 individuals with SCI had a correlation of 0.41 with self-assessed rating of interference of spasticity with function (single item; three possible responses).[25] In this same group, correlations between the Ashworth score and various other clinical measures of spasticity ranged from 0.20 to 0.55.[25] In a more recent study of 47 individuals with SCI, the Ashworth score had correlations of 0.36 and 0.70 with self-ratings of spasticity during the Ashworth test (4-point scale) and with self-rating of spasticity in general (4-point scale), respectively.[7]

The SCI-SET had a poor correlation with the FIM Motor subscale ($r = 0.21$). Although it might be suggested that the impact of spasticity on daily life should be reflected in a measure of functional performance such as the FIM,[20] this result is not surprising in hindsight. Inspection of the individual FIM Motor subscale items identifies items that might be affected by spasticity; however, these items are also likely to be affected by various other deficits or complications resulting from SCI. For example, differences in impairment ratings (ASIA score), neurological levels, and other characteristics likely mask relationships between spasticity and functional independence. In particular, partial or complete paralysis of skeletal muscle due to SCI is likely to have a much greater effect on function than spasticity in most individuals. Therefore, unless there are also changes in muscle strength or function, a change in spasticity severity or frequency may not be reflected by FIM scores. A recent study observing reductions in spasticity measured clinically and through self-assessment did not find an improvement in ADL, as assessed by the FIM.[29] In response to reports of poor correlations between observed reductions in spasticity and improvements in function, Pierson[13, p.S41] has questioned: "Does change in spasticity not affect function because no effect actually occurs, because the patient was inappropriately selected for spasticity reduction, or because the functional measures used are insensitive or unreliable?" Our findings of poor correlations between the SCI-SET and the FIM Motor subscale suggest that general measures of function may be insensitive as indicators of change in spasticity. It is anticipated that, as a valid and reliable measure of the impact of spasticity on daily life, the SCI-SET will provide a more appropriate indication of change following spasticity intervention in individuals with SCI.

There are three possible limitations of our series of studies that deserve mention. First, as with any study using a convenience sample, there is the potential to misrepresent the population of interest. We are confident, however, that our samples were representative of the general SCI population; a range of education levels and a variety of employment statuses were included and the proportions of injury levels, injury severities, and gender were similar to that of the general SCI population (see Table 3.1).[43] Second, the selection of a 7-day recall period for the SCI-SET may lead to an inability to accurately capture short-term changes in spasticity (if any) that might occur with an intervention. So long as any short-term benefits are perceived by individual, however, the 7-day recall period of the SCI-SET should allow for the individual to report the perceived

change. Furthermore, because spasticity has been shown to vary depending on the day of the week, there are two additional benefits of a 7-day recall period: 1) Arguably, inclusion of each day of the week is most appropriate to obtain an accurate assessment of the effects of spasticity on overall daily life. 2) A questionnaire with a shorter recall period (e.g., 3 days) would only be useful for measurement of changes over time if administered on the same days of the week with successive administrations. Of course, any questionnaire designed with a 7-day recall may not be appropriate for use in studies designed to assess only the acute effects of an intervention. Lastly, it is possible that individual participants in the present study applied different assessments when considering the impact of spasticity during the previous 7 days. If questioned, the interviewer instructed the participant to consider the "overall" impact. The high test-retest reliability of the SCI-SET suggests that individuals were consistent in their assessment methods over time. Although not included within the SCI-SET instructions, future users may wish to clarify: "For each of the following, please choose the answer that best describes how your spasticity symptoms have affected that area of your life [overall *or* on average] during the past 7 days."

3.6 CONCLUSIONS

The importance of the inclusion of self-assessment of spasticity by individuals with SCI has been recognized, but no appropriate measure has been available. The SCI-SET fills a need for a reliable and valid self-report measure of the impact of spasticity on daily life in people with SCI, taking into account both the problematic and useful effects of spasticity. As an adjunct to conventional spasticity assessment, the SCI-SET could be used in clinical and research settings and, in particular, as a tool to facilitate the role of the individual with SCI as an active contributor to his/her medical management decisions. As scale validation is an ongoing process and cannot be "proven" by any single study,[33] we recommend continued validation studies of the SCI-SET using broader samples and other validation techniques. In particular, it would be valuable to examine the responsiveness to change of the SCI-SET during an intervention known to have a significant impact on spasticity.

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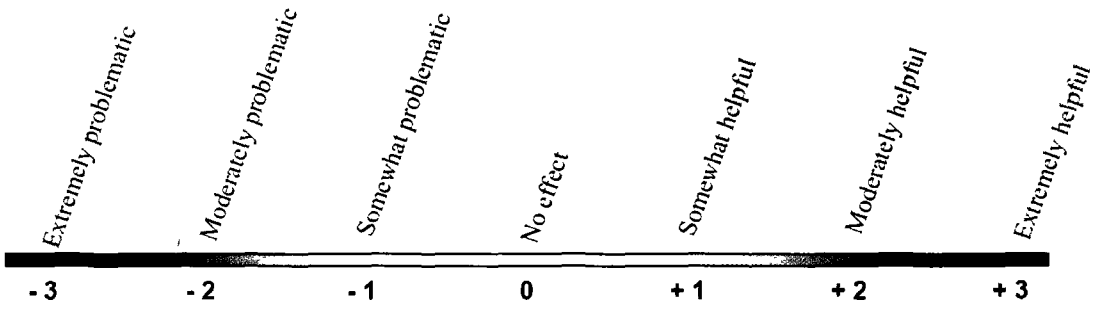
3.8 APPENDIX A: THE SPINAL CORD INJURY SPASTICITY EVALUATION TOOL

Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET)

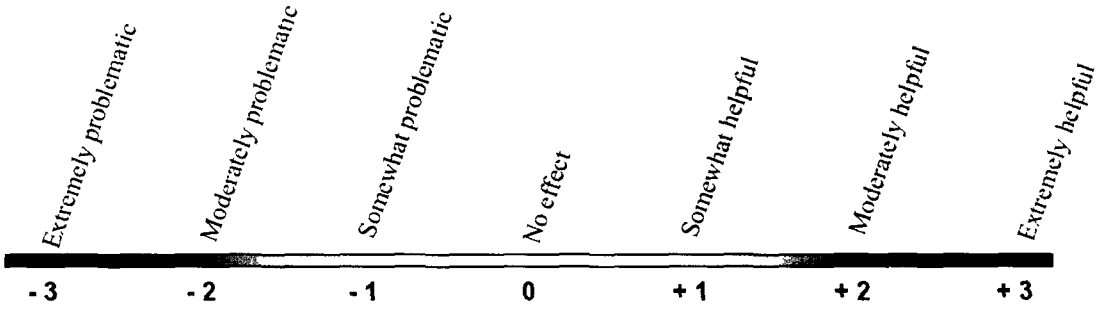
For each of the following, please choose the answer that best describes how your spasticity symptoms have affected that area of your life **during the past 7 days**. When I talk about "spasticity symptoms", I mean: a) uncontrolled, involuntary muscle contraction or movement (slow or rapid; short or prolonged), b) involuntary, repetitive, quick muscle movement (up and down; side to side), c) muscle tightness, and d) what you might describe as "spasms". Please let me know when a question is not applicable to you.



DURING THE **PAST 7 DAYS**, HOW HAVE YOUR SPASTICITY SYMPTOMS AFFECTED:

1. your showering?	-3	-2	-1	0	+1	+2	+3	N/A
2. your dressing/undressing?	-3	-2	-1	0	+1	+2	+3	N/A
3. your transfers (to and from bed, chair, vehicle, etc.)?	-3	-2	-1	0	+1	+2	+3	N/A
4. your sitting positioning (in your chair, etc.)?	-3	-2	-1	0	+1	+2	+3	N/A
5. the preparation of meals?	-3	-2	-1	0	+1	+2	+3	N/A
6. eating?	-3	-2	-1	0	+1	+2	+3	N/A
7. drinking?	-3	-2	-1	0	+1	+2	+3	N/A
8. your small hand movements (writing, use of computer, etc.)?	-3	-2	-1	0	+1	+2	+3	N/A
9. your ability to perform household chores?	-3	-2	-1	0	+1	+2	+3	N/A
10. your hobbies/recreational activities?	-3	-2	-1	0	+1	+2	+3	N/A
11. your enjoyment of social outings?	-3	-2	-1	0	+1	+2	+3	N/A
12. your ability to stand/weight-bear?	-3	-2	-1	0	+1	+2	+3	N/A
13. your walking ability?	-3	-2	-1	0	+1	+2	+3	N/A
14. your stability/balance?	-3	-2	-1	0	+1	+2	+3	N/A
15. your muscle fatigue?	-3	-2	-1	0	+1	+2	+3	N/A
16. the flexibility of your joints?	-3	-2	-1	0	+1	+2	+3	N/A
17. your therapy/exercise routine?	-3	-2	-1	0	+1	+2	+3	N/A
18. your manual wheelchair use?	-3	-2	-1	0	+1	+2	+3	N/A

Page 1 of 2



DURING THE **PAST 7 DAYS**, HOW HAVE YOUR SPASTICITY SYMPTOMS AFFECTED:

19. your power wheelchair use?	-3	-2	-1	0	+1	+2	+3	N/A
20. your lying positioning (in bed, etc.)?	-3	-2	-1	0	+1	+2	+3	N/A
21. your ability to change positions in bed?	-3	-2	-1	0	+1	+2	+3	N/A
22. your ability to get to sleep?	-3	-2	-1	0	+1	+2	+3	N/A
23. the quality of your sleep?	-3	-2	-1	0	+1	+2	+3	N/A
24 your sex life?	-3	-2	-1	0	+1	+2	+3	N/A
25. the feeling of being annoyed?	-3	-2	-1	0	+1	+2	+3	N/A
26. the feeling of being embarrassed?	-3	-2	-1	0	+1	+2	+3	N/A
27. your feeling of comfort socially?	-3	-2	-1	0	+1	+2	+3	N/A
28. your feeling of comfort physically?	-3	-2	-1	0	+1	+2	+3	N/A
29. your pain?	-3	-2	-1	0	+1	+2	+3	N/A
30. your concern with falling?	-3	-2	-1	0	+1	+2	+3	N/A
31. your concern with getting injured?	-3	-2	-1	0	+1	+2	+3	N/A
32. your concern with accidentally injuring someone else?	-3	-2	-1	0	+1	+2	+3	N/A
33. your ability to concentrate?	-3	-2	-1	0	+1	+2	+3	N/A
34. your feelings of control over your body?	-3	-2	-1	0	+1	+2	+3	N/A
35. your need to ask for help?	-3	-2	-1	0	+1	+2	+3	N/A

Number of (+) items: _____ Negative score: _____

Number of (-) items: _____ Positive score: _____

Number of (0) items: _____ **Total score:** _____

Applicable items (#): _____

Average score: _____

CHAPTER 4

SPASTICITY IN INDIVIDUALS WITH CHRONIC SPINAL CORD INJURY. PART 1: EFFECTS OF SINGLE SESSIONS OF BODY-WEIGHT SUPPORTED TREADMILL TRAINING AND TILT-TABLE STANDING.

Submitted to: Archives of Physical Medicine and Rehabilitation (October 2007)

Authors: Melanie M. Adams and Audrey L. Hicks

4.0 SPASTICITY IN INDIVIDUALS WITH CHRONIC SPINAL CORD INJURY. PART 1: EFFECTS OF SINGLE SESSIONS OF BODY-WEIGHT SUPPORTED TREADMILL TRAINING AND TILT-TABLE STANDING

4.1 ABSTRACT

Study Design: Random cross-over
Objective: To compare the effects of single sessions of body-weight supported treadmill training (BWSTT) and tilt-table standing (TTS) on clinically-assessed spasticity and motor neuron excitability in individuals with chronic spinal cord injury (SCI).
Setting: MacWheelers Exercise Program, a community exercise program for people with SCI in Hamilton, Ontario, Canada.
Method: Seven community-dwelling individuals with chronic SCI and self-reported spasticity performed single sessions of BWSTT and TTS and were assessed for changes in symptoms of spasticity: Modified Ashworth Scale, Spinal Cord Assessment Tool for Spinal reflexes, and soleus maximum H-reflex to maximum M-wave ratio (H/M ratio).
Results: A single session of TTS appeared to reduce extensor spasms (ES=0.68). There were indications that BWSTT had greater beneficial effects compared to TTS for muscle tone (ES=0.69), flexor spasms (ES=0.57), and the H/M ratio (motor neuron excitability; ES=0.50).
Conclusions: These results suggest that individuals with SCI and spasticity may benefit from a single session of weight-bearing activity and that BWSTT may offer superior benefits to TTS for some aspects of spasticity.

