ON SPASTICITY IN SPINAL CORD INJURY: THE CHALLENGE OF MEASUREMENT AND THE ROLE OF NOVEL INTERVENTION (SEGWAY)

by

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A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF APPLIED SCIENCE

in

The Faculty of Graduate Studies

(Experimental Medicine)

THE UNIVERSITY OF BRITISH COLUMBIA

(Vancouver)

December 2009

ABSTRACT

Spasticity is a common sequale of spinal cord injury (SCI), and can have both beneficial and detrimental effects on mobility, functional independence and self-esteem. Clinical measurement of spasticity suffers from questions of credibility and contextual isolation. Recently self-report measures of spasticity have gained recognition as a viable alternative to independent examiner techniques. This pilot study endeavored to discern whether agreement was present between the clinical 'gold standard' measure (the modified Ashworth scale or MAS) and a recently validated self-report tool (the Spinal Cord Injury Spasticity Evaluation Tool or SCI-SET).

Spearman rank correlational analysis of measurement of spasticity using MAS and SCI-SET demonstrated some agreement, particularly with respect to the upper extremity musculature (ρ=.564, p=0.001). This relationship was much weaker comparing the lower extremity (ρ=.249, p=.161). They appear to measure similar, yet distinct aspects of the patients' spasticity. While the MAS is quick and offers an objective interpretation, perhaps the SCI-SET better reflects the multifaceted nature of spasticity and how it affects the individual, and may enable some interpretation regarding the upper and lower extremities. This information is helpful for clinicians to compile a more comprehensive picture of spasticity as it affects the individual.

The Segway Personal Transporter® is a novel, yet practical mobility tool which has yet to garner widespread support in the SCI population. It requires minimal functional ability to operate, and is appropriate for use in individuals with disabilities. Previous work suggests a possible link between the Segway and physiologic benefits to spasticity, pain and fatigue. A one month intervention program targeted these outcome

measures to determine (1) if they exist and (2) whether the effects are immediate or long-term in nature.

The Segway provides evidence for short term reductions in clinical ratings of spasticity ($p=.001$) and self-report pain ($p=.027$). Self-evaluations of fatigue approached significance ($p=.12$). There is some evidence to suggest that these beneficial outcomes may have lasting effects. The Segway may provide an adjunct to current therapy options for treating spasticity by introducing a stimulus to the system which overrides some underlying mechanism(s). As this was pilot work, further investigation of a longitudinal nature with a larger sample size is required to substantiate these findings.

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ACKNOWLEDGEMENTS

I would like to acknowledge my thesis committee, Dr. Bonnie Sawatzky, Dr. Heather Finlayson and Dr. Richard Beauchamp for their support throughout this process. A special thanks to Bonnie, for providing me with the perfect balance of guidance and independence which has enabled me to learn so much on the path of achieving this goal. To Heather, who always made herself available, even at the last minute. A warm thanks to Ian Denison PT, Chris Grant MD, and Silas Wiefelspuett PT, who cheerfully gave a great deal of their time and energy in order to see this project through.

I would also like to thank my participants who made this research possible. They shared their stories and gave so much of themselves, their energy and their gas mileage, and always did so with tremendous grace. They rose over and above the challenges placed in front of them, and they continue to inspire me with their determination and zest for life.

DEDICATION

To Bonnie, for the giving of her time and intellect, and for pointing out frankly when I didn't make any sense at all! To my friends and fellow graduate students for their ideas, suggestions and mainly for listening when I needed them most.

To my family, ever-encouraging, always loving. To my grandparents, who always supported my pursuit of higher education. To my mother for her endless wisdom, gift of vocabulary, and mostly for her patience and spiritual reflection. To my father, for his quiet strength, which is always waiting for me, right on time. And to my sister, who hugged me through this, even across the miles.

And finally to the man who has my hand and who is better than a light and safer than a known way.

CHAPTER 1- INTRODUCTION

1.1 Overview of Thesis

This thesis consists of four chapters. The first chapter provides the overall purpose of the work, followed by background information regarding spinal cord injury (SCI) implications and secondary complications. The following two chapters present pilot work pertaining to a correlational study of a clinical measure of spasticity and a recently validated bidirectional self-report tool (Chapter 2) and the results of a one-month rehabilitation program using the Segway Personal Transporter with outcome measures related to spasticity, pain and fatigue (Chapter 3). These chapters are manuscript style and methodology is addressed with respect to each individual investigation. The literature review is followed by a description of the research questions and their respective hypotheses. The chapters conclude with the significance of the studies. Chapter four integrates the findings from each study and concludes with implications for both rehabilitation and research.

1.2 Purpose

The purpose of this thesis was to: (1) compare the current 'gold standard' clinical measure of spasticity in individuals with SCI with a newly designed self-report tool and (2) discuss the findings of a one-month intervention study employing the Segway PT device with respect to spasticity, pain and fatigue.

1.3 Introduction to Spinal Cord Injury

1.3.1 Spinal Cord Injury

The human spinal cord is a multifaceted network of bidirectional neural communication between the brain and its motor, sensory and autonomic targets. It is also a site of reflex integration between proprioceptors and their motor and autonomic effectors. Due to the diversity of functions controlled by level and structure, injury or disease to the spinal tracts results in varying forms and extent of dysfunction, depending on the affected structures (Jacobs et al., 2004). Thereafter, these individuals face unique physical, social and psychological alterations to their lifestyles.

A spinal cord injury (SCI) involves damage to the cord resulting in some disruption of neural transmission. Paralysis and/or weakness of musculature, accompanied by altered sensation in the area below the level of injury remains the most obvious effect of SCI (Somers et al, 1992, Eng et al., 2008). In addition, individuals who sustain spinal cord injuries are affected with varying degrees of bladder, bowel and sexual dysfunction. They may experience respiratory compromise, loss of temperature regulatory mechanisms, circulatory impairment, and other autonomic nervous system dysfunction (Gerhart et al., 1991).

1.3.2 Incidence of SCI

Approximately 41, 000 Canadians are living with SCI, and 1, 100 new cases occur yearly (www.rickhansen.com). Prevalence ratings are distributed bimodally with respect to age; injuries in persons aged 15-24 years are usually the result of high-energy trauma, such as motor vehicle accidents, accidents resulting from sporting activities, or

acts of violence. Injuries in persons older than 55 years usually result from low-energy trauma, such as falls from the standing position (Goodrich et al., 2008). Highly specialized acute and long-term medical care has dramatically increased life expectancy rates, bringing individuals with paraplegia almost on par with the general population, while those with tetraplegia continue to live approximately 10-20 years below average (Yeo et al., 1998).

1.3.3 Cost

The cost to the Canadian health system is between $1.25 million and $25 million, over the lifetime of each injured person, depending on the severity of the injury. Annual costs to the Canadian health care system for individuals with spinal cord injury are estimated at $750 million (www.rickhansen.com). The financial burden also affects the individual, with tetraplegics in particular may be required to pay in excess of $100, 000 for initial acute care and rehabilitation. Yearly follow up costs may surpass $20, 000 for those individuals who do not require mechanical ventilation, and over $50,000 for those who do (Gerhart et al., 1991).

1.3.4 Classification Schemes: Traumas, Plegias, and Impairments

The nature of spinal cord injury can be classified as either traumatic in origin (e.g., motor vehicle accidents, falls, violent incidences, diving) or non-traumatic (e.g., tumors, spinal stenosis, vascular ischemia) (Jacobs et al., 2004). Traumatic SCI accounts for the larger proportion of SCI injuries, however, the exact proportion compared to non-traumatic SCI is difficult to ascertain because reporting of nontraumatic SCI has been inconsistent (Eng et al., 2008).

Rarely does disease or trauma to the spine result in total anatomical or physiological transection of the cord. Advances in modern medicine have allowed more than half of the survivors to retain varying degrees of motor, sensory or autonomic sparing at different spinal cord levels (Marino et al., 1999). A *complete* spinal cord injury involves total and permanent loss of sensory and motor function in the level of the lowest sacral segment. Conversely, in an *incomplete* spinal cord injury there is partial preservation of sensory and/or motor function more than 3 segments below the neurological level of injury, including the lowest sacral segment (Maynard et al., 1997, Waters et al., 1991). Partial sparing of ascending or descending pathways in incomplete injuries can result in varying patterns of neurological deficit, and may often produce asymmetrical paralysis. Injuries to the spinal cord can result in *tetraplegia,* impairment or loss of motor and/or sensory function at the highest thoracic segment (T1) or above, or *paraplegia*, impairment or loss of motor and/or sensory function in the thoracic segments (T2 and inferior) or lower (Hsieh et al., 2008).

The functional outcome in spinal cord injury is determined by the level of neurological injury. The American Spinal Injury Association (ASIA) system defines the neurologic level of injury as the most caudal segment on both sides of the body that tests as normal for both sensory and motor function (Donovan et al., 1990). Accurate descriptions of SCI lesions are often confounded by spontaneous recovery of sensorimotor function. Therefore the benchmark of one-year post injury exists as the clinical standard for determining neurological completeness (Jacobs et al., 2004). Table 1.1 illustrates the ASIA SCI classification system.

Table 1.1 American Spinal Injury Association Impairment Scale

A	*Complete:* no motor or sensory function is preserved in the sacral segment S4-S5.
B	*Incomplete:* Sensory but not motor function is preserved below the neurological level and extends through the sacral segments S4-S5
C	*Incomplete:* Motor function is preserved below the neurological level, and the majority of key muscles below the neurological level have a muscle grade less than 3.
D	*Incomplete:* Motor function is preserved below the neurological level, and the majority of key muscles below the neurological level have a muscle grade greater than or equal to 3.
E	*Normal:* Motor and sensory function is normal.

1.3.5 Spasticity in SCI

Upper motor neurons (UMNs) originate in the brain and brain stem and comprise the long tracts of the spinal cord. They project to lower motor neurons via the corticospinal (pyramidal) tract (Kandel et al., 1995). Lesions to the upper motor neurons occur at spinal levels of T10 and above and result in decentralization of the nervous system by disruption of the pathways between the higher centres and the motor subsystems (Adams et al., 2005, Satkunam, 2008). While motor, sensory and autonomic reflex activities are preserved, they are no longer modulated by the brain (Jacobs et al., 2004).The resulting 'release phenomena' is an increase in abnormal and stereotyped responses due to the loss of tonic inhibition from supraspinal to spinal neuronal centres (Dietz, 2000). Evidence of this occurrence has been demonstrated in decerebrate cats in which ordinary head and neck movements produce postural reflex activity that would not occur in an intact animal (Kandel et al., 1995). One of the most prominent occurrences of UMN syndrome is exaggerated sensorimotor reflexes below the level of injury. This enhanced reflex activity is part of the phenomenon of spasticity.

1.3.5.1 Definition of Spasticity

Spasticity is a common, but not inevitable sequale of spinal cord injury (Adams, et al., 2005). Lance (1980) refers to spasticity as 'a velocity dependent increase in muscle tone characterized by exaggerative tendon jerks resulting from hyperexcitability of the stretch reflex as one component of upper motor neuron syndrome'. Discrepancy exists within the literature pertaining to the definition of spasticity; that is, while some authors include symptoms such as clonus, hyperactive tendon reflexes and spasms within the umbrella term 'spasticity' (Dietz, 2000, Sköld et al., 1999, St George et al., 1993), others discuss these same symptoms as related to but distinct from spasticity, which is specifically an increased muscle tone (Bohannon et al., 1993, Sheean et al., 2002, Elovic et al., 2001, Maynard et al., 1990). More recently spasticity has been divided into various subcomponents: (1) *intrinsic tonic spasticity*, which involves exaggeration of the tonic component of the stretch reflex (manifesting as increased muscle tone), (2) *intrinsic phasic spasticity*, pertaining to exaggeration of the phasic component of the stretch reflex (manifesting as increased tendon reflexes and clonus) and (3) *extrinsic spasticity*, which is the increased exteroceptive reflexes (flexor reflex) and pathologic radiation of reflexes between spinal segments (increased spinal reflexes) over time (Sköld et al., 1999, Decq, 2003). While not part of the spasticity syndrome, increased exteroceptive reflexes, as well as loss of motor function such as muscle power and coordination are often integrated with spasticity (Young et al., 1986).

Functionally, spasticity may involve slow voluntary movements characterized by abnormal stereotyped patterns of total synergy, and co-contraction of agonist and antagonist muscles, preventing fractionate movement patterns (Nuyens et al., 1994). Tonic spasticity can be painful, often interfering with activities of daily living (ADLs), self-care and sleep, while phasic spasticity in patients with SCI can lead to other secondary

health complications including falls or pressure sores (Craven et al., 2009). It may also prevent the individual from returning to independent living and gainful employment (Canadian Paraplegic Association, 1997). External factors such as medication, pain, urinary tract infections, constipation, fatigue and mental state may influence spasticity (DeSouza et al., 1987).

ASIA classification of SCI severity and level of injury may predict the likelihood of developing spasticity (Adams et al., 2005). This is based on the assumption that higher (cervical) injury level and greater loss of function (ASIA A and B) will have an increased tendency to develop spasticity. However, in a survey of individuals with SCI, self-reported problematic spasticity was more common in individuals with incomplete injury (ASIA grades B to D) than with a complete injury.

Spasticity is not always a negative outcome. Often increased spasticity is beneficial in maintaining muscle tension, which would facilitating transfers and weight bearing, as well as reduce muscular atrophy and possibly prevent osteoporosis (Sommers et al., 1992, Kirschblum, 1999, Hseih et al., 2008). Additionally, by maintaining muscle tone, spasticity can serve to improve circulation by increasing venous return, thus reducing the risk of deep vein thrombosis (Kita et al., 2000, Jozefxzyk, 2002). It has also been suggested to enable lower body dressing and improve performance of activities of daily living (ADLs) (St George et al., 1993). Spasticity may also serve as a diagnostic tool in that it may provide a warning mechanism to identify pain or problems in areas where there is no sensation (increased spasticity being a sign of exposure to a noxious stimuli—infection, bowel impaction, urinary retention, etc) (SCI Peer Support Discussion Forum www.apparalyzed.com).

Consensus in the literature suggests that the goal of treatment should not be to modify the excitability and rigorousness of reflexes, but to overcome functional impairments that are related to spasticity (Dietz, 2000, Ward, 2008). The aim of

treatment is to reduce abnormal sensory inputs in order to decrease disproportionate and uncontrolled alpha motor neuron activity (Gracies et al., 1997a, Ward, 2008).

1.3.5.2 Prevalence and Impact for People with SCI

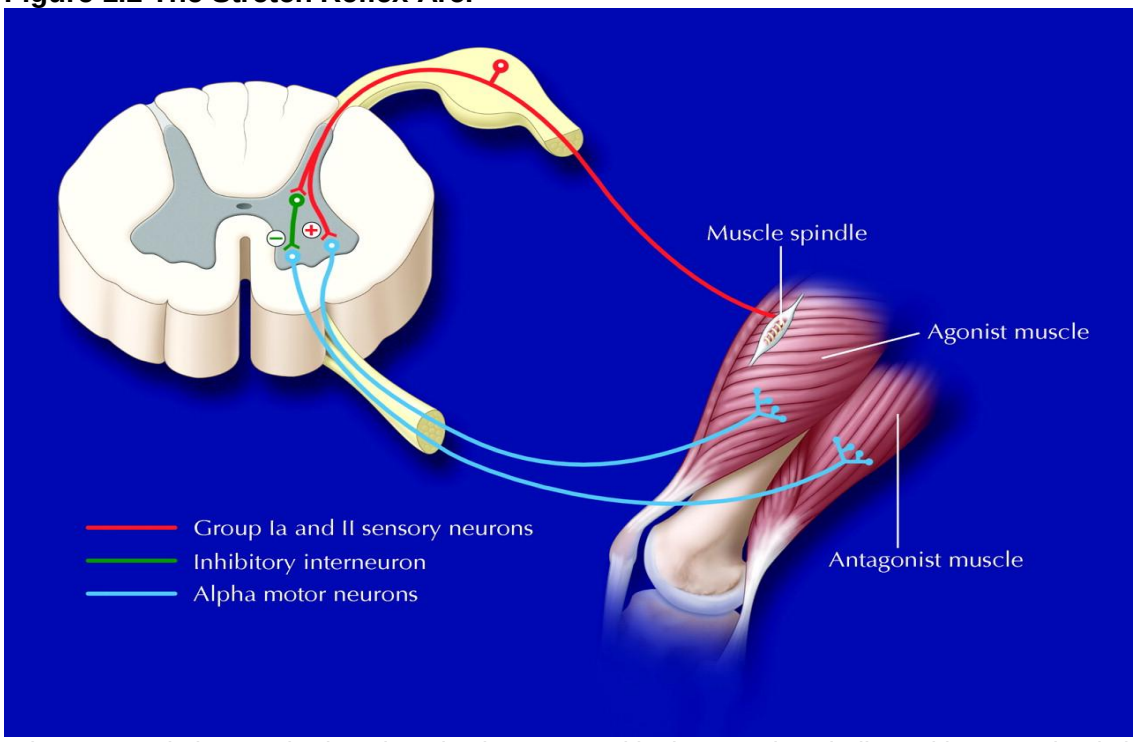
One-third of all individuals with SCI experience spasticity secondary to SCI. (Lewis 1993). Higher estimates, suggest these numbers are closer to 53% (Walter et al., 2002) to 78% (Levi et al., 1995). Furthermore, approximately 41% (Levi et al., 1995) of these individuals with spasticity secondary to chronic SCI list it as one of the major medical obstacles to community and workplace re-integration (Canadian Paraplegic Association, 1997).

1.3.5.3 Pathophysiology

The pathophysiology of spasticity is poorly understood, however, it is assumed to be a sensorimotor phenomenon involving a disruption of integration of sensory input to motor responses in the nervous system (Ivanhoe et al., 2004). The decrease in supraspinal inhibition results in a loss of inhibitory drive to alpha motor neurons, making them hyperexcitable to sensory input (Dietz, 2000).

The stretch reflex is a monosynaptic reflex pathway that originates in the muscle spindles, which are embedded parallel to muscle fibres. Once the spindles deform with stretch, an impulse is sent via a 1a afferent to the spinal cord where it synapses either with interneurons or directly with an alpha motor neuron innervating the muscle from which the stimulus derived (Lundy-Ekman, 2002). See figure 1.2 for an illustration of the stretch reflex arc.

Figure 1.2 The Stretch Reflex Arc.



When a muscle is stretched, an impulse is generated in the muscle spindle and is transmitted via the sensory neuron to the grey matter of the spinal cord. Here the sensory neuron synapses with the motor neuron, and the transmitted impulse results in muscle contraction. While agonist muscles contract in response to stretching, antagonist muscles must relax. Reprinted with permission from the Canadian Medical Association Journal (CMAJ).

Decq (2003) differentiates intrinsic spasticity into tonic and phasic components. The *tonic* component of the stretch reflex is associated with increased muscle tone results from a maintained stretch of the central region of the muscle fibres initiated by polysynaptic connections between type Ia and type II afferents with interneurons within the ventral horn of the cord, and these interneurons subsequently synapsing with alpha motor neurons to facilitate sustained contraction of the muscle being stretched. (Lundy-Ekman, 2002). Increased muscle tone in response to passive stretch in SCI is thought to be attributable to the hyperexcitability of the tonic component of the stretch reflex. The resulting hypertonia is velocity dependent, with increased stretching velocities leading to greater amounts of reflex activity (Dietz, 2000). Tonic stretch reflex hyperexcitability may be due to a lower threshold of firing, an increased gain of the stretch reflex or a

combination of the two (Sehgal et al. 1998. Enhanced sensitivity to neural transmitters (Decq, 2003) and changes in muscle characteristics (atrophy, fibrosis and decreased elasticity, loss of sarcomeres) may alter contractile properties and contribute to increased passive tension (Lundy-Ekman 2002, Seghal et al., 1998, Gracies 1997).

Intrinsic *phasic* spasticity, resulting from exaggeration of the phasic component of the stretch reflex, manifests in tendon hyper-reflexia and clonus (Decq, 2003). Tendon hyper-reflexia is an exaggerated muscle response to an externally applied tap of deep tendons (St George et al., 1993). Reduced pre-synaptic inhibition of group Ia fibres is thought to play a role in this hyper-reflexia (Dietz, 2001). Clonus, or involuntary rhythmic muscle contraction, can result in distal joint oscillation which is most evident at the ankle (St George et al, 1993, Elovic, 2001). It is elicited by a sudden rapid stretch of the muscle and results in recurrent activation of the stretch reflexes. Interruption of descending influence in SCI leads to disinhibition of the stretch reflex, causing exaggeration of the phasic stretch reflex pathway (Sheean et al., 2002).

Involuntary muscle spasms can also occur as a response to external influences, known as extrinsic spasticity (Sheean et al., 2002, Decq, 2003, St George et al., 1993). Flexor spasms, triggered by flexor reflex afferent input from the skin, muscle, subcutaneous tissues and joints is the most common form of extrinsic spasticity (Adams et al., 2005). The disruption of normal descending influences in SCI lowers flexor reflex afferent thresholds such that polysynaptic reflexes involved in flexion withdrawal are overly apparent (Sheean et al., 2002).

1.3.5.4 Other Mechanisms of Spasticity

Decreased threshold sensitivity of the stretch reflex is thought to be due to an imbalance of the excitatory and inhibitory influences, specifically a lack of supraspinal inhibition (Adams et al., 2005). Other proposed mechanisms of spasticity include fusimotor hyperactivity, loss of presynaptic inhibition (GABA released, collateral branch inhibition, Renshaw inhibition), and abnormal excitability of the spinal segmental and intersegmental interneurons from loss of supraspinal influences (inhibitory and excitatory) and changes in muscles themselves (Dietz, 2000, Satkunam, 2008).

1.3.5.5 Treatment

At present, management of spasticity often involves a combination of approaches (Adams et al., 2005). Conservative treatment is initially preferred, with gradual administration of more invasive treatments as needed. It is commonly understood that no one approach is likely to be universally successful for all individuals (Kirshblum, 1999, Adams et al., 2005).

Elimination of noxious stimuli, including urinary tract infections (UTI), ingrown toe nails, decubitus ulcers, infection anywhere in the limb (pneumonia, pancreatitis, etc), impacted stool, poor positioning in the wheelchair, fractures in paralytic limbs, stress and neuroleptic agents which tend to increase tone is often the first step (Merritt, 1981, Satkunam, 2008).

1.3.5.6 Stretching/Physiotherapy

Habitual movement of joints and soft tissue elongates joint capsules, muscles, subcutaneous tissue and ligaments through a wide range of motion many times a day. If for any reason the range of motion is restricted (as in tonic spasticity), contracture of the connective tissue will develop due to unopposed forces. Specifically, in areas where little or no motion occurs, collagen is laid down in a denser meshwork of sheets and bands with shorter distances between attachment sites, which leads to restriction of movement (Kottke et al., 1966). Additionally, positional shortening of the series elastic component of the musculotendinous unit and a decrease in the number of sarcomeres within the muscle fibres may increase muscle tension and tone (Katz et al., 2000). Orthoses are employed to maintain a spastic limb in a neutral position. Serial casting may also be used to attempt to change the position through gentle stretching with the application of successive casts. Appropriate limb positioning can reduce spasticity and improve comfort. This includes correct seating posture in a wheelchair, bed position and upper limb positioning (Satkunam, 2008).

Therapeutic movement programs which focus on activation of residual motor functions and improving range-of-motion of a joint are thought to prevent secondary complications such as contractures (permanent shortening of the muscle, or in essence, a state of constant spasticity (Dietz, 2000) and deformity of the limb. This is of particular import with respect to tonic spasticity, where there is a decreased amount of stretch of the muscle (Satkunam, 2008). Aggressive management in the early stages of spasticity after SCI is anticipated to prevent permanent deformities and joint contracture (Satkunam, 2008). A program consisting of prolonged stretching on awakening and/or prior to sleep has been cited by individuals with spinal cord injury as a means to reduce spasticity (Merritt, 1981).

The application of topical cold (cryotherapy) may reduce stretch reflex excitability by decreasing the sensitivity of cutaneous receptors, slowing nerve conduction and afferent firing rate of the muscle spindle (Katz et al., 2000). Functional electrical stimulation (FES) may help improve muscle imbalance by stimulating a weak muscle to oppose the activity of a stronger, spastic muscle. It is most often used to assist the ankle dorsiflexors during walking, of the wrist and hand extensors in opposition to spastic flexors (Katz et al., 2000). The use of biofeedback may enable the individual with spasticity to train themselves to consciously reduce muscle tone, however, there is minimal research to support this or previously mentioned physical modalities (Katz et al., 2000, Satkunam 2008).

Although physical therapy may reduce the effects of spasticity, this reduction is almost always temporary and may not affect voluntary movements in a useful manner (Katz et al., 2000).

1.3.5.7 Pharmacologic Interventions

Drug therapies for spasticity in SCI are indicated when spasticity is diffuse and severe in nature (Satkunam 2008). The majority of antispasmolytics act centrally (at the neuromuscular junction) with the exception of one (Dantrolene) which acts peripherally. Conversely, neuromuscular blockers act by interfering with transmission at the neuromuscular end plate and have no CNS activity. The goal of these medications is to induce selective neurological impairment. A number of randomized clinical trials have shown antispasticity medications to be efficacious in the management of spasticity in spinal disorders (Nance et al., 1994, Gruenthal et al., 1997, Gracies 1997a).

1.3.5.8 Oral Medications

A variety of oral medications is employed to manage spasticity and associated pain. Gamma-amino butyric acid (GABA) and Glycine are the two major inhibitory neurotransmitters of the central nervous system. GABA is usually found in small interneurons within the spinal cord that are responsible for presynaptic inhibition (Merritt, 1981). When GABA is released, it binds to receptors in the post-synaptic membrane either ligand-gated chloride ion channels ($GABA_A$) or G-protein coupled receptors ($GABA_B$)

Baclofen is the most commonly prescribed medication for spasticity in SCI (Satkunam, 2003). It binds to the $GABA_B$ receptor at the presynaptic terminal of the Ia afferent to inhibit excitatory neurotransmitter release. This is accompanied by inhibition of gamma motor neurons that reduces the sensitivity of the muscle spindles to stretch. *Benzodiazpines* bind to the $GABA_A$ receptors and lead to postsynaptic inhibition. *Tizanidine* and *Clonidine* are imidazoles that act on the alpha-2 noradrenaline receptors. They induce presynaptic noradrenergic inhibition.

While oral agents successfully manage spasticity in many individuals, these medications are costly and may have minimal efficacy with respect to more severe cases (Kunkel, 1993). Side effects, such as sedation, drowsiness, insomnia, dry mouth, nausea, fatigue, weakness, ataxia, hypotension and others vary between pharmacologic agents (Adam et al., 2005). Toxicity, addiction and cognitive impairment often preclude practical application of these agents (Merritt, 1981).

1.3.5.9 Chemodenervation

Intramuscular injections have been employed to interrupt neuronal signaling. These treatments allow for selective inhibition of problematic spasticity while preserving maximal sensation and valuable voluntary function (Merritt, 1981).Administration of phenol or ethanol to the nerve trunk simulates a local anesthetic by blocking sodium channels to reduce nerve depolarization (Jozefczyk, 2002). The mechanism of action involves denaturing of protein and fibrosis of neural tissue, disrupting conduction of the reflex arc, leading to muscle relaxation (Kirschblum, 1999, Gracies et al., 1997a). Injection techniques for phenol include either motor nerve block resulting in complete loss of tone, or motor point block in which small motor braches are injected with multiple small doses to achieve a graded, though highly variable response (Satkunam 2008). While inexpensive, there is some permanent denervation with every injection (Ward, 2003), in addition of other side effects such as injection site pain, risk of phlebitis, tissue necrosis, sensory dysesthesia, and muscular weakness (Gracies et al. 1997a, Jozefczyk, 2002).

Unlike ethanol and phenol, whose actions are mediated by protein denaturation at the nerve, botulinum toxins (BTX) disrupt the docking and fusion of acetylcholine vesicles at the pre-synaptic membrane, thereby impeding the release of the neurotransmitter acetylcholine (Ach) into the neuromuscular junction (Kita et al., 2000, Elovic, 2001). The effect is the creation of a local neuromuscular blockade. BTX injections generally last approximately 3-4 months, but vary between individuals. Muscle function returns gradually by way of regeneration or sprouting of new neuromuscular junctions of blocked nerves. BTX is dose-dependent and reversible secondary to the regeneration process Suggested dosages depend on the muscle group being injected,

however 400 units per treatment is commonly understood to be the maximal dose (Tsui et al., 1994).

BTX has been shown to be effective in reducing muscle hypertonia (Brashear et al., 2002b) and motor unit activity (Burgar, 1994), improving tone (Bohlega et al., 1995) and joint mobility, (Bohlega et al., 1995) and is associated with functional improvements in gait due to reduced co-contraction of muscle antagonistic to movement (Hesse et al., 2000, Gracies, 2004). Although BTX has yet to be investigated extensively in the SCI population, there is evidence in the literature of its potential use in these individuals (Kirschblum, 1999, Al-Khodairy et al., 1998, Barnes et al., 2003). However, BTX is an expensive (roughly $375 per 100 units with patients often needing up to 400 units per treatment), and may not be covered under various health plans. Dosages are individual and muscle specific, which often requires a trial-and-error approach. Finally, due its temporary nature repetitive injections are required approximately every 3 months to maintain a continuous effect indefinitely.

1.3.5.10 Intrathecal Medications

As oral baclofen does not easily cross the blood brain barrier, it must be taken in very high doses to achieve effective CSF levels (Satkunam, 2008). Intrathecal Baclofen administration by way of a battery operated pump inserted in the lower quadrant of the anterior abdominal wall has resolved this dilemma in some patients by delivering high concentrations of the drug near the site of action (Katz et al., 2000). At only 1% of the oral dose, one of the main advantages to this procedure is the reduction in negative systemic side effects as compared with oral administration (Kirschblum, 1999). A catheter is inserted subcutaneously from the pump to the lumbar spine where it is inserted into the dural sac. The reservoir containing the drug is programmed by

telemetry to deliver various dosing regimes. Candidates for this surgery experience severe functional impairment from spasticity and have not successfully responded to conservative therapies and other medications, or are unable to tolerate side effects of oral medication.

There is evidence of successful management of spasticity in SCI using intrathecal baclofen (Gracies et al., 1997a, Ward, 2003). Recent reviews of the literature suggest improvements in quality of life, facilitation of transfers and self-care, in addition to reducing spasm frequency and/or spasm related comfort (Emery, 2003, Ward, 2003). However, the surgery is invasive, with additional risks of infection, and the long terms effects of intrathecal treatment with Baclofen are not yet known (Ward, 2003). Possible complication as a result of surgical implantation of the pump and catheter include dislodgement, disconnection, migration, catheter kinking, blockage, infection, pump failure and accidental under- or overdose (Kita et al., 2000, Kirschblum, 1999).

1.3.5.11 Cannabinoids

There is growing interest in the efficacy of cannabinoids as a means of managing spasticity. The active ingredient in cannabinoids is delta-9-tetrahydrocannabinol (delta-9-THC). CB1 cannabinoid receptors are present on central and peripheral neurons and appear to be responsible for the euphoric and anticonvulsive effects of cannabis. CB1 receptor agonists that are thought to contribute to the suppression of muscle spasm/spasticity associated with multiple sclerosis or spinal cord injury (Satkunam 2008). CB2 receptors, present mainly on immune cells and appear to be responsible for the anti-inflammatory and possibly other therapeutic effects of cannabis (Pertwee et al., 1999).

Use of medicinal marijuana to control spasticity has been studied with positive outcomes in patients with multiple sclerosis (Wade et al., 2006, Collin et al, 2007). Unfortunately, much is yet unknown about the therapeutic potential of cannabis or CB1 receptor agonists on spasticity in SCI. Although many individuals with SCI indicate that marijuana smoking limits their spasticity, few scientific investigations of this phenomenon have been done. Dunn et al. (1974) reported that 5 of 8 subjects indicated that they experience decreased spasticity during marijuana intoxication. Malec et al. (1982) found that 88% percent of respondents who indicated they used marijuana for spasticity management reported total elimination or reduction of spasticity. However, some argue that while reporting positive subjective findings, these studies fail to show objective improvement in spasticity (Satkunam, 2008). The concern that perceived reduction in spasticity is used as a rationalization for marijuana use has been raised. On the contrary, Malec and colleagues (1982) found that perceived spasticity reduction was independent of variables related to marijuana use (current and previous marijuana use, current and previous use among social reference group, and age). Further research pertaining to cannabis use is needed specific to the spinal cord population.

1.3.5.12 Surgical Interventions

Lastly, surgical neurolysis may be employed to treat spasticity on a local level; however these are reserved for selected cases (Hseih et al., 2008). Selective rhizotomies (cutting of the posterior roots to interrupt the peripheral reflex arc), while encouraging in children with cerebral palsy, is not frequently used in individuals with SCI (Kirschblum, 1999). Orthopaedic techniques such as tenotomy (the release of a tendon from a severely spastic muscle), tendon lengthening (reducing the angle of pull to improve joint alignment), and tendon transfer (moving the tendon attachment closer to

the muscle) are all performed with the goal of improving usefulness of voluntary function (Jozefczyk, 2002). Intensive therapy is often necessary to maximize long-term functional gains, and post-surgical side effects such as sensory changes, muscle wasting frequent voiding difficulties, loss of erectile function and secondary infections are a concern (Katz et al., 2000).

1.3.5.13 Remedial Management Summary

Given the variable nature of spasticity, it is unlikely that one agent is beneficial in all situations. Often pharmacologic interventions are costly and require time and patience on the part of the individual to determine appropriate doses. In addition, all drugs have potentially serious side effects, such as sedation, somnolence, dry mouth, headache, ataxia and respiratory and cardiovascular depression. Rarely, development of individual antibody resistance can occur (Al-Khodairy et al., 1998). These factors may limit dose optimization (Satkunam, 2008) and their negative effects must be carefully weighed based on psychological impact. Continued use of medications should be contingent on a clearly beneficial overall effect (Katz et al., 2000).

1.4 Measurement of Spasticity

1.4.1 Introduction to Measurement of Spasticity

Various methods are used to measure the character and degree of spasticity. The three most common approaches in assessing spasticity are biomechanical, neurophysiological, and clinical methods. In general these approaches are employed in

isolation of one other and have little practical crossover application (Lunenberger et al., 2005). For example, although various techniques to asses the mechanical manifestation of spasticity such as electrogoniometry and dynamometry (combined with surface electromyography) are frequently used in research investigations, they are rarely utilized in routine clinical settings (Sköld et al., 1999, Lechner et al., 2006). Recently self-report measures of spasticity have gained credibility as a viable alternative to independent examiner techniques (Collin et al., 2007).

1.4.1.1 Biomechanical Measurement Techniques

Biomechanical assessments of spasticity attempt to equate muscle activity as an indicator of spasticity. They do so by employing a controlled perturbation while quantifying the mechanical response to the movement with torque and position transducers and electromyography (EMG). However, EMG recordings are susceptible to inherent variability and high levels of noise, and have been poorly correlated with intensity of spasticity (Katz et al., 2000). Although biomechanical methods of quantifying spasticity have been demonstrated to deliver viable approaches, they often require extensive time on behalf of both the researcher and the participant, and further involve expensive equipment (Lunenberg et al., 2005, Wood et al., 2005).

1.4.1.2 Neurophysiologic Measurement Techniques

A wide variety of electrophysiological reflex studies have been created in an attempt to explore the neuronal circuits within the spinal cord (DeSouza et al., 1987). The premises for these techniques are often based on animal models which may or may not be applicable to human subjects. Further, most of these techniques study the neural

circuitry in isolation or at rest, ignoring the effect of movement on biomechanical and neurophysiological features (Katz et al., 2000). These methods have been criticized in terms of lack translation and/or application to rehabilitation settings (Lunenberger et al., 2005) and are generally impractical for clinical application (Sköld et al., 1999).

1.4.1.3 Clinical Assessments

Clinical assessments of spasticity involve observer appraisal (usually by a physician or a therapist) of the resistance of the spastic limb to manual movement (Lechner et al., 2006). They entail manual examination of a single agonist group over a specific range of motion with or without the employ of gravity. Clinically-based assessments have the benefit of minimal equipment cost and set up time, and of reflecting typical manifestations of spasticity in realistic settings. However, they have been criticized for providing a single component of spasticity: the resistance to passive movement, or 'tone' of the muscle in question (Priebe et al., 1996, Sköld et al., 1998, Sköld et al., 1999), rather than a measure of overall spasticity (Pandyan et al., 1999) and for being subjective in nature (Sköld et al., 2000).

1.4.1.4 Wartenberg Pendulum Test

The Wartenberg Pendulum test uses gravity to assess the limb's impedance to imposed movement (rapid stretch) in the quadriceps and hamstrings in a supine position (Ness et al., 2009). When the lower limb segment falls from a fully extended position, it sways about the vertical like a pendulum. Knee kinematics can be assessed with an electrogoniometer and accelerometer. Sinusoidal patterns of angular motion are created using a mathematical model to help differentiate normal from spastic limbs (Katz et al.,

2000). Although it has been validated for use in individuals with SCI (Nance et al., 1994), it is limited for use in thigh muscles only. Moreover, this method is time consuming for clinicians and suffers from the assumption that the mechanical properties of the agonists and antagonist knee musculature are equal. In reality these vary with the level of muscle excitation and length (Katz et al., 2000).

1.4.1.5 Ashworth and modified Ashworth Scales

The most widely accepted clinical scale to measure muscle tone is the modified Ashworth Scale (Pandyan et al., 1999, Sköld et al., 1999, Gregson et al., 2000, Satkunam, 2003, Ivanhoe et al., 2004). The Ashworth scale was originally designed as a simple clinical tool to test the efficacy of an anti-spastic drug in patients with multiple sclerosis (Ashworth, 1964) by quantifying the reflex activity elicited in the muscle groups that oppose the passive movement (Bohannon et al., 1987). The assessor is required to move the limb passively about a joint in one second and grade the resistance on a five point ordinal scale (Pandyan et al., 1999, Ivanhoe et al., 2004). Each grade corresponds to a level of spasticity and where in the passive range of motion the resistance is experienced by the examiner. Grade 0 corresponds to no spasticity, and grade 4 the limb is rigid.

An adapted version, called the modified Ashworth Scale (see Table 1.2), was introduced by Bohannon and Smith (1987) with an additional category of '+1' falling between 1 and 2, which aimed to increase the sensitivity of the scale by identifying the phenomena of 'catch', a sudden increase in muscle stiffness in response to a brisk muscle stretch (van der Salm et al., 2005). The individual is placed in a standardized position (supine) and the test is performed by movement provocation of an extremity at as fast a speed as possible (~50º/s) (Sköld et al., 1998).

Table 1.2 The modified Ashworth Scale

0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of range of motion
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of motion
2	Marked increase in muscle tone throughout most of the range of motion, but the affected part is easily moved
3	Considerable increase in muscle tone, passive movement is difficult
4	Affected part is rigid in flexion or extension (abduction or adduction, etc)

1.4.1.6 The Tardieu Scale

The Tardieu scale has recently been suggested as a more appropriate alternative to the Ashworth Scale for measuring spasticity, as it incorporates multiple velocities (slow, speed of gravity, and fast) in the assessment of muscle tone (Haugh et al., 2006). Tardieu (1954) argues that a slow velocity (below that which would trigger the stretch reflex), and a high velocity (that would elicit it), are necessary to record the quality and strength of the reaction. Grading for the Tardieu is based on observation of the angle at which catch and clonus can be detected in response to stretch in the muscle being tested (van der Salm et al., 2005). A modified version of the Tardieu scale has been developed which aims to quantify the presence of dynamic muscle tone by establishing the proportion of change between the slow passive stretch and a fast

velocity stretch. Such a calculation is termed the R2–R1 difference (Mackey et al., 2004). It is considered an ordinal measure of spasticity (Ivanhoe et al., 2004). Several authors have suggested that a higher level of sensitivity occurs based on smaller graduations in the dynamic range of movement measurement of the scale (Wallen et al., 2004, Filipetti et al., 2003, Ivanhoe et al., 2004), however it has yet to supersede the modified Ashworth in terms of frequency of use.

In a review of the literature on using the Tardieu Scale, Haugh et al. (2006) noted a significant dearth of support to legitimize its use. There are only two scientific studies which have investigated the reliability of the Tardieu scale, in both cases comparing it to the MAS in children with cerebral palsy. Mackey et al. (2004) looked at intrarater reliability for biceps brachii muscle group. Fosang et al. (2003) assessed both intra and interrater reliability of the hamstrings, gastrocnemius, and hip adductor muscle groups. These papers produced conflicting results, with Fosang et al. (2003) suggesting that the interrater reliability of the Tardieu was greater than the intrarater reliability, whereas Mackey et al. (2004) found large intersessional variability in the Tardieu scale measures of R2 and R1 as well as the R2–R1 difference. They also highlighted the difficulties of applying three standardized angular velocities to the upper limbs of individuals with differing levels of tone.

Evidence of validity for the Tardieu scale is also minimal, however, it is noteworthy that additional error may be introduced by the clinician within the ranges of the three angular velocities (i.e. fast velocities for some participants being equivalent to slow velocities for other participants (Mackey et al., 2004). Further investigation into these constructs is required before the scale is adopted as a common measure of spasticity (Haugh et al., 2006). Table 1.3 provides a comparison of previously mentioned clinical-observer methods to measure spasticity.

Table 1.3 Comparison of Clinical Scales to measure spasticity

Clinical Scale	Description	Metric Properties
Wartenberg Pendulum Test	Clinician employs gravity to assess the limb's impedance to imposed movement Limited to thigh muscles only	*Nance (1994)* Pearson correlation with the AS (r=.88) in SCI *Bohannon et al. (2009)* Spearman rho correlation (ρ)=.57 in stroke patients (SP)
Ashworth Scale (AS)	Clinician manually assess the resistance of an agonist group to movement. Grading occurs on a 5 point scale (0-4)	**Interrater Reliability** *Bohannon et al. (1987)* kappa (k)=0.85 in SP *Lee et al. (1989)* Kendall's tau (Kt)=0.92, Spearman ρ=.89 *Bodin et al. (1991)* k=.83 in SP *Sloan et al. (1992)* k=.74 in SP *Nuyens et al. (1994)* k= .24-.84 in multiple sclerosis **Intrarater Reliability** *Allison et al. (1996)* r=.55-.74 in traumatic brain injury
Modified Ashworth Scale (MAS)	AS with the addition of a 1+ category to enhance sensitivity at the lower end of the scale.	**Interrater Reliability** *Haas et al. (1996)* k=.21-.61 in SCI *Gregson et al. (2000)* weighted kapp (kw)=.45-.95 in SP *Craven et al. (2009) k*=.22-.60 in SCI **Intrarater Reliability** *Gregson et al. (2000)* kw=.59-.94 in SP *Craven et al. (2009)* k=.60-1.0 in SCI
Tardieu Scale	Clinician assess resistance to stretch of an agonist over three different velocities (slow, speed of gravity, fast). Grading is based on the angle at which catch and clonus can be detected.	**Interrater Reliability** *Fosang et al. (2003)* ICC with MAS=.70 in children with cerebral palsy (CP) **Intrarater Reliability** *Fosang et al. (2003)* ICC with MAS range=.38-.93 in children with CP

		Mackey et al. (2004). Mean V1=89 deg/s±51, mean V2=166deg/s±78, mean V3=298deg/s±165 in children with CP

1.4.1.7 Self Evaluation

The presence and severity of spasticity has been shown to vary greatly over the course of the day, commonly being most pronounced in the morning and evening (Sköld et al., 2000). Self-assessments are sensitive to these changes (Sköld et al., 2000, Elovic et al., 2004, Ditunno et al., 1994). As such, spasticity should be assessed by the individual with SCI as he or she can evaluate the impact of their spasticity on daily life (Sköld et al., 1999, Kirschblum et al., 1999, Adams et al., 2007). Routine clinical work often incorporates self opinion or personal accounts of the degree and impact of spasticity (Lechner et al., 2006). As such, researchers are increasingly persuaded that patient-report outcome measures are uniquely appropriate for spasticity assessment (Collin et al., 2007). Self-ratings have begun to be included among the outcome measures of spasticity in the research, however this has yet to become the norm. These measures include both spasticity severity using Likert scales (Sköld et al., 2000) or single item ratings (Priebe et al., 1996, Parise et al., 1997, Benz et al., 2005), or spasticity impact on daily pain and function (Priebe et al., 1996) or daily life (Lechner et al., 2006). Different days of the week (Pierson et al., 1997, Sköld et al., 2000, Adams et al., 2007), visceral activity (Sjolund et al., 2002), concurrent illness and emotional state (Sköld et al., 1999, Marciniak et al., 2008) are also likely to play a role in experiences and functional impacts of spasticity.

By contrast, examiner-based physical assessments may fail to detect spasticity in people with SCI who report it (Adams et al., 2007). Assessment of 1 or more

symptoms of spasticity by an examiner do not correlate well with self-assessments of spasticity. Priebe et al. (1996) found a weak correlation ($r \leq .40$) between several self-report scales and clinical examination scores. Work by Lechner and colleagues (2006) also demonstrated this with a weak correlation ($p=.36$) between clinical scale (the Ashworth) and self-rated general spasticity. Nor does examiner-based spasticity appear to relate to improved function (Pierson et al.1997, Sherwood et al., 2000) or with each other (Priebe et al., 1996). Manual physical examination of specific muscle groups identified spasticity in only 60% of patients who reported it (Sköld et al., 1999). For an overview of the attributes and clinical utilities of various self-report and clinical measures of spasticity in SCI see Hsieh et al. (2008).

Visual analogue scales (VAS) allow the responder to select the degree to which a construct of interest is graded from one extreme to the opposite (Hsieh et al., 2008). VAS has been used in various studies on SCI patients to measure intensity of different qualities of pain (Song et al., 1993, New et al., 1997, Strömer et al., 1997.) Previous studies have had participants rate their spasticity from 'no spasticity' to 'most imaginable spasticity' either within the previous hour (Sköld et al., 2000) or after completion of a specific test activity (Lechner et al., 2006). Likert scales are numerical scales in which respondents specify their level of agreement to a statement (Likert, 1932). These have also been employed in individuals with SCI for spasticity (Gruenthal et al., 1997, Lechner et al., 2006). In general VAS (Sköld et al., 2000) numerical (Lechner et al., 2006) and single-item scales such as the Penn Spasm Frequency Scale (Priebe et al., 1996, Benz et al., 2005) self-report generally present either an absence of or a negative impression of spasticity. At present, no tools allow for a beneficial rating of spasticity.

Self-report and clinical examination scores appear to represent different dimensions of the clinical problem of spasticity (Lechner et al., 2006). With the exception of work by Sköld and colleagues (2000), self-report measures are usually rendered with

respect to global perceptions of spasticity, while clinical measures directly rate each specific muscle group provoked by movement. Patients may attribute other symptoms such as pain as part of the spasticity syndrome, while clinicians only consider phenomena related to muscle tone and manually elicited physical resistance (Sköld et al., 2000, Lechner et al., 2006). Sensory projection neurons and segmental reflex pathways may share interneurons. Thus, processes in the sensory systems stimulating spinal pain pathways may correspond to those causing spasticity in motor systems (Ashby, 1975). Sensations caused by parathesia (pain, prickle, tension, constriction) were described by Sjölund (2002) as 'sensory spasticity' and may be similar to the phenomena of phantom limb pain, which would have no influence on a clinical rating of passive resistance to movement. Accordingly, Lechner and colleagues (2006) found that 25% of their subject population selected equal or greater symptoms associated with sensory spasticity than from muscle tone-related phenomenon. Of these subjects, all but one was ASIA grade A and had no sensory function below their lesion level.

1.4.2 The Ashworth Scale: A Closer Look

The Ashworth and the modified Ashworth scales have garnered controversy regarding their metric properties. While the original Ashworth was devised to research the effects of Caisporadol on muscle tone in patients with multiple sclerosis (Ashworth 1964), these scales have been applied to a myriad of disease conditions (brain injury, stroke, MS, SCI, and other neurodegenerative conditions) which have implications of spasticity. The inclusion of individuals with varying neurological conditions (Ghotbi et al., 2009) and those with cognitive impairments (Bohannon et al., 1987) may limit the generalizability of these findings. In addition, these studies have utilized a variety of statistical techniques including Pearson product moments (r), Spearman's rank

correlation coefficient (ρ), Cohen's Kappa (k), and weighted kappa (kw) among other models, which further obscures comparison.

1.4.2.1 Reliability: Inter-rater and Intra-rater

Reliability is a determination of the consistency of the measurements. It is essential to interpret the results of a measure and to provide answers regarding differences between measures and to speculate on confidence of findings (Craven et al., 2009). For the Ashworth, inter-rater reliability (the extent to which the scores of two independent raters would agree) of k=.85 was achieved in the elbow flexors in individuals with intracranial lesions (Bohannon et al., 1987). Subsequent values of k=.74 (Sloan et al, 1992) and k=.83 (Bodin et al., 1991) for the elbow flexors and k=.75 for the wrist flexors (Bodin et al., 1991) reproduced this reliability in stroke patients. Gregson and colleagues (2000) also studied stroke patients reported good and very good inter-rater reliability for the modified Ashworth with respect to the elbow, wrist and knee flexors (weighted kappa (kw) =.73-.96), but only moderate inter-rater agreement kw=.45-.51) for the ankle plantarflexors. Allison et al. (1996) observe an inter-rater reliability for the plantarflexors of r=.73 in 30 adults with traumatic brain injury (TBI). In individuals with SCI, the MAS has only been assessed in the lower extremity. Haas et al. (1996) found an extremely broad range of k= 0.21-0.61 for hip flexors, extensors, adductors and ankle plantar flexors, while Craven et al. (2009) reported ranges between k=.22 -.60 for MAS inter-rater reliability.

Several authors suggest that the Ashworth scales are less reliable for the muscles of the leg (Sloan et al., 1992, Haas et al., 1996). This is thought to be related to difficulty experienced by the examiners in supporting larger limbs due to their mass (Pandyan et al., 1999) and would be problematic when drawing conclusions of global

spasticity encompassing both the upper and lower extremity scores (Lee et al 1989, Gregson 2000). By contrast, Nuyens et al (1994) concluded that in patients with multiple sclerosis, the inter-rater reliability of the Ashworth scale was better for distal muscle groups than for the proximal muscle groups. It has been postulated that the shorter lever arm and relatively small range of movement about the ankle limits the rater's ability to distinguish between grades (Allison et al., 1996), however they may still be able to perceive a distinction of normal versus abnormal tone (Gregson et al., 2000).

Intra-rater reliability is the extent to which the same rater would consistently assign the same value. Craven and colleagues (2009) reported good intrarater reliability of the MAS (k range=.60 -1.0) in individuals with SCI. Gregson et al. (2000) examined intra-rater reliability of the modified Ashworth scale, finding strong correlation (kw=.77-.94) reliability for the elbow, wrist, and knee flexors. Values were lower for the ankle plantarflexors ranging from kw=..59-.64. Allison et al. (1996) achieved values of r=.55-.74 for ankle plantarflexor intra-rater reliability. The limited reliability of the measurement of the ankle plantarflexors is consistent with previous work using the Ashworth (Haas et al 1996) and modified Ashworth (Sloan et al., 1992) scales. Mean kappa values between the Ashworth and the modified Ashworth scales of k=.37 (Haas et al., 1996) and differences in the reliability of different muscle groups and from one side to the other have lead some to suggest that an inappropriate level of confidence has been placed on these scales and to call for abandonment of the MAS from the rehabilitation science community (Craven et al., 2009). At minimum, these uncertainties should encourage investigators to proceed with caution when applying these scales in research (Haas et al., 1996, Pandyan et al., 1999, Craven et al., 2009).

Previous work in this area is also significantly flawed as they made the mistake of summing the individual scores (Nuyens et al., 1994, Lechner et al., 2006). Single Ashworth scores cannot be summated because they provide only an ordinal level of

measurement (Pandyan et al., 1999). Moreover, these scores represent a stand-alone measure of tone, and summated scores will serve mask unreliability. This is relevant to clinical intervention studies, as unreliability alone may account for differences in pre- and post values, and be wrongly attributed to an intervention effect. Methodology (Lee et al., 1989) and choice of statistical analysis (Haas et al., 1996) have also been questioned (Gregson et al., 2000). Development and implementation of standardized guidelines for administration could contribute to the consistency of the scales (Hseih et al., 2008).

Recently, a group of researchers have created a new version of the modified Ashworth. The Modified modified Ashworth Scale (MMAS) omits the ambiguous category '+1" and redefines the grade '2' (Ansari et al., 2006). In patients with stroke and multiple sclerosis, excellent interrater agreement was established for the hip adductors and knee extensors (kw =0.82, p< .0001) and good agreement was found for the ankle plantarflexor (kw =0.74, p< .0001) (Ghotbi et al., 2009). Interestingly, the raters in this study were both physical therapy students who had minimal experience treating patients with spasticity, and no practice sessions were offered. If the MMAS is reliable, this agreement between students suggests that even the most novel examiners may be appropriate. The validity of this scale must be explored before it may be used for clinical and research purposes.

1.4.2.2 Validity

Validity can be defined as the ability of scale (or system) to measure accurately whatever it is intending to measure (Wilkin et al., 1992). It must be investigated with reference to: (1) the theoretical basis and underlying assumptions upon which the scale is designed (construct and content validity), and (2) comparison of the relationship

between the scale in question and previously developed measured (criterion validity-concurrent or predictive).

1.4.2.2.1 Construct and Content Validity

Both the construct and the content validity of the Ashworth and modified Ashworth have been challenged because they attribute any changes in passive resistance exclusively to spasticity. This overlooks the contributions of viscoelastic soft tissues surrounding the joints that may alter with prolonged immobilization (Pandyan et al., 1999, Ivanhoe et al., 2004). Moreover, alpha motor neuron activity will be influenced by changes in reflex excitability and compromised proprioception which are often affected by extraneous factors (i.e. temperature, noxious stimuli, etc) (Rhymer et al., 1994). The Ashworth scales are unable to distinguish between central and peripheral mechanisms of spasticity (Ivanhoe et al., 2004, Haugh et al., 2006).

Furthermore, the Ashworth and modified Ashworth scales are vulnerable to subjective interpretation by the examiners as they do not quantify passive resistance in absolute. Examiners may thus draw biased conclusions about the findings and their implications (Pandyan et al., 1999). There may be a further problem with the additional level of measurement in the MAS. There is no clinical definition for the reflex phenomena called ‘catch and release’. By attempting to increase the sensitivity of the scale, the 1+ category also increases the probability of errors, and reduces the scale to a nominal category, which creates statistical analysis challenges (Pandyan et al., 1999, Ivanhoe et al., 2004). Scales based on physical movement rely on the assumption that the linear velocity and range of motion regulated by the examiner are the same between trials (Pandyan 1999), which in reality is not perfectly reproducible.

1.4.4.2.2 Criterion Validity

There is some evidence to support criterion validity of the Ashworth Scales. Several studies have attempted to investigate its predictive criterion validity. Parise et al. (1997) found no relationship between the Ashworth score of spasticity and the presence of early or late biceps femoris flexion reflexes measured with electromyography in individuals with neuromuscular disorders. Pandyan and colleagues (2001) developed a device to biomechanically measure passive resistance in the elbow and compared it to the modified Ashworth scale in a stroke population. They found a poor correlation (k=.37). However, both studies investigated individuals with differing conditions (those with a myriad of neuromuscular disorders and acute stroke, respectively) which may make them difficult to generalize with respect to SCI. Sköld et al (1998) demonstrated a range of moderate-to-good correlation (r=.32-.91) between simultaneous surface electromyography and modified Ashworth ratings of spastic muscle activity elicited by movement provocation. This study would have been stronger had the authors not summed the MAS scores and more clearly referenced the EMG parameters which are necessary for comparison.