4.2 INTRODUCTION

A spinal cord injury (SCI) is most commonly sustained by individuals who are relatively young in age.[1] With progress in medical management, the likelihood of surviving the critical 2-year period post-SCI is improving.[2] Therefore, it is becoming increasingly important to focus on the long-term effective management of the consequences of SCI that can impact quality of life. Spasticity has recently been defined as: "disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary contraction of muscles".[3] Therefore, the term 'spasticity' is no longer limited to only 'muscle hypertonicity' (as was initially suggested by Lance[4]) but, rather, is acknowledged to include numerous positive features of the upper motor neuron syndrome (e.g., hypertonicity, spasms, and clonus). It has been reported that 65% to 78% of sample populations of individuals with chronic SCI have spasticity.[5,6] Spasticity may be considered beneficial by individuals with SCI for some aspects of daily life, such as sitting or standing stability and the performance of transfers and some activities of daily living.[6,7] There are also, however, numerous potentially problematic effects of spasticity, including inhibition of effective walking and self-care, pain, fatigue, disturbed sleep, compromised safety, and negative self-image.[6,7] As a result, many individuals with SCI seek strategies to manage their spasticity.

There are various possible approaches to the management of spasticity following SCI.[7] It is generally agreed that physical therapy/rehabilitation is an essential component of the management of spasticity as a first line of defence, as well as in a long-term regimen during and after the implementation of pharmacological or surgical strategies.[8-10] There is evidence suggesting that single sessions of such active or passive "activity-based strategies" have immediate effects on spasticity in individuals with SCI.[11,12] A case report has provided evidence that passive standing assisted by a tilt-table can reduce lower limb spasticity.[13] The combination of weight-bearing and stretching has been found more effective than either weight-bearing or stretching alone, with effects lasting up to 4 hrs in some cases.[14] Motorized repetitive passive movement[15] and activity-based therapies assisted by functional electrical stimulation (FES) have also been shown to reduce spasticity immediately following the activity.[16-18] It is important to recognize, however, that there have also been reports of increased spasticity by some study participants immediately following activity-based therapy.[16]

The mechanisms explaining the spasticity-reducing effects of activity-based management strategies remain poorly understood; suggestions have included mechanical changes at the muscle, tendon, and soft-tissue level[19] and plastic changes within the central nervous system (e.g., reduction of motor unit activity/excitability[13,20]). There is evidence that motor neuron excitability plays a role in spasticity following SCI[21] and it has been shown to be modified following single[22] and multiple[23] sessions of activity-based intervention.

Body-weight supported treadmill training (BWSTT) is an activity during which individuals with impaired walking ability can undergo independent or assisted ambulation. During BWSTT, the lower limb musculature of an individual with motor-complete or -incomplete SCI bears weight, undergoes stretch, and exhibits

electromyographic activity.[24-26] A reduction in the amplitude of the soleus maximum H-reflex / maximum M-wave ratio (H/M ratio; an indication of motor neuron excitability) assessed during the stance and swing phases of gait has been noted after a single bout of BWSTT in a group of 4 individuals with American Spinal Injury Association (ASIA) D SCI.[27] Therefore, BWSTT may offer a unique combination of the characteristics of more traditional activity-based therapies that have been shown to reduce spasticity in individuals with SCI. For example, tilt-table standing (TTS) allows individuals who have deficits in independent standing ability to undergo prolonged passive "standing". Compared to a seated position, TTS places load on the lower limbs and imposes stretch on the skeletal muscles that cross the hip, knee, and ankle joints. Although these two therapies may provide some similar stimuli relevant to spasticity management, increased skeletal muscle activity has only been reported during BWSTT.[24-26] To our knowledge, however, there are no studies examining the immediate effects of BWSTT and TTS on multiple measures of spasticity in individuals with SCI who are wheelchair-dependent. The differences in financial and personnel costs of these two activities could play an important role in their use as activity-based interventions. Therefore, the purpose of this study was to compare the effects of single sessions of BWSTT and TTS on clinically-assessed spasticity and motor neuron excitability in individuals with chronic SCI and self-reported spasticity. It was hypothesized that, although both BWSTT and TTS would have beneficial effects on spasticity-related outcomes, BWSTT would be more beneficial than TTS overall. A companion paper[28] reports on the effects of multiple sessions of BWSTT and TTS on these and other spasticity-related outcome measures.

4.3 METHODS

4.3.1 Participants

Participants for this study were recruited from the Hamilton Health Sciences outpatient SCI rehabilitation program. Inclusion criteria included chronic (> 1 year) complete or incomplete paraplegia or tetraplegia, self-reported "stable" spasticity, use of a wheelchair as a primary mode of mobility, and consistent medication and physical activity/physiotherapy routines during the previous 6 mos. Exclusion criteria included participation in BWSTT during the previous 6 mos and/or any medical contraindications to performance of BWSTT or TTS. This study received ethics approval from the Hamilton Health Sciences Research Ethics Board and all participants provided informed consent prior to beginning the study.

4.3.2 Study Design and Overall Protocol

Each participant performed both 30 min of BWSTT and 30 min of TTS in random order, 8 wks apart. Prior to their first session, each participant had an introductory session: participants were weighed and were familiarized with the BWSTT, TTS, and testing protocols and were randomized with regard to first activity type.

Immediately prior to the BWSTT and TTS sessions, baseline testing occurred in the following sequence: 1) Participants entered a temperature-controlled room, underwent testing of femoral artery blood flow in both legs, and were asked to complete self-report questionnaires related to spasticity (results not reported here). 2) Participants wheeled to a second testing room for assessment of: a) H/M ratio, b) muscle tone, c) flexor spasms, d) extensor spasms, and e) clonus, in both legs. The same measures were carried out immediately following each activity session.

Baseline and post-activity testing was performed by the same examiner and in the same order within activity conditions. Participants emptied their bladders prior to arrival to the laboratory and were asked to refrain from caffeine, nicotine, marijuana, and physical activity 20 hrs before baseline testing. Participants were at least 90 mins postprandial at the start of data collection.

4.3.3 Activity Conditions

4.3.3.1 Body-Weight Supported Treadmill Training (BWSTT)

Body-weight supported treadmill training was performed using a motor-driven treadmill (Woodway USA Inc., Foster, CT) with a harness and overhead pulley system capable of supporting a percentage of the participant's body weight. Body-weight support (BWS) and speed of the treadmill were chosen to enable appropriate gait with full knee extension during stance. When necessary, lower limb movement was assisted by a therapist positioned at each side of the participant. Assistance was particularly important in extension of the knee and hip joints during the stance phase of the gait cycle. Participants were encouraged to place their entire body weight over a fully extended leg during the stance phase of the walking cycle and to swing their arms and/or use the parallel supports for balance only. Decisions regarding the training parameters were made during the introductory session according to our protocol, outlined previously;[29] briefly, the lowest BWS and the highest comfortable treadmill speed were used that allowed the individual to ambulate without buckling at the knees.

4.3.3.2 Tilt-Table Standing (TTS)

Tilt-table standing was performed on a motorized tilt-table (Midland Manufacturing Co., Inc., Columbia, SC) at the greatest tolerated tilt angle (up to a table maximum of 80^{o}). Velcro straps at the knees, hips, and torso (if necessary) were used to support the individual in an upright position. Participants were allowed to increase gradually to their maximum tilt angle and were allowed to reduce their tilt angle during the session if they felt light-headed.

4.3.4 Outcome Measures

The multi-dimensional nature of spasticity has lead to the suggestion that no single outcome measure should be used when conducting assessments of spasticity;

rather, a range of different approaches should be included.[3,11,30-32] Therefore, outcome measures for the present study were chosen to represent clinical assessments of tone, spasms, and clonus. Measures of the soleus H/M ratio were also performed, as elevated motor neuron excitability has been implicated as a mechanism to explain spasticity.[21]

4.3.4.1 Modified Ashworth Scale (MAS): Muscle Tone

Despite its acknowledged limitations,[33] the Modified Ashworth Scale (MAS) is considered to be among the most commonly used spasticity assessment instruments.[34] Participants were tested in a supine position after having transferred to a flat surface (plinth). Muscle groups assessed for passive resistance to tone were left and right hip flexors, extensors, and adductors, knee flexors and extensors, and ankle plantarflexors and dorsiflexors. A metronome was used to standardize the speed of limb motion at 1 sec per direction. Each joint was moved through its range of motion three times, with tone assessed during the third range of motion performance. The scale was altered from a scale ranging from 0 to 4 (with the "1+") to a scale ranging from 0 to 5, as has been done previously.[35] The single scores from the muscle groups of both lower limbs were then summed for each participant, providing an overall MAS score.[36]

4.3.4.2 Spinal Cord Assessment Tool for Spastic reflexes (SCATS)

The Spinal Cord Assessment Tool for Spastic reflexes (SCATS) is a valid clinical measure designed to assess spasm and clonus severity in individuals with SCI; a score from 0-3 is given for each of flexor spasms, extensor spasms, and clonus following a standardized perturbation.[37] Participants were tested in a supine position immediately following MAS testing. Left and right lower limb scores were summed for each of the assessed spasticity symptoms.

4.3.4.3 *Soleus H/M Ratio: Motor Neuron Excitability*

The H-reflex reflects the motor neurons that can be excited by antidromic stimulation mediated through the Ia fibres.[38] A greater H-reflex, therefore, is considered to be representative of greater motor neuron excitability.[39] Normalization of the maximum H-reflex to the maximum M-wave (total pool of motor neurons that can be excited by stimulation[38]) reduces the effect of possible measurement variability, making comparisons between subjects and between groups justifiable.[39] To elicit the soleus H-reflex and M-wave, a single rectangular biphasic pulse, with a pulse width of 500μs, was applied to the tibial nerve in the popliteal fossa using a constant-voltage stimulator (Digitimer Devices, Hertfordshire, UK). The interelectrode distance was 3 cm.[40] The Ag-AgCl gel, self-adhesive recording electrodes (Kendall Meditrace 530, Chicopee, MA) were placed in a bi-polar arrangement; one electrode was secured over the right soleus muscle and the other over the right Achilles tendon. Prior to electrode application, the leg was shaved and the skin was abraded and cleaned with alcohol. The recording electrodes remained affixed during the activity session, ensuring identical placement for baseline

and post-activity testing. During data collection, participants remained seated in their chairs, with knee and ankle angles between 110°-130° and 90°-110°, respectively. Participants were asked to maintain consistent head and torso positions and to avoid clenching their teeth during testing.[39] The sampling frequency of the electromyographic activity was 4 KHz and the data were bandpass filtered at 1 Hz to 1 KHz. The data were collected using customized LabVIEW software (National Instruments, Austin, TX). The stimulus frequency was less than 0.1 Hz to prevent depression of the H-reflex.[41] Stimulus intensity was increased in steps of ~5 mV until Hmax and Mmax were approached and then increased in steps of ~1 mV until peak values were obtained. The peak-to-peak amplitudes of the H-reflex and M-wave were determined by automatic calculation following manual identification of the time window containing the wave of interest. The three greatest measurements of Hmax and the three greatest measurements of Mmax were averaged.[41] The H/M ratio was calculated by dividing the average Hmax by the average Mmax. An average of the right and left soleus H/M ratios for each participant was used for all analyses.

4.3.5 Data Treatment and Statistical Analyses

The amount of BWSTT and TTS training time were compared using a paired samples t-test. To check for possible carryover effects of activity, independent samples t-tests were performed for each outcome measure, with Bonferroni correction for multiple comparisons: the group of individuals who performed BWSTT as their first activity (n=4) was compared to the group of individuals who performed TTS as their first activity (n=3). In studies with small sample sizes, it can be difficult to identify statistically significant effects using traditional ANOVA methods. Furthermore, a p-value does not provide an indication of the size of the effect and may falsely suggest that an intervention is ineffective. Therefore, Cohen's *d* effect sizes (ES) were calculated to compare baseline scores to post-activity scores within condition and to compare change scores between activity conditions for each outcome measure; relevant medium (~0.50+) or large (~0.80+) effect sizes are discussed.[42] Data are presented as mean±SD.