Correlations among clinical scales also remain equivocal. Priebe and colleagues (1996) compared several clinical scales in individuals with SCI. Employing an estimated correlation technique for ordinal categories assuming normality, the Ashworth score correlated modestly with patellar tendon taps (.553). Both ankle clonus with Achilles tendon tap (.663) and patellar tendon tap with adductor tendon tap demonstrated poor correlations (Priebe et al., 1996). Benz and colleagues (2005) found significant correlations between the Spinal Cord Assessment Tool for Spastic reflexes (SCATS) extensor spasms and the Ashworth scores for hip and knee flexors and for weak correlations for the ankle plantarflexors (ρ=.98, .88, .61 respectively), however, the

SCATS has yet to obtain widespread use within the research community. Several studies have demonstrated that reductions in spasticity as measured clinically are not necessarily correlated with improvements in function (Lechner et al., 2006, Adams et al., 2007). Finally, multijoint flexor and extensor spasms, which are prevalent in SCI, are not accounted for in the Ashworth Scale and MAS (Benz et al., 2005).

More recently, Sköld et al. (1999) reported a relationship between self-reported spasticity and impaired range of motion for hip abduction and flexion bilaterally. They recommend self-reporting of spasticity with regular intervals over several consecutive days. Repeated modified Ashworth ratings would also be beneficial in determining the character of the individual experience of spasticity. Using a Spasm severity scale (SSS) Lechner and colleagues (2006) compared 'present spasticity' ($SSS_{present}$, that experienced during testing) with 'general spasticity' ($SSS_{general,}$ that ordinarily experienced throughout the day). They found a good correlation (r=.70) between the Ashworth Scale and $SSS_{present}$, but a weak correlation of r=.36 between the Ashworth Scale and $SSS_{general}$. This suggests that while Ashworth scores may offer insight into the patient's perceptions of their spasticity at the time of testing, they cannot be used as a measure to reflect the patient's generally perceived spasticity. A correlational analysis of $SSS_{present}$ and $SSS_{general}$ would have enabled further discernment.

Only Priebe and colleagues (1996) have drawn comparisons between self-report measures of spasticity. A sample of veterans with SCI showed weak correlations between Penn Spasm Frequency Scale and self-report scales of interference with function (estimated polychoric correlation =.407) and pain (estimated polychoric correlation= .312).

Currently, the majority of measures of spasticity provide instantaneous representations of the condition. Moreover, they tend to represent spasticity as a hindrance, or to have no effect, however, several authors have commented on its

potential beneficial application (Sommers et al., 1992, St George, et al., 1993, Kirschblum, 1999). Additional research is essential to understand the specific impact of spasticity in functional limitation in the SCI population (Hsieh et al., 2008).

1.4.3 The Spinal Cord Injury-Spasticity Evaluation Tool

A promising new instrument is the Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET) developed by Adams et al. (2007). As spasticity can be both helpful and harmful to the individual, the authors also aimed to provide a broader scope to spasticity in SCI which would capture its' bidirectional nature. While it is common to ask respondents to rate positive and negative changes to overall health (Shields 2005), this has yet to be done with respect to spasticity.

The SCI-SET is a 35-item, 7-day recall questionnaire that targets aspects of daily life relevant to the SCI population which allows respondents to rate the impact of their spasticity. Responses can 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 SCI-SET asks questions pertaining to activities of daily living (showering, eating, dressing), emotional health (feelings of embarrassment, being annoyed), independence (control over your body, concern of falling, need to ask for help) and social activities (hobbies, recreation, sex 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’”. A copy of the SCI-SET questionnaire can be found in Appendix I.

1.4.3.1 Scoring

The SCI-SET provides a total item score and an average score. The total item score is calculated by adding together all applicable scores (positive and negative), which retain their signs in order to be representative of the global impact of spasticity. The average score is calculated dividing the total score by the number of applicable items (N/A scores are not included in the denominator). This provides a measure of the overall experiential impact of spasticity. A positive score indicates the individual perceives their spasticity as beneficial, while a negative score would suggest they find it detrimental. A score of zero would indicate that they do not perceive their spasticity to have an impact.

1.4.3.2 Metric Properties

To test the validity and reliability, Adams et al. (2007) interviewed 61 participants (n=8-in person, n=53-by telephone) with chronic SCI and “stable” spasticity. The SCI-SET showed moderate correlations with measures of self-assessed spasticity impact (r= -.61), self-assessed spasticity severity (r= -.48), Quality of Life Index- SCI Version III health and functioning subscale, or QLI, (r= .68), and Penn Spasm Frequency Scale (PSFS) (r= -.66). It correlated weakly with the motor subscale of the Functional Independence Measure motor subscale (FIM) (r=.21).The SCI-SET has not yet been validated or compared with any non-self report measures of spasticity such as biomechanical measures (EMG) or clinical assessment tools (MAS).

1.4.4 Measurement Summary

Although quantification of spasticity by means of biomechanical and electrophysiological methods is available, this remains undesirable due to lack of standardization and questionable relevance (Lunenberger et al., 2005). Some authors dispute the notion that spasticity can even be measured clinically other than to simply state whether it is present of not (Wade et al., 1985) and others affirm that it is easy to recognize but difficult to quantify in these settings (McLellan, 1983). Despite its metric shortcomings, the MAS has been utilized frequently in research involving individuals with various neuromuscular conditions (brain injury, multiple sclerosis, stroke and SCI), enabling comparison with previous work. It provides a quantitative, objective method of tracking functional change, and is the most commonly used clinical measure of tone, and thus enables translation of results to relevant clinical settings.

An estimation of the impact and severity of spasticity based on one evaluation measure will likely under represent the magnitude and severity of spasticity in the SCI population (Priebe et al., 1996). Due to the variable nature of spasticity, true measurement of spasticity requires a series of assessment tools in order to present an accurate, legitimate and comprehensive picture of individual experiences (Priebe et al., 1996, Lechner et al., 2006, Adams et al., 2007, Hsieh et al., 2008). The SCI-SET may provide a useful interpretation of the bi-directional nature of spasticity, however it has yet to be validated clinically.

1.5 Other complications of SCI

1.5.1 Pain

Chronic pain remains a significant problem for many individuals with spinal cord injury (SCI). A recent review of the literature found that prevalence rates for SCI-related pain ranged between 48 and 94% of the SCI population, depending on population characteristics (acute, chronic) and measurement factors, such as intensity and daily interference (Sawatzky et al. 2008). As many as 40% of these individuals rate their pain as severe in nature (Sjolund 2002). Singh and colleagues (2000) estimate that in as many as 25% of patients with SCI, there is sufficient pain to interfere with normal functioning. Often pain is present from the time of injury onwards, however in some individuals it can commence more than a decade subsequent to the time of injury. (Bloch 1986). Regardless of population frequency, several authors (Turner et al., 1999, Cardenas et al., 2006, Sawatzky et al., 2008) have cited the refractory nature of SCI-related pain in terms of its chronicity, interference with functioning, and resistance to medical interventions.

The International Association for the Study of Pain Taskforce on Spinal Cord Injury classifies pain based on three tiers; the first tier divides pain into nociceptive or neuropathic pain. Nociceptive pain is then divided into musculoskeletal or visceral, and neuropathic pain is divided into above injury level (with respect to the life situation of the individual), at injury level (located in a segmental pattern at the level of the injury) and below injury level (located diffusely below the level of injury).The final tier then indicates the specific pathology and all structures speculated to be involved (Siddall et al., 2000).

Table 1.4 IASP Tiers of SCI Pain

Tier 1	Tier 2	Tier 3
Nociceptive	Musculo-skeletal Visceral	Presumed mechanism (structure, pathology)
Neuropathic	Pain Level (at, above, below)	

Individuals with an incomplete SCI often experience musculoskeletal and neuropathic pain above, at or below the level of the injury (Sjolund, 2002, Cardenas et al., 2006). A longitudinal study that followed 100 newly injured patients found that musculoskeletal pain patterns and at level neuropathic pain were more prevalent early on in the injury. Conversely, below level neuropathic pain was the most common pain condition 3-6 months after injury (Siddall et al., 1999). 64% of respondents to a mail out survey reported below level pain (Turner 1999) and another study found that more than one-third of the below level SCI pain began one-year post-injury, and 30% more than 10 years subsequent to injury (Stormer et al., 1997).

Interventions for musculoskeletal pain generally focus on alleviation of underlying pathology or aggravating conditions such as poor posture or overuse. Management for chronic pain often incorporates physical therapy and exercise, relaxation training, psychotherapy, and dietary improvement (Siddall et al., 1999).

Oral pharmacological agents including antidepressants, anticonvulsants, and analgesics are often used to treat chronic SCI-related pain. Historically, antidepressants have been considered first-line drugs for neuropathic pain after SCI; however the evidence for the efficacy of antidepressants for treating chronic pain in SCI is lacking (Cardenas et al., 2005). Anticonvulsants, particularly gabapentin (GABA), are increasingly employed for neuropathic pain. Levendoglu et al. (2004) performed a randomized controlled trial using GABA and a placebo, finding GABA to be more successful than placebo in treating neuropathic pain in individuals with complete

paraplegia. Recently, intrathecal infusion of opiates mixed with local anesthetics has been advocated, though the results of limited available research remain ambiguous (Sjolund, 2002). Narcotics for pain may be addictive, often have side effects, and serve to mask the pain, rather than treat the origin of it (Turner et al., 1999) .Cardenas et al. (2005) suggest that alternative therapies should be explored as additional treatment options in this population.

1.5.2 Fatigue

While no universal definition of fatigue exists, with respect to neuromuscular loss of function, fatigue has been described as difficulty in initiating or sustaining voluntary activities (Chaudhuri et al., 2004). It is often characterized as a mismatch between the energy required to perform routine tasks and the energy available to do so (Hammell et al., 2009). Approximately 60% of individuals with SCI report fatigue of sufficient severity to interfere with function (Fawkes-Kirby et al., 2008). Barat et al. (2006) identified two kinds of fatigue in SCI: 'muscular fatigue', characterized as a physiological phenomenon of paralyzed muscles; and 'chronic fatigue' associated with 'aging, physiological and psychological deconditioning' contributing to decreased quality of life. Fatigue further exacerbates the physical consequences of SCI by compromising the ability to partake in life activities (Hammell et al., 2009). An inherent psychological aspect may also be involved, as feelings of lassitude may be present with no obvious loss of muscle force production or objective measure of fatigue (Anton et al., 2008). Additional dimensions of cognitive (sense of being overwhelmed, coping), emotional (frustration, guilt regarding partner's involvement, depression) and physical (tension, stress) fatigue have been identified (Hammell et al., 2009). Environmental factors such as physical barriers and a lack of wheelchair access, weather and the expectations that arise from a sociocultural

context that values independence may also contribute to feelings of fatigue (Hammell et al., 2009).

Declines in muscular force production abilities, and early fatigue onset are detrimental to maintaining an active lifestyle in SCI patients. As activities of daily living are more challenging to perform, this then limits the development of cardiopulmonary fitness. A sedentary lifestyle is thought to exacerbate this situation, leading to disuse atrophy and decreased aerobic capabilities (Glaser et al., 1989, Hoffman, 1986). Moreover, various secondary medical complications that can increase suffering and augment financial requirements for medical care are often prevalent in this debilitative cycle (Nichols et al., 1979, Brenes et al., 1986). A longitudinal study of physical capacity and physical strain during activities of daily living conducted by Janssen et al. (1996) determined that even small changes in maximal power output capacity could be associated with drastic changes in functional capacity in men with SCI. Of those with low capacity (15-40W), an improvement of 5-10 W could result in total independence during activities of daily living.

Studies on wheelchair users with SCI indicate that those who maintain a more active lifestyle by regularly participating in physical activity programs can increase their muscular strength, VO2 max, and physical performance to well exceed levels of their inactive peers (Glaser et al.,1989, 1996, Hoffman et al.,1986). Petajan (1998) has suggested that improving aerobic fitness and muscle strength can enhance exercise tolerance and reduce fatigue in spinal cord injury. In addition to these fitness gains, habitual physical activity may lead to improvements in health, psychosocial condition, rehabilitation potential, functional independence and quality of life (Hjeltnes et al., 1990, Noreau et al., 1992, Noreau et al., 1995).

1.5.3 Other Impacts (Depression)

There have been conflicting findings regarding the relationship between SCI and depression. Dysphoria and depression have often been anticipated following spinal cord injuries; however, findings remain ambiguous (Elliot et al., 1996). Judd (1991) reports that 20 percent of SCI patients score within the 'depressed' range on the Beck Depression Inventory, but these scores showed improvement over the course of time. Cushman et al. (1991) examined the prevalence of self-reported depressed mood in a population of newly spinal cord injured patients. Their results showed similar rankings to an able-bodied population.

Measures of life satisfaction in people with SCI have a tendency to be lower, on average, than for the general population (Ditunno et al., 1994); however, work by Carpenter et al. (2007) suggests that according to the Satisfaction with Life Scale and Happiness Scale, individuals with SCI were generally happy and satisfied with life. In a study of chronic spinal cord injured community residents, the occurrence of depressive symptoms was higher than for the able-bodied residents (Fuhrer et al., 1993). Greenberg and Good (1998) suggest that feelings of dysphoria are more likely to be affiliated with perceptions of reduced opportunity, rather than the physical measures of the extent of the disability. They consider this to be situational in nature, and suggest it can be combated with improvements in social and community integration.

Approximately 95 percent of people with SCI are able to return to the community, either to a personal or assisted living residence (Ditunno et al., 1994) and this is considered to be a positive determinant of successful adaptation. The majority of individuals who have been injured for many years rated their quality of life as "good" or "excellent" (Whiteneck et al.,1992). Variables associated with improved mood after 15 years of injury were greater tolerance for sitting, more years of education and greater

satisfaction with finances and employment (Krause, 1992). Further examination of the impact of spinal cord injury on mood is required.

1.5.4 The Role of Physical Activity

Physical activity is often associated with decreases in pain, fatigue and depression in individuals with SCI (Tawashy et al., 2009). Habitual exercise for this population is thought to minimize joint deterioration and incipient neurological deficits that appear over time (Jacobs et al., 2004). While heavily encouraged, exercise programs face specific challenges and limitations compared to programs for those without disability. Acute exercise and training responses are less robust than for those without SCI (Davis et al., 1993, Thomas et al., 1997), and carry significantly greater risks of lasting negative effects or injury (Hartkopp et al., 1998, Jacobs et al., 2001). Exercise performance after SCI is plagued with circulatory dysregulation (significantly lower resting stroke volume and higher resting heart rates) and insufficiency (diminished venous return and cardiac end-diastolic volumes) (Jacobs et al., 2004). Autonomic dysreflexia involves increases in blood pressure mediated via sympathetic vasoconstriction elicited by noxious (Burton et al., 2008) and non-noxious (Marsh et al., 2004, Burton et al., 2008) stimuli. This results in fluctuations in heart rate and cardiac output that may not be proportional to or indicative of a response to exercise. Higher resting catecholamine (stress hormone) levels and exaggerated catecholamine responses to physical work have been seen in individuals with mid-thoracic (T5) cord injuries (Schmid et al., 1998).

In addition, activities of daily living are understandably prioritized. Due to the high energy cost of many of these activities for people with SCI (van der Woude et al., 1997, Hammell et al., 2009), this often results in limited energy and motivation for additional

physical activity. The psychological consequences and ensuing social isolation are well documented (Hammell et al., 1994).

Exercise programs involving individuals with SCI must be mindful of the additional physiologic considerations for this population. They must also respect the energy demands of daily living while encouraging safety and independence.

1.6 Mobility

1.6.1 SCI and Upright Standing

While a great deal of anecdotal evidence supports the notion that standing is beneficial for individuals with SCI, little in the way of empirical evidence exists (Kunkel et al., 1993) and the majority of studies examining the physiological benefits of standing thus far are plagued by small sample sizes (Kunkel et al., 1993, Bohannon, 1993).

In addition to improvements in digestion, breathing, and circulation (reduced swelling in the legs and feet) from being vertical, several authors have posited benefits regarding sleep (Dunn et al., 1998, Eng et al., 2001), bladder and bowel function (Leo 1983, Cybulski et al., 1986, Dunn et al., 1998, Eng et al., 2001, Shields et al., 2005), decreased calcium in the urine (Issekutz et al., 1966, Kaplan et al., 1981), increased bone density (Goemaere et al., 1994), decreased muscle spasms (Duffus & Wood, 1983, Little et al., 1988) and improved skin integrity (Cybulski et al., 1986, Eng et al., 2001),

Upright standing supported in a frame allows body weight to be transmitted through the bones and joints of the paretic lower extremities. It is thought that such intervention might be a means of controlling spasticity by increasing muscle activity and

stretch, and anecdotal reports of improvements are common (Eng et al., 2001). Odeen et al. (1981) examined the effects of passive standing and whole muscle elongation on spasticity in paraplegics (n=9). Following 30 minutes of standing with the feet in a dorsiflexed position, they found a 30% decrease in resistance to passive stretch, as compared to a 17% reduction produced by a similar stretch which was administered in the supine position. Kunkel et al. (1993) examined the effect of standing in a frame on muscle tone (assessing passive resistance) in six paralyzed males (mean age 49 years). They found no difference in pre and post- six month program measures. However, in addition to their small sample size, the cohort they analyzed was a mixture of men with SCI and multiple sclerosis. Therefore it is not surprising that half were identified as increased tone/hyperreflexive/conic, while the other half were had decreased tone/hypo- or areflexive and absent of tone. Additionally, the authors fail to mention when during the study tone was assessed. In light of the variable cohort and nature of spasticity, it is difficult to apply these clinical findings.

Subjective interpretations of the benefits of standing are much easier to come by, and self-evaluations have yielded primarily positive outcomes (Kunkel et al., 1993, Dunn et al., 1998). Kunkel et al. (1993) reported a psychologically beneficial effect of upright standing. Although it had a marginal influence on appetite, sleep and relaxation, subjects reported feeling healthier during the study, and suggested they would recommend it to other individuals with paralysis. Also, the majority of their subjects continued a regular standing program eight weeks after completing the study. Dunn and colleagues (1998) investigated subject's perceptions of leg spasticity with use of a standing device. While they did not define spasticity (i.e. how the individuals graded their own spasticity) in their questionnaire, they suggested that their subjects were responding to either leg spasms or resistance to leg movement. Their results showed that 42% of subjects reported a decrease in leg spasticity with use of a standing device. They also found a significant

relationship between this response and the amount of time spent upright in the device. Additionally, 38% of their subjects reported an increased ability to straighten their legs with use of their standing device. Using a case-study approach, Shields et al. (2005) reported that their participant was consistently 'very satisfied' with his standing device, and would highly recommend that people with SCI stand as long as they are able. He further believed his standing program (described in the previous section) had a beneficial effect on his lower extremity spasticity. To this end, he frequently and intentionally performed his standing program before tasks such as dressing and showering, which would present significantly greater challenges, should spasms occur. He also stated that on the days he did no standing, his spasms were more frequent and more bothersome.

Results of a survey of 38 individuals with SCI who engaged in prolonged standing on average 40 minutes 3 to 4 times per week are less straightforward (Eng et al., 2001). While some subjects reported reduced muscle spasms with passive standing (n=9), others reported increased spasticity (n=5). Increases in pain (n=17) and fatigue (n=14) were also reported. These contradictions should serve to remind researchers of the individually-specific nature of spasticity, and to exercise caution regarding positive assumptions when implementing programs often perceived as beneficial.

1.6.2 Current Options in Mobility

1.6.2.1 Orthoses, Crutches and Wheelchairs

Several options exist for mobility in individuals with spinal cord injury. These are determined not only by the individual's level of neurological function and physical

dimensions, but must also account for their goals in life and the surrounding environment (Ozer, 1988).

Braces are often employed to compensate for weak muscles to create a stable base of support. These orthoses can take the form of hip-knee-ankle-foot orthosis (HKAFO) which consists of a trunk section connected to two knee-ankle-foot orthoses (KAFOs) by two lateral hip joints. Movement with an HKAFO is achieved by a swing through or hopping type gait with the assistance of either a walker or forearm crutches with both legs moving together as one unit. KAFOs provide knee stability for those individuals with intact hip flexors and proprioception at the hip, and ankle-foot orthosis (AFOs) for individuals with preserved proprioception and extension of the knee. Braces provide individuals with SCI the opportunity to stand and move with crutches, however the time associated with donning and doffing them may limit their appeal among users (Shields et al., 2005). Arm crutches are often employed to improve stability and enhance the base of support. In general, a manual wheelchair is required for long distances.

Most persons with a functional level of C6 or lower use some form of a manual wheelchair, while those with injuries to the C5 or above will require a power chair (Ozer, 1988). The appropriate design of the wheelchair is paramount, and should reflect the needs of the individual. The development of assistive technology devices has been a leading factor in the increased independence of persons with disabilities (DeRuyter, 1995). Greater recognition of the need for proper positioning has led to an increase and improvement in commercially available equipment. In addition, technological advancements in chair design and materials have facilitated greater personalization in the form of additional features, such as those for sporting events, and varying mobility ranges. Manufacturers now offer a variety of solid backs and seat, as well as arm rests, trunk supports, and cushions. Lightweight materials such as titanium, Kevlar and composites have reduced the energy requirements for operation of equipment (Cooper

et al., 2006). Unfortunately, funding for wheelchairs continues to be an ongoing concern, and cost is often a priority when design and features are considered.

1.6.2.2 Standing Wheelchairs

The use of a standing wheelchair has been argued to have additional benefits to seated chairs. An upright posture has been shown to improve personal interactions, build self-esteem and self-image, and improve morale (Cybulski et al., 1986, Eng et al., 2001, Sawatzky et al., 2007). This is profoundly demonstrated through comments such as 'It feels so wonderful to get vertical," (Eng et al., 2001).

Regardless of the advocated benefits, standing wheelchairs for individuals with SCI are not widely prescribed (Dunn et al., 1998). Concern regarding complications such as leg fractures, foot ulcers and fainting have been cited as potential reasons for this, however few accounts pertaining to these potential detriments have been reported by users of standing wheelchairs (Dunn et al., 1998). Additionally, the excessive weight and front leg rests (angled at approximately 70º for safety) have limited the maneuverability and possibly deter their widespread use (Shields et al., 2005). Shields et al. (2005) reported high compliance to a standing wheelchair in their 2-year case study of a 25 year old male with T10 complete paraplegia who stood for short bouts (mean=11.57 minutes) 3.86 days per week. While he perceived positive physiologic benefits (improvements in bowel motility and decreased spasticity), the participant commented that the standing chair, with its lack of suspension or wheel camber, was heavy and difficult to maneuver.

Participating in a regular standing program is difficult for individuals with higher injury levels (Eng et al., 2001). Those with tetraplegia may have greater physical or medical barriers (e.g. assistance required for transfers into and out of a standing frame)

that lead to more sedentary lifestyles. Unfortunately, those with higher spinal cord lesions are more prone to complications such as urinary tract infections, spasticity and contractures (Maynard et al., 1990, Eng et al., 2001). Researchers advocate the need to address this group's special requirements for a standing device that is physically less demanding to use (Eng et al., 2001).

1.6.2.3 Exercise Guidelines for SCI

Training recommendations for individuals with paraplegia involve three to five weekly exercise sessions of 20–60 minutes in duration at an intensity of 50-80%$VO2_{peak}$ (ACSM, 2009). Previous work regarding upright standing tolerance in the SCI population suggests a myriad of tolerance, ranging from 20 to 45 minutes (Kunkel et al., 1993, Bohannon, 1993, Ragnarsson et al., 1981, Kaplan et al., 1981, Odeen et al., 1981). Eng et al. (2001) found that survey respondents reported engaging in an average of 40 minutes per session, 3 times a week using either a standing frame or a combination of braces with an assistive device such as a walker as a method to improve and maintain their health. They therefore defined their standing program as a minimum of 20 minutes per day. Dunn et al.(1998) also conducted a study of 100 individuals with SCI. Thirty nine percent of their subjects spent 15 minutes or less per day upright, 33% used the device between 15 and 45 minutes per day, 15% used it 45 minutes to 1.5 hours per day and only 8% used it greater than 1.5 hours per day. Significant correlation exists between lower levels of injury and increased amount of upright time (Dunn et al., 1998, Eng et al., 2001). In a case study of a 25-year old male with a T10 ASIA A classification who used a standing wheelchair for a period of 2 years, Shields et al. (2005) reported that the client chose to stand for short bouts (mean =11.57 min) at an average of 3.9 days per calendar week. Reported benefits from prolonged standing occurred relatively

quickly (within 1 week), but were somewhat temporary, lasting only one day (Eng et al., 2001). However, this amount of standing activity has been reported to be sufficient in achieving some of the benefits previously documented in the literature such as reduced reflex activity and improved well-being (Odeen et al., 1981, Bohannon, 1993, Kaplan, et al., 1981). Recently, Ness et al. (2009) had subjects with chronic, motor-incomplete SCI complete a 12-session (3days/week for 4 weeks) intervention of upright whole body vibration.

1.6.3 The Segway Personal Transporter

The Segway® Personal Transporter (PT) is an electric-powered, self-balancing mobility device which enables the user to negotiate variable terrain. It was developed in 2001, and is currently in operation within airports, government and law enforcement institutions and by private citizens. A small platform supported by 2 parallel wheels 20cm above the ground supports a standing rider. A flat handlebar with a steering device in the form of a twist grip system allows for easy maneuvering. A closed loop dynamic stability control system called LeanSteer™ technology is comprised of a build-in gyroscope which senses the rider's center of gravity. This 'inner-ear like' balancing agent is the key to the Segway and is accomplished with the use of five special gyroscopes called sold-state angular rate sensors. Only three are really needed to detect forward, backward and side-to-side motion, but the extra two add stability and reliability to the Segway. This allows stationary upright balancing, as well as forward motion when the rider assumes a forward lean position. Likewise leaning back will cause the device to stop or move backward. Velocity of the Segway is determined by the angle of forward or backward lean, such that greater lean corresponds to higher speeds (www.segway.com).

The Segway can travel up to 12.5mph/20kph, and as far as 38km on a single battery charge (www.segway.com). Its' capabilities navigating uneven terrain may help to make it a more attractive alternative to a standard wheelchair. At a cost of $6 000, it may be a more feasible device alternative for many prospective consumers.

Only two previous studies have looked at the effect of Segway use in people with disabilities. Sawatzky and colleagues (2007) attempted to correlate several functional outcome measures such as strength, range of motion, and balance with the ability to use a Segway to help clinicians determine functional ability levels needed to operate a Segway safely. There was no correlation between functional scores and Segway skill in that all subjects who entered the study were able to operate the Segway. With the aid of braces, even individuals with complete spinal cord injuries (paraplegia) were able to use the Segway, suggesting that strength and dexterity requirements are minimal. Psychosocial benefits were also reported. Participants felt that the Segway improved their independence and helped to minimize their disability to others, and in so doing increased their feelings of self-esteem.

In a second study, Sawatzky et al. (2009) compared the subjects' current mobility aids to the Segway, examining what goal might the Segway fulfill for people with disabilities and how well it would achieve that goal. Using the Wheelchair Outcome Measure (WhOM), subject's rated the Segway significantly higher than their current mobility device(s), suggesting that they found the Segway more appropriate for mobility. Psychosocial benefits were again reported, with participants feeling 'less disabled' while using the Segway. Several anecdotal accounts from subjects pertained to improvements in balance and reductions in spasticity immediately following Segway training. One subject who had severe hypertonicity in his left hand, and required assistance to open the hand in order to hold the Segway handle, could open and close it easily and independently after only 15 minutes on the Segway. In fact, most of the participants in

the study commented that they were able to use the Segway for longer periods than were able to typically stand or experienced less pain immediately following their time on the Segway. Thus, the researchers postulated that perhaps consistent use of the Segway could also increase this stamina over time. The question then remains, do participants glean physiological benefits from Segway training and if so, are these changes measurable and significant?

Given the significant compromises in balance that are often experienced by individuals with SCI (Cybulski et al., 1986), they may require cumbersome and expensive mobility equipment for longer distances. As the Segway technology is 'self-balancing', could individuals with SCI benefit not only from the upright standing discussed earlier, but also experience decreases in secondary complications such as spasticity, pain and fatigue without the familiar compromise of energy expenditure? And, as a Segway retails within the range of $ 6000 USD, could this be a more economical mobility alternative for this population?