4.4 RESULTS

4.4.1 Participants

Six males and one female (age: 37.1±7.7 yrs) with chronic (5.0±4.4 yrs post-injury) complete or incomplete paraplegia or tetraplegia and self-reported spasticity participated in this study (Table 4.1). Current medication and physical activity/physiotherapy routines had been consistent for at least six months prior to the start of the study. The four participants with ASIA C SCI were capable of some independent ambulation. Three participants (#2, 3, and 4) were not involved in any formal or regular lower body exercise or therapy. Three participants (#5, 6, and 7) were involved in outpatient physiotherapy prior to the study. One participant (#1) used

ambulation with forearm crutches for some walking within his home and occasional standing with assistance.

4.4.2 Activity Conditions

Only one participant (#4) could not tolerate 30 min of BWSTT or TTS; 20 min of TTS and 25 min of BWSTT were performed by this individual (Table 4.2). Group average duration of BWSTT and TTS did not differ, at 29.3±1.9 min and 28.6±3.8 min, respectively ($p=0.36$). BWSTT training parameters included a group mean treadmill speed of 0.21±0.09 m/s and a group mean provision of 51.1±24.3% BWS. Tilt-table standing was performed at a group mean table angle of 62.8±10.0°.

Table 4.1 Participant characteristics

Participant ID	Sex	Age (yrs)	Severity	Level	Post-injury (yrs)	Cause of SCI	Daily Spasticity Medications
1	M	40	C	T2	2	Fall	None
2	M	37	A	C6	14	MVA	40mg Baclofen
3	M	32	B	T5	7	MVA	80mg Baclofen Marijuana (2-3mg/day)
4	F	24	C	C5	5	MVA	None
5	M	39	A	T5	3	MVA	None
6	M	49	C	T10	3	Bleed	60mg Baclofen
7	M	39	C	T2	1	MVA	80mg Baclofen 5mg Tizanidine

Table 4.2 Body-weight supported treadmill training (BWSTT) and tilt-table standing (TTS) durations and parameters.

ID	BWSTT			TTS	
	Duration (min)	Avg. Speed (m/s)	Avg. BWS (%)	Duration (min)	Avg. Tilt Angle (°)
1	30	0.28	8.0	30	70.0
2	30	0.28	50.3	30	63.3
3	30	0.33	32.0	30	56.7
4	25	0.07	40.0	20	43.0
5	30	0.19	72.0	30	70.0
6	30	0.17	24.0	30	66.7
7	30	0.14	40.0	30	70.0

4.4.3 Spasticity: Tone, Flexor Spasms, Extensor Spasms, and Clonus

The mean scores for all outcome measures are presented in Table 4.3. There was no evidence of carryover effects, nor any statistically significant differences between baseline scores within outcome measures for either condition. There was a tendency for extensor spasms to decrease following TTS (ES=0.68; Figure 4.1), but not following BWSTT. A comparison of the change scores suggested that there was a greater reduction in muscle tone after BWSTT compared to TTS (ES=0.69; Figure 4.2). It also appeared that BWSTT had a tendency to reduce flexor spasms compared to the tendency of TTS to increase flexor spasms (ES=0.57; Figure 4.2).

Table 4.3 Summary of scores (mean±SD) for all outcome measures at baseline and post-activity for the BWSTT and TTS conditions.

Outcome Measures	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
Ashworth	29.6±16.0	25.6±13.0	29.4±14.4	29.1±13.5
Flexor Spasms	2.9±1.2	2.7±1.1	2.4±1.6	3.0±1.5
Extensor Spasms	3.7±1.5	3.3±1.6	4.6±1.1	3.7±1.4
Clonus	2.6±2.5	2.4±2.3	1.9±1.7	1.7±1.5
H/M Ratio	0.61±0.25	0.60±0.22	0.64±0.26	0.66±0.24

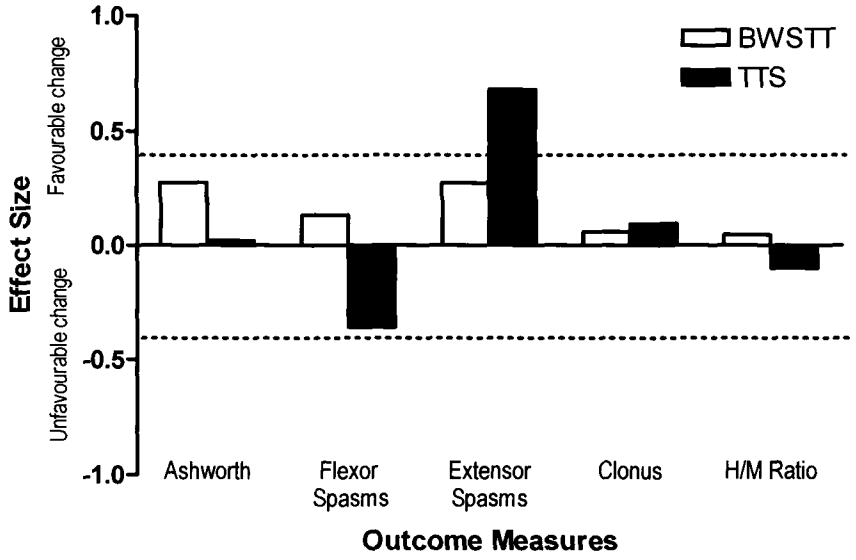


Figure 4.1 Effect sizes of the differences between baseline and post-activity outcome measure scores for BWSTT (open bars) and TTS (solid bars) conditions. Positive and negative effect sizes represent favourable and unfavourable changes, respectively, in the outcome measures. Absolute effect sizes greater than 0.45 (dashed line) are discussed in the text.

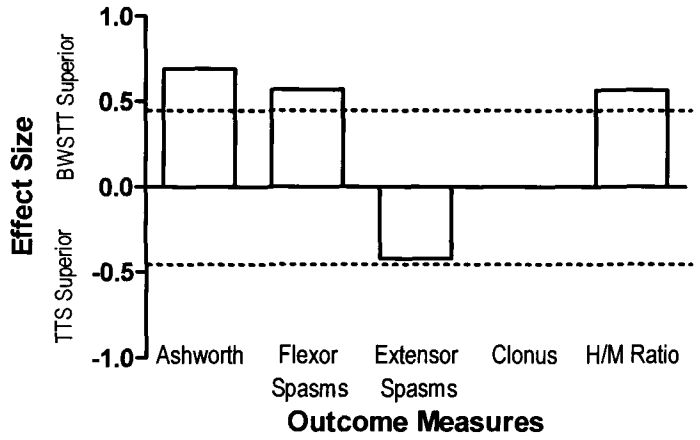


Figure 4.2 Effect sizes of the differences between outcome measure change scores following a single session of BWSTT and a single session of TTS. Positive effect sizes represent a superiority of BWSTT whereas negative effect sizes represent a superiority of TTS in terms of the induction of a favourable change. Absolute effect sizes greater than 0.45 (dashed line) are discussed in the text.

Soleus H/M Ratio: Motor Neuron Excitability

There was a suggestion that the change in H/M ratio following activity was different between the two conditions (ES=0.50; Figure 4.2). This difference seemed to be the result of tendencies of the H/M ratio to change in opposite directions following BWSTT and TTS (Figure 4.1).

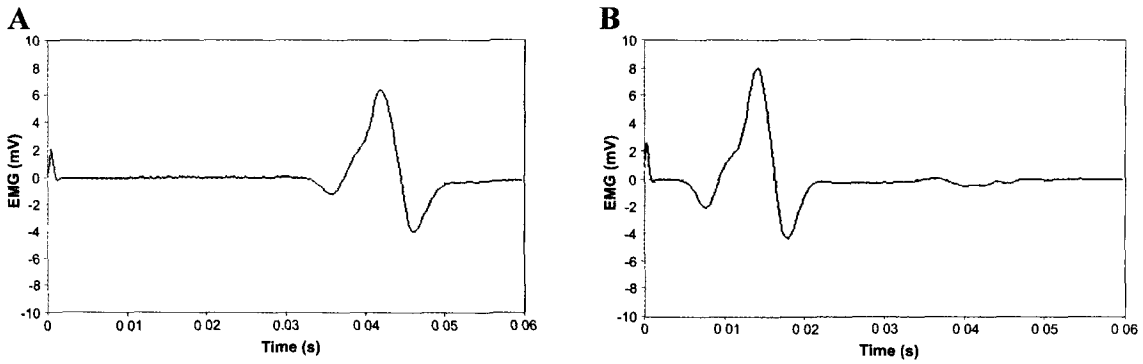


Figure 4.3 Representative maximum H-reflex (A) and maximum M-wave (B) tracings from one participant.

4.5 DISCUSSION

To our knowledge, this is the first study comparing the immediate effects of BWSTT and TTS on various measures of spasticity in individuals with chronic ASIA A to C SCI. A single session of TTS had an effect on reducing extensor spasms. There were also indications of differences between the effects of BWSTT and TTS on spasticity-related outcome measures (Figures 4.1 & 4.2). Overall, a single session of weight-bearing activity may be of benefit to some individuals with SCI as a strategy to modify spasticity symptoms; it should not be assumed, however, that all individuals with SCI will respond similarly to BWSTT or TTS. A greater understanding of the effects of single sessions of activity may assist individuals with SCI in the selection and proper application of the various spasticity management strategies.

During BWSTT, an average of 51.1±24.3% of BWS was provided. Our participants performed TTS at a mean tilt angle of 62.8±10.0°. In a previous study examining the effects of TTS at 85°, the load on the soles of the feet was estimated to be ~70% of the body weight (i.e., ~30% BWS).[14] Therefore, it is reasonable to deduce that, on average, more than 30% of BWS was provided during TTS in our study. The group mean activity durations were similar between conditions. Therefore, the main difference in training stimuli provided by the two conditions was likely the lower limb movement and resulting muscle activity that has been shown to occur during BWSTT.[24-26]

Overall, the results from this study are consistent with the general findings in the literature for the effects of single sessions of activity-based therapy on lower limb spasticity in humans with SCI[11] (i.e., ambulation training,[27] standing and/or stretch,[13,14] passive movements,[15,43] and FES-assisted cycling[18] and walking[16]). These include reports of beneficial effects, lack of effect, and even detrimental effects of activity, with many of these studies reporting different effects depending on the outcome measure or the study participant.[13,14-16,18,22,43]

Participation in a single session of TTS by the individuals in the present study had an immediate effect on the reduction of extensor spasms. This finding is in agreement with a previous clinical observation that extensor spasms, in particular, tend to be reduced following TTS.[13] The present study is novel in its demonstration of possible differing effects of BWSTT and TTS on various measures of spasticity (through the comparison of change scores; Figure 4.2). There are indications that BWSTT may be superior to TTS for several spasticity-related outcomes (reduction of muscle tone, flexor spasms, and motor neuron excitability). In some cases, these observed differences were due to outcome changes in opposite directions by the two activity conditions (Table 4.3 & Figure 4.1). Given the numerous previous reports of poor correlations between different measures of spasticity, our finding that the various outcome measures did not respond similarly to activity is not surprising.[21,30,36,37]

Mechanisms to explain the effects of activity, whether beneficial or detrimental, remain elusive. It has been suggested, however, that decreases in spasticity observed immediately following the application of electrical stimulation may be due muscle fatigue.[44] We did not assess fatigue in our participants, but anecdotal reports, particularly by those with some preserved motor function, suggested that performance of BWSTT

was typically perceived as being more demanding than TTS. We examined the soleus H/M ratio to determine if either activity had an effect on motor neuron excitability and, although this ratio tended to decrease to a greater extent following BWSTT compared to TTS, neither activity appeared to have an independent effect. Therefore, while motor neuron excitability may play a role in spasticity after SCI, it is unlikely to be the sole mechanism explaining its presence and adaptation.

Although several interesting findings related to the effects of BWSTT and TTS on spasticity were identified by the present study, the small and heterogeneous sample would have hindered our ability to detect statistically significant changes over time or between conditions using traditional ANOVA methods. More specific recommendations may stem from larger studies that are designed to examine mediating characteristics of responses to activity, as it is likely that certain outcome measures and individuals respond more favourably to certain activity types. Sample size calculations based on the data from the current study suggest that, for any outcome with a medium or large effect size, inclusion of 27 (MAS) to 49 (H/M ratio) participants in future similar studies would reveal statistically significant effects if assessed using ANOVAs (power = 0.80).[45]

4.6 Conclusions

There are indications that participation in weight-bearing activity by individuals with SCI and spasticity may offer benefits, with a tendency for BWSTT to offer superior benefits to TTS for a number of outcomes. Continued investigation of activity-based therapies for the management of spasticity after SCI is required; in particular, future studies are needed to further elucidate the types of activities that are most likely to provide benefits to the various sub-populations of individuals with SCI.