1.7.Summary

From the literature we see that individuals with SCI deal with a considerable array of complications, such as spasticity and pain, which can be difficult to manage. Given the implications of pharmacologic side effects and invasive surgical procedures, most clinicians and patients prefer conservative management programs if possible (Kirshblum, 1999). However, in order to track these management programs, clinicians must understand their impacts on the individual. Current methods of clinical measurement focusing on examiner-based representation may be biased. A more patient-centered approach that relies on self-evaluation should be considered as relevant, if not of great import in determining therapeutic venues.

Issues of adequate mobility are also problematic for these individuals. New technologies such as the Segway PT offer an alternative mode of transportation for individuals with mobility impairment. Anecdotal evidence suggests that there may be a link between the Segway and improved standing tolerance, as well as declines in spasticity and pain. Preliminary research is needed to confirm whether these effects are measureable and significant.

The purpose of this thesis was to examine the effect of using the Segway as a possible method for reducing spasticity in individuals with SCI. An additional goal was to examine a global self-report measure of spasticity in comparison with a common clinical tool.

1.8 Research Questions

Several research questions were posed and two studies were conducted in order to address the purpose of the thesis:

1.8.1 Research Questions #1 (Chapter 2)

Do clinical measures of physical properties in spasticity correlate to patients' interpretations of its overall impact based on a recently validated self-report measure?

Hypothesis 1: There will be a correlation between clinical measure of spasticity as measured by the modified Ashworth Scale and self report using the Spinal Cord Injury Spasticity Evaluation Tool.

1.8.2 Research Questions #2 (Chapter 3)

What physiologic benefits such as reductions in spasticity, pain and fatigue can be derived from Segway training for individuals with SCI, and are these potential benefits short or long term in nature?

Hypothesis 1: There will be a short-term intervention effect of the Segway PT on reduction of spasticity in the indicated muscles as measured by the modified Ashworth Scale.

Hypothesis 2: There will be a longer term (one month) intervention effect of the Segway PT on reduction of spasticity, pain and fatigue as measured by self-report tools including the Spinal Cord Injury Spasticity Evaluation Tool, the Pain Outcomes Questionnaire-VA, the Fatigue Severity Scale and a Daily Log.

1.9 Significance

Should we find that a correlation exists between the current clinical 'gold standard' to measure spasticity (the modified Ashworth scale) and the self-report impact tool (SCI-SET), this will strengthen the evidence to support use of self-report measures in clinical settings. A single measure of spasticity may not encompass all the factors that are necessary to consider. Additionally, physical tests may not provide therapists with the appropriate understanding of impact upon which to establish clinical, home- and community-based programs. A single clinical snapshot of specific muscles does not

necessarily reflect the impact to the individual over an extended period of time (i.e. the week, month, etc).

Intervention studies have yet to encompass both clinical and self- report measures on both a daily and weekly basis. This will allow the investigators to draw comparisons regarding both instantaneous and longitudinal outcome measures which, until now have been lacking in the literature. The bidirectional nature of the recently validated SCI-SET tool has yet to be used in rehabilitation research, and this study will be the first to incorporate it into an intervention model.

Furthermore, should use of the Segway lead to measureable physiologic improvements, it may serve as an adjunct to current therapies, allowing individuals to reduce other time-consuming programs and direct their energies to other fulfilling ventures. It may also provide evidence-based support for extended health coverage of these devices, which are presently regarded skeptically by the insurance industry.

1.10 References

1. ACSM Exercise management for Persons with Chronic Diseases and Disabilities. 3rd ed. Durstin JL, Moore GE, Painter PL, Roberts SO (eds). Chapter 39, Figoni SF.Spinal Cord Disabilities: Paraplegia and Tetraplegia. pp.298-303. Human Kinetics: Champaign, IL.

2. Adams MM, Hicks AL. (2005). Spasticity after spinal cord injury. Spinal Cord. 43:577-86.

3. Adams MM, Martin Ginis KA, Hicks AL. (2007). The Spinal Cord Injury Spasticity Evaluation Tool: Development and Evaluation. Archives of Physical Medicine and Rehabilitation. 88:1185-1192.

4. Al-Khodairy AT, Gobelet C, Rossier AB. (1998). Has botulinum toxin type A a place in the treatment of spasticity in spinal cord injury patients? Spinal Cord. 36:854-858.

5. Allison SC, Abraham LD, Petersen CL. (1996). Reliability of the modified Ashworth Scale in the assessment of plantar flexor spasticity in patients with traumatic brain injury. International Journal of Rehabilitation Research. 10:67-78.

6. American Spinal Injury Association: Standards for Neurological Classification of Spinal Injury Patients. Revised April 1990.

7. Ansari NN, Naghdi S, Moammeri H, Jalaie S. (2006). Ashworth scales are unreliable for the assessment of muscle spasticity. Physiotherapy Theory and Practice. 22:119-125.

8. Anton HA, Miller WC, Townson AF. (2008). Measuring Fatigue in Persons with Spinal Cord Injury. Archives of Physical Medicine and Rehabilitation. 89:538-542.

9. Apparelyzed: Spinal Cord Injury Peer Support Network. /www.apparelyzed.com/index.html. Accessed Nov. 2, 2009.

10. Ashby P, Verrier M. (1975). Neurophysiological changes following spinal cord lesions in man. Canadian Journal of Neurological Science. 2:91-100

11. Ashworth B. (1964). Preliminary trial of carisoprodal in multiple sclerosis. Practitioner. 192:540-542.

12. Barat M, Dehail P, de Souze M. Fatigue after spinal cord injury. (2006).Annals de Readaptation et de Medicine Physique. 49: 365–369.

13. Barnes M. (2003). Botulinum toxin- mechanisms of action and clinical use in spasticity. Journal of Rehabilitation Medicine. 41:S56-59.

14. Benz EN, Hornby TG, Bode RK, Scheidt RA, Schmit BD. (2005). A physiologically based clinical measure for spastic reflexes in spinal cord injury. Archives of Physical Medicine and Rehabilitation. 86:52-59.

15. Bloch RF, Basbaum M. (1986). Management of Spinal Cord Injuries. Williams & Wilkins: Baltimore.

16. Bodin PG, Morris ME. (1991). Interrater reliability of the modified Ashworth scale for wrist flexor spasticity. Proceedings from the 11th Congress of the World Federation of Physical Therapy. London: (II): 505-507.

17. Bohannon RW, Smith MB. (1987). Inter rater reliability of a modified Ashworth Scale of muscle spasticity. Physical Therapy. 67: 206-207.

18. Bohannon RW. (1993). Tilt table standing for reduced spasticity after spinal cord injury. Archives of Physical Medicine and Rehabilitation. 74:1121-1122.

19. Bohlega S, Chaud P, Jacob PC(1995). Botulinum toxin A in the treatment of lower limb spasticity in hereditary spastic paraplegia. Movement Disorders; 10:399(Abstract).

20. Brashear A, Zafonte R, Corcoran M, Galvez-Jimenez N, Gracies JM, Gordon MF, McAfee A, Ruffing K, Thompson B, Williams M, Lee CH, Turkel C. (2002b). Inter- and intrarater reliability of the Ashworth Scale and the Disability Assessment Scale in patients with upper-limb poststroke spasticity. Archives of Physical Medicine and Rehabilitation. 83(10):1349-54.

21. Brenes G, Dearwater S, Shapera R, LaPorte RE, Collin E. (1986). High density lipoprotein cholesterol considerations in physically active and sedentary spinal cord injured patients. Archives of Physical Medicine and Rehabilitation. 67:445-450.

22. Burgar CG. (1994). Electrophysiologic identification of motor points during injection of botulinum toxin: a pilot study. Archives of Physical Medicine and Rehabilitation. 75(4):1031.

23. Burton AR, Brown R, Macefield VG.(2008). Selective activation of muscle and skin nociceptors does not trigger exaggerated sympathetic responses in spinal-injured subjects. Spinal Cord. 46:660-5.

24. Canadian Paraplegic Association (1997). Spinal Cord Injury Workforce Participation National Survey. Ottawa, ON, Canada.

25. Cardenas DD, Jensen MP. (2006) Treatments for Chronic Pain in Persons With Spinal Cord Injury: A Survey Study. Journal of Spinal Cord Medicine. 29:109-117.

26. Carpenter C, Forwell SJ, Jongbloed LD, Backman CL. (2007). Community Participation After Spinal Cord Injury. Archives of Physical Medicine and Rehabilitation. 88:427-33.

27Chaudhuri A, O'Behan P. (2004). Fatigue in neurologic disorders. Lancet. 363:978-988.

28. Collins C, Davies P. Mutiboko IK, Ratcliffe S. (2007). Randomized controlled trial of cannabis-based medicine in spasticity caused by multiple sclerosis. European Journal of Neurology. 14: 290–296

29. Cooper RA, Boninger ML, Spaeth DM, Ding D, Guo S, Koontz AM, Fitzgerald SG, Cooper R, Kelleher A, Collins DM. (2006). Engineering better wheelchairs to enhance community participation. IEEE Transactions on Neural Systems and Rehabilitation Engineering. 14:438-55.

30. Craven BC, Morris AR. (2009). Modified Ashworth scale reliability for measurement of lower extremity spasticity among patients with SCI. Spinal Cord. E-pub ahead of press, Accessed Sept. 29. 1-7

31. Cushman LA, Dijkers M. (1991). Depressed mood during rehabilitation of persons with spinal cord injury. Journal of Rehabilitation. 57:35-38.

32. Cybulski GR, Jaegar RJ. (1986). Standing performance of persons with paraplegia. Archives of Physical Medicine and Rehabilitation. 67:103-108.

33. Davis GM. (1993). Exercise capacity of individuals with paraplegia. Medicine and Science in Sports and Exercise. 25:423-432.

34. Decq P. (2003). Pathophysiology of Spasticity. Neurochirurgie. 49:163-184.

35. DeRuyter F. (1995). Evaluating outcomes in assistive technology: do we understand the commitment**?** Assistive Technology.7(1):3-8.

36. DeSouza LH, Musa IM. (1987). The measurement and assessment of spasticity. Clinical Rehabilitation. 1:89-96.

37. DeVivo MJ, Richards JS, Stover SL, Go BK. (1991). Spinal cord injury. Rehabilitation adds life to years. Western Journal of Medicine. 154:602-606.

38. Dietz V.(2000). Spastic movement disorder. Spinal Cord. 38:389-93.

39. Dietz V. (2001). Spinal cord lesion: effects of and perspectives for treatment. Neural Plasticity. 8:83-90.

40. Ditunno JF, Formal CS. (1994). Chronic spinal cord injury. New England Journal of Medicine. 330:550-556.

41. Duffus A, Wood J. (1983). Standing and walking for the T6 Paraplegic. Physiotherapy. 69:45-46.

42. Dunn M, Davis R. (1974) Perceived effects of marijuana on spinal cord injured males. Paraplegia. 12:175.

43. Dunn RB, Walter JS, Lucero Y, et al. (1998). Follow up assessment of standing mobility device users. Assistive Technology. 10: 84-93

44. Elliott TR, Frank RG. (1996). Depression following spinal cord injury. Archives of Physical Medicine and Rehabilitation. 77:816-823.

45. Elovic E. (2001). Principles of pharmaceutical management of spastic hypertonia. Physical Medicine and Rehabilitation Clinics of North America. 12:793-816.

46. Elovic EP, Simone LK, Zafonte R. (2004). Outcome assessment for spasticity management in the patient with traumatic brain injury. Journal of Head Trauma and Rehabilitation. 19:155-77.

47. Emery E. (2003). Intrathecal baclofen. Literature review of the results and complications. Neurochirurgie. 49:276-288.

48. Eng JJ, Levins SM, Townson AF, Mah-Jones D, Bremner J, and Huston G. (2001). Use of Prolonged Standing for Individuals with Spinal Cord Injury. Physical Therapy. 81:1392-1399.

49. Eng JJ, Miller WC. Rehabilitation: from bedside to community following spinal cord injury (SCI). Spinal Cord Injury Rehabilitation Evidence. (http://www.icord.org/scire/pdf/SCIRE_CH1.pdf). Accessed September 9, 2008.

50. Fawkes-Kirby TM, Wheeler MA, Anton HA, Miller WC, Townson AF, Weeks CAO. (2008). Clinical correlates of fatigue in spinal cord injury. Spinal Cord. 46: 21–25.

51. Filipetti P, Decq P. (2003). Interest of anesthetic blocks for assessment of the spastic patient. A series of 185 motor blocks. Neurochirurgie. 49:226-238.

52. Fosang AL, Galea MP, McCoy AT, Reddihough DS, Story I. (2003). Measures of muscle and joint performance in the lower limb of children with cerebral palsy. Developmental Medicine and Child Neurology. 45:664 – 670.

53. Fuhrer MJ, Rintala DH, Hart KA et al. (1993). Depressive symptomatology in persons with spinal cord injury who reside in the community. Archives of Physical Medicine and Rehabilitation. 74:255-260.

54. Gerhart KA. (1991). Spinal Cord Injury Outcomes in a Population-Based Sample. The Journal of Trauma. 31:1529-1535.

55. Ghotbi N, Ansari NN, Naghdi S, Hasson S, Jamshidpour B, Amri S. (2009). Inter-rater reliability of the Modified Modified Ashworth Scale in assessing lower limb muscle spasticity. Brain Injury. 23:815-819.

56. Glaser RM, Davis GM.Wheelchair-dependent individuals. In: Franklin BA, Gordon S, Timmins GC (eds). (1989). Exercise in modern medicine. Baltimore: Williams & Wilkins: 237-267..

57. Goemaere S, Van Laere M, De Neve P, Kaufman JM. (1994). Bone mineral status in paraplegic patients who do or do not perform standing. Osteoporosis International.4:138-43.

58. Goodrich JA, Riddle T. (2008). Lower Cervical Spine Fractures and Dislocations. E-medicine. http://emedicine.medscape.com/article/1264065-overview. Accessed December 17, 2009.

59. Gracies JM, Nance P, Elovic E, McGuire J, Simpson DM. (1997a). Traditional pharmacological treatments for spasticity. Part II: General and regional treatments [review]. (Muscle Nerve Suppl). 6:S92-120.

60. Gracies JM, Wilson L, Gandevia SC, Burke D. (1997b). Stretched position of spastic muscle aggravates their co-contraction in hemiplegic patients. Annals of Neurology. 42(30):438-439.

61. Gracies JM. (2004) Physiological Effects of Botulinum Toxin in Spasticity. Movement Disorders. 19:S120-128.

62. Greenberg JP, Good DC. (1998). Functional Assessment in Neurologic Disability, in Lazar RB (ed): Principles of Neurologic Rehabilitation. New York, McGraw-Hill.

63. Gregson JM, Leathly MJ, Moore PA, Smith TL, Sharma AK, Watkins CL. (2000). Reliability of measurements of muscle tone and muscle power in stroke patients. Age and Ageing. 29:223-228.

64. Gruenthal M, Mueller M, Olson WL, Priebe MM, Sherwood AM, Olson WH. (1997). Gabapentin for the treatment of spasticity in patients with spinal cord injury. Spinal Cord. 35:686-9.

65. Haas BM, Bergstrom E, Jamous A, Bennie A. (1996). The inter rater reliability of the original and of the modified Ashworth scale for assessment of spasticity in patients with spinal cord injury. Spinal Cord. 34:560-564.

66. Hammell KR. (1994). Psychosocial outcome following spinal cord injury. Paraplegia. 32:771-9.

67. Hammell KW, Miller WC, Forwell SJ, Forman BE, Jacobsen BA. (2009). Fatigue and spinal cord injury: a qualitative analysis. Spinal Cord. 47:44-49.

68. Hartkopp A, Murphy RJ, Mohr T, Kjaer M, Biering-Sorenson F. (1998). Bone fracture during electrical stimulation of the quadriceps in a spinal cord injured subjects. Archives of Physical Medicine and Rehabilitation. 79:1133-1136.

69. Haugh AB, Pandyan AD, Johnson GR. (2006). A systematic review of the Tardieu Scale for the measurement of spasticity. Disability and Rehabilitation. 28:889-907.

70. Hesse S, Brandl-Hesse B, Seidel U, Doll B, Gregoric M. (2000). Lower limb muscle activity in ambulatory children with cerebral palsy before and after the treatment of Botulinum toxin A. Restorative Neurology and Neuroscience. 17:1-8.

71. Hjeltnes N, Jansen T. (1990). Physical endurance capacity, functional status and medical complications in spinal cord injured subjects with long-standing lesions. Paraplegia. 28:428-432.

72. Hoffman MD. ((1986). Cardiorespiratory fitness and training in quadriplegics and paraplegics. Sports Medicine. 3:312-330.

73. Hsieh JTC, Wolfe DL, Connoly S et al. Spasticity following spinal cord injury. . Spinal Cord Injury Rehabilitation Evidence. (http://www.icord.org/scire/pdf/SCIRE_CH1.pdf). Accessed September 9, 2008.

74. Hsieh JTC, Wolfe DL, Miller WC, Curt A and the SCIRE Research Team. (2008). Spasticity outcome measures in spinal cord injury: psychometric properties and clinical utility. Spinal Cord. 46:86-95.

75. Ivanhoe CB, Reistetter TA. (2004). Spasticity: The Misunderstood Part of the Upper Motor Neuron Syndrome. American Journal of Physical Medicine and Rehabilitation. 83(Suppl):S3-S9.

76. Jacobs PJ and Nash MS. (2001). Modes, benefits and risks of voluntary and electrically induces exercise in persons with spinal cord injury. Journal of Spinal Cord Medicines. 24:10-18.

77. Jacobs PJ, Nash MS. (2004). Exercise Recommendations for Individuals with Spinal Cord Injury. Sports Medicine. 34:727-751.

78. Janssen TW, VanOers CAJM, Rosendale EP, Willemsen EM, Hollanders AP, Van der Woude LHV. (1996). Changes in physical strain and physical capacity in men with spinal cord injuries. Medicine and Science in Sports and Exercise. 28(5): 551-559.

79.Jozefczyk PB. (2002). The management of focal spasticity. Clinical Neuropharmacology. 25:158-173.

80. Judd FK, Brown DJ, Burrows GD. (1991). Depression, disease and disability: Application to patients with traumatic spinal cord injury. Paraplegia. 29:91-96.

81. Kandell ER, Schwartz JH, Jessell TM. (1995) Essentials of neural science and behaviour. New York: McGraw-Hill.

82. Kaplan PE, Roden W, Gilbert E, Richards L and Goldschmidt JW. (1981). Reduction of hypercalciuria in tetraplegia after weight-bearing and strengthening exercises. Paraplegia. 19:289-293.

83. Katz RT, Dewalkd JPA, Schmidt BD. (2000). Ch. 29: Spasticity. Physical Medicine and Rehabilitation (2nd ed). Braddon RL (editor). 592-611.

84. Kita M, Goodkin DE. (2000). Drugs used to treat spasticity. Drugs. 59:487-495.

85. Kirshblum S. (1999). Treatment alternatives for spinal cord injury related spasticity. Journal of Spinal Cord Medicine. 22:199-217.

86. Kottke FJ, Pauley DL, Ptak RA. (1966). The Rationale for Prolonged Stretching for Correction of Shortening of Connective Tissue. Archives of Physical Medicine and Rehabilitation. 47(6): 345-352.

87. Krause JS. (1992). Longitudinal changes in adjustment after spinal cord injury: A 15-year study. Archives of Physical Medicine and Rehabilitation. 73:564-568.

88. Krupp L, LaRocca N, Muir-Nash J, Steinberg AD. (1989). The Fatigue Severity Scale: application to patients with multiple sclerosis and systemic lupus erythematosus. Archives of Neurology. 46: 1121–1123.

89. Kunkel CF, Scremin AME, Eisenberg B, Garcia JF, Roberts S, Martinez S. (1993). Effect of "standing" on spasticity, contracture, and osteoporosis in paralyzed males. Archives of Physical Medicine and Rehabilitation. 74:73–78.

90. Lance JW. (1980). What is spasticity? The Lancet. 335: 606.

91. Lechner HE, Frotzler A, Eser P. Relationship between self- and clinically rated spasticity in spinal cord injury. Archives of Physical Medicine and Rehabilitation. 87:15-19.

92. Lee K-C, Carson C, Kinnin KE et al. (1989) The Ashworth Scale: a reliable and reproducible measure of spasticity. Journal of Neuro and Rehabil? 3:205-209.

93. Leo K. (1985). The effects of passive standing. Paraplegia News. Nov. 45-47.

94. Levendoglu F, Ogun CO, Ozerbil O, Ogun TC, Ugurlu H. (2004). Gabapentin is a first line drug for the treatment of neuropathic pain in spinal cord injury. Spine. 29:743–751.

95. Levi R, Hultling C, Nash MS, Seiger A. (1995).The Stockholm spinal cord injury study: 1. medical problems in a regional SCI population. Paraplegia. 33:308-315.

96. Lewis KS, Mueller WM. (1993). Intrathecal baclofen for severe spasticity secondary to spinal cord injury. Annals of Pharmacotherapy. 27:767-774.

97. Likert, Rensis (1932). A Technique for the Measurement of Attitudes. Archives of Psychology 140: 1–55.

98. Little JW, Merritt JL. (1988). In: DeLisa, JA, ed. Rehabilitation Medicine. Philadelphia: Lippincott: 430–447.

99. Lundy-Ekman L. (2002). Neuroscience Fundamentals for Rehabilitation. W.B. Saunders Co. Toronto.

100. Lünenburger L, Colombo G, Reiner R, Dietz V. (2005). Clinical assessments performed during robotic rehabilitation by the gait training robot Lokomat. Proceedings of the 9th International Conference on Rehabilitation Robotics. June 28-July 1. Chicago; 345-348.

101. Mackey AH, Walt SE, Lobb G, Stott NS. (2004). Intraobserver reliability of the modified Tardieu scale in the upper limb of children with hemiplegia. Developmental Medicine in Child Neurology. 2004;46: 267 – 272.

102. Malec J, Harvey RF, Cayner JJ. (1982). Cannabis Effect on Spasticity in Spinal Cord Injury. Archives of Physical Medicine and Rehabilitation. 63:116-118.

103. Marciniak C, Rader L, Gagnon C. (2008). The use of botulinum toxin for spasticity after spinal cord injury.
American Journal of Physical Medicine and Rehabilitation. 87(4):312-7.

104. Marino RJ, Ditunno JF, Donovan WH, Maynard Jr F. (1999). Neurologic recovery after traumatic spinal cord injury: data from the Model Spinal Cord Injury Systems. Archives of Physical Medicine and Rehabilitation. 80:1391-6.

105. Marsh DR, Weaver LC. (2004). Autonomic dysreflexia, induced by noxious or innocuous stimulation, does not depend on changes in dorsal horn substance p. Journal of Neurotrauma. 21: 817–828.

106. Maynard FM, Karunas RS, Waring WP 3rd. (1990).Epidemiology of spasticity following traumatic spinal cord injury. Archives of Physical Medicine and Rehabilitation. 71:566-569.

107. Maynard FM, Bracken MB, Creasey G, Ditunno JF, Donovan WH et al. (1997). International Standards for Neurological Function and Classification of Spinal Cord Injury. Spinal Cord. 35:266-274.

108. McArdle WD, Katch FI, Katch VL. (1995). Exercise Physiology: Energy, Nutrition and Human Performance (4th ed). Lippincott Williams & Wilkins: Philadelphia PA.

109. McLellan DL.(1983). The drug treatment of spasticity. International Rehabilitation Medicine. 5:141-2.

110. Merritt JL. (1981). Management of Spasticity in Spinal Cord Injury. Mayo Clinic Proceedings. 56:614-622.

111. Nance PW, Bugaresti J, Shellenberger K, Sheremata W, Martinez- Arizala A. (1994). Efficacy and safety of tizanidine in the treatment of spasticity in patients with spinal cord injury. North American Tizanidine Study Group. Neurology. 44: S44–S51.

112. Ness LL, Field-Fote EC. (2009b). Effect of whole-body vibration on quadriceps spasticity in individuals with spastic hypertonia due to spinal cord injury. Restorative Neurology and Neuroscience (in press).

113. Nichols PJR, Norman PA, Ennis JR. (1979). Wheelchair users's shoulder? Scandinavian Journal of Rehabilitative Medicine. 11:29-32.

114. Noreau L, Shepard RJ. (1992). Physical fitness and productive activity in paraplegics. Sports Medicine Training and Rehabilitation. 3:165-181.

115. Noreau L, Shepard RJ. (1995). Spinal cord injury, exercise and quality of life. Sports Medicine. 20:226-50.
Pandyan AD, Johnson GR, et al. (1999). A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measure of spasticity. Clinical Rehabilitation. 13:373-383.

116. Nuyens GE, De Weerdt WJ, Ketelaer P, Feys H, De Wolf L, Hantson L, Nieuwboer A, Spaepen A, Carton H. (1994). Inter-rater reliability of the Ashworth scale in multiple sclerosis. Clinical rehabilitation. 8:286-292.

117. Pandyan AD, Price CI, Rodgers H, Barnes MP, Johnson GR. (2001). Biomechanical examination of a commonly used measure of spasticity. Clinical Biomechanics..16:859-65.

118. Parise M, Garcia-Larrea L, Mertens P, Sindou M, Mauguiere. (1997). Clinical use of polysynaptic flexion reflexes in the management of spasticity with intrathecal Baclofen. Electroencephalography and clinical Neurophysiology. 105:141-148.

119. Pertwee RG. (1999). Cannabis and cannabinoids: pharmacology and rationale for clinical use. Forschende Komplementarmedizin. 6(Suppl 3):12-5.

120. Petajan JH. (1998). Fatigue and rehabilitation of neurologic disorders. In Lazar RB (ed.) Principles of Neurologic Rehabilitation. Chicago: McGraw-Hill. Pp.367-399.

121. Phillips L, Ozer MN, Axelson P, Chizeck H. (1987). Spinal Cord Injury: A Guide for Patient and Family. Raven Press: New York.

122. Pierson SH. (1997). Outcome measures in spasticity management. Muscle and Nerve (Suppl). 6:S36-60.

123. Priebe MM, Sherwood AM, Thornby JI, Kharas NF, Markowski J. (1996). Clinical assessment of spasticity in spinal cord injury: a multidimensional problem. Archives of Physical Medicine and Rehabilitation. 77: 713-716.

124.Odeen I, Knutson E. (1981). Evaluation of the effects of muscle stretch and weight load in patients with spastic paraplegia. Scandinavian Journal of Rehabilitative Medicine. 13:117-121.

125. Ozer, MN. (1988). The Management of Persons with Spinal Cord Injury. Demos Publications, New York. Chapter3, pp. 69.

126. Ragnarsson KT, Krebs M, Naftchi NE, et al. (1981). Head-up tilt effect on glomerular filtration rate, renal plasma flow, and mean arterial pressure in spinal man. Archives of Physical Medicine and Rehabilitation. 62:306 –310.

127. Rhymer WZ, Katz RT. (1994). Mechanism of spastic hypertonia. Physical Medicine and Rehabilitation. 8:441-54.

128. Rick Hansen Foundation.www.rickhansen.com. About SCI and Links. Accessed Nov. 2, 2009.

129. Satkunam L. (2003). Rehabilitation medicine:3. Management of adult spasticity. Canada Medical Association Journal. 169:1173-119.

130. Satkunam L. (2008). Spasticity. 5th Canadian Comprehensive Review Course in Physical Medicine and Rehabilitation. April (Where was this Heather?)

131. Sawatzky B, Denison I, Langrish S, Richardson S, Hiller K, Slobogean B. (2007). The Segway Personal Transporter as an Alternative Mobility Device for People with Disabilities: A Pilot Study. Archives of Physical Medicine and Rehabilitation. 88:1423-1428.

132. Sawatzky B, Bishop CM, Miller WC, SCIRE Research Team. (2008). Classification and measurement of pain in the spinal cord-injured population. Spinal Cord. 46:2-10.

133. Sawatzky B, Denison I, Tawashy A. (2009). The Segway for People with Disabilities: Meeting Clients' Mobility Needs. American Journal of Physical Medicine and Rehabilitation 88(6):484-90.

134. Schmid A, Huonker M, Stahl F, Barturen JM, König D, Heim M, Lehmann M, Keul J. (1998). Free plasma catecholamine's in spinal cord injured persons with different injury levels at rest and during exercise. Journal of the autonomic nervous system. 68:96-100.

135. Segway Inc. Segway Smart Motion™. The science behind the technology. Available at: http://segway.com/personal-transporter/how_it_works.html. Accessed July 31/07.

136. Sehgal N, McGuire JR. (1998). Beyond Ashworth: Electrophysiologic quantification of spasticity. Physical Medicine and Rehabilitation Clinics of North America. 9:949:979 ix.

137. Sheean G. (2002). The Pathophysiology of spasticity. European Journal of Neurology. 9(Suppl 1):3-9.

138. Sherwood AM, Grave DE, Priebe MM. (2000). Altered motor control and spasticity after spinal cord injury: Subjective and objective assessment. Journal of Rehabilitation Research and Development. 37:41-52.

139. Shields RK, Dudley-Javoroski S. (2005). Monitoring standing wheelchair use after spinal cord injury: A case report. Disability and Rehabilitation. 27(3):142-146.

140. Siddall PJ, Taylor DA, McClelland JM, Rutkowski SB, Cousins MJ. (1999) Pain report and the relationship of pain to physical factors in the first 6 months following spinal cord injury. Pain. 81:187-197.

141. Siddall PJ, Yezierski RP, Loeser JD. (2000). Pain following spinal cord injury, clinical features, prevalence and taxonomy. IASP Newsletter. 3:3-7.

142. Sjölund BH. (2002). Pain and rehabilitation after spinal cord injury: the case of sensory spasticity? Brain Research Reviews. 40(1-3):250-6.

143. Sherwood AM, Grave DE, Priebe MM. (2000). Altered motor control and spasticity after spinal cord injury: Subjective and objective assessment. Journal of Rehabilitation Research and Development. 37:41-52.

144. Sköld C, Harms-Ringdahl K, Hultling C, Levi R, Seiger A. (1998). Simultaneous Ashworth measurements and EMG recordings in tetraplegic patients. Archives of Physical Medicine and Rehabilitation. 79:959-965.

145. Sköld C, Levi R, Seiger A. (1999). Spasticity after traumatic spinal cord injury: nature, severity and location. Archives of Physical Medicine and Rehabilitation. 80:1548-1557.

146. Sköld C.(2000). Spasticity in spinal cord injury: self- and clinically rated intrinsic fluctuations and intervention-induced changes. Archives of Physical Medicine and Rehabilitation. 81:144-9.

147. Sloan RL, Sinclair E, Thompson J, Taylor S, Pentalnd B. (1992). Inter-rater reliability of the modified Ashworth scale for spasticity in hemiplegic patients. International Journal of Rehabilitation Research. 15:158-161.

148. Sommers, MF. (1992). Spinal Cord Injury: Functional Rehabilitation. Appleton & Lange, Connecticut. Ch 2, pp.27.

149. Song ZK, Cohen MJ, Ament PA, Ho WH, Vulpe M, Schandler SL. (1993). Two-point discrimination thresholds in spinal cord injured patients with dysesthetic pain. Paraplegia. 31:485-93.

150. St George CL. (1993). Spasticity. Mechanisms and nursing care. Nursing Clinics of North America. 28:819-827.

151. Strömer S, Gemer HJ, Grthringer W, Metzmacher K, Follinger S, Wienke C. (1997) Chronic pain/dysaesthesiae in spinal cord injury patients: results of a multicentre study. Spinal Cord 1997;35:446-55.

152. Tardieu G, Shentoub S, Delarue R. (1954). A la recherche d'une technique de measure de la spasticite. Rev Neurol (Paris). 91:143 – 144.

153. Tawashy AE, Eng JJ, Lin KH, Tang PF, Hung C. (2009). Physical activity is related to lower levels of pain, fatigue and depression in individuals with spinal-cord injury: a correlational study. Spinal Cord. 47:301-6.

154. Thomas AJ, Davis GM, Sutton JR. (1997). Cardiovascular and metabolic responses to electrical stimulation-induces leg exercise in spinal cord injury. Methods of information in medicine. 36:372-375.

155. Tsui JKC, O'Brien CF. (1994). Clinical trials for spasticity. In Jankovic J, Hallet M (eds). Therapy with botulinum toxin, 1st ed. Marcel Dekker Inc. New York: pp523-533.

156. Turner J, Cardenas D. (1999).Chronic pain problems in individuals with spinal cord injuries. Seminars in Clinic Neuropsychiatry. 4:186-194.

157. Wade DT, Makela PM, House H, Bateman C, Robson P. (2006). Long-term use of a cannabis-based medicine in the treatment of spasticity and other symptoms in multiple sclerosis. Multiple Sclerosis. 12: 639:645.

158. Wallen MA, O'Flaherty SJ, Waugh MCA. (2004). Functional outcome of intramuscular botulinum toxin type a in the upper limbs of children with cerebral palsy.: A phase II trial. Archives of Physical Medicine and Rehabilitation. 85:192-200.

159. Walter JS, Sacks J, Othman R, Rankin AZ, Nemchausky B, Chintam R, Wheeler JS.(2002). A database of self-reported secondary medical problems among VA spinal cord injury patients: its role in clinical care and management. Journal of Rehabilitation Research and Development. 39:53-61.

160. Ward, AB. (2003). Long-term modification of spasticity. Journal of Rehabilitation Medicine. 41(Suppl):S60-65.

161. Ward AB. (2008). Spasticity treatment with botulinum toxins. Journal of Neural Transmission. 115:607-616.

162. Waters RL, Adkins RH, Yakura JS. (1991). Definition of Complete Spinal Cord Injury. Paraplegia. 29:573-581.

163. Whiteneck GG, Charlifue SW, Frankel HL et al. (1992). Mortality, morbidity and psychosocial outcomes of persons spinal cord injured more than 20 years ago. Paraplegia. 30:617-630.

164. Wilkin D, Hallam L, Doggett M. (1992). Measures of need and outcome for primary health care. Oxford Medical Publications. Oxford.

165. Wood DE, Burridge JH, van Wijck FM, McFadden C, Hitchcock RA, Pandyan AD, Haugh A, Salazar-Torres JJ, Swain ID. (2005). Biomechanical approaches applied to the lower and upper limb for the measurement of spasticity: a systematic review of the literature. Disability and Rehabilitation. 27:19-32.

166. Van der Salm A, Veltink PH, Hermen HJ, Ijzerman MJ, Nene AV. (2005). Development of a new method for objective assessment of spasticity using full range passive movements. Archives of Physical Medicine and Rehabilitation. 86:1991-1997.

167. Van der Woude LH, Botden E, Vriend I, Veeger D. (1997). Mechanical advantage in wheelchair lever propulsion: effect on physical strain and efficiency. Journal of Rehabilitation Research and Development. 34:286-94.

168. Yeo, JD, Walksh J, Rutkowski S, Soden R, Craven M, Middleton J. (1998). Mortality following spinal cord injury. Spinal Cord. 36:329-336.

169. Young RR, Shahani BT. (1986). Spasticity in spinal cord injury patients. In: Bloch RF, Basbaum M (eds). Management of spinal cord injuries. Baltimore: Williams and Wilkins p.241-83.

CHAPTER 2 –SPASTICITY: THE CHALLENGE OF MEASUREMENT

2.1 Introduction: Measurement of Spasticity[1]

Spasticity is defined by Lance (1980) as 'a velocity-dependent increase in muscle tone, associated with increased muscle stretch reflexes, as part of the upper motor neuron syndrome'. As many as 78% of individuals with spinal cord injury (SCI) experience some form of spasticity (Levi et al., 1995). Functionally, spasticity presents as an increased resistance to passive stretch of agonist muscles. This results in atypical gross patterns for slow voluntary movements, in addition to inappropriate firing of antagonistic muscles (De Souza et al., 1987, Nuyens et al., 1994). This can be perceived as both problematic and beneficial by persons with SCI (Lechner et al., 2006, Adams et al., 2007).

Muscle spasms may be painful and their involuntary nature makes them unpredictable. This often interferes with activities of daily living (ADLs), self-care and sleep (De Souza et al., 1987), can also lead to other secondary health complications including falls or pressure sores (Craven et al., 2009). Spasticity may prevent the individual from returning to independent living and gainful employment (Canadian Paraplegic Association, 1997) and is a potential source of embarrassment for individuals who are sensitive to the stigmatization of disability. Hand and arm function are often negatively affected, and these play a key role grasping and manipulating, which are important in tasks such as opening doors, personal grooming, communicating through writing/typing. Tasks such as these are extremely important for personal independence

[1] A version of this chapter will be submitted for publication. Boutilier G, Finlayson H, Grant C, Sawatzky BJ. (2009). Correlation of the Modified Ashworth Scale and the SCI-SET for upper and lower extremity muscles in spinal cord injury.

(Marino et al., 1998). Indeed, 75% of persons with tetraplegia would prefer restoration of their upper limb function to that of any other lost function (Anderson, 2004).

Conversely, spasticity can have positive implications. Often increased spasticity is beneficial in maintaining muscle tension, which may reduce muscular atrophy and possibly prevent osteoporosis (Sommers et al., 1992, Kirschblum, 1999, Hseih et al., 2008). Increased tone may facilitate transfers and weight bearing, as well as performance of crutch walking and enable lower body dressing (St George et al., 1993). Consensus in the literature suggests that the goal of treatment should not be to eliminate spasticity in its entirety, but to attempt to overcome detrimental functional impairments that are related to spasticity (Dietz, 2000, Ward, 2008). How we define these functional impairments warrants scrutiny and consideration.

Various clinical, biomechanical, neurophysiologic and self-report methods are used to measure the character and degree of spasticity. In general these approaches are employed in isolation of one other and have little practical crossover application (Lunenberger et al., 2005). For example, although various techniques to asses the mechanical manifestation of spasticity such as electrogoniometry and dynamometry (combined with surface electromyography) are frequently used in research investigations, they are rarely utilized in routine clinical settings (Sköld et al., 1999, Lechner et al., 2006). Recently self-report measures of spasticity have gained credibility as viable alternatives to independent examiner techniques (Collin et al., 2007). The complex nature of the condition of spasticity and discrepancies in its definition contribute to confusion in terms of measuring it validly and reliably (Joasefcyzk, 2002, Adams et al., 2005).

2.1.2 The modified Ashworth Scale

Originally designed as a simple clinical tool to test the efficacy of an anti-spasmodic drugs in patients with multiple sclerosis (Ashworth, 1964), the modified Ashworth scale (MAS) has achieved widespread use in clinical settings as a means of measuring spasticity (Pandyan et al., 1999, Sköld et al., 1999, Gregson et al., 2000, Satkunam, 2003, Ivanhoe et al., 2004).The scale grades the resistance of a relaxed limb to rapid passive stretch in 6 stages. A rating of "0" relates to normal muscle tone and "4" signifies rigidity of the limb (Bohannon et al., 1987). Table 2.1 depicts the MAS.

Table 2.1 The modified Ashworth Scale

0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of range of motion
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of motion
2	Marked increase in muscle tone throughout most of the range of motion, but the affected part is easily moved
3	Considerable increase in muscle tone, passive movement is difficult
4	Affected part is rigid in flexion or extension (abduction or adduction, etc)

The modified Ashworth scale is simple to administer, is well tolerated, and requires no special equipment (Hsieh et al., 2008). However, despite its' widespread use, it appears to have significant psychometric shortcomings. Findings for inter- and intra-rater reliability of the MAS remain controversial (Gregson et al., 2000, Craven et al., 2009). The validity of the scale has also come into question, as it has been criticized for making assumptions regarding the condition of spasticity based on a single component, muscle tone (Pandyan et al., 1999). This fails to account for the contributions of surrounding viscoelastic structures (Ivanhoe et al., 2004) or changes in alpha-motor

neuron reflex excitability (Rhymer et al., 1994). The MAS only addresses examiner-perceived passive resistance to movement (Sköld et al., 2000), and therefore may be vulnerable to subjective interpretations (Pandyan et al., 1999) and human error in testing (specifically velocity reproduction) (Craven et al., 2009).

While considered the 'clinical gold standard', there are still important limitations in using the modified Ashworth and it must not be used as exclusive measures of spasticity (Pandyan et al., 1999).

2.1.3 The Spinal Cord Injury-Spasticity Evaluation Tool

2.1.3.1 Introduction

A promising new instrument is the Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET) (Adams et al., 2007). As spasticity can be both helpful and harmful to the individual, this tool is the first to capture the bidirectional nature of the condition to offer a broader scope to spasticity in SCI. While it is common to ask respondents to rate positive and negative changes to overall health (Shields et al., 2005), this has yet to be done with respect to spasticity. No previous measures have specifically addressed the functional impact of spasticity in individuals with SCI (Hsieh et al., 2008).

The SCI-SET is a 35-item, 7-day recall questionnaire that targets aspects of daily life relevant to the SCI population which allows respondents to rate the impact of their spasticity on their daily activities and feelings. Items include such tasks as personal care, social activities, therapy regimes, as well as self-esteem, concentration and sleep, and safety. Responses can 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 SCI-SET is scored by summing the responses from all applicable items and dividing the sum

by the number of applicable items. The instructions developed for the SCI-SET include 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”. A positive item score would indicate that the individual perceives their spasticity as beneficial for this item, while a negative score would suggest they find it detrimental, and a score of zero would indicate that they do not perceive their spasticity to have an impact on the item in question.

The SCI-SET provides a total item score and an average score. The total item score is calculated by adding together all applicable scores (positive and negative), which retain their signs in order to be representative of the global impact of spasticity. The average score is calculated by dividing the total score by the number of applicable items (N/A scores are not included in the denominator). This provides a measure of the overall experiential impact of spasticity while eliminating the influence of any questions which may not have relevance to the individual.

2.1.3.2 Significance

The challenge for clinicians and researchers is to develop simple, reliable and robust measures that capture change in function and quality of life and to link them to changes in impairments such as spasticity. Adams and colleagues (2007) evaluated the SCI-SET using functional measures and other self-report items, however, no objective clinical measures of spasticity were included for comparison. Suggesting the incongruence of the Ashworth scales with the intentions of measuring overall spasticity experienced by the participants, the authors opted not to incorporate it. Exclusion of the most widely employed clinical tool for measuring spasticity represents a major void in the strength of the tool, and criterion validity of the SCI-SET must be established or refuted.

While the research community has identified the weaknesses and assumptions of the Ashworth scales (Pandyan et al., 1999, Satkunam, 2008), it is important to recognize that it still remains the primary clinical measure (and therefore the clinician's interpretation) of spasticity today. Therefore, it is relevant and necessary to investigate whether there is a connection between clinical interpretations of passive resistance ('spasticity') by the examiner and the patient's perceptions of the impact of spasticity on his or her daily life.

2.2 Purpose and Objectives

The purpose of this measurement study was to determine whether a correlation exists between clinical scores of spasticity and a recently-validated bidirectional spasticity self-report measure, which is the first of its kind.

2.3 Hypothesis

There will be a correlation between clinical measure of spasticity as measured by the modified Ashworth Scale and self report using the Spinal Cord Injury Spasticity Evaluation Tool.

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2.4 Methods

2.4.1 Study Design

This study was a cross-sectional study with repeated measures which was part of a one month dynamic standing clinical intervention. Subjects were assessed at the local rehabilitation centre three times; once at the beginning, once mid-month, and once at the end of the month. Two measures of spasticity were taken at each point in time: 1) an examiner-based clinical measure (MAS), and 2) a self-report Likert scale questionnaire of the impact of spasticity (SCI-SET).

2.4.2 Eligibility and Recruitment

To be eligible for the study, subjects were adults aged 19 to 65 years and had a chronic spinal cord injury (≥ 1 year post injury). They were proficient in English, and had adequate cognitive capacity to enable them to follow instructions. Subjects were able to stand independently or with braces, and to walk a few steps, either independently or with assistance, and had a history of spasticity.

Subjects were from a convenience sample recruited via referral from physical and occupational therapists at local rehabilitation centers in Vancouver, BC. Poster advertisements were also placed in inpatient and outpatient clinics for subject self-recruitment. Information letters were distributed to physicians and therapists to pass on to clients who met the eligibility criteria. Potential subjects were then able to contact the investigators directly for further information.

Subjects were asked to provide information on their type and year of injury, as well as any other relevant medical history, current medications, therapeutic management and exercise programs.

2.4.3 Data Collection

Verbal and written informed consent was obtained prior to testing, and the participants were well versed in their ability to leave the study at any time. This investigation received approval from the Clinical Research Ethics Board of the University of British Columbia.

Subjects participated in an intervention which involved 30 minutes of upright standing on a dynamic mobility device (the Segway® Personal Transporter) three times per week for four weeks (Sawatzky et al., 2009b). Subsequently MAS and SCI-SET data was collected. Testing sessions were done on Day 1 (T1), Day 6 (T2) and Day 12 (T3) of training. Subjects were asked to void their bladder prior to commencement of testing each day, as a full bladder is known to affect spasticity.

2.4.4 Modified Ashworth Testing

Participants were asked to identify primary muscle groups (right or left) in the upper and lower extremity in which they experienced spasticity, and these groups were assessed. Spasticity was measured by one of either a physiatrist or a physiatry resident. Both were well versed in using the MAS, and consistency between raters was established prior to testing. A copy of the MAS examination findings sheet developed by the investigators can be found in Appendix II. The day of the week and time of

assessment were maintained during the study to minimize an effect of time of day on spasticity.

Subjects were transferred onto a plinth and their shoes were removed. During testing of the knee extensors the proximal thigh was supported at 90º of flexion and the knee rested in full flexion as the distal limb was moved through the available extension of the knee. Each muscle group was tested twice and the MAS score was recorded. A detailed description of the modified Ashworth technique used to measure tone is found in Gregson et al. (2000), and is in accordance with that described by Nuyens et al. (1994). Of note, the examiner was careful not to move the limb through any range other than to place it in the appropriate anatomical position for testing. Examiners were also careful to avoid passively stretching the limb prior to grading, as this can initiate viscoelastic changes that may render the testing indeterministic (Pandyan et al., 1999). Testing sessions for each participant were scheduled at approximately the same time of day.

Modified Ashworth scores cannot be summed to produce an overall value (Gregson et al., 2000). As such, each individual testing session was acknowledged in isolation and scores were not summed. MAS scores were converted to ordinal data by allocating any number with a '+' value to .5. Participants were blinded completely to the scores, and the examiner did not have access to this information once the test had been performed and scores were recorded.

2.4.5 SCI-SET

In accordance with a standardized procedure, participants were next asked to complete the Spinal Cord Injury Spasticity Evaluation Tool. Subjects completed these on their own once the examiners had left the room to avoid influencing the subjects'

responses. An exception was made for one subject who had difficulty holding a writing instrument, and therefore the investigator circled the answers as the subject instructed. If subjects had questions, they were asked to continue with the remaining items until the examiners returned. To prevent any coercion or 'leading' statements, the principal examiner's (GB) standard response was 'answer as best you can as this pertains to you'.

2.5 Data Analysis Procedures

Data were analyzed to determine correlational agreement between the measure of clinical spasticity (modified Ashworth) and self-report spasticity impact (SCI-SET). The modified Ashworth scale is non-parametric in nature. Therefore, a Spearman rank correlation coefficient significance test was performed, as it does not assume normality when measuring of the association between two paired samples. SPSS v.16.0 statistical software was used for data analysis.

2.6 Results

Nine volunteers (7 males, 2 females), aged 33-61 participated in this study. All had a chronic spinal cord injury (≥ 1 year post injury) of varying degrees (ASIA A-D, 8 incomplete, 1 complete). Eight subjects completed the study. One subject withdrew voluntarily due to a family emergency, and their incomplete data was removed from the analysis. Of the eight remaining subjects, no data collection sessions were missed due to transportation or timing issues or subject illness on the day of testing. Subjects reported a myriad of affected muscle groups; however several did not experience any

upper extremity spasticity and this was noted. Baclofen (oral and intrathecal) was the most frequently prescribed spasticity medications, and subjects remained on these medications during the study. Table 2.2 summarizes the subject demographics.

Table 2.2 Subject Demographics. Subjects are categorized descriptively by sex, age, injury level, ASIA classification, year(s) since injury and their top upper and lower extremity muscle groups (right or left) in which they experience spasticity. R=right, L= left, Fin Flx= finger flexor, An Plfl= ankle plantarflexor, Hip Add= hip adductor, Kn Ext= knee extensor, Wr Ext= wrist extensor.

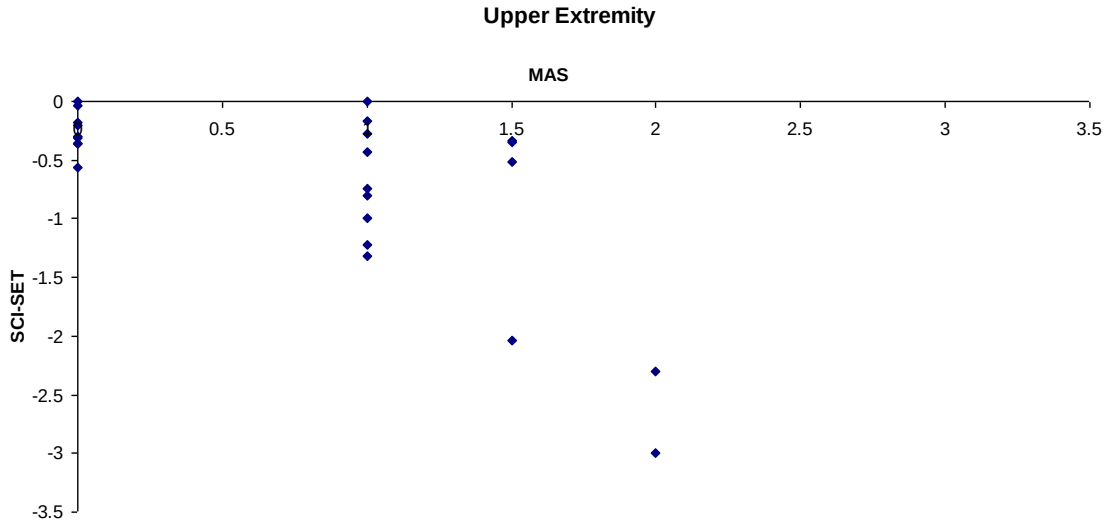
Sub	Sex	Age	Injury Level	ASIA	Year(s) Since Injury	Daily Meds	UE	LE
1	M	48	C5	C/D	24	Flouexetine	L FIN FLEX	L AN PLFL
2	M	35	T11	A	7	Baclofen, botox (rectus femr), Novotrimnol, Vesicare		L HIP ADD
3	M	33	C5	C	15	Baclofen (oral)	R FLEX DIG	R HIP EXT
4	M	41	T5	B	6	Baclofen (intrathecal), Pariet, Citalopram		R AM PLFL
5	M	54	C6	D	29	Baclofen (oral), Diazepam	R FIN FLEX	L KN EXT
6	F	54	C5	C	4	Botox (pectoralis)	R FIN FLEX	L HIP ADD
7	M	36	T6	C/D	18	NSAIDS		L HIP ADD
8	F	61	C5	D	1	Baclofen (oral), vitamins	L WR EXT	R HIP ADD

2.6.1 Correlational Agreement

Several subjects did not experience upper extremity spasticity (S2, S4, S7), while all were reported to have lower extremity spasticity. Subjects perceived their spasticity as overwhelmingly detrimental, as evidenced by the negative SCI-SET scores which ranged from -3 to zero. No correlational agreement was found between primary muscle groups and SCI-SET (ρ=.373, p=0.073). However, post hoc analysis revealed some interesting findings. Significant correlational agreement (ρ=.564, p=0.001) was seen for subjects' upper extremity MAS and SCI-SET scores. No correlation was present for

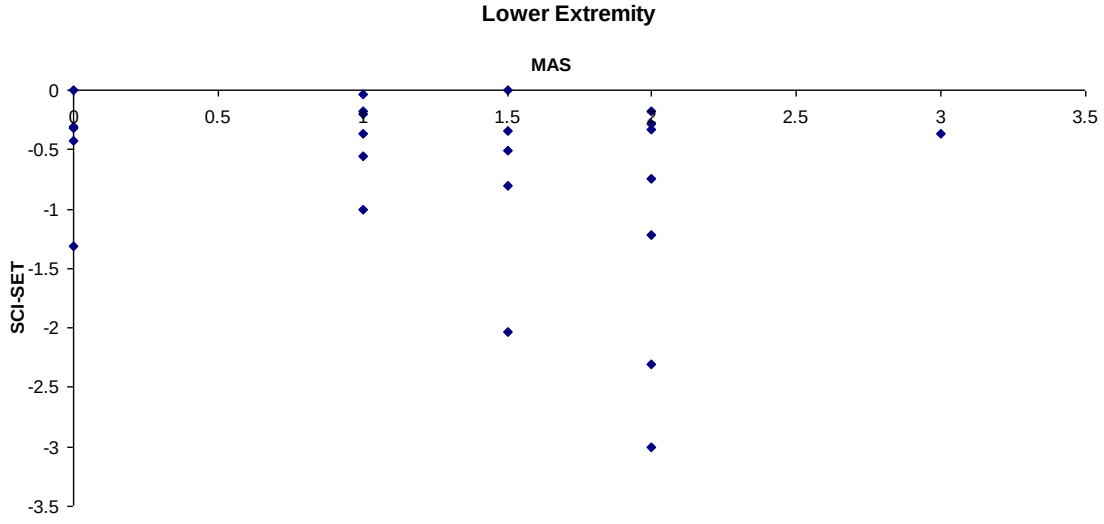
lower extremity MAS and SCI-SET scores (ρ=.249, p=.161). Figures 2.4 and 2.5 illustrate these findings respectively.

Figure 2.4. Upper extremity MAS and SCI-SET score correlation.



A correlational agreement of ρ=.564 (p=0.001) was found in upper extremity spasticity.

Figure 2.5. Lower extremity MAS and SCI-SET score correlation.



A correlational agreement of ρ=.249 (p=0.161) was found in lower extremity spasticity.

2.7 Discussion

The modified Ashworth provides a relatively objective physical measure of muscle tone. By contrast, the SCI-SET provides a picture of how the individual perceives the impact of their spasticity. A moderate correlation of ρ=.564 was found between the upper extremity MAS scores and the SCI-SET scores, while the lower extremity MAS scores showed a weak correlation (ρ=.249). Higher MAS scores were associated with more negative SCI-SET scores in the upper extremity, while the MAS/lower extremity showed no such relationship.

The SCI-SET is a tool which asks respondents to consider a broad range of activities and emotions. While not specifically segregated into categories, items regarding tasks like eating/drinking, meal preparation, manual wheelchair use and small hand movements clearly pertain to upper extremity function, while those focusing on weight bearing, transfers and stability would be relevant to lower extremity function. We suggest the relatively robust relationship between upper extremity MAS scores and SCI-SET scores may be indicative of this psychological link to upper limb competencies and the consequential effect on independence. Hand and arm movements are often essential in performance of activities of daily living (Marino et al., 1998). In fact, restoration of upper limb function was identified as the number one priority for individuals with tetraplegia (Anderson, 2004). Tasks that require greater dexterity, eye- hand coordination and fine motor skills likely impact the individuals' perceptions of the extent of limitations imposed by their spasticity. The ability to independently execute life skills such as eating, personal care and hygiene, writing/typing, driving, meal preparation, and having physical contact with others enhance personal autonomy, and promote a sense of empowerment.