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CHAPTER 5

SPASTICITY IN INDIVIDUALS WITH CHRONIC SPINAL CORD INJURY. PART 2: EFFECTS OF MULTIPLE SESSIONS OF BODY-WEIGHT SUPPORTED TREADMILL TRAINING AND TILT-TABLE STANDING.

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5.0 SPASTICITY IN INDIVIDUALS WITH CHRONIC SPINAL CORD INJURY. PART 2: EFFECTS OF MULTIPLE SESSIONS OF BODY-WEIGHT SUPPORTED TREADMILL TRAINING AND TILT-TABLE STANDING

5.1 ABSTRACT

Study Design: Random cross-over.
Objective: To compare the effects of multiple sessions of body-weight supported treadmill training (BWSTT) and tilt-table standing (TTS) on clinically-assessed and self-reported spasticity, motor neuron excitability, and related constructs in individuals with chronic spinal cord injury.
Setting: MacWheelers Exercise Program, a community exercise program for people with spinal cord injury (SCI) in Hamilton, Ontario, Canada.
Method: Seven individuals with chronic SCI performed thrice-weekly BWSTT for 4 wks and thrice-weekly TTS for 4 wks and were assessed for changes in symptoms of spasticity 24-48 hrs following the twelfth session of activity: 1) Spasticity assessed clinically (Modified Ashworth Scale, Spinal Cord Assessment Tool for Spinal reflexes) and through self-report (Spinal Cord Injury Spasticity Evaluation Tool, Penn Spasm Frequency Scale); 2) Self-reported constructs related to spasticity: quality of life (Quality of Life Index Spinal Cord Injury Version – III) and functional mobility (FIM Motor Subscale); and 3) soleus maximum H-reflex to maximum M-wave ratio.
Results: Flexor spasms (ES=0.79) clonus (ES=0.66), and self-reported mobility (ES=1.27) tended to benefit more from 4 wks of BWSTT than of TTS, whereas extensor spasms had greater reductions following TTS (ES=1.32). Participation in BWSTT appeared to have a favourable effect on quality of life (ES=0.50).
Conclusions: Both BWSTT and TTS appear to provide specific benefits with respect to spasticity outcomes. These results suggest that individuals with SCI and spasticity may benefit from multiple sessions of weight-bearing activity for optimal spasticity management.

5.2 INTRODUCTION

Individuals with a spinal cord injury (SCI) are at risk of developing numerous complications that can have detrimental effects on quality of life.[1] Spasticity has recently been defined as "disordered sensori-motor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary contraction of muscles".[2] Within the symptoms of spasticity, therefore, are numerous positive features of the upper motor neuron syndrome (e.g., hypertonus, spasms, and clonus). Spasticity may be considered beneficial for some aspects of daily life by individuals with SCI, such as through an increase in sitting or standing stability and the facilitation of the performance of some activities of daily living and transfers.[3,4] There are also, however, numerous potentially problematic effects of spasticity, including inhibition of effective walking and self-care, pain, fatigue, disturbed sleep, compromised safety, and negative self-image.[3,4] As there are reports that 65% to 78% of sample populations of individuals with chronic SCI have symptoms of spasticity,[3,5] it is important to examine the effectiveness of therapeutic alternatives to help manage these complications.

There are various possible approaches to the management of spasticity following SCI.[4] The importance of considering the role of physical therapy / rehabilitation strategies is recognized, as these have the additional advantages of causing few adverse effects while possibly providing further health-related benefits. Motor neuron excitability, which is believed to play a role in spasticity after SCI,[6] has been shown to be modifiable by such activity-based interventions.[7,8] A companion paper[9] reports on the effects of single sessions of body-weight supported treadmill training (BWSTT) and tilt-table standing (TTS) on hypertonus, spasms, clonus, and motor neuron excitability in seven individuals with chronic SCI. Body-weight supported treadmill training and TTS are therapies during which individuals with impaired lower limb motor function can undergo assisted-ambulation and -standing, respectively. Both activities provide opportunities for weight-bearing and stretch of the lower limbs compared to a seated position. Unlike during TTS, however, the lower limbs also are moved during BWSTT, with the musculature exhibiting electromyographic activity.[10-12]

There is evidence to support the beneficial effects of single sessions of activity-based therapy, such as BWSTT and TTS, for the modification of various spasticity outcomes in individuals with SCI.[13-18] In many cases, however, it is reported that the effects are short-lasting (i.e., less than 24 hrs, typically 1 to 8 hrs).[13,19] It is important to develop knowledge about whether certain strategies have longer-lasting effects than others and/or whether a culmination of repeated, properly timed, short-term effects leads to a perception by the individual of effective day-to-day spasticity management.

There are reports that participation in multiple sessions of active or passive movement activities by individuals with SCI can modify their spasticity symptoms.[20] Studies in which participants were asked to describe their satisfaction with regular passive standing have identified that some individuals perceive a benefit with respect to spasticity reduction.[21-23] Motorized repetitive passive movement [7,17] and activity-based therapies assisted by functional electrical stimulation (FES; e.g., cycling[24,25] and walking[26,27]) have also been shown to reduce spasticity. It is also important to recognize, however, that

there have been reports of increased spasticity by some study participants following long-term active or passive activity-based interventions.[19,22,24,25,28] Furthermore, studies of multiple sessions of activity-based therapy often do not state when, in relation to the final session of activity, the final assessments were performed; a measure performed within 24 hrs of the final session may represent acute / short-lived effects rather than actual longer-term, "maintained" changes.

It remains uncertain whether regular participation in activity-based therapy has a maintained effect on the management of spasticity. The characteristics of BWSTT make it a likely activity to have beneficial effects on spasticity management, but its costly equipment and personnel requirements necessitate the assessment of similar, less-costly possible alternatives. Therefore, the purpose of this study was to compare the effects of multiple sessions of BWSTT and of TTS on clinically-assessed and self-reported spasticity, motor neuron excitability, and theoretically-related constructs in individuals with chronic SCI and self-reported spasticity. It was hypothesized that, although both BWSTT and TTS would have beneficial effects on spasticity-related outcomes, BWSTT would be more beneficial than TTS overall.

5.3 METHODS

5.3.1 Participants

The participants were recruited from the outpatient SCI rehabilitation program associated with Hamilton Health Sciences. Eligibility for this study required that participants had chronic (> 1 year) complete or incomplete paraplegia or tetraplegia, self-reported presence of "stable" spasticity and consistent medication and physical activity/physiotherapy routines during the previous 6 mos and relied on a wheelchair as a primary mode of mobility. Participation in BWSTT during the previous 6 mos and/or any medical contraindications to performance of BWSTT or TTS were considered exclusion criteria. All participants provided informed consent prior to beginning the study and ethics approval was obtained from the Hamilton Health Sciences Research Ethics Board.

5.3.2 Study Design and Overall Protocol

A random cross-over design was used to compare the effects of activity within and between BWSTT and TTS conditions. Prior to their first activity session, each participant was weighed and familiarized with BWSTT, TTS, and the testing protocols. The order of the activity conditions was randomized. Participants were scheduled to perform 12 sessions of BWSTT over 4 wks (3 sessions/wk) and 12 sessions of TTS over 4 wks (3 sessions/wk), with a 4 wk detraining period between the two activity conditions. As much as possible, the total amount of time spent ambulating and standing was matched. The goal was for the first activity session of each condition to be 30 min of BWSTT or TTS, with the greatest tolerated activity duration performed every session thereafter (up to a maximum of 45 min). If a session was missed, efforts were made to make-up the missed session. Similar to other studies examining the effects of BWSTT,[29]

participants in this study were not prevented from performing their regular therapy during the study period.

Immediately prior to the first BWSTT and TTS sessions, participants were asked to answer all questions within the self-report questionnaires. Subsequently, the examiner-administered spasticity measures were assessed in the following order: 1) maximum H-reflex to maximum M-wave ratio (H/M ratio; an index of motor neuron excitability), 2) muscle tone, 3) flexor spasms, 4) extensor spasms, and 5) clonus, in both legs. The responses to BWSTT and TTS were assessed 24 - 48 hrs following the final session of each 4-wk activity condition.

The same examiner performed all testing in the same order for each assessment session within activity conditions. To control for the possible effects of seasonal/temperature variations on the outcome measures, testing for all participants occurred between the months of May to mid-September. Within each participant, all testing sessions were scheduled for the same day of the week and the same time of day. Participants were asked to refrain from caffeine, nicotine, marijuana and physical activity 20 hrs before testing and to empty their bladders prior to arrival to the laboratory. Participants were at least 90 mins postprandial at the start of data collection and were asked to standardize the time and type of breakfast eaten prior to testing.

5.3.3 Activity Conditions

5.3.3.1 Body-Weight Supported Treadmill Training (BWSTT)

Body-weight supported treadmill training took place using the Woodway Loco-system (Woodway USA Inc., Foster, CT), capable of supporting a percentage of the participant's body weight with a harness and overhead pulley system. Initial body-weight support (BWS) and speed of the treadmill were chosen to enable appropriate gait with full knee extension during stance. Decisions to modify the training parameters over the 4 wk BWSTT period were made according to our training protocol, outlined previously;[30] briefly, our protocol included an increase in speed and duration of walking and/or a decrease in the amount of BWS provided if and when possible. Over the course of each session, two therapists were positioned at each side of the treadmill to assist with leg movement when necessary. In particular, assistance was provided during the stance phase of the gait cycle to facilitate extension of the knee and hip joints. Participants were encouraged to swing their arms while walking and to place their entire body weight over a fully extended leg during the stance phase of the gait cycle.

5.3.3.2 Tilt-Table Standing (TTS)

Tilt-table standing allows individuals who have deficits in independent standing ability to undergo prolonged passive "standing". Compared to a seated position, TTS places load on the lower limbs and imposes stretch on the skeletal muscles that cross the hip, knee, and ankle joints. Standing was performed on a motorized tilt-table (Midland Manufacturing Co., Inc., Columbia, SC) at the greatest tolerated tilt angle (up to a table

maximum of 80°). Velcro straps at the knees, hips, and torso (if necessary) were used to support the individual in an upright position. Participants were allowed to increase gradually to their maximum tilt angle and were allowed to reduce their tilt angle during a session if they felt light-headed.

5.3.4 Outcome Measures

As spasticity is multi-dimensional, it has been suggested that a number of different outcome measures should be combined to obtain a representative assessment of this complication.[2,19,20,31,32] In the present study, therefore, outcome measures were selected to represent clinical assessments of tone, spasms, and clonus and of self-reported impact of spasticity on daily life and spasm frequency. Furthermore, measures capturing self-reported quality of life and functional mobility were administered because of the demonstrated relationship of these constructs with spasticity.[33] As elevated motor neuron excitability has been implicated as a mechanism to explain spasticity,[6] measures of the soleus H/M ratio were also performed.

5.3.4.1 Modified Ashworth Scale (MAS): Muscle Tone

The Modified Ashworth Scale (MAS) is used very commonly when clinical assessment of spasticity is indicated.[35] After having transferred to a flat surface (plinth), participants in the present study were tested in a supine position. Muscle groups assessed for passive resistance to tone were left and right hip flexors, extensors, and adductors, knee flexors and extensors, and ankle plantarflexors and dorsiflexors. The tone of each muscle group was assessed during the third of three range of motion excursions of the joint of interest. The speed of limb motion was standardized at 1 sec per direction. As has been done previously,[28] the scale was altered from a scale ranging from 0 to 4 (with the "1+") to a scale ranging from 0 to 5. An overall MAS score for each participant was obtained by summing the single scores from all tested muscle groups of both lower limbs.[36]

5.3.4.2 Spinal Cord Assessment Tool for Spastic reflexes (SCATS)

The Spinal Cord Assessment Tool for Spastic reflexes (SCATS) outlines standard perturbations and rating scales from 0-3 for the assessment of flexor spasms, extensor spasms, and clonus in individuals with SCI.[37] Participants were tested in a supine position immediately following MAS testing. An overall score for each assessed symptom was obtained by summing the ratings for the left and right lower limbs of each participant.