On the other hand, the self esteem and sense of autonomy in individuals with SCI may be less affected by gross motor and locomotor challenges, especially in light of emerging technologies that minimize disability and maximize performance (Boutilier et al., 2009). The lack of correlation between MAS scores and SCI-SET for the lower extremity muscle may illustrate this phenomenon. Several subjects (S6, S7) gave positive scores to tasks pertaining to weight bearing and transferring. While MAS scores would indicate merely the presence of spasticity , the SCI-SET may actually capture some of the benefits of spasticity in lower extremities, i.e. enhanced ability to bear weight, greater stability/balance, and improved transfers with such positive ratings. Requiring assistance for mobility impairments may be less stigmatizing and/or discouraging than requiring similar assistance for intimate personal care needs, and the SCI-SET may be sensitive to this subtlety.

SCI-SET scores represent global interpretations of the functional impact of spasticity. By contrast, the modified Ashworth scores assess tone for upper and lower extremity muscles. The clinician's expectation is that similar MAS scores represent a similar level of spasticity which leaves no room for differential assignment related to functional performance (i.e. whether this is beneficial or detrimental). This quantitative measure may have little relevance in light of the vastly different types of tasks performed by muscle groups in the lower extremities versus those in the upper extremity. Future exploration of the psychological impact of various types of impairments using the SCI-SET and MAS may clarify these complex issues.

Self-report measures of spasticity are a representation of the individual's experiences, which are not necessarily limited to a single component of physical tone. These perceptions may include what Sjolund (2002) terms 'sensory spasticity': other symptoms such as pain, prickle, parasthesia, tension, and constriction which may be identified as contributing to spasticity. As sensory projection neurons and segmental

reflex pathways may share interneurons, processes in the sensory systems inducing central spinal pain may correspond to those causing spasticity in motor systems (Ashby 1975). This phenomenon, which may be similar to that of phantom limb pain, would have no bearing on MAS ratings of spasticity, and yet is still a valid representation of the physical experience of spasticity for the patient.

We asked our participants to self-identify primary muscle groups (right or left) in the upper and lower extremities in which they most notably experience spasticity. This patient-centered approach contributed to the validity of our study by making it more representative of the individual nature of the condition, and to our knowledge this has not been done before. Our decision to include a heterogeneous population (ASIA A-D) may allow further generalization to other populations with mobility impairments. Viewing spasticity as beneficial is a relatively new concept and tools such as the SCI-SET which account for the potential benefits that may be accrued may also facilitate the positive attitude which has allowed so many individuals with SCI to breach so-called 'barriers'. The possibility of measuring positive features of spasticity is fascinating and may contribute to a more precise and optimistic outlook in future research.

Treatment goals for individuals with SCI should revolve around attainment of a high quality of life, and in some cases spasticity can positively contribute to an improved life satisfaction. The fact that these two instruments appear to measure similar, yet distinct aspects of the patients' spasticity is helpful in that the cumulative results enable clinicians to have a more comprehensive picture of spasticity as it affects daily tasks. While the MAS is quick and offers an objective interpretation, perhaps the SCI-SET better reflects the multifaceted nature of spasticity and how it affects the individual. It is time the research reflects spasticity appropriately, which entails the inclusion of self-report phenomena on par with clinical, biomechanical or neurophysiologic measures.

2.8 Limitations

The small size of this pilot study, may limit the power of the statistical test to detect significant change. Perhaps a larger sample would strengthen the relationships found. It may have also allowed for a more in-depth analysis by item of the SCI-SET. Responses could be examined to look for relationships by gender and functional levels, as was suggested by Sköld et al (1999). A bias towards lower extremity tone in our sample may under represent individuals' experiences of tone in the upper extremity in single-item analysis. Given our low sample size, the investigators were not satisfied that such analyses would provide useful insight.

A randomized double blind study might have strengthened the findings, but in reality it is impossible to blind the clinician as they are diagnosing spasticity. However, they handed over all documentation to the research immediately following testing, and were not privy to the result of any MAS scores during the course of the study. We acknowledge that self-report tactics may still suffer from the participants' hope and expectations of positive outcomes. The experimenter attempted to minimize any source of recall bias by immediately collecting it from the participants.

Use of a single rater for the MAS would have been preferable, though this was not feasible for our work. Both clinicians were well-trained in using the MAS and use it on a regular basis and are highly familiar with its' clinical implications. The large discrepancy in experience between the two raters may have detracted from standardization of the outcome. However, a study by Ghotbi et al. (2009) suggested that physiotherapy students with novel experience treating patients with spasticity could still generate highly reproducible results with a modified version of the Ashworth scale. Using an accelerometer may have addressed any inter-rater variability by standardizing of the speed of movement of the limb. Craven et al. (2009) employed the use of a one-cycle

per second metronome in an attempt to provide feedback to the examiner in to improve consistency; however that study had yet to be published at the commencement of our study.

2.9 Bridging Summary

An ideal measurement of spasticity should incorporate the impartiality of an external examiner together with the subjective experience of the patient. By itself, a physical measure such as the modified Ashworth scale is likely inadequate to explore the full scope of spasticity; however, it is a useful component of a comprehensive assessment.

While the SCI-SET has only been validated in a single study by its authors, we have provided some evidence to support its' criterion validity with respect to the MAS. As such, we feel that this tool has potential to provide a more representative and balanced picture of the personal impact of spasticity. Using patient-centered self-report information such as that provided by the SCI-SET as a primary outcome measure, rather than an observer-rated scale has been questioned due to its' subjective nature. The results of this investigation strongly defend the inclusion of the patients' experience as an adjunct to the existing clinical measures as it may provide greater depth of information.

In determining the effectiveness of an intervention strategy, both objective physiological measurement and patient self-report/satisfaction would seem indicated. Future work is needed to determine whether the SCI-SET is responsive to functional change, and we advocate for its' inclusion in intervention studies targeting spasticity.

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2.10 References

1. Adams MM, Hicks AL. (2005). Spasticity after spinal cord injury. Spinal Cord. 43:577-86.

2. Adams MM, Martin Ginis KA, Hicks AL. (2007). The Spinal Cord Injury Spasticity Evaluation Tool: Development and Evaluation. Archives of Physical Medicine and Rehabilitation. 88:1185-1192.

3. Anderson KD. (2004). Targeting recovery: priorities of the spinal cord-injured Population. Journal of Neurotrauma. 21:1371-1383.

4. Ashworth B. (1964). Preliminary trial of carisoprodal in multiple sclerosis. Practitioner. 192:540-542.

5. Benz EN, Hornby TG, Bode RK, Scheidt RA, Schmit BD. (2005). A physiologically based clinical measure for spastic reflexes in spinal cord injury. Archives of Physical Medicine and Rehabilitation. 86:52-59.

6. Bohannon RW, Smith MB. (1987). Inter rater reliability of a modified Ashworth Scale of muscle spasticity. Physical Therapy. 67: 206-207.

7. Collin C, Davies P. Mutiboko IK, Ratcliffe S. (2007). Randomized controlled trial of cannabis-based medicine in spasticity caused by multiple sclerosis. European Journal of Neurology. 14: 290–296.

8. Craven BC, Morris Ar. (2009). Modified Ashworth scale reliability for measurement of lower extremity spasticity among patients with SCI. Spinal Cord. [Epub ahead of print]:1-7.

9. DeSouza LH, Musa IM. (1987). The measurement and assessment of spasticity. Clinical Rehabilitation. 1:89-96.

10. Dietz V.(2000). Spastic movement disorder. Spinal Cord. 38:389-93.

11. Gregson JM, Leathly MJ, Moore PA, Smith TL, Sharma AK, Watkins CL. (2000). Reliability of measurements of muscle tone and muscle power in stroke patients. Age and Ageing. 29:223-228.

12. Haas BM, Bergstrom E, Jamous A, Bennie A. (1996). The inter rater reliability of the original and of the modified Ashworth scale for assessment of spasticity in patients with spinal cord injury. Spinal Cord. 34:560-564.

13. Hsieh JTC, Wolfe DL, Miller WC, Curt A and the SCIRE Research Team. (2008). Spasticity outcome measures in spinal cord injury: psychometric properties and clinical utility. Spinal Cord. 46:86-95.

14. Ivanhoe CB, Reistetter TA. (2004). Spasticity: The Misunderstood Part of the Upper Motor Neuron Syndrome. American Journal of Physical Medicine and Rehabilitation. 83(Suppl):S3-S9.

15. Jozefczyk PB. (2002). The management of focal spasticity. Clinical Neuropharmacology. 25:158-173.

16. Kirshblum S. (1999). Treatment alternatives for spinal cord injury related spasticity. Journal of Spinal Cord Medicine. 22:199-217.

17. Lechner HE, Frotzler A, Eser P. Relationship between self- and clinically rated spasticity in spinal cord injury. Archives of Physical Medicine and Rehabilitation. 87:15-19.

18. Levi R, Hultling C, Nash MS, Seiger A. (1995).The Stockholm spinal cord injury study: 1. medical problems in a regional SCI population. Paraplegia. 33:308-315.

19. Marino RJ, Shea JA, Stineman MG. (1998). The capabilities of upper extremity instrument: reliability and validity of a measure of functional limitation in tetraplegia. Archives of Physical Medicine and Rehabilitation. 79:1512-1521.

20. Nuyens GE, De Weerdt WJ, Ketelaer P, Feys H, De Wolf L, Hantson L, Nieuwboer A, Spaepen A, Carton H. (1994). Inter-rater reliability of the Ashworth scale in multiple sclerosis. Clinical rehabilitation. 8:286-292.

21. Pandyan AD, Johnson GR, et al. (1999). A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measure of spasticity. Clinical Rehabilitation. 13:373-383.

22. Platz T, Eickhof C, Nuyens G, Vuadens P. (2005). Clinical scales for the assessment of spasticity, associated phenomena and function: a systematic review of the literature. Disability and Rehabilitation. 27:7-18.

23. Priebe MM, Sherwood AM, Thornby JI, Kharas NF, Markowski J. (1996). Clinical assessment of spasticity in spinal cord injury: a multidimensional problem. Archives of Physical Medicine and Rehabilitation. 77: 713-716.

24. Rhymer WZ, Katz RT. (1994). Mechanism of spastic hypertonia. Physical Medicine and Rehabilitation. 8:441-54.

25. Satkunam L. (2003). Rehabilitation medicine:3. Management of adult spasticity. Canada Medical Association Journal. 169:1173-119.

26. Satkunam L. (2008). Spasticity. 5th Canadian Comprehensive Review Course in Physical Medicine and Rehabilitation. Toronto; April 2.

27. Sawatzky BJ. Denison I., Tawashy A, Boutilier, G. (2009b). The Segway Personal Transporter as an Alternative Mobility Device for People with Disabilities. Presented at the 4th International State-of-the-art Congress Rehabilitation: Mobility, Exercise & Sports. Amsterdam, the Netherlands. April 2009.

28. Shields RK, Dudley-Javoroski S. (2005). Monitoring standing wheelchair use after spinal cord injury: A case report. Disability and Rehabilitation. 27(3):142-146.

29. Sköld C, Levi R, Seiger A. (1999). Spasticity after traumatic spinal cord injury: nature, severity and location. Archives of Physical Medicine and Rehabilitation. 80:1548-1557.

30. Sköld C.(2000). Spasticity in spinal cord injury: self- and clinically rated intrinsic fluctuations and intervention-induced changes. Archives of Physical Medicine and Rehabilitation. 81:144-9.

31. Sommers, MF. (1992). Spinal Cord Injury: Functional Rehabilitation. Appleton & Lange, Connecticut. Ch 2, pp.27.

32. St George CL. (1993). Spasticity. Mechanisms and cursing care. Nursing Clinics of North American. 28:819-827.

33. Vanden Berghe A, Van Laere M, Hellings S, Vercauteren M. (1991). Reconstruction of the upper extremity in tetraplegia: functional assessment, surgical procedures, and rehabilitation. Paraplegia. 29:103-112.

34. Ward AB. (2008). Spasticity treatment with botulinum toxins. Journal of Neural Transmission. 115:607-616.

CHAPTER 3- THE ROLE OF NOVEL INTERVENTION (SEGWAY)

3.1 Introduction[2]

Spinal cord injuries (SCIs) affect over 41,000 Canadians and there are 1,100 new cases each year (www.rickhansen.com). In addition to loss of function, numerous secondary complications, such as spasticity, pain and fatigue may also arise. (Eng et al., 2008) Facing these difficulties may have a considerable impact on the individual's emotional well-being and sense of self-efficacy and self-esteem (Ditunno et al., 1994).

3.1.1 Spasticity in SCI

Damage to the corticospinal tract which is responsible for voluntary movement results in a loss of central input to modulate or inhibit reflex activity (Dietz, 2000), producing in an exaggerated or inappropriate reflex response to a normal stimulus known as *spasticity* (Ozer et al., 1987). Spasticity is defined by Lance as '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 upper motor neuron syndrome' (Lance,1980). It occurs in as much as 78 % of the SCI population (Hsieh et al., 2008). Spasticity is often considered problematic, inhibiting movement and performance of activities of daily living (ADLs). When elicited suddenly and forcefully, it may lead to falls, impact transfers, standing and walking, and interfere with sleep (Little et al., 1989). Moreover, it is thought to contribute

[2] A version of this chapter will be submitted for publication. Boutilier G, Finlayson H, Denison I., Wiefelspuett S, Grant C, Sawatzky BJ. (2009b). The Segway PT as a novel intervention reduces spasticity, pain and fatigue and improves overall well being in individuals with spinal cord injury.

to poor self esteem, body image, pain, fatigue and sexual dysfunction (Adams et al., 2007). On the other hand, it may have a beneficial effect in terms of extension movements to improve postural support in sitting, standing, transfers, walking, dressing and performance of ADLs (St George et al., 1993, Sköld et al., 2000).

No universal approach is successful in the management of spasticity (Kirshblum, 1999). Therapeutically, any combination of modalities including stretching, cryotherapy, serial casting or bracing is employed (Katz 2000). Oral, intrathecal and motor point injections are commonly used in moderation (Satkunam, 2008). Vigilant monitoring and elimination of noxious stimuli, including urinary tract infections (UTI), ingrown toe nails, decubitus ulcers, and/or infection anywhere in the limb is imperative (Satkunam, 2008). Spasticity is also painful for some individuals with SCI.

3.1.2 Pain

Chronic pain remains a significant problem for many individuals with spinal cord injury (SCI). Prevalence rates for SCI-related pain are as high as 94% of the SCI population, depending on population characteristics (acute, chronic) and measurement factors, such as intensity and daily interference (Sawatzky et al., 2008). This pain can be nociceptive (musculoskeletal or visceral) or neuropathic in nature (Siddall et al., 2000). Interventions generally focus on alleviation of underlying pathology or aggravating conditions, often incorporating physical therapy and exercise, relaxation training, psychotherapy, and dietary improvement (Ozer et al., 1987). Oral or intrathecal pharmacological agents including antidepressants, anticonvulsants, and analgesics are often used to treat chronic SCI-related pain. Unfortunately, narcotics for pain may be addictive, often have unwanted side effects, and serve to mask the pain, rather than

treat the origin of it (Collin et al., 2007). Dealing with the effects of chronic SCI pain may lead to emotional stress and physical exhaustion.

3.1.3. Fatigue

Fatigue is characterized as a mismatch between the energy required to perform routine tasks and the energy available to do so (Hammell et al., 2009). Prevalence rates of fatigue that interferes with functional ADLS in the SCI population are approximately 60% (Fawkes-Kirby et al., 2008). A recent review of literature identified two kinds of fatigue in SCI: 'muscular fatigue', characterized as a physiological phenomenon of paralyzed muscles; and 'chronic fatigue' associated with 'aging, physiological and psychological deconditioning' contributing to decreased quality of life (Barat et al., 2006). Additional dimensions of cognitive (sense of being overwhelmed, coping), emotional (frustration, guilt regarding partner's involvement, depression) and physical (tension, stress) fatigue have been identified (Hammell et al., 2009). Environmental factors such as physical barriers and a lack of wheelchair access, weather and the expectations that arise from a sociocultural context that values independence may also contribute to feelings of fatigue (Hammell et al., 2009).

Hammell et al. (2009) interviewed 29 individuals, 21 with SCI, in addition to two family members, two care givers and four therapists. They employed the nine-item Fatigue Severity Scale (FSS) which indicates clinically significant fatigue when mean scores are greater than or equal to '4' (Miller, 2009). Mean FSS scores were 4.65 (sd=1.42). Additional qualitative comments indicated that fatigue was perceived to exert a profound effect on the lives of many people with SCI, and was consistently associated with pain, depression and hopelessness, side effects of medications, poor quality sleep,

spasticity, poor posture, diet, and the effort required to accomplish routine and self-care tasks.

Studies on wheelchair users with SCI indicate that those who regularly participate in physical activity programs can increase their muscular strength, VO2 max, and physical performance to well exceed levels of their inactive peers (Hoffman, 1986, Glaser et al., 1996). This in turn enhances exercise tolerance and minimizes fatigue (Petajan et al., 1998). Besides these fitness gains, habitual physical activity may lead to improvements in health, psychosocial condition, rehabilitation potential, functional independence and quality of life (Hjeltnes et al., 1990, Noreau et al., 1992, Noreau et al., 1995).

3.1.4 Physical Activity and Mobility

Physical activity is often associated with decreases in pain, fatigue and depression in individuals with SCI (Tawashy et al., 2009). Habitual exercise for this population is thought to minimize joint deterioration and incipient neurological deficits that appear over time (Jacobs et al., 2004). Bearing in mind the additional challenges and limitations of this population with respect to possible circulatory and respiratory impairments (Jacobs et al., 2001), exercise programs for individuals with SCI must consider the energy demands of daily living while encouraging safety and independence.

Options for mobility in individuals with SCI are determined by the individual's functional abilities and physical dimensions, as well as the surrounding environment and their mobility goals (Ozer, 1988). Leg braces, forearm crutches, and manual and/or power wheelchairs are commonly used in this population. Standing wheelchairs may be preferable to seated chairs. An upright posture has been shown to improve personal interactions, build self-esteem and self-image, and improve morale (Cybulski et al.,

1986, Eng et al., 2001, Sawatzky et al., 2007). Digestion, breathing, and circulation are thought to be enhanced with vertical position (Dunn et al., 1998). Additional physiologic benefits such as reductions in spasticity (Duffus et al., 1983, Little et al., 1988), improved bladder and bowel function (Leo 1983, Cybulski et al., 1986, Dunn et al., 1998, Eng et al., 2001, Shields et al., 2005), decreased calcium in the urine (Issekutz et al., 1966, Kaplan et al., 1981), increased bone density (Goemaere et al 1994), improved skin integrity (Cybulski et al., 1986, Eng et al., 2001) and sleep (Dunn et al., 1998, Eng et al., 2001) have been attributed to passive standing in SCI. However, the majority of studies examining the physiological benefits of standing thus far are plagued by small sample sizes (Kunkel et al., 1993, Bohannon et al., 1993) and therefore these results should be interpreted with caution/the evidence remains inconclusive (Kunkel et al., 1993, Eng et al., 2001). Regardless of the advocated benefits of upright posture, standing wheelchairs for individuals with SCI are not widely prescribed, possibly due to their heavy weight, lack of suspension or wheel camber, and difficulty maneuvering (Dunn et al., 1998, Shields et al., 2005).

With the advent of the Segway on the market which has shown to be user friendly for people with disabilities (Sawatzky et al. 2007), this may provide an opportunity to promote standing while concurrently acting as a mobility device for people with SCI.

3.1.5 The Segway Personal Transporter

The Segway Personal Transporter® is an electric-powered, self-balancing mobility device which enables the user to negotiate variable terrain. It consists of small platform supported by 2 parallel wheels 20 cm above the ground that support a standing rider. A flat handlebar with a steering device in the form of a twist grip system allows for

easy maneuvering. A closed loop dynamic stability control system called LeanSteer™ technology is comprised of 5 angular rate gyroscopes which sense the rider's center of gravity. When the rider leans forward the Segway moves forward and when the rider leans back the Segway moves backwards, or stops. Forward velocity of the Segway is determined by the angle of forward lean, such that greater lean corresponds to higher speeds (www.segway.com). The Segway can travel up to 12.5mph /20kph and as far as 38km on a single battery charge. Its' capabilities for such robust tasks as navigating uneven terrain may make it a more attractive alternative to a standard wheelchair. Additionally, at a cost of $6, 000, as comparable to other scooters (~$4 - 6,000) and power wheelchairs (~$9, 000) it may be a feasible alternative for many individuals.

Only two previous studies (Sawatzky and colleagues et al., 2007, 2009) have looked at the effect of Segway use in people with disabilities. They found that all subjects, regardless of ability, were able to use the Segway provided they could stand with or without assistance. Psychosocial benefits were also reported with respect to increased independence and helped to minimize their disability to others, and in so doing increased their feelings of self-esteem. In a second study, satisfaction of current mobility aids (wheelchairs, crutches, walkers) were compared to the Segway using the Wheelchair Outcome Measure (WhOM). All subjects preferred the Segway to their existing devices (Sawatzky et al., 2008). Several anecdotal reports from subjects were of improvements in balance and reductions in spasticity immediately following Segway training. The question then remains, is there an additional therapeutic physiologic effect of the Segway for these individuals to the existing mobility benefits?

3.2 Purpose and Objectives

The purpose of this investigation was to determine if physiologic benefits such as spasticity, pain and fatigue reduction can be derived from Segway training, and whether these potential benefits have an immediate or a more long term benefit.

3.3 Hypotheses

Hypothesis 1: There will be an immediate (within day) intervention effect of a one month dynamic standing program on reduction of spasticity in the indicated muscles as measured by the MAS.

Hypothesis 2: There will be a long-term intervention effect of a one month dynamic standing program on reduction of spasticity, pain and fatigue as measured by the MAS, as well as self-report.

3.4 Methods

3.4.1 Study Design

3.4.1.1 Segway Protocol

Since there are no standard training programs specific to the Segway, we chose to use the ACSM exercise training recommendations for individuals with paraplegia. ACSM suggests exercise three to five times per week of 20–60 minutes in duration at an

intensity of 50-80%$VO2_{peak}$ (ACSM, 2009). As individuals with both para- and tetraplegia were being recruited for the study, the research team consensus was that the protocol should lean towards the lower end of these recommendations in order to prevent subject fatigue or involuntary withdrawal due to exhaustion. Therefore, the finalized program consisted of 30 minutes per day, 3 times a week for the period of one month.

In conceiving a Segway training program, the research team attempted to devise a program which would foster confidence by allowing individuals to progress at their own rate. Overhead harnesses were made available to participants who requested additional safety. Each participant wore a helmet while on the Segway, and had at least one spotter with them at all times. The training routes were varied in order to maintain the interest of the subject and to remain challenging, yet achievable. Both indoor and outdoor obstacles, tasks and targets were integrated into the individualized programs of the participants.

All training was conducted at the ICORD Blusson Spinal Cord Center and GF Strong Rehabilitation Center in Vancouver, BC.

3.4.1.2 Ethics

This study was approved by the Clinical Research Ethics Board of the University of British Columbia.

3.4.1.3 Study Participants

Participants were recruited via therapist referrals at a rehabilitation outpatient clinic in the local community. Poster advertisements were also placed in various rehabilitation centers for subject self-recruitment.

For this pilot project, we endeavored to recruit 20 individuals to participate. In order to maintain homogeneity of condition among the participants, inclusion criteria were individuals with chronic (≥ 1 year post injury) SCI who had the ability to rise from sitting to standing with no more than moderate assistance from one person, and ability to stand (using upper extremity support). To improve the generalizability of our findings, we recruited individuals with varying ability levels. As such, participants with ASIA scores of A-D were included, as were those with both paraplegia and tetraplegia. Subjects were required to have a history of spasticity in one or more muscle groups at least one month prior to the study, and report some occurrence of associated pain. Information about daily use of medications and assistive devices for mobility was also recorded.

All subjects gave written and verbal informed consent to participate. They were instructed to maintain their regular exercise habits until completion of the study, and to notify investigators of any medication changes.

3.4.2 Outcome Measures

Spasticity was assessed using a clinical scale (the modified Ashworth Scale) in addition to a new bidirectional self-report tool (The Spinal Cord Injury Spasticity Evaluation tool). Self-evaluations of pain and fatigue were measured using The Pain Outcomes Questionnaire (VA) and the Fatigue Severity Scale, respectively. All of these were performed bi-weekly. Daily records regarding the frequency and intensity of spasticity and pain were kept, in addition to information pertaining to fatigue, sleep, appetite and digestion, bladder/bowel health and overall well-being.

3.4.2.1 The modified Ashworth Scale

Spasticity using the modified Ashworth Scale (MAS) was assessed by one of two examiners (a physiatrist and a physiatry resident). Both were well versed in using the MAS. In preliminary work we found that they were very consistent in the measurement technique and grading. The MAS grades the resistance of a relaxed limb to rapid passive stretch in 6 stages. A rating of “0” relates to normal muscle tone and “4” signifies rigidity of the limb (see Table 3.1). In the present study, we tested muscle groups including elbow flexors and extensors, wrist flexors and extensors, and finger flexors with the patient in a seated position. We also tested hip flexors, extensors and adductors, knee flexors and extensors, and plantar flexors with the subject positioned on a plinth in a supine position.

Table 3.1 The modified Ashworth Scale

0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of range of motion
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of motion
2	Marked increase in muscle tone throughout most of the range of motion, but the affected part is easily moved
3	Considerable increase in muscle tone, passive movement is difficult
4	Affected part is rigid in flexion or extension (abduction or adduction, etc)

3.4.2.2 The SCI-SET

The SCI-SET is a 35-item, 7-day recall questionnaire that targets aspects of daily life relevant to the SCI population which allows respondents to rate the overall global

impact of their spasticity (Adams et al., 2007). It has been validated with respect to (1) self-assessment of spasticity severity, (2) self-assessment of spasticity impact, (3) the Penn Spasm Frequency Scale (PSFS), (4) the Functional Independence Measure motor subscale (FIM), and (5) the Quality of Life Index (QLI) SCI Version–III health and functioning subscale (satisfaction). Internal consistency (.90) and interclass correlation (.91) have also been established (Adams et al., 2007). Responses in the SCI-SET are bidirectional and can range from -3 (extremely problematic) to +3 (extremely helpful), with the option of choosing "0" if spasticity had no effect on the activity/aspect of life in question. The SCI-SET provides a total item score and an average score. The total item score is calculated by adding together all scores (positive and negative which retain their signs) to give an overall picture of how spasticity affects the individual. A positive total score would indicate that the individual perceives their spasticity as a benefit, while a negative total score suggests that it is a greater hindrance. An average score can then be calculated by excluding any non-applicable items (i.e. questions on power chair use if the individual does not use a power chair) in order to be truly representative of each individual respondent.

3.4.2.3 The Pain Outcome Questionnaire-Veterans Affairs (POQ-VA)

The POQ-VA is a primary pain outcomes tool comprised of 19 items that numerically recalls pain history, average pain intensity, pain interference, emotional distress, pain-related fear, satisfaction with treatment and medical use (Clark et al., 2003). Queries ask participants to rate their pain on average during the previous week ("Does your pain affect your ability to walk?"), while others ask specifically about immediate experiences ("How would you rate your strength and endurance TODAY?"). Scores range from 0 (e.g. no pain interference, very poor strength/endurance) to 10 (e.g.

significant pain interference/very high strength/endurance). Positive and negative polarity of the scale is varied across the instrument to prevent systematic response bias. The POQ-VA provides a total score, along with 5 sub categories pertaining to activities of daily living, negative affects, mobility, vitality and fear. A copy of the POQ-VA is found in Appendix III.

While not specifically validated for the SCI population yet, the POQ-VA has been shown to be reliable, valid and sensitive to changes associated with pain management in a heterogeneous group of veterans undergoing a variety of treatments (Sawatzky et al., 2008).

3.4.2.4 The Fatigue Severity Scale (FSS*)*

Originally developed for use in patients with multiple sclerosis, the FSS is comprised of a unidimensional Likert scale containing nine items which are rated from 1 to 7 with respect to the effects of fatigue on function (Krupp et al., 1989). “1” signifying ‘no effect of fatigue’, to “7” signifying s significant effect of fatigue. Questions such as “I am easily fatigued” or “fatigue causes frequent problems for me” provide the examiner with a broad picture of the global effects of fatigue; while the impact of a specific activity on the symptoms of fatigue is conveyed in questions such as “fatigue interferes with response to the questions (adding up all the answers and dividing by nine). Scores are calculated by totaling the responses and dividing by nine to provide a mean score. Mean scores of >=4 is considered clinically fatigued (Miller, 2009). A copy of the FSS can be found in Appendix IV.

Previous work has found mean FSS scores for SCI participants to fall within the range of 4.1±1.8 (Fawkes-Kirby et al., 2008) to 4.7±1.4 (Hammell et al., 2009). These scores are slightly lower than those for multiple sclerosis (5.2±1.5) and postpolio

syndrome (5.1±1.7), but significantly higher than FSS scores reported among individuals with no known pathology (2.2±1.1) (Packer et al., 1994). Values for internal consistency (Cronbach α=.89) and test-retest reliability (ICC=.84) led Anton and colleagues (2008) to conclude that the FSS was a reliable, valid measure of fatigue in individuals with SCI. Its' focus on the impact of a specific activity on the symptoms of fatigue make the FSS an ideal tool for rehabilitation settings. The FSS was used in this study to assess general fatigue in the study's subject population.

3.4.2.5 Daily Log

While all other outcome measures were performed on a bi-weekly basis, the research team recognized the importance of obtaining from the participants their interpretations of the effects of the program on a daily basis. The daily log provided the investigators with subject's daily ratings of spasticity, pain, fatigue, as well as quality of relaxation, sleep, digestion, bladder/bowel health, and overall wellbeing. The intent of this was to identify any notable changes over the course of the one-month program. These were documented on a 10-point Likert scale, for example 1="I feel the best I have ever felt", to 10= "I feel the worst I have ever felt"). A copy of the Daily Log can be found in Appendix V.

Subjects were asked to provide responses each day that reflected their perceptions for that day. The log was returned to the investigators at the completion of that week, and subjects were given a new log. This was done in an attempt to minimize responder effect bias by preventing subjects from reviewing their previous responses.

3.4.3 Research Protocol

3.4.3.1 Familiarization

The principal investigator performed the initial interview with study volunteers. This consisted of acquiring demographic information, including age, level and year of injury, and current method of mobility. Potential participants were also asked open-ended questions to describe the muscle groups in which they experienced spasticity. Subsequently, volunteers took part in a 45-minute familiarization session on the Segway with a physical therapist and one other member of the research team. At this time, participants were shown how to perform some simple tasks (getting on/off, going forwards/backwards, turning, etc). They were then assisted onto the Segway, and the remainder of the session was spent practicing basic tasks of pendulum movements, negotiating and stopping on objects, and familiarizing themselves with the steering mechanism.

3.4.3.2 Testing Session 1 (T1)

Participants returned several days later to begin the study. A physician performed a clinical history examination pertaining to their injury and any other relevant medical information. All participants were asked to describe their usual standing habits and any current exercise and/or standing regimes and pharmacological interventions. It was requested that they attempt to avoid alterations to their daily routine and to notify investigators of any necessary changes. Participants underwent a baseline MAS test. Participants were blinded to the results of this test. Scores were also withheld from the examiner once they had performed each test.

Subsequently, participants rode the Segway for 30 minutes in the gymnasium of the rehabilitation centre. Simple tasks like navigating indoor hallways and stopping on floor targets were employed to maintain interest and provide the subject with small challenges. Upon completion of the 30-minute training session, a follow up MAS was performed by the same physician. The participants then completed the SCI-SET, POQ-VA and FSS.

Participants were asked to maintain a daily log with information on spasticity, pain, fatigue, relaxation and sleep patterns, appetite, and wellness. This was completed at home at approximately the same time each day. In an attempt to minimize any recall bias, each week they returned the 7-day log, and were given a new daily log to fill out for the upcoming week.

3.4.3.3 Follow up Sessions (T2 and T3)

Subsequent Segway training sessions followed at a frequency of 3 times per week. Training sessions were scheduled on a regular basis as was convenient for the subject over the course of the month. Reassessment of the baseline measures were done again at Day 6 (T2) and Day 12 (T3) of testing. A timeline is provided for clarification.

3.4.3.4. Timeline of Intervention. Each box represents one day of training over a four week period. Testing sessions are T1(baseline), T2 (Day 6), T3 (Day 12). MAS are taken pre/post Segway training session. All other measures are taken post training session.

WEEK 1	**T1** **MAS** **SEGWAY SESSION 1** **MAS, SCI-SET** **POQ-VA, FSS**	**SEGWAY SESSION 2**	**SEGWAY SESSION 3**
WEEK 2	**SEGWAY SESSION 4**	**SEGWAY SESSION 5**	**T2** **MAS** **SEGWAY SESSION 6** **MAS, SCI-SET** **POQ-VA, FSS**
WEEK 3	**SEGWAY SESSION 7**	**SEGWAY SESSION 8**	**SEGWAY SESSION 9**
WEEK 4	**SEGWAY SESSION 10**	**SEGWAY SESSION 11**	**T3** **MAS** **SEGWAY SESSION 12** **MAS, SCI-SET** **POQ-VA, FSS**

3.5 Data Analysis

Due to the non-parametric nature of the MAS, Wilcoxon signed rank tests were performed to analyze pre- and post intervention MAS values (1x2) and over time (1x3). A 2x3 analysis of variance (ANOVA) with repeated measures was employed to examine changes over time for the SCI-SET, POQ-VA, and FSS data. Daily log values were averaged to provide weekly means and then compared. SPSS v16.0 software was used.

3.6 Results

Nine subjects (aged 33-61) enrolled in the one month training program. One subject withdrew voluntarily due to a family emergency, and their incomplete data was removed from the analysis. Eight subjects completed all sessions on schedule. Demographic information is shown in Table 3.2.

Table 3.2 Subject Demographics. Subjects are categorized descriptively by sex, age, injury level, ASIA classification, year(s) since injury, daily medication, current physical activities and mobility aids.

Sub	Sex	Age	Injury Level	ASIA	Year(s) since Injury	Daily Meds	Current Activities	Mobility Aids
1	M	48	C5	C/D	24	Flouexetine	Walking	Cane, L AFO
2	M	35	T11	A	7	Baclofen (oral) Botox (rectus fem), Novotrimnol, Vesicare	Brace walking	Forearm crutches, HKAFOs, Manual chair
3	M	52	C5	C	7	GABAp, Baclofen (oral) Nortripaline	Walking, Gym	Cane
4	M	33	C5	C	15	Baclofen (oral)	Gym, yoga, stretching	Manual chair, Forearm crutches
5	M	41	T5	B	6	Baclofen (intrathecal), Pariet, Citalopram	Walking	Walker, HKAFOs, Manual chair
6	M	54	C6	D	29	Baclofen (oral), Diazepam	Walking	Cane
7	F	54	C5	C	4	Botox (pectoralis)	Standing frame	Power chair, Walker
8	M	36	T6	C/D	18	NSAIDS	Gym, WC training	Manual chair, L AFO, Forearm crutches
9	F	61	C5	D	1	Baclofen (oral), vitamins	Walking, stretching	Manual chair, Walker

Participants experienced spasticity in a myriad of upper and lower extremity muscles, with the plantarflexors being most common. Self-identified spastic muscle groups are shown in Table 3.3.

Table 3.3 Self-identified Spasticity. Subjects' top three muscle groups (right or left) in which they experience spasticity. R=right, L= left, Fin Flex= finger flexor, An Plfl= ankle plantarflexor, Hip Ext= hip extensor, Hip Add= hip adductor, Kn Flex= knee flexor, Kn Ext= knee extensor, Wr Ext= wrist extensor, For pron= forearm pronator.

SUB	M1	M2	M3
1	L FIN FLEX	L AN PLFL	L FOR PRON
2	L HIP ADD	R HIP ADD	R AN PLFL
3	R HIP EXT	R KN FLEX	R FIN FLEX
4	R HIP EXT	L AN PLFL	R AN PLFL
5	L KN EXT	L AN PLFL	R AN PLFL
6	R FIN FLEX	L HIP ADD	R HIP ADD
7	L HIP ADD	R HIP ADD	R AN PLFL
8	L WR EXT	R WR EXT	R FIN FLEX

3.6.1 Immediate (Pre-Post) Intervention Effects

Results of clinically-measured pre-post intervention spasticity using MAS were significantly reduced for Muscle 1 (p=.001), Muscle 2 (p=.001) and Muscle 3 (p=.001). All subjects experienced improvements in MAS ratings of two or more muscles in the pre-post tests, with scores dropping as much as three grades (score of '3' to score of '1' in S1) after a single trial, while others had no change; no subjects had an increase in MAS following Segway training. Two subjects (S1 and S2) showed improvements in every pre-post intervention for all three trials. Data for all subjects is shown in Table 3.4.

Table 3.4 Pre-post intervention MAS Scores. Same-day scores across all 3 trials (T1, T2, T3) for the three self-identified muscles (M1, M2, M3). Improvements are shown in bold.

SUB	TEST	M1_PRE	M1_POST	M2_PRE	M2_POST	M3_PRE	M3_POST
S1	T1	3	**1**	3	**1**	2	**1**
	T2	3	**2**	3	**2**	1	**0**
	T3	3	**1.5**	2	**1.5**	1	**0**
S2	T1	1.5	**1**	1.5	**1**	3	**2**
	T2	1.5	**0**	1.5	**0**	1.5	**1**
	T3	1.5	**1**	1.5	**0**	1.5	**1**
S3	T1	2	**0**	3	**2**	1	1
	T2	2	**1.5**	2	2	1	**0**
	T3	0	0	2	**1**	1.5	**1**
S4	T1	1.5	**0**	0	0	3	3
	T2	1	**0**	1.5	**0**	2	**1**
	T3	0	**0**	1	**0**	1	**0**
S5	T1	2	2	2	**1.5**	1.5	**1**
	T2	2	2	1	1	1.5	**1**
	T3	3	**2**	2	**1**	2	**1.5**
S6	T1	3	**2**	2	2	2	2
	T2	3	**2**	2	**1.5**	2	**1.5**
	T3	3	**1.5**	2	**1.5**	2	**1.5**
S7	T1	1.5	**1**	2	**1.5**	3	**1.5**
	T2	1	**0**	1.5	**0**	1.5	1.5
	T3	1.5	**1**	1.5	**1**	2	**1.5**
S8	T1	2	**1.5**	1.5	1.5	3	**2**
	T2	3	**2**	1	**0**	3	**2**
	T3	2	**1.5**	2	**1.5**	3	**2**

3.6.2 Between Session (Over Time) Intervention Effects

3.6.2.1 MAS

Modified Ashworth scores of spasticity over one month were less consistent. Statistical analysis was not significant for Muscle 1 (p=.114), Muscle 2 (p=.211) or Muscle 3 (p=.354). However, all participants with the exception of S8 demonstrated a reduction in their pre-intervention MAS values over time (T1-pre to T3-pre) for at least one muscle group. Several experienced no change (S2, S6, S7, S8) in at least one

muscle group, and S2, S3, S4, S5 and S8 had an increase, in at least one muscle group.

Between session responses are shown in Table 3.5.

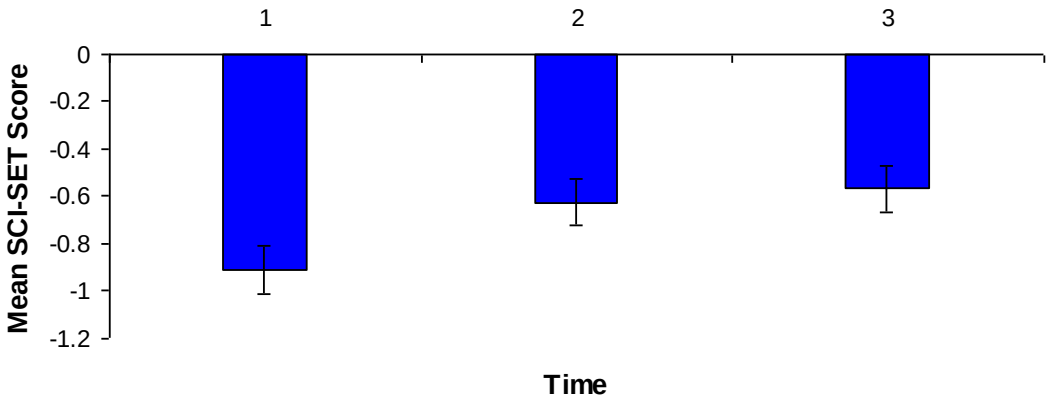
Table 3.5 Between session intervention MAS Scores. Pre-intervention MAS scores over time (T1 and T3) for the three self-identified muscles (M1, M2, M3). Improvements are shown in bold.

	M1		M2		M3	
SUB	T1	T3	T1	T3	T1	T3
S1	3	**2**	3	**1**	2	**1**
S2	1.5	3	1.5	1.5	3	**1.5**
S3	2	**1**	2	**1**	1	1.5
S4	1.5	3	0	2	3	**1**
S5	2	**1.5**	2	1.5	1.5	2
S6	3	**2**	2	2	2	2
S7	1.5	3	2	**1.5**	3	**2**
S8	2	3	1.5	3	3	3

3.6.2.2 SCI-SET

Differences in mean SCI-SET scores between testing sessions were not statistically significant (p=.133), but all subjects showed improvements in the scores over time except one (S3). Mean scores improved from -0.91 (±0.30 Standard error of the mean [SEM]) at baseline (T1) to -0.63 (±0.24 SEM) for mid-month (T2) and again at T3 -0.57 (±0.24 SEM). Figure 3.1 presents a graphical representation.

Figure 3.1 Mean SCI-SET Scores

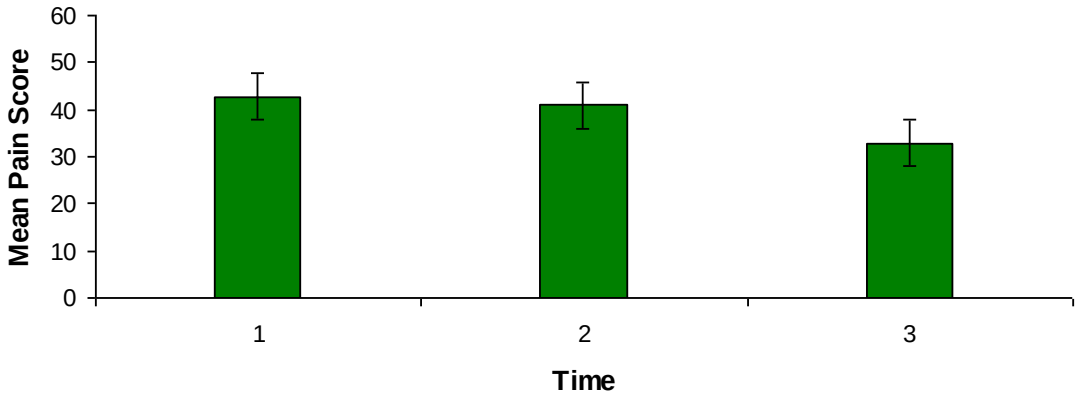


Mean SCI-SET scores over time for T1, T2 and T3. Scores were not significantly reduced between T1 and T3 (p=.133). Error bars indicate standard error of the mean.

3.6.2.3 POQ-VA

Reductions in mean pain scores were statistically significant (p=.027) from T1 to T3. Over time, mean POQ-VA scores decreased from T1 (42.75±8.49 SEM), to T2 (40.88±10.10 SEM) and further for T3 (32.88±7.17 SEM). See Figure 3.2 for POQ-VA mean scores.

Figure 3.2. Mean Total Pain (PTOT) Scores

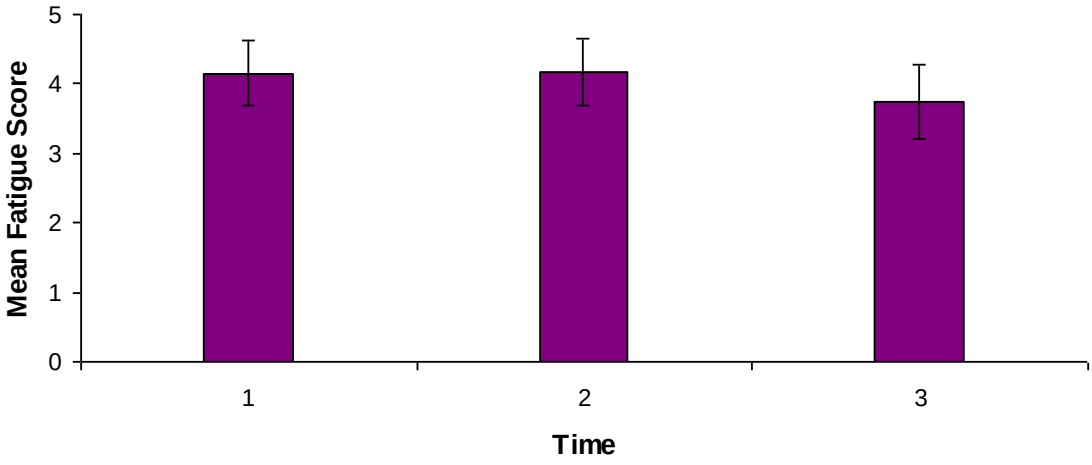


Total pain mean scores over time for T1, T2 and T3. Scores were significantly reduced (p=.027) between T1 and T3. Error bars indicate standard error of the mean.

3.6.2.4 Fatigue results

ANOVA values for FSS scores over time were not statistically significant (p=.122), however mean FSS scores demonstrated an improvement from T1 (4.2±0.47 SEM) to T3 (3.7±0.54SEM). Six subjects (S1, S2, S4, S5, S6, S7) all reported feeling less fatigue by the completion of the study as per the FSS. One subject (S3) had increased fatigue, and one (S8) had no appreciable change between the first and final testing sessions. See Figure 3.3 for mean FSS scores.

Figure 3.3 Mean FSS Scores

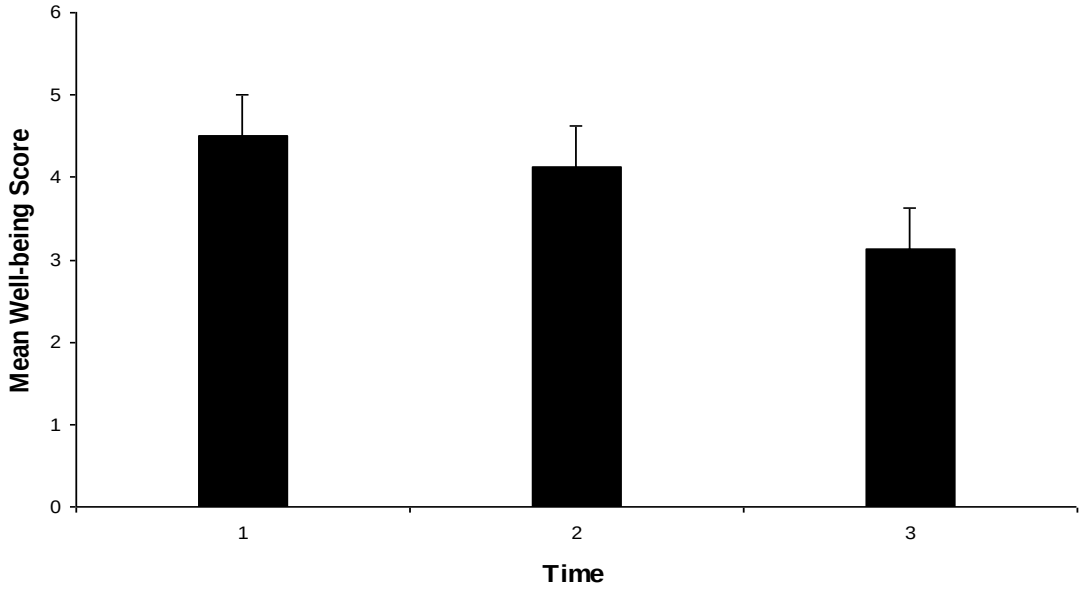


Mean Fatigue Severity Scale scores over time for T1, T2 and T3. Scores were not significantly reduced (p=.122) between T1 and T3. Error bars indicate standard error of the mean.

3.6.2.5 Daily Log

Improvements in overall well being over the course of the intervention were statistically significant (p=.001). Score improved from 4.5±0.42 SEM) to 3.1±0.48 SEM). Daily ratings of pain showed that six (S2, S3, S5, S6, S7, S8) of the eight subjects reported that the intensity of their pain decreased, while four subjects (S2, S3, S6, S8) noted reduced frequency of pain episodes. Fatigue ratings varied, with four subjects noting decreases (S2, S4, S6, S7), three showing increases (S1, S3, S5) and one with no change (S8). Daily self-evaluations of spasticity frequency and intensity remained constant. No notable changes were observed in self-ratings of sleep, appetite, digestion or bladder or bowel function over the course of the intervention. See Figure 3.4 for mean Overall Well Being daily log scores.

Figure 3.4. Mean Daily Log Overall Well Being Scores



Daily Log mean scores over time (T1, T2, T3). Error bars indicate standard error of the mean. Improvements are indicated as scores decrease (1=I feel the best I have ever felt, to 10= I feel the worst I have ever felt.)

3.7 Discussion

A dynamic standing program using the Segway showed statistical improvements for several outcome measures including MAS for spasticity (immediate effect), POQ-VA for pain (p=.027) and Daily Log for overall well-being (p=.001). Although other variable failed to achieve statistical significance (possibly attributable to small sample size) several interesting changes were apparent.

3.7.1 Spasticity

With respect to spasticity, the immediate effect of the intervention was evident in the entire pool of participants across all three muscles (p=.001). MAS scores decreased immediately following the intervention (pre- post-Segway intervention) in at least two of the three muscle groups they identified as being spastic. Scores improved by as much as three MAS grades after a single trial, or remained constant; no scores increased. Two subjects (S1 and S2) showed improvements in every pre-post intervention for all three trials.

Whether or not a dynamic standing program can reduce spasticity over time remains yet to be determined. There were no apparent changes in MAS scores across trials for preliminary values (T1-pre vs. T3-pre) for each of the three affected muscle groups. Several subjects noted decreases in frequency (S2, S4, S6) and intensity (S2, S6) of spasticity in the daily log and no subjects showed an increase. Although not statistically significant, SCI-SET scores indicated a reduced negative effect of spasticity in our subjects, tending to become less negative over the course of the study in all subjects except one (S3). Despite the fact that our participants scored the impact of their

spasticity as overwhelmingly negative, several did allot positive scores to a number of items. In both instances this was related to some form of weight bearing activity (transfers or walking), which lends credibility to the notion that responses for the SCI-SET may vary based on the upper versus the lower extremity (Boutilier et al., 2009). If there is a connection between upper extremity spasticity and loss of independence, highly negative SCI-SET scores could reflect this. Conversely, lower extremity spasticity may be of assistance in weight bearing, and thus be perceived as less disempowering, receiving more positive scores (Boutilier et al., 2009). Should this distinction exist, this knowledge would be useful in disseminating the degree of impact that spasticity has on the individual. Positive ratings would have been overlooked had we used a traditional self-report Spasm Frequency Scale or single-item scale to measure overall spasticity. In light of these findings, we have minimal, yet relevant support for the application of this bidirectional scale to measure self-report impact of spasticity. The SCI-SET may offer greater sensitivity in determining functional impact than the MAS, and we recommend that clinicians and researchers incorporate this measure as a fundamental means of interpreting the implications of spasticity in individuals with SCI.

While the MAS has been criticized for measuring one entity of spasticity (muscle tone), it provides an objective, responsive measure of spasticity, and it remains the most common tool in clinical practice and research to measure spasticity (Pandyan et al., 1999). Therefore it is necessary to include it. Rather than testing certain muscle groups commonly found in the literature (which are by-and-large the lower extremity groups) we chose a patient-centered approach, having subjects self-identify three muscle groups including the upper extremity in which they most notably experience spasticity. Given the varied preservations and functional abilities of individuals with SCI, we feel that this was more representative of the individual nature of the condition. Choosing to include a

heterogeneous population (ASIA A-D) makes these findings more generalizable across the population of individuals with SCI who experience spasticity.

3.7.2 Pain

Self-evaluations of pain were significantly reduced in all subjects (p=.027). Analysis of the sub-categories showed variations among subjects, however 6 subjects reported feel less negatively affected (i.e. “feelings of dysphoria and associated symptoms”). This phenomena was further supported by daily log contributions, which reported reductions in pain intensity in 75% (6 of 8) of the subjects. Half of the subjects also reported decreases in the frequency of pain bouts. Both S1 and S4 failed to report improvements in their daily log for either frequency or intensity of pain, however, both scored these very low across trials (1 and 0 respectively), indicating that pain likely plays a minimal role in their daily lives.

Though the POQ-VA does not differentiate the various types of pain, we do anticipate that it would account for both physical (nociceptive and neuropathic) and psychological aspects in this population. The immediate reductions in spasticity may also provide some explanation, as sensory projection neurons and segmental reflex pathways often overlap (Ashby, 1975). As such, decreased tonic reflex hyperexcitatbility may influence central spinal pain sensory systems. Decreased pain may also be a result of positional changes (including muscle stretch and visceral organ realignment), increased postural muscle activation and variations in cutaneous feedback with Segway training. Requiring less energy to operate than a manual wheelchair, cane or forearm crutches (Sawatzky et al., 2009), we hypothesize that pain which interferes with function would be less of an issue with the Segway, and this may account for some of the improvements in pain scores. Finally, perhaps these notable improvements in pain are

attributable not only to a physical effect, but as well a positive mental state and socialization experiences.

3.7.3 Fatigue

Fatigue Severity Scale scores were diminished in six of the eight subjects, with the group mean score falling from 4.2 (baseline), 4.2 (mid-study) to 3.7(final). As an FSS score of ≥=4 is considered clinically significant, this discrepancy suggests that subjects were clinically fatigued after the first and second interventions, but not the final score. Perhaps it is not surprising to note that greater changes were seen in fatigue from T2 to T3. Becoming acquainted with the Segway, subjects may have been engaging unfamiliar postural muscles which may have atrophied with seating. If fatigue is equated with exertion (which is no doubt oversimplifying), this may explain minimal changes within the first two weeks of their involvement, however, with continued use these muscle groups may have gained sufficient strength to impact the final test results. Additional benefits such as increases in physical activity, mental alertness and social enjoyment as well as reductions in prolonged seating time may also contribute to improvements in fatigue scores.

Three subjects (S1, S3, and S5) reported increases in fatigue as measured by the daily log; however only in the case of S3 was this consistent with increases in FSS score. Interestingly, S3 had no change in SCI-SET scores, and was the only subject to report anecdotally that his spasms were not affected positively by his involvement in the study. However, he did show improvements in MAS scores (both immediately and over time) and his pain (POQ-VA, frequency and intensity) and overall well being all improved as a result of his participation. We were additionally surprised by the increased accounts of daily fatigue, as compared with FSS. We might have anticipated the fatigue ratings

immediately following the intervention to be higher due to exertion, with weekly fatigue levels being less affected.

Several of our outcomes measures may be interconnected, and this relationship must be acknowledged as we are interpreting them as mutually exclusive. Qualitative interviews from a study of people with SCI by Hammell et al. (2009) identified pain and secondary sequale including spasticity as contributing to fatigue. Participants in this study suggested that increases in either of these two variables would impact their fatigue levels. While we have no reason to expect 100% correlation between these outcome measures, we appreciate that they are interrelated phenomena in individuals with SCI and therefore some of the variability may be captured by all three.

3.7.4 Daily Log

All of our subjects with the exception of one (S8) reported improvements in overall well-being at the completion of the study and this was significant (p=.001). However, S8 is a recently injured older adult (just 1 year post injury), and therefore, while physically stable, may yet be coping with life adjustments and emotional turmoil. Daily reports of spasticity, pain and fatigue have been discussed previously. Sleep, appetite, digestion, bladder or bowel health remained relatively constant in self-report ratings. This is in contrast to the conclusions of Eng et al. (2001) who found bladder and bowel health to be one of the most dramatically influenced outcome measure of a standing program.