5.3.4.3 Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET)

The Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET) is a reliable and valid self-report measure of the impact of spasticity on daily life in people with SCI,

taking into account both the problematic and potentially useful effects of spasticity.[38] Respondents are asked to rate the impact of spasticity during the past 7 days on 35 items addressing aspects of daily life on a scale ranging from "-3" (extremely problematic) to "+3" (extremely helpful), with the option of selecting "0" (no impact of spasticity) or "N/A" (the aspect of daily life in question does not apply). Average scores, which can range from -3.00 to +3.00, were calculated by summing the scores from all applicable items and dividing by the number of applicable items.

5.3.4.4 Penn Spasm Frequency Scale

The Penn Spasm Frequency Scale[39] is a self-report measure of spasm frequency that is commonly used in studies involving individuals with SCI.[31] Participants in the present study were asked to rate their spasms during the past 7 days on a 5-point scale (0 = no spasms; 4 = spasms occurring more than 10 times per hour).

5.3.4.5 Quality of Life Index Spinal Cord Injury Version – III

The presence of spasticity is believed to have an impact on quality of life.[33] The Quality of Life Index[40] SCI Version – III (QLI) is a self-report questionnaire that asks respondents to rate several components of quality of life that are relevant specifically to individuals with SCI.[41] Of the QLI subscales, the Health and Functioning subscale was selected as most likely to be related to spasticity. This subscale has 15 items and has been shown to have adequate internal consistency ($\alpha > 0.80$).[40] Because of the known limitations when scales use multiplicative composite scores,[42] the importance-based weighting component of the QLI was omitted for use in the present study. Participants were asked to rate how satisfied they have been with each item over the past 7-days on a 6-point Likert-type scale (1 = very dissatisfied; 6 = very satisfied). Individual item ratings were summed to produce a total score that could range from 15 to 90.

5.3.4.6 FIM Motor Subscale

Improvement in function is considered to be important when evaluating success of a spasticity intervention.[43,44] The FIM is an 18-item subjective scale that assesses burden of care[45] and includes some items that are likely to be related to the assessment of the effects of spasticity intervention.[33] The Motor Subscale of the FIM is most likely to be relevant to spasticity (i.e., 13 items assessing self-care, sphincter control, transfers, and locomotion).[45] A self-assessment version of the FIM has been shown to be a reliable and valid measure of perceived functional independence in the SCI population.[46] Participants in the present study were asked to rate their levels of ability for the independent performance of each motor tasks during the previous 7 days on a 7-point Likert-type scale (1 = low ability; 7 = high ability). A total score for each participant, ranging from 13 to 91, was obtained by summing the individual item ratings.

5.3.4.7 Soleus H/M Ratio: Motor Neuron Excitability

The excitation of motor neurons via antidromic stimulation of Ia fibres is reflected by the H-reflex[47] Greater motor neuron excitability, therefore, is considered to be represented by a greater H-reflex.[48] To minimize the possible effects of measurement variability when comparing the maximum H-reflex between subjects and/or between groups, the H-reflex is normalized to the maximum M-wave (total pool of motor neurons that can be excited by stimulation[47]; H/M ratio).[48] To elicit the soleus H-reflex and M-wave in the present study, a constant-voltage stimulator (Digitimer Devices, Hertfordshire, UK) was used to apply a single rectangular biphasic pulse with a pulse width of 500μs to the tibial nerve in the popliteal fossa. The interelectrode distance was 3cm.[49] The Ag-AgCl gel, self-adhesive recording electrodes (Kendall Meditrace 530, Chicopee, MA) were placed in a bi-polar arrangement; one electrode was secured over the right soleus muscle and the other over the right Achilles tendon. Electrode location was recorded and repeated for each successive testing session. Prior to electrode application, the leg was shaved and the skin was abraded and cleaned with alcohol. Participants remained seated in their chairs, with knee and ankle angles between 110°-130°, and 90°-110°, respectively. During data acquisition, participants were asked to avoid clenching their teeth and to maintain consistent head and torso positions.[48] The sampling frequency of the electromyographic activity was 4 KHz and the data were bandpass filtered at 1 Hz to 1 KHz. The data were collected using customized LabVIEW software (National Instruments, Austin, TX). To prevent depression of the H-reflex, the stimulus frequency was less than 0.1 Hz.[50] Stimulus intensity was increased in steps of ~ 5 mV until Hmax and Mmax were approached and then increased in steps of ~ 1 mV until peak values were obtained. Manual identification of the time window containing the wave of interest allows for subsequent automatic calculation of the peak-to-peak amplitudes of the H-reflex and M-wave. The three greatest measurements of Hmax and the three greatest measurements of Mmax were averaged.[50] The H/M ratio was calculated by dividing the average Hmax by the average Mmax. An average of the right and left soleus H/M ratios for each participant was used for all analyses.

5.3.5 Data Treatment and Statistical Analyses

A paired samples t-test was used to compare the amount of BWSTT and TTS training time. To check for possible carryover effects of activity, independent samples t-tests were performed for each outcome measure, with Bonferroni correction for multiple comparisons: the group of individuals who performed BWSTT as their first activity (n=4) was compared to the group of individuals who performed TTS as their first activity (n=3). In studies with small sample sizes, it can be difficult to identify statistically significant effects using traditional ANOVA methods. Furthermore, a p-value does not provide an indication of the size of the effect and may falsely suggest that an intervention is ineffective. Therefore, to compare baseline scores to post-activity scores within condition and to compare change scores between activity conditions for each outcome measure,

effect sizes (ES) were calculated; relevant medium (~0.50+) or large (~0.80+) effect sizes are discussed.[51] Data are presented as mean±SD.

5.4 RESULTS

5.4.1 Participants

Six males and one female (age: 37.1±7.7 yrs) with chronic (5.0±4.4 yrs post-injury) complete or incomplete paraplegia or tetraplegia and self-reported spasticity participated in this study (Table 5.1). Current medication and physical activity/physiotherapy routines had been consistent for at least six mos prior to the start of the study and remained consistent throughout the duration of the study. The four participants with ASIA C SCI were capable of some independent ambulation at the beginning of the study. Three participants (#2, 3, and 4) were not involved in any formal or regular lower body exercise or therapy. Three participants (#5, 6, and 7) were involved in outpatient physiotherapy prior to the study. One participant (#1) used ambulation with forearm crutches for some walking within his home and occasional standing with assistance. All participants were asked to maintain consistent exercise and/or therapy routines throughout the study duration, as much as possible.

5.4.2 Activity Conditions

All participants completed the study protocol with attendance at 12 sessions of BWSTT and 12 sessions of TTS within the respective four-week training blocks (Table 5.2). Total training time of BWSTT and TTS did not differ, at 453.7±72.3 min and 471.6±52.0 min, respectively (p=0.23). BWSTT training parameters included a group mean treadmill speed of 0.23±0.10 m/s and a group mean provision of 50.7±24.7% BWS. Tilt-table standing was performed at a group mean table angle of 68.6±11.3°.

Table 5.1 Participant characteristics

Participant ID	Sex	Age (yrs)	Severity	Level	Post-injury (yrs)	Cause of SCI	Daily Spasticity Medications
1	M	40	C	T2	2	Fall	None
2	M	37	A	C6	14	MVA	40mg Baclofen
3	M	32	B	T5	7	MVA	80mg Baclofen Marijuana (2-3mg/day)
4	F	24	C	C5	5	MVA	None
5	M	39	A	T5	3	MVA	None
6	M	49	C	T10	3	Bleed	60mg Baclofen
7	M	39	C	T2	1	MVA	80mg Baclofen 5mg Tizanidine

Table 5.2 Body-weight supported treadmill training (BWSTT) and tilt-table standing (TTS) durations and parameters.

ID	BWSTT			TTS	
	Duration (min)	Avg. Speed (m/s)	Avg. BWS (%)	Duration (min)	Avg. Tilt Angle (°)
1	487	0.33	4.8	487	78.8
2	510	0.33	71.7	515	71.6
3	495	0.33	68.6	510	66.9
4	320	0.10	55.0	418	46.8
5	460	0.19	72.9	456	62.8
6	513	0.17	34.3	525	78.7
7	391	0.19	47.8	390	74.9

5.4.3 Clinically-Assessed Spasticity: Tone, Flexor Spasms, Extensor Spasms, and Clonus

The mean scores for all outcome measures are presented in Table 3. There was no evidence of carryover effects, nor any statistically significant differences between baseline scores within outcome measures for either condition. A potential beneficial effect of TTS on extensor spasms was supported by a strong effect size (ES=0.95; Figure 5.1). There were also indications that the change scores differed between BWSTT and TTS conditions for extensor spasms and flexor spasms; while TTS had a tendency to decrease and BWSTT to increase extensor spasms following 12 sessions of activity (ES=1.32), the opposite was true for flexor spasms (ES= 0.79; Figure 5.2). Clonus also appeared to benefit more from BWSTT, as TTS tended to increase clonus following 4 wks of activity compared to the tendency of BWSTT to decrease clonus (ES=0.66; Figure 5.2).

5.4.4 Self-Reported Spasticity: Impact on Daily Life and Spasm Frequency

Participation in BWSTT or TTS did not result in group changes in scores on the SCI-SET or the Penn Spasm Frequency Scale, likely due to the lack of uniformity in individual responses. Five and four individuals reported a reduction in the problematic effects of spasticity on daily life following BWSTT and TTS, respectively. More participants reported a worsening of the problematic effects of spasticity on daily life following TTS (n=3) compared with BWSTT (n=1). With regard to spasm frequency, more participants reported less frequent spasms following BWSTT (n=3) compared with TTS (n=1), while one individual in each condition reported more frequent spasms following activity.

Table 5.3 Summary of mean±SD scores for all outcome measures at baseline and post-activity for the BWSTT and TTS conditions.

Outcome Measures	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
Ashworth	29.6±16.0	29.9±8.5	29.4±14.4	30.1±14.5
Flexor Spasms	2.9±1.2	2.4±2.0	2.4±1.6	3.0±1.4
Extensor Spasms	3.7±1.5	4.0±2.1	4.6±1.1	3.6±1.0
Clonus	2.6±2.5	1.7±1.8	1.9±1.7	2.0±1.3
SCI-SET	-0.77±0.46	-0.64±0.46	-0.72±0.36	-0.64±0.41
Penn	2.6±0.8	2.3±0.8	2.3±1.0	2.1±0.4
QLI	62.0±12.4	68.1±12.6	61.7±12.0	64.7±12.9
FIM – Motor	73.6±11.4	76.3±11.7	77.4±10.2	76.1±12.5
H/M Ratio	0.61±0.25	0.63±0.26	0.64±0.26	0.65±0.27

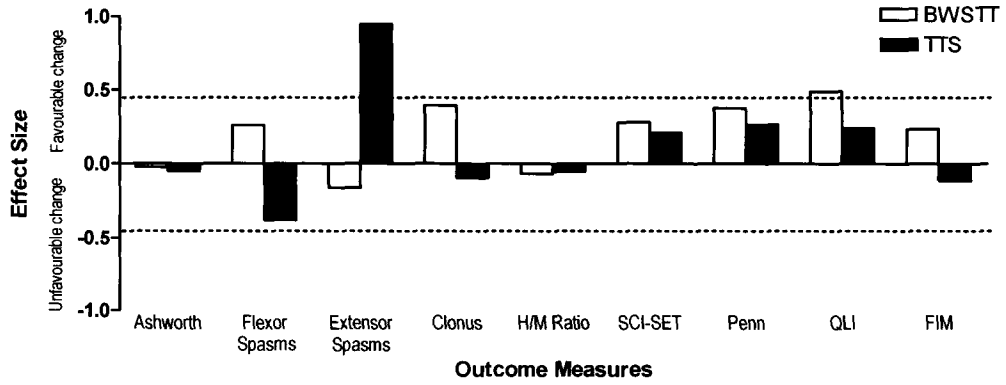

Figure 5.1 Effect sizes of the differences between baseline and post-activity outcome measure scores for BWSTT (open bars) and TTS (solid bars) conditions. Positive and negative effect sizes represent favourable and unfavourable changes, respectively, in the outcome measures. Absolute effect sizes greater than 0.45 (dashed line) are discussed in the text.

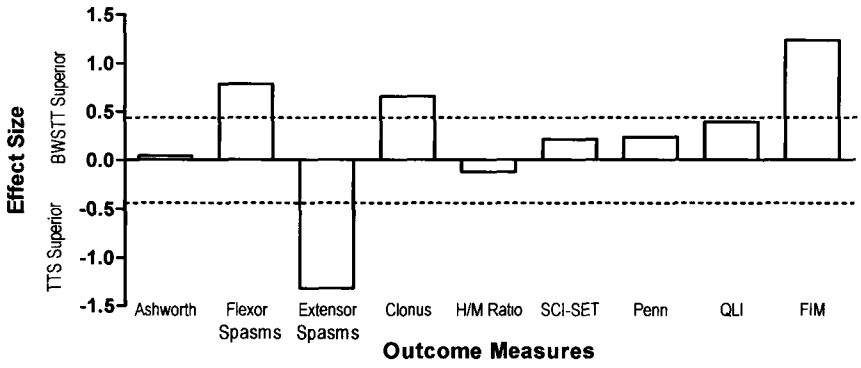

Figure 5.2 Effect sizes of the differences between outcome measure change scores following multiple sessions of BWSTT and of TTS. Positive effect sizes represent a superiority of BWSTT whereas negative effect sizes represent a superiority of TTS in terms of the induction of a favourable change. Absolute effect sizes greater than 0.45 (dashed line) are discussed in the text.

5.4.5 Self-Reported Related Constructs: Quality of Life and Functional Mobility

A medium effect size supported the potential beneficial effect of BWSTT on quality of life (ES=0.50; Figure 5.1). There was an indication that change in the group FIM Motor Subscale scores differed between activity conditions (ES=1.24; Figure 5.2); whereas scores tended to decrease following TTS, they tended to increase following BWSTT. There was also a trend, however for a higher mean FIM motor subscale score at baseline TTS compared to baseline BWSTT (p=0.10).

5.4.6 Soleus H/M Ratio: Motor Neuron Excitability

There were no observed changes in the H/M ratio following participation in either BWSTT or TTS, nor any observed differences in effects between activity conditions (Table 1; Figures 5.1 & 5.2).

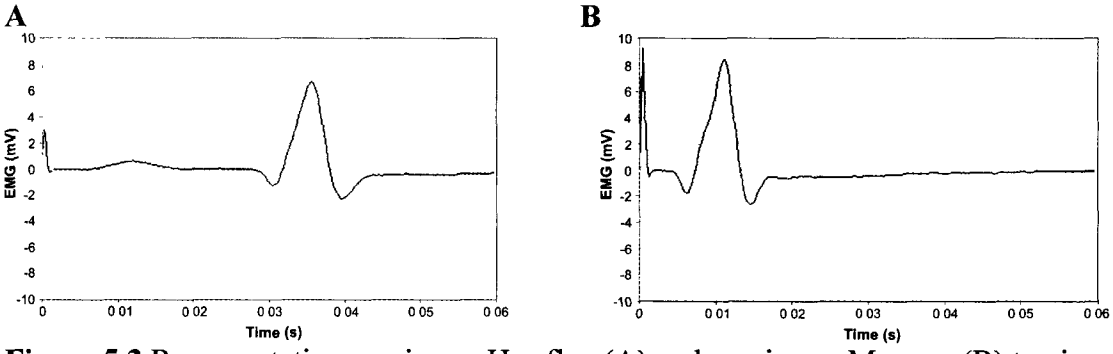

Figure 5.3 Representative maximum H-reflex (A) and maximum M-wave (B) tracings from one participant.

5.5 DISCUSSION

To our knowledge, this is the first study comparing the effects of multiple sessions of BWSTT and TTS on various measures of spasticity in individuals with chronic ASIA A to C SCI. All study participants successfully attended 12 sessions of BWSTT and 12 sessions of TTS within the respective 4 wk periods. In general, participation in weight-bearing activity tended to improve quality of life over 4 wks. There were also indications for reductions in some spasticity-related measures as well as possible differences between the effects of BWSTT and TTS. The heterogeneous nature of SCI and spasticity may lead to differing responses to activity by different individuals; although activity-based interventions have many potential health-related benefits, firm conclusions about the overall effects of BWSTT and TTS on spasticity-related outcome measures remain elusive.

The largest difference in training duration within participants between the BWSTT and TTS conditions was only 15min, with one exception: participant #4, who participated in TTS as the first activity type, performed 98 fewer minutes of BWSTT. This participant felt that the work required by BWSTT was greater than with TTS and, therefore, was consistently unable to match her BWSTT activity session durations with those of her previously completed TTS activity sessions. Despite this difference in one participant, there was no statistically significant difference between the group mean activity durations for the two conditions. During BWSTT, an average of 50.7±24.7% of BWS was provided. Our participants performed TTS at a mean tilt angle of 68.6±11.3°. In a previous study examining the effects of TTS at 85°, the load on the soles of the feet was estimated to be ~70% of the body weight (i.e., ~30% BWS).[15] Therefore, it is reasonable to deduce that, on average, more than 30% of BWS was provided during TTS in our study. Therefore, the main difference in training stimuli provided by the two conditions was likely the lower limb movement and resulting muscle activity that has been shown to occur during BWSTT.[10-12] Generally speaking, the majority of the participants in this study (6 out of 7) indicated anecdotally that they preferred BWSTT to TTS. The one participant who preferred TTS (#3) suggested that this was because he did not perceive benefits of BWSTT and the harness was uncomfortable, whereas TTS gave him a better stretch.

There was no evidence of change in muscle tone (MAS) following 4 wks of BWSTT or TTS. Others have found similar results following BWSTT or overground locomotor training for 12 wks,[52] FES-assisted locomotor training for 3 mos,[26] and FES-assisted cycling for 6 mos[28]. This could reflect limitations of the MAS[34] or a true inability of activity to reduce muscle tone. Results did suggest, however, that participation in TTS led to a reduction in extensor spasms. This finding is in agreement with a previous clinical observation that extensor spasms, in particular, tend to be reduced following TTS.[13]

Through the comparison of change scores, the present study demonstrated possible differing effects of BWSTT and TTS on various clinical measures of spasticity (Figure 5.2). There are indications that BWSTT was superior to TTS in terms of management of flexor spasms and clonus, but that TTS was superior to BWSTT with

regard to management of extensor spasms. It is difficult to explain these findings, but the mechanics of the activities and the specific pathophysiologies of the different symptoms of spasticity may play a role.[4] For example, it has been noted that proprioceptive stimuli from the hip are the most likely trigger for extensor spasms in individuals with SCI.[53] Therefore, perhaps activation of the hip flexor muscles during BWSTT (compared to during TTS) is perceived as a noxious stimulus and triggers spasticity.

Despite findings that participation in BWSTT and/or TTS may modify clinical measures of spasticity, either alone or differently from one another, we were unable to detect changes in the soleus H/M ratio following activity. A previous study that found an increase in low-frequency depression of the soleus H-reflex at rest after walking on a treadmill for 4 mos also was unable to find a change in the H/M ratio.[8] Although this may cast doubt on the role of motor neuron excitability in spasticity, it is most likely that various mechanisms play a role depending on the symptom, the timeline, and the activity.

The importance of considering the perception of the individual with regard to research and clinical outcomes is becoming more apparent and accepted. In particular, the best judge of spasticity is considered to be the individual, as he/she is better able to assess the impact of spasticity on daily life.[3,54-56] This message is supported by the suggestion that short-term benefits of a single session of activity may not be obvious to an examiner beyond a few hours.[13,19] It is possible, therefore, that even if the direct physiological effects of activity are short-lived, the perception of the individual may be that properly-timed activity sessions could improve function and quality of life overall. Of note, 5 and 4 out of 7 participants in the present study identified smaller problematic effects of spasticity on daily life at the end of the BWSTT and TTS conditions, respectively.

There was moderately strong evidence that self-reported quality of life improved following participation in multiple sessions of BWSTT. This finding is encouraging given the previous finding of no changes in quality of life following one year of intrathecal pharmacological treatment for spasticity.[57] The improvement in quality of life might have been related to the identified tendency of BWSTT to increase self-reported functional mobility compared to TTS, but it must be noted that this trend may simply reflect regression of FIM Motor Subscale scores toward the group mean. To enhance sensitivity in detecting change, the inclusion of five additional SCI-specific mobility and locomotor items with the FIM has been suggested.[58]

Overall, our finding that the various outcome measures did not respond similarly to training is not surprising given the numerous reports of poor correlations between different measures of spasticity.[6,31,36,37] The variability of results within the present study, including some negative findings, is consistent with the general findings in the literature for the effects of multiple sessions of activity-based therapy on lower limb spasticity in humans with SCI[20] (i.e., ambulation training,[8,29] standing,[22] passive movements,[7], and FES-exercise (including cycling[19,24,25,28] and walking[18,26,27]). These include reports of beneficial effects, lack of effect, and even detrimental effects, with many of these studies reporting different effects depending on the measure and/or the individual.[7,8,18,19,22,24-29] Therefore, the observation that some spasticity measures indicated unfavourable responses to training by some participants is disappointing, but also not surprising given

the similar previous reports. It is likely that the variable nature of spasticity and of SCI has contributed to the variability in responses to intervention in this population. There is some evidence that a higher degree of spasticity is experienced by individuals with tetraplegia and with an incomplete lesion,[5,19,59] although these findings are not unanimous.[60] Little information exists concerning any effects of injury level or severity on the responses to activity-based spasticity interventions. Anecdotal reports by the participants from the present study suggested that the effects of BWSTT and TTS on spasticity may differ between individuals; whereas some participants perceived short-term decreases in spasticity following a training session (up to ~4 hrs), others indicated a decrease in spasticity frequency, but an increase in intensity when spasticity did occur. These reports coincide with our finding that responses to activity, either single- or multiple-session, were inconsistent between individuals.[9] A previous study indicating similar variable effects of intervention on measures of spasticity was unable to detect participant characteristics to be used as predictors.[61] The small sample size in the present study similarly prevents the determination of whether injury level and severity (or other characteristics) were related to differing individual responses.

While the present study identified several interesting findings related to the effects of BWSTT and TTS on spasticity-related measures, the small and heterogenous nature of our sample made it difficult to draw conclusions based on group changes. The medium and large effect sizes for some of our results suggest that, with a greater number of study participants, the interventions may, in fact, have demonstrated statistically significant effects using traditional ANOVA methods. Sample size calculations based on the data from the current study suggest that, for any outcome with a medium or large effect size, inclusion of 9 (extensor spasms, FIM) to 50 (QLI following BWSTT) participants in future similar studies would reveal statistically significant effects if assessed by ANOVAs (power = 0.80).[62]

It is also possible that our intervention durations were not long enough for the activities to exert their benefits. Mechanisms to explain the effects of activity on spasticity after SCI, whether beneficial or problematic, remain elusive. It has been suggested, however, that observed decreases in spasticity immediately following the application of electrical stimulation may be due muscle fatigue.[63] It is conceivable that, once fatigue resolves following unaccustomed activity, physiological changes in response to activity could be perceived as noxious by the nervous system and, therefore, act as a trigger for spasticity in some individuals. Although not investigated in individuals with SCI, delayed-onset muscle soreness (DOMS) is typically present 24-48 hours after "atypical" exercise in the able-bodied population.[64] The participants in the present study exerted efforts that were uncustomary for them and, in some cases, reported an increase in spasm severity during the intervention period (predominantly in the BWSTT condition). Perhaps a longer intervention period would have allowed for the development of favourable physiological adaptations to activity, eventually making the activity sessions customary and beneficial to spasticity management. A recent examination of a single bout of BWSTT on pain found that, while some participants reported decreases in pain, others reported increased pain immediately following the session.[65] Although these assessments of pain would not reflect DOMS (as they were done immediately post-

activity), they may reflect the strenuous nature of BWSTT in some individuals with SCI for whom the activity is novel. A previous study investigating the effects of electrical stimulation-induced quadriceps contraction twice-daily, six days per week, found increased spasticity after 4 wks, but this was no longer evident after 8 wks (although only 8 participants completed the 8 wk protocol).[19] The progression of stimulation intensity over time was not discussed in this study, but it is possible that relatively similar absolute intensities of conditioning were administered during the study period. Therefore, as participants became stronger and more accustomed to the training, the "relative" intensity of the sessions would have progressively lessened. This may partially account for the finding that spasticity was no longer increased following 8 wks.

5.6 Conclusions

There are indications that some individuals with SCI and spasticity may benefit from participation in weight-bearing activity and that BWSTT and TTS may have different effects for certain outcomes. Compared to more invasive management strategies (e.g., pharmacological, surgical), activity-based interventions offer the potential for further health-related benefits and fewer possible problematic side-effects. Therefore, this area of research would benefit from investigations of longer activity periods and of individual characteristics that may affect responses to participation.

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CHAPTER 6

CONCLUSIONS AND FUTURE DIRECTIONS

5.0 CONCLUSIONS AND FUTURE DIRECTIONS

Spasticity is a relatively common neurological consequence that can be perceived as being both problematic and helpful to those who experience the symptoms. Although the pathophysiologies of muscle hypertonicity, spasms, and clonus remain to be fully elucidated, the existing literature outlines various methods for the assessment and management of spasticity after SCI. There continues to be the need, however, for a reliable and valid method of spasticity assessment by the *individual* and for management strategies that offer alternatives to pharmacological or surgical interventions. This thesis adds to the spasticity literature by providing a comprehensive overview of spasticity after SCI, including its pathophysiology and possible management strategies (Chapter 2). A new self-report measure of the impact of spasticity on daily life has been developed and shown to be both reliable and valid (Chapter 3). In addition, studies were conducted to examine the effects of both single sessions and multiple sessions of activity-based therapies on various measures of spasticity; BWSTT and TTS have been shown to offer potential benefits, with the two activities demonstrating differences in effects for some outcomes (Chapters 4 and 5). Overall, this thesis affords an increase in knowledge related to spasticity after SCI and has the potential to influence an improvement in quality of life for individuals who are affected by this complication.

6.1 CONTRIBUTIONS TO THE UNDERSTANDING OF SPASTICITY AFTER SCI

Spasticity is a symptom resulting from disruption of the upper motor neurons and, therefore, is a complication experienced by individuals who have various conditions such as stroke, multiple sclerosis, and SCI. Because the causes and locations of the lesions within the central nervous system vary with the different conditions, however, the presentations and pathophysiologies of the symptoms of spasticity can also vary depending on the population. Therefore, although the scientific literature included information about spasticity,[e.g.,1-5] up-to-date and comprehensive information about spasticity after SCI was lacking. The review presented in Chapter 2[6] of this thesis addresses this need.

As expected, the review of spasticity after SCI confirmed that the symptoms of spasticity are experienced by many individuals with SCI and are possible contributors to reduced quality of life.[3,4,7-12] This review highlighted the emerging suggestions in the literature that muscle hypertonicity, spasms, and clonus may be best represented as distinct symptoms under the "spasticity umbrella".[13] Also emphasized was the potential of spasticity to be both problematic and helpful in the daily lives of those who experience the symptoms and, therefore, efforts aimed at simply reducing spasticity may not be the most appropriate management approach. Physical rehabilitation, pharmacological, and surgical management strategies were discussed with regard to their applicability and limitations in the SCI population. Overall, this review served to support the anecdotal comments by our previous study participants that assisted weight-bearing activity may be beneficial to spasticity management. It was also identified, however, that there was a

need for a reliable and valid measure of the impact of spasticity on daily life that reflects the perceptions of the individual.

6.2 Contributions to the Advancement of Spasticity Assessment

Various clinical, biomechanical, and electrophysiological methods exist for the assessment of spasticity in the SCI population.[14-18] Although each of these examiner-based measures can provide information relevant to the quantification of spasticity symptoms, none is suited for assessment of the overall impact of spasticity on daily life in individuals with SCI. This need is addressed through the development and preliminary evaluation of the SCI-SET (Chapter 3[19]). The SCI-SET was shown to be a reliable and valid self-report measure of the impact of spasticity on daily life in individuals with SCI that takes into account both the problematic and helpful effects of spasticity.

The SCI-SET is a 35-item, 7-day recall questionnaire that targets aspects of daily life relevant to the SCI population. The scale demonstrated high internal consistency and test-retest reliability, as well as moderate to strong indications of construct validity. A key feature of the SCI-SET is the acknowledgement of spasticity as a factor affecting activity and participation restrictions, rather than simply a physical impairment. The bidirectional response scale (-3 to +3) is also new to spasticity assessment instruments. Given the emphasis on the importance of considering both the problematic and the potentially beneficial effects of spasticity on daily life,[2,3,13,20] this feature of the SCI-SET makes it particularly useful.

As it was designed to be comprehensive and easy to understand by respondents, the SCI-SET could be useful in both research and clinical settings. In particular, the SCI-SET may help individuals with SCI to consider and communicate their thoughts about the impact of spasticity on their daily life, facilitating their involvement in spasticity-related management decisions. Because published self-reports of spasticity are limited and because psychometric analysis is lacking for many clinical scales,[21] the SCI-SET is a potentially very valuable addition to the current literature.

6.3 Contributions to the Advancement of Spasticity Management

There are several possible approaches to the management of spasticity following SCI.[6] The importance of considering the role of physical therapy / rehabilitation strategies is recognized, as these have the additional advantages of causing few adverse effects and possibly providing further health-related benefits. There is evidence to support the beneficial effects of single sessions[22-27] and multiple sessions[14-18] of activity-based therapy for the modification of various spasticity outcomes in individuals with SCI. As two weight-bearing activities, both BWSTT and TTS provide loading and stretch of the lower limbs compared to a seated position. Unlike TTS, however, the lower limbs of individuals with SCI also undergo movement and electromyographic activity during BWSTT.[28-30] Within Chapters 3 and 4 of this thesis, novel research examining the effects of single and multiple sessions of BWSTT and TTS on various measures of spasticity is discussed.[31,32]

6.3.1 Effects of Single Sessions of Weight-Bearing Activity

Despite the relatively small (and heterogeneous) sample, collective analysis of the data produced some interesting findings: 1) a single session of TTS had an immediate effect on the reduction of extensor spasms and 2) BWSTT appeared to be superior to TTS for the reduction of muscle tone, flexor spasms, and motor neuron excitability. These results support a previous clinical observation that extensor spasms tend to be reduced following TTS[22] and add to existing literature by identifying the possible differences between the two weight-bearing conditions. The finding that the various outcome measures did not respond similarly to activity is not surprising given: 1) the numerous reports of poor correlations between different measures of spasticity[33-36] and 2) the many reports of different effects of activity-based intervention on spasticity depending on the outcome measure or the study participant.[22,24-27,37,38]

6.3.2 Effects of Multiple Sessions of Weight-Bearing Activity

Investigation of the effects of participation in weight-bearing activity for 4 wks revealed that: 1) quality of life tended to improve following BWSTT and 2) extensor spasms were reduced following TTS. There were also indications that flexor spasms, clonus, and self-reported mobility benefited more from 4 wks of BWSTT than of TTS, but that extensor spasms had greater reductions following TTS. Neither the MAS nor motor neuron excitability appeared to be modified by 4 wks of activity. It is difficult to explain the different effects of activity on the various outcome measures, but the mechanics of BWSTT and TTS and the specific pathophysiologies of the different symptoms of spasticity may play a role.[6] The heterogeneous natures of both SCI and spasticity may also lead to differing responses to activity by different individuals. Existing literature describing the effects of multiple sessions of activity-based therapy on lower limb spasticity in humans with SCI includes reports of beneficial effects, lack of effect, and even detrimental effects; interestingly, many of these studies have reported different effects of the intervention depending on the measure and/or the individual.[27,38-47] Therefore, this thesis further confirms the importance of including a combination of outcome measures of spasticity in order to best obtain a representative assessment. In particular, the perception of the individual should be considered in addition to examiner-based measures. The study described within Chapter 5 of this thesis was the first to implement the SCI-SET and, therefore, was able to acquire information about perceived changes in the impact of spasticity on daily life. Of our 7 participants, 5 and 4 identified smaller problematic effects of spasticity on daily life at the end of the BWSTT and TTS conditions, respectively. The group as a whole also demonstrated a tendency for improved quality of life following participation in multiple sessions of BWSTT, which speaks to the potential multifaceted benefits of weight-bearing activity as a spasticity-management intervention. Given the previous finding of no changes in quality of life following one year of intrathecal pharmacological treatment for spasticity, activity-based management may be an attractive alternative for some individuals.[48]

6.4 Study Limitations and Future Directions

The information and findings within this thesis add to the existing literature on spasticity after SCI; future clinical practice and scientific research can benefit from the new reliable and valid SCI-SET and the novel information about the effects of single and multiple sessions of BWSTT and TTS on spasticity. As discussed within each chapter, a few limitations of the presented research should be considered when designing new studies or implementing knowledge into practice. For example, future users of the SCI-SET may want to implement a standardized statement to remind participants to consider the impact of *spasticity* (and not other SCI-related impairments) when responding to questionnaire items. Continued validation and examination of the responsiveness to change of the SCI-SET would also be of benefit. While the goals of the intervention studies were to 1) assess changes in spasticity after the intervention, and 2) compare the specific effects of BWSTT and TTS, our small and heterogeneous sample precluded our ability to identify group changes using traditional ANOVA techniques. Future studies may detect statistical significance using ANOVA methods by using larger sample sizes and/or providing a longer intervention period (when investigating effects of multiple sessions). As shown in this thesis, responses to intervention may differ depending on the participant, the activity, and the time-period. Therefore, it will also be particularly important to focus on sub-populations of individuals with SCI in order to identify the characteristics that may mediate the effects of different activity types and durations. Furthermore, the particular characteristics of activity that induce changes in spasticity outcomes and the mechanisms explaining any effects of activity remain unclear. Mechanical changes at the muscle, tendon, and soft-tissue level[8] and plastic changes within the central nervous system[1,22] are among the current suggestions. It is also important to consider new possible mechanisms to explain the observed variable responses to intervention, such as DOMS resulting from participation in novel activity.

6.5 Overall Conclusions

Spasticity is prevalent in the majority of the SCI population, but how muscle hypertonicity, spasms, and clonus impact quality of life may be quite unique to each individual. When assessing spasticity, it is important to include a combination of measures that can represent the set of symptoms of interest and/or their impacts. The newly developed SCI-SET fills a need for a reliable and valid self-report measure of the impact of spasticity on daily life in people with SCI, taking into account both the problematic and useful effects of spasticity. The treatment and/or management of spasticity can take various forms, depending on its severity and impact on daily function. Activity-based management of spasticity symptoms is often considered to be more "palatable" than some of the pharmacological and surgical options currently available, and weight-bearing activity in particular (in the form of BWSTT or TTS) was investigated in this thesis. Both interventions led to some favourable adaptations, but their effects may differ depending on the spasticity outcome and the timeline. This thesis highlights the importance of persisting with efforts to broaden the scope of non-invasive

spasticity management options and encouraging individuals with SCI to become active contributors to their medical management decisions. Continued validation of the SCI-SET and further examination of the effects of BWSTT and TTS on the various spasticity symptoms in sub-populations of individuals with SCI will further our understanding of this prevalent complication.

6.6 REFERENCES

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APPENDIX B: THE PENN SPASM FREQUENCY SCALE

How would you rate your spasms **during the past 7 days**?

SPASM SCORE	FREQUENCY OF SPASMS
0	No spasms
1	Mild spasms induced by stimulation
2	Infrequent full spasms occurring less than once per hour
3	Spasms occurring more than once per hour
4	Spasms occurring more than 10 times per hour

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APPENDIX C: THE FUNCTIONAL INDEPENDENCE MEASURE – MOTOR SUBSCALE

1	2	3	4	5	6	7
Low ability			Moderate ability			High ability

During the past 7 days, what was your level of ability to:

1. perform toileting tasks (i.e., personal hygiene)?	_____
2. perform feeding tasks (i.e., reaching and grabbing, setting the table)?	_____
3. perform grooming tasks (i.e., brushing hair, brushing teeth, shaving)?	_____
4. perform bathing tasks (i.e., reaching and grabbing, washing hair/body, balancing)?	_____
5. dress your upper body?	_____
6. dress your lower body?	_____
7. perform your bladder management program (i.e., set up, clean up, positioning)?	_____
8. perform your bowel management program (i.e., set up, clean up, positioning)?	_____
9. transfer to and from such places as your bed, a chair, or your wheelchair?	_____
10. transfer to and from the toilet?	_____
11. transfer to and from the tub and/or shower?	_____
12. walk and/or wheel?	_____
13. use the stairs?	_____

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APPENDIX D: THE QUALITY OF LIFE INDEX SPINAL CORD INJURY – VERSION III (SATISFACTION COMPONENT)

For each of the following, please choose the answer that best describes how satisfied you have been with that area of your life during the past 7 days. Please mark your answer by circling the number. There are no right or wrong answers.

DURING THE PAST 7 DAYS, HOW *SATISFIED* HAVE YOU BEEN WITH:	Very Dissatisfied	Moderately Dissatisfied	Slightly Dissatisfied	Slightly Satisfied	Moderately Satisfied	Very Satisfied
1. Your health?	1	2	3	4	5	6
2. Your health care?	1	2	3	4	5	6
3. The amount of pain that you had?	1	2	3	4	5	6
4. The amount of energy you had for everyday activities?	1	2	3	4	5	6
5. Your ability to take care of yourself without help?	1	2	3	4	5	6
6. Your ability to go places outside your home?	1	2	3	4	5	6
7. Your ability to clear your lungs	1	2	3	4	5	6
8. The amount of control you had over your life?	1	2	3	4	5	6
9. Your chances of living as long as you would like?	1	2	3	4	5	6
10. Your sex life?	1	2	3	4	5	6
11. Your ability to take care of family responsibilities?	1	2	3	4	5	6
12. How useful you were to others?	1	2	3	4	5	6
13. The amount of worries in your life?	1	2	3	4	5	6
14. The things you do for fun?	1	2	3	4	5	6
15. Your chances for a happy future?	1	2	3	4	5	6

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APPENDIX E: RAW DATA – EFFECTS OF SINGLE SESSIONS OF BWSTT AND TTS

1) Clinical Spasticity: Modified Ashworth Scale (Muscle Hypertonicity)

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	60	50	55	51
2	25	28	8	7
3	12	10	27	28
4	26	30	31	33
5	21	15	21	20
6	22	20	36	32
7	41	26	28	33
Mean	**29.57**	**25.57**	**29.43**	**29.14**
SD	**15.97**	**12.96**	**14.36**	**13.48**
ES	0.28		0.02	

Change Scores

ID	BWSTT	TTS
1	-10	-4
2	+3	-1
3	-2	+1
4	+4	+2
5	-6	-1
6	-2	-4
7	-15	+5
Mean	**-4.00**	**-0.29**
SD	**6.86**	**3.25**
ES	**0.69**	

2) Clinical Spasticity: SCATS Flexor Spasms

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	4	4	3	5
2	2	1	3	3
3	2	2	1	1
4	2	3	2	2
5	2	2	3	2
6	5	3	0	3
7	3	4	5	5
Mean	**2.86**	**2.71**	**2.43**	**3.00**
SD	**1.21**	**1.11**	**1.62**	**1.53**
ES	0.13		0.36	

Change Scores

ID	BWSTT	TTS
1	0	+2
2	-1	0
3	-	0
4	+1	0
5	0	-1
6	-2	+3
7	+1	0
Mean	**-0.14**	**0.57**
SD	**1.07**	**1.40**
ES	**0.57**	

3) Clinical Spasticity: SCATS Extensor Spasms

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	6	5	6	6
2	3	4	4	2
3	5	4	5	4
4	4	4	6	4
5	4	4	4	3
6	2	1	4	4
7	2	1	3	2
Mean	**3.71**	**3.29**	**4.57**	**3.71**
SD	**1.50**	**1.60**	**1.13**	**1.38**
ES	0.27		**0.68**	

Change Scores

ID	BWSTT	TTS
1	-1	0
2	+1	-2
3	-1	-1
4	0	-2
5	0	0
6	-1	-2
7	-1	+1
Mean	**-0.43**	**-0.86**
SD	**0.79**	**1.21**
ES	0.42	

4) Clinical Spasticity: SCATS Clonus

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	2	2	0	0
2	2	2	1	1
3	2	2	4	3
4	6	6	2	2
5	0	0	0	0
6	0	0	2	2
7	6	5	4	4
Mean	**2.57**	**2.43**	**1.86**	**1.71**
SD	**2.51**	**2.30**	**1.68**	**1.50**
ES	0.06		0.09	

Change Scores

ID	BWSTT	TTS
1	0	0
2	0	0
3	0	-1
4	0	0
5	0	0
6	0	0
7	-1	0
Mean	**-0.14**	**-0.14**
SD	**0.38**	**0.38**
ES	0.00	

5) Motor Neuron Excitability: Soleus H/M Ratio

Individual Data

ID	BWSTT		TTS	
	Baseline	**Post-Activity**	**Baseline**	**Post-Activity**
1	0.833	0.736	0.815	0.930
2	0.117	0.127	0.103	0.170
3	0.823	0.795	0.811	0.781
4	0.486	0.601	0.670	0.636
5	0.631	0.615	0.540	0.672
6	0.775	0.744	0.832	0.773
7	0.634	0.605	0.685	0.669
Mean	**0.614**	**0.603**	**0.637**	**0.662**
SD	**0.252**	**0.224**	**0.257**	**0.239**
ES	0.05		0.10	

Change Scores

ID	BWSTT	TTS
1	-0.097	+0.116
2	+0.010	+0.067
3	-0.027	-0.030
4	+0.115	-0.034
5	-0.016	+0.132
6	-0.032	-0.059
7	-0.029	-0.015
Mean	**-0.011**	**+0.025**
SD	**0.064**	**0.078**
ES	**0.50**	

Note: Cohen's *d* Effect size (ES) calculation

$$ES = \frac{mean_1 - mean_2}{\sqrt{(SD_1^2 + SD_2^2)/2}}$$

ES = 0.20 – Small
ES = 0.50 – Medium
ES = 0.80 – Large

APPENDIX F: RAW DATA – EFFECTS OF SINGLE SESSIONS OF BWSTT AND TTS

1) Clinical Spasticity: Modified Ashworth Scale (Muscle Hypertonicity)

Individual Data

ID	BWSTT		TTS	
	Baseline	**Post-Activity**	**Baseline**	**Post-Activity**
1	60	36	55	54
2	25	36	8	11
3	12	12	27	25
4	26	35	31	32
5	21	27	21	18
6	22	31	36	29
7	41	32	28	42
Mean	**29.57**	**29.86**	**29.43**	**30.14**
SD	**15.97**	**8.51**	**14.36**	**14.46**
ES	0.02		0.05	

Change Scores

ID	BWSTT	TTS
1	-24	-1
2	+11	+3
3	0	-2
4	+9	+1
5	+6	-3
6	+9	-7
7	-9	+14
Mean	**+0.29**	**+0.71**
SD	**12.75**	**6.65**
ES	0.04	

2) Clinical Spasticity: SCATS Flexor Spasms

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	4	4	3	5
2	2	2	3	2
3	2	1	1	2
4	2	2	2	2
5	2	0	3	2
6	5	6	0	3
7	3	2	5	5
Mean	**2.86**	**2.43**	**2.43**	**3.00**
SD	**1.21**	**1.99**	**1.62**	**1.41**
ES	0.26		0.38	

Change Scores

ID	BWSTT	TTS
1	0	+2
2	0	-1
3	-1	+1
4	0	0
5	-2	-1
6	+1	+3
7	-1	0
Mean	**-0.43**	**+0.57**
SD	**0.98**	**1.51**
ES	**0.79**	

3) Clinical Spasticity: SCATS Extensor Spasms

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	6	6	6	5
2	3	4	4	3
3	5	5	5	4
4	4	6	6	4
5	4	4	4	3
6	2	3	4	4
7	2	0	3	2
Mean	**3.71**	**4.00**	**4.57**	**3.57**
SD	**1.50**	**2.08**	**1.13**	**0.98**
ES	0.16		**0.95**	

Change Scores

ID	BWSTT	TTS
1	0	-1
2	+1	-1
3	0	-1
4	+2	-2
5	0	-1
6	+1	0
7	-2	-1
Mean	**0.29**	**-1.00**
SD	**1.25**	**0.58**
ES	**1.32**	

4) Clinical Spasticity: SCATS Clonus

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	2	2	0	2
2	2	0	1	1
3	2	4	4	3
4	6	2	2	2
5	0	0	0	0
6	0	0	2	2
7	6	4	4	4
Mean	**2.57**	**1.71**	**1.86**	**2.00**
SD	**2.51**	**1.80**	**1.68**	**1.29**
ES	0.39		0.09	

Change Scores

ID	BWSTT	TTS
1	0	+2
2	-2	0
3	+2	-1
4	-4	0
5	0	0
6	0	0
7	-2	0
Mean	**-0.86**	**+0.14**
SD	**1.95**	**0.90**
ES	**0.66**	

5) Motor Neuron Excitability: Soleus H/M Ratio

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	0.833	0.862	0.815	0.900
2	0.117	0.098	0.103	0.083
3	0.823	0.829	0.811	0.838
4	0.486	0.546	0.670	0.634
5	0.631	0.628	0.540	0.703
6	0.775	0.786	0.832	0.650
7	0.634	0.659	0.685	0.745
Mean	**0.614**	**0.630**	**0.637**	**0.650**
SD	**0.252**	**0.261**	**0.257**	**0.268**
ES	0.06		0.05	

Change Scores

ID	BWSTT	TTS
1	+0.029	+0.085
2	-0.019	-0.020
3	+0.006	+0.027
4	+0.060	-0.036
5	-0.003	+0.163
6	+0.011	-0.183
7	+0.025	0.060
Mean	**+0.016**	**+0.014**
SD	**0.025**	**0.109**
ES	0.03	

6) Self-Assessed Spasticity: SCI-SET

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	-1.21	-1.21	-1.15	-1.26
2	-0.34	-0.22	-0.44	-0.29
3	-0.53	-0.35	-0.47	-0.21
4	-0.91	-0.56	-0.82	-1.09
5	-1.52	-1.25	-1.19	-0.66
6	-0.53	-0.79	-0.74	-0.68
7	-0.35	-0.12	-0.24	-0.29
Mean	**-0.771**	**-0.643**	**-0.720**	**-0.639**
SD	**0.455**	**0.457**	**0.364**	**0.410**
ES	0.28		0.21	

Change Scores

ID	BWSTT	TTS
1	+0.006	-0.107
2	+0.125	+0.147
3	+0.178	+0.257
4	+0.353	-0.265
5	+0.266	+0.531
6	-0.265	+0.059
7	+0.235	-0.059
Mean	**+0.128**	**+0.081**
SD	**0.205**	**0.262**
ES	0.20	

7) Self-Assessed Spasticity: Penn Spasm Frequency Scale

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	3	3	3	3
2	3	2	1	2
3	3	2	2	2
4	1	1	2	2
5	2	3	2	2
6	3	2	4	2
7	3	3	2	2
Mean	**2.6**	**2.3**	**2.3**	**2.1**
SD	**0.8**	**0.8**	**1.0**	**0.4**
ES	0.38		0.26	

Change Scores

ID	BWSTT	TTS
1	0	0
2	-1	+1
3	-1	0
4	0	0
5	+1	0
6	-1	-2
7	0	0
Mean	**-0.29**	**-0.14**
SD	**0.76**	**0.83**
ES	0.19	

8) Self-Assessed Related Construct: QLI

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	46	47	44	48
2	80	83	65	73
3	56	76	72	75
4	68	57	47	47
5	48	67	61	61
6	69	78	75	78
7	67	69	68	71
Mean	**62.0**	**68.1**	**61.7**	**64.7**
SD	**12.4**	**12.0**	**12.0**	**12.9**
ES	**0.50**		0.24	

Change Scores

ID	BWSTT	TTS
1	+1	+4
2	+3	+8
3	+20	+3
4	-11	0
5	+19	0
6	+9	+3
7	+2	+3
Mean	**+6.1**	**+3.0**
SD	**10.9**	**2.5**
ES	0.39	

9) Self-Assessed Related Construct: FIM Motor Subscale

Individual Data

ID	BWSTT		TTS	
	Baseline	Post-Activity	Baseline	Post-Activity
1	75	82	76	77
2	83	85	85	84
3	82	82	84	85
4	81	81	85	85
5	50	51	56	50
6	74	74	80	80
7	70	79	76	72
Mean	**73.6**	**76.3**	**77.4**	**76.1**
SD	**11.4**	**11.7**	**10.2**	**12.5**
ES	0.23		0.11	

Change Scores

ID	BWSTT	TTS
1	+7	+1
2	+2	-1
3	0	+1
4	0	0
5	+1	-6
6	0	0
7	+9	-4
Mean	**+2.7**	**-1.3**
SD	**3.7**	**2.5**
ES	**1.27**	

Note: Cohen's *d* Effect size (ES) calculation

$$ES = \frac{mean_1 - mean_2}{\sqrt{(SD_1^2 + SD_2^2)/2}}$$

ES = 0.20 – Small
ES = 0.50 – Medium
ES = 0.80 – Large