We are cautiously optimistic regarding the improvements we found in overall well being. Perhaps again interdependence exists between overall wellness and other outcome measures such as spasticity, pain and/or fatigue in that improvement in one area may spill over into others. Another possibility is that subjects received positive

feedback and were encouraged by their participation in an intervention study on a novel device that received some news coverage and attention from pedestrians and other researchers at the centre where the study took place. This may have contributed to the subject's sense of self-esteem and personal enjoyment. Many expressed their disappointment at the completion of the study and expressed interest in increasing the duration of their involvement. A longer duration of investigation with a larger sample size might yield some answers to these complex possibilities.

3.7.5 Protocol

As we recruited individuals with a myriad of ability levels, it was necessary that the Segway protocol reflect the varying standing tolerances of subjects. Additionally, in light of the time commitment required, we were concerned that recruitment and feasibility of the study would have been compromised had we extended the duration of the sessions. Positive feedback from several participants who requested extending the 30-minute session would support lengthening the study to explore the effect over a longer period of time. However, several subjects appeared fatigued near the end of their sessions, and others noted a slight increase in back and/or shoulder pain during their training sessions which may be attributable to atrophy of postural muscles as a result of prolonged sitting. One participant (S8) was unable to complete the entire 30 minute session, often requiring several minutes of rest (getting off the device and sitting down included) every 10-15 minutes during the length of her involvement in the study. It is interesting to note that this individual was the most recently injured (just over one year post), so perhaps they had not yet experienced as significant regains strength as some of the others. This may be reflected in her fatigue scores (FSS and daily log) in that

while they did not increase over the course of the intervention, neither did they follow the propensity of others to decrease.

3.7.6 Passive versus Dynamic Standing

It may be debated that the positive results from this study are merely due to the fact that these participants had to stand. Standing frames produce passive stretch for muscles and viscoelastic joint structures, and rely on skeletal support systems to transmit body weight. While reductions in spasticity have been associated with standing frames in SCI (Odeen et al., 1981, Kunkel et al., 1993, Eng et al., 2001), these studies rely on subjective self-report measures and none have drawn a link to the examiner-based assessment (MAS), nor have they compared various self-report ratings. Additionally, standing frames have occasionally been implicated in increases in spasticity (Eng et al., 2001). All of the patients we enrolled were already participating in standing programs on a weekly basis, or did some household ambulation, yet most subjects reported to the investigators that their spasms were reduced with Segway use.

In addition to passive stretch and weight bearing, the Segway involves the vestibular system to a much greater degree. Muscle spindles and joint receptors relay proprioceptive feedback to the cord for integration, and cutaneous receptors in the feet transmit information regarding the position of the platform. Visual information is required for steering, and dynamic muscle activations are constantly occurring to produce postural adjustments. Thus, the individual is challenged, and yet still an allowance for deficits exists. Further, standing frames are static and impractical for use outside a rehabilitation facility. Conversely, the Segway enables freedom of movement and independence in addition to these physiologic improvements. There may be alternative

explanations to these effects which may implicate vibration or involve the vestibulospinal system.

Although considerably encouraged by the results of this study, the investigators are reluctant to speculate at this time as to the underlying mechanisms of the phenomena observed. Some evidence exists to support the use of whole body vibration (WBV) to reduce spasticity. Ness and Field-Fote (2009b) have pilot data that suggest that a 12-session intervention of upright WBV decreased spasticity in the quadriceps muscle (measured using a Pendulum test). Ahlborg et al. (2006) reported improvements in muscle strength and reductions in spasticity of the knee extensor muscles with WBV. However, preliminary data suggest that the frequency of vibration of the Segway lies within the range of 2-5Hz, which is much lower than the 50Hz used in these studies, therefore, the evidence for this explanation remains unconvincing.

Another fascinating area to explore involves the role of dynamic stability and implication of an override of reflex activity. The vestibulospinal pathways that regulate extensor tone do not require cortical input, and therefore may remain intact in individuals with SCI (Liechti et al., 2008). Vestibulospinal activity can be initiated by inputs activated by a change in relative head position and/or afferent inputs from the limbs (Horak et al., 2001). A standing frame requires relatively little voluntary motor activity or cortical modulation for an individual who is passively supported. Conversely, maintaining a dynamic equilibrium (such as on the Segway) requires significant activation of leg muscles and co-contraction strategies to generate postural adjustments (Horak et al., 1986), particularly with respect to a hip control strategy, for which the vestibular system is intimately involved (Horak et al., 1990). These dynamic adjustments may modify the descending drive to the spinal cord (e.g. vestibulospinal) via spino-bulbo-spinal pathways. Descending activity within pathways such as the vestibulospinal system can

change or modulate the excitability of spinal reflex pathways involved in spasticity (Liechti et al., 2008).

Many tasks of daily living require significant energy output (van der Woude et al., 1997), and are likely to be facilitated by standing. If minimal reductions in power output during activities of daily living can lead to significant changes in functional capacity (Janssen et al., 1996), then the reduced effort to use the Segway may allow the user to preserve energy which would otherwise be expended for mobility. If the Segway could replace time-consuming stretching or exercise programs, as several subjects suggested, while facilitating the completion of ADLs, the time and energy saved could be redirected to recreational or occupational targets. In light of the importance of physical activity in the lives of individuals with SCI, this energy-saving option may reduce the limitations and barriers to participation in physical activity programs and community events. It is noteworthy that at least three of our subjects have since purchased Segways for personal use.

With positive benefits universally reported by the subjects, this dynamic standing program is promising. No harmful effects have been documented in previous work (Sawatzky 2007, 2009) or in the present study. Improvements in overall health in addition to physiologic improvements affords support for this exciting new mobility device and may further its' acceptance in both the scientific and rehabilitation communities.

3.8 Limitations

As seen in our data, statistically significant decreases in spasticity and pain, as well as improvements in sense of overall well being were seen in a small sample of individuals with SCI. However, ratings of fatigue, the impact of spasticity, and muscle scores over time were not significant. Our study had a small sample size. Our goal was

20 but we only managed to recruit eight subjects. This was partly due to the stringent eligibility criteria, many volunteers were excluded because they did not have a spinal cord injury, did not have spasticity or were injured too recently. A multi-centre study would help provide a larger sample as well as broaden the results generalizability to other regional areas.

To be able to participate in our study, subjects had to be able to stand or walk with or without assistance. As mentioned previously, all did stand or walk but we did not record specifically how much was done during the week. In retrospect, the investigators might have asked participants to keep a continuous record of their standing regime over the course of the study in order to fully differentiate the physiologic implications of the Segway from their regular standing programs. In addition, information about their standing for at least one month pre- and post- may have been useful. Finally, one-month follow up values for both clinical and self-report measures would provide the investigators with some idea of any changes the participant's may have experienced without the Segway intervention. We would suggest single subject design, in which investigators could take baseline values, followed by intervention data and then a second baseline value during a specified time frame.

Two examiners were used for feasibility of the study; however the inter-rater reliability of the modified Ashworth scale remains questionable. Data produced by both examiners, however, yielded the similar findings, which added credence to the outcome. Ideally a single observer for MAS should be used but for our study was not feasible.

Should vibration play a role, we acknowledge that over varying surfaces, vibration felt by the rider would fluctuate (Griffin, 1990, Vorrink et al., 2008). And as the rider must mount, dismount and stop and start often, constant velocity is not an option. However, the examiners endeavored to minimize this effect by maintaining the device on

the lowest speed (6km/hr) while the subjects trained. By placing a ceiling on the speed we hoped to at least prevent the apparatus from vibrating beyond a certain amount.

Self- reports may have a tendency to elicit honest, yet positively biased responses ('self-deceptive enhancement') (Paulhus, 1991) In addition, the perceived benefits to the Segway may have been overestimated as there remains a novelty factor to the device, as it is not yet legal for use on the streets and sidewalks of the city where the study was performed.

3.9 Conclusions

In addition to providing an enjoyable socialization experience, the Segway PT may provide short term reductions in spasticity as measured by MAS. However, long term benefits in spasticity are not as apparent. There is some evidence to suggest benefits in pain and fatigue may have lasting effects over a month, but further investigations of a longitudinal nature are required to support this notion. Perhaps the Segway serves as therapeutic device by introducing a stimulus to the system which overrides spasticity in some capacity. Future research is needed to explore these mechanisms in detail, specifically the effect of vibration or reflex-modulation to determine whether neural contributions are involved in the physiologic changes seen with the Segway. Moreover, this research may wish to incorporate a control group or single subject repeated measures design to eliminate the effect of standing. We further suggest that future research investigate whether the physiologic effects of the Segway extend to other populations with mobility impairments, and if Segway training might replace other therapeutic programs, such as stretching.

3.10 References

1. ACSM Exercise management for Persons with Chronic Diseases and Disabilities. 3rd ed. Durstin JL, Moore GE, Painter PL, Roberts SO (eds). Chapter 39, Figoni SF.Spinal Cord Disabilities: Paraplegia and Tetraplegia. pp.298-303. Human Kinetics: Champaign, IL.

2. Adams MM, Hicks AL. (2005). Spasticity after spinal cord injury. Spinal Cord. 43:577-86.

3. Adams MM, Martin Ginis KA, Hicks AL. (2007). The Spinal Cord Injury Spasticity Evaluation Tool: Development and Evaluation. Archives of Physical Medicine and Rehabilitation. 88:1185-1192.

4. Ahlborg L, Andersson C, Julin P. (2006). Whole-body vibration training compared with resistance training: effect on spasticity, muscle strength and motor performance in adults with cerebral palsy. Journal of Rehabilitation Medicine. 38:302-308

5. Anton HA, Miller WC, Townson AF. (2008). Measuring Fatigue in Persons with Spinal Cord Injury. Archives of Physical Medicine and Rehabilitation. 89:538-542.

6. Ashworth B. (1964). Preliminary trial of carisoprodal in multiple sclerosis. Practitioner. 192:540-542.

7. Bohannon RW. (1993). Tilt table standing for reduced spasticity after spinal cord injury. Archives of Physical Medicine and Rehabilitation. 74:1121-1122.

8. Boutilier G, Finlayson H, Denison I., Wiefelspuett S., Grant C, Sawatzky BJ. (2009). Correlation of the SCI-SET and Modified Ashworth Scale for upper and lower extremity muscles in spinal cord injury. Submitted for publication to….

9. Clark ME, Gironda RJ, Young RW. (2003). Development and validation of the Pain Outcomes Questionnaire-VA. Journal of Rehabilitation Research and Development. 40:381-396.

10. Cybulski GR, Jaegar RJ. (1986). Standing performance of persons with paraplegia. Archives of Physical Medicine and Rehabilitation. 67:103-108

11. Dietz V.(2000). Spastic movement disorder. Spinal Cord. 38:389-93.

12. Ditunno JF, Formal CS. (1994). Chronic spinal cord injury. New England Journal of Medicine. 330:550-556.

13. Duffus A, Wood J. (1983). Standing and walking for the T6 Paraplegic. Physiotherapy. 69:45-46.

14. Dunn RB, Walter JS, Lucero Y, et al. (1998). Follow up assessment of standing mobility device users. Assistive Technology. 10: 84-93.

15. Eng JJ, Levins SM, Townson AF, Mah-Jones D, Bremner J, and Huston G. (2001). Use of Prolonged Standing for Individuals with Spinal Cord Injury. Physical Therapy. 81:1392-1399.

16. Eng JJ, Miller WC. Rehabilitation: from bedside to community following spinal cord injury (SCI). Spinal Cord Injury Rehabilitation Evidence. (http://www.icord.org/scire/pdf/SCIRE_CH1.pdf). Accessed September 9, 2008.

17. Fawkes-Kirby TM, Wheeler MA, Anton HA, Miller WC, Townson AF,Weeks CAO. (2008). Clinical correlates of fatigue in spinal cord injury. Spinal Cord. 46: 21–25.

18. Glaser RM. The Physiology of Exercise. (1996). In: Apple DJ (ed). Rehabilitation Research and Development Service- Physical Fitness: A Guide for Individuals with Spinal Cord Injury. Baltimore: Department of Veterans Affairs. 3-21.

19. Griffin MJ. (1990). Handbook of human vibration. San Diego CA. Academic Press

20. Hammell KW, Miller WC, Forwell SJ, Forman BE, Jacobsen BA. (2009). Fatigue and spinal cord injury: a qualitative analysis. Spinal Cord. 47:44-49.

21. Hjeltnes N, Jansen T. (1990). Physical endurance capacity, functional status and medical complications in spinal cord injured subjects with long-standing lesions. Paraplegia. 28:428-432.

22. Hoffman MD. ((1986). Cardiorespiratory fitness and training in quadriplegics and paraplegics. Sports Medicine. 3:312-330.

23. Horak FB, Nashner L (1986) Central programming of postural movements: adaptation to altered support surface configurations. Journal of Neurophysiology. 55:1369–1381

24. Horak FB, Nashner LM, Diener HC.(1990). Postural strategies associated with somatosensory and vestibular loss. Experimental Brain Research. 82:167-77.

25. Horak FB, Earhart GM, Dietz V. (2001). Postural responses to combinations of head and body displacements: vestibular-somatosensory interactions. Experimental Brain Research. 141:410–414

26. Hsieh JTC, Wolfe DL, Connoly S et al. Spasticity following spinal cord injury. . Spinal Cord Injury Rehabilitation Evidence. (http://www.icord.org/scire/pdf/SCIRE_CH1.pdf). Accessed September 9, 2008.

27. Issekutz, B, Blizzard JJ, Birkhead NC, Rodahl K. (1966). Effect of prolonged bed rest on urinary calcium output. Journal of Applied Physiology. 21: 1013:1020.

28. Jacobs PJ and Nash MS. (2001). Modes, benefits and risks of voluntary and electrically induces exercise in persons with spinal cord injury. Journal of Spinal Cord Medicines. 24:10-18.

29. Jacobs PJ, Nash MS. (2004). Exercise Recommendations for Individuals with Spinal Cord Injury. Sports Medicine. 34:727-751.

30. Janssen TW, VanOers CAJM, Rosendale EP, Willemsen EM, Hollanders AP, Van der Woude LHV. (1996). Changes in physical strain and physical capacity in men with spinal cord injuries. Medicine and Science in Sports and Exercise. 28(5): 551-559.

31. Kaplan PE, Roden W, Gilbert E, Richards L and Goldschmidt JW. (1981). Reduction of hypercalciuria in tetraplegia after weight-bearing and strengthening exercises. Paraplegia. 19:289-293.

32. Krupp L, LaRocca N, Muir-Nash J, Steinberg AD. (1989). The Fatigue Severity Scale: application to patients with multiple sclerosis and systemic lupus erythematosus. Archives of Neurology. 46: 1121–1123.

33. Kunkel CF, Scremin AME, Eisenberg B, Garcia JF, Roberts S, Martinez S. (1993). Effect of "standing" on spasticity, contracture, and osteoporosis in paralyzed males. Archives of Physical Medicine and Rehabilitation. 74:73–78.

34. Lance JW. (1980). What is spasticity? The Lancet. 335: 606.

35. Liechti M, Müller R, Lam T, Curt A.(2008). Vestibulospinal responses in motor incomplete spinal cord injury. Clinical Neurophysiology. 119: 2804–2812

36. Little JW, Merritt JL. (1988). In: DeLisa, JA, ed. Rehabilitation Medicine. Philadelphia: Lippincott: 430–447.

37. Little JW, Michlesesn P, Umlauf R, Brittel C. (1989). Lower extremity manifestations of spasticity in chronic spinal cord injury. American Journal of Physical Medicine and Rehabilitation. 68:32-36.

38. Leo K. (1985). The effects of passive standing. Paraplegia News. Nov. 45-47.

39. Miller WC. (2009). Personal correspondence. November 9, 2009.

40. Ness LL, Field-Fote EC. (2009b). Effect of whole-body vibration on quadriceps spasticity in individuals with spastic hypertonia due to spinal cord injury. Restorative Neurology and Neuroscience (in press).

41. Noreau L, Shepard RJ. (1992). Physical fitness and productive activity in paraplegics. Sports Medicine Training and Rehabilitation. 3:165-181.

42. Noreau L, Shepard RJ. (1995). Spinal cord injury, exercise and quality of life. Sports Medicine. 20:226-50.

43. Packer TL, Sauriol A, Brower B. (1994). Fatigue secondary to chronic illness: postpolio syndrome, chronic fatigue syndrome and multiple sclerosis. Archives of Physical Medicine and Rehabilitation. 75:1122-1126.

44. Pandyan AD, Johnson GR, et al. (1999). A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measure of spasticity. Clinical Rehabilitation. 13:373-383.

45. Paulhus DL. (1991). Measurement and control of response bias. In: Robinson JP, Shaver PR, Wrightsman LS, eds. Measures of Personality and Social Psychological Attitudes. . Academic Press: Orlando.

46. Petajan JH. (1998). Fatigue and rehabilitation of neurologic disorders. In Lazar RB (ed.) Principles of Neurologic Rehabilitation. Chicago: McGraw-Hill. Pp.367-399.

47. Phillips L, Ozer MN, Axelson P, Chizeck H. (1987). Spinal Cord Injury: A Guide for Patient and Family. Raven Press: New York.

48. Odeen I, Knutson E. (1981). Evaluation of the effects of muscle stretch and weight load in patients with spastic paraplegia. Scandinavian Journal of Rehabilitative Medicine. 13:117-121.

49. Ozer MN, Britell CW, Phillips L. (1987). Chronic Pain, Spasticity and Autonomic Dysreflexia. In: Spinal Cord Injury: A guide for Patient and Family. New York, Raven Press:135-146.

50. Ozer, MN. (1988). The Management of Persons with Spinal Cord Injury. Demos Publications, New York. Chapter3, pp. 69.

51. Satkunam L. Spasticity. 5th Canadian Comprehensive Review Course in Physical Medicine and Rehabilitation. Toronto: April 2008.

52. Sawatzky B, Denison I, Langrish S, Richardson S, Hiller K, Slobogean B. (2007). The Segway Personal Transporter as an Alternative Mobility Device for People with Disabilities: A Pilot Study. Archives of Physical Medicine and Rehabilitation. 88:1423-1428.

53. Sawatzky B, Bishop CM, Miller WC, SCIRE Research Team. (2008). Classification and measurement of pain in the spinal cord-injured population. Spinal Cord. 46:2-10.

54. Sawatzky B, Denison I, Tawashy A. (2009). The Segway for People with Disabilities: Meeting Clients' Mobility Needs. American Journal of Physical Medicine and Rehabilitation. 88:484-90.

55. Sawatzky BJ, Denison I., Tawashy A, Boutilier G. (2009). The Segway Personal Transporter as an Alternative Mobility Device for People with Disabilities. Presented at the 4th International State-of-the-art Congress Rehabilitation: Mobility, Exercise & Sports. Amsterdam, the Netherlands. April 2009.

56. Shields RK, Dudley-Javoroski S. (2005). Monitoring standing wheelchair use after spinal cord injury: A case report. Disability and Rehabilitation. 27(3):142-146.

57. Siddall PJ, Yezierski RP, Loeser JD. (2000). Pain following spinal cord injury, clinical features, prevalence and taxonomy. IASP Newsletter. 3:3-7.

58. Sköld C.(2000). Spasticity in spinal cord injury: self- and clinically rated intrinsic fluctuations and intervention-induced changes. Archives of Physical Medicine and Rehabilitation. 81:144-9.

59. St George CL. (1993). Spasticity. Mechanisms and cursing care. Nursing Clinics of North American. 28:819-827.

60. Tawashy AE, Eng JJ, Lin KH, Tang PF, Hung C. (2009). Physical activity is related to lower levels of pain, fatigue and depression in individuals with spinal-cord injury: a correlational study. Spinal Cord. 47:301-6.

61. Van der Woude LH, Botden E, Vriend I, Veeger D. (1997). Mechanical advantage in wheelchair lever propulsion: effect on physical strain and efficiency. Journal of Rehabilitation Research and Development. 34:286-94.

62. Vorrink SNW, Van der Woude LH, Messenberg A, Cripton PA, Hughes B, Sawatzky BJ. (2008). Comparison of wheelchair wheels in terms of vibration and spasticity in people with spinal cord injury. Journal of Rehabilitation Research and Development. 45:1269-1280.

63. Yeo JD, Walksh S, Rutkowski S, Soden R, Craven M, Middleton J. (1998) Mortality following spinal cord injury. Spinal Cord. 36:329-336.

CHAPTER 4- IMPLICATIONS AND CONCLUSIONS

4.1 General Findings

Spasticity has a multifaceted impact on daily living in the lives of individuals with SCI. It may be perceived by these individuals as beneficial or problematic, depending on the extent of spasms and the goals of movement. This research has attempted to provide support for a bidirectional self-evaluation measurement tool of spasticity and was a first attempt to establish its value within an intervention study. Additionally, an effort was made to incorporate a comprehensive range of measurement tools within a single intervention study. These measures were used to evaluate the potential physiologic and psychological benefits of a new method of mobility that has been gaining popularity within the spinal cord injury community.

A correlation between the clinical measures of muscles of the upper extremity and the self-report SCI-SET may afford some evidence to suggest a psychological link between upper extremity function and feelings of independence and empowerment. The lack of correlation between clinical measures and self-report ratings of spasticity in the lower extremity muscle groups may suggest that spasticity in these muscles may be perceived as more beneficial, which is plausible in light of their contributions to weight bearing. Regardless, we advocate the need for clinicians to afford equal weighting to these separate components of the individual variability. The instantaneous examiner-based representations of passive resistance to movement provided by the modified Ashworth scale provide a quantitative value upon which to track change. However, self-evaluations, both present and weekly recall enable clinicians to have a more comprehensive picture of spasticity as it affects daily tasks. The potential for beneficial

outcomes of spasticity on quality of life should have an impact upon decisions regarding management of individual cases, and on the research upon which these may be based.

The Segway PT® is a novel, yet practical mobility tool which has yet to garner widespread support in the SCI population. It requires minimal functional ability (Sawatzky et al. 2007), and is an energy-efficient, environmentally-friendly option. Short term reductions in spasticity, pain and fatigue were demonstrated in a population of individuals with SCI of various ASIA classifications. Additional research is needed to appreciate whether these benefits could be extended to longer periods with increased daily use of the Segway. Moreover, these investigations should attempt to uncover the underlying mechanisms which have facilitated these positive changes.

4.2 Measurement Standards

Two diverse measures were compared to investigate whether there was any common variance. Lack of absolute correlation between the two measures suggests they may measure similar yet distinct components of the overall phenomenon of spasticity. Clinician's interpretations of passive resistance to movement represent a single benchmark entity of tone. Conversely, patients' qualitative descriptions of spasticity may be more representative of 'sensory spasticity' which includes pain and other varied sensations in addition to increased muscle tone and resistance to movement (Sjölund, 2002). The clinician's interpretation is that similar MAS scores represent similar levels of tone; however this may not necessarily translate to the actual impact the patient perceives.

4.2.1 The MAS

The MAS has been utilized frequently in research involving individuals with various neuromuscular conditions (brain injury, multiple sclerosis, stroke and SCI), enabling comparison with previous work. Despite the equivocal nature of its metric properties, it provides a quantitative, objective method of tracking functional change. The MAS is the most commonly used clinical tool used to measure tone, which gives the clinician information regarding the physical implications of spasticity. In light of this, choosing the MAS as one of our outcome measures facilitates the dissemination of our results to clinical settings.

However, the impact of spasticity is not manifested solely in a physical realm. Self-esteem, personal independence, social interactions and emotional well-being all contribute to how an individual reflects upon their spasticity (Sköld et al., 2000, Sanger et al., 2003). Modified Ashworth tests are simple to perform and ascribe a quantitative value to each individual muscle group. While this is helpful in discerning affected groups and potential obstacles the individual may face, the MAS is charged with omitting other relevant physical factors that contribute to spasticity, namely reflex activity and viscoelastic joint structures (Pandyan et al., 1999). Physical measurements of muscle tone at a single point in time are merely one component of spasticity. The MAS may not adequately encompass the nature of the condition, as it may be less sensitive to and prove to be less relevant to our overall understanding of symptoms and treatment recommendations.

4.2.2 The SCI-SET

Strengths of the SCI-SET lie not only in its bidirectional nature, but also in its application of a 7-day recall scheme. Many self-report measures, as well as clinical measures such as the modified Ashworth provide a 'snapshot in time' of spasticity. However, it is clear that conditions such as spasticity are of such a varied nature that one single snapshot is not adequate to represent the effect in its entirety. Something as simple as transferring from chair to examination table (which was done for the modified Ashworth testing) could influence the clinical rating of spasticity. Recounting the weekly impact of the condition helps to minimize the contributions of these diurnal fluctuations. Most studies employ visual analogue and single item scales to detect self-report spasticity (Lechner et al., 2006). However, it is likely that many subjects, finding no other alternative, could score their spasticity as having no impact ("0"), when the item in question is either beneficial of not applicable. An example of this would be asking about use of a manual chair when the individual requires a power chair. The inclusion of a non-applicable category was helpful in light of the diversity of individual conditions included in the study, and prevented skewing the data.

Bidirectional tools such as the SCI-SET may have the unfortunate consequence of creating a 'wash out' effect, in which the positive scores simply cancel out the negative scores, rendering the tool less able to articulate these differences. A separate calculation of positive and negative scores is an alternate scoring on the SCI-SET and may be helpful in future research. In addition, based on our correlation findings, we hypothesize that items pertaining to lower extremity function such as weight bearing and transferring may accrue greater positive scores than those related to upper extremity hand functioning. To address this, we would recommend the SCI-SET be modified to allow scoring to reflect sub categories such as spasticity-related impairment in ADLs,

mobility, emotional health, etc as well as the upper extremity versus lower extremity tasks. Finally, our respondents found some of the wording in the SCI-SET confusing, such as the question that asked about manual wheelchair use, which could incorporate various aspects such as steering, push strategies, or the individual's energy level, among others. We suggest further clarification of the phrasing to improve the usefulness of this progressive tool.

4.2.3 Measurement Conclusions

Based on the results of a correlational analysis there may be some overlap in terms of information regarding spasticity which may be gleaned through assorted assessment techniques. Given the variable nature of the condition, it stands to reason that equal weighting of clinical measures, such as the modified Ashworth scale, in combination with the patient's account of their symptoms and the effects of spasticity on their motor function may be the most effective method of evaluation in routine clinical practice. Some support for the criterion validity of the SCI-SET may lend credibility to its application in both clinical and rehabilitation settings as a bidirectional tool for the measurement of the impact of spasticity.

4.2.4 The Segway Protocol

While no guidelines exist for the duration of a Segway training program, several participants remarked that they were encouraged by the physiologic benefits to their day to day lives, and would prefer the length of the study be extended to several months in order to examine whether further benefits would be achieved. Based on the time

commitment required of the participants, we were concerned that recruitment and feasibility of the study would have been compromised. This positive feedback would support lengthening the study to explore the extent of improvement which could be achieved over the course of a longer period. However, several subjects appeared fatigued near the end of their sessions, and others noted a slight increase in back and/or shoulder pain during their training sessions. This is likely due in part to atrophy of stabilizer muscles used in standing as a result of sitting for extended periods of time. As we recruited a variety of individuals with a myriad of ability levels, it was appropriate to be sensitive to the standing tolerances of subjects. One participant (S8) was unable to complete the entire 30 minute session, often requiring several minutes of rest (getting off the device and sitting down included) every 10-15 minutes during the length of her involvement in the study. Another elected to continue using the harness throughout the training program. The researchers believe this was more of psychological 'safety net' as opposed to a physical necessity. Given these circumstances, we feel that 30 minutes per session was an appropriate duration and we would suggest that it is best to exercise caution regarding the duration of the protocol in these individuals, as their functional capacity levels vary. None of the subjects reported any experiences of autonomic dysreflexia or cardiac/ventilatory changes with standing.

The participants enjoyed the outdoor training setting, as it made the sessions fun and interesting, while adding an element of reality to the program. Traversing small bumps and uneven terrain is often difficult or impossible in a wheelchair, however can be fairly straightforward with the Segway.

4.3 Suggested Modifications

To minimize any inter-rater unreliability, future work with MAS should aim to utilize a single examiner. Standardizing the velocity of the modified Ashworth technique with the introduction of an electrogoniometer and a metronome (Craven et al., 2009) would lend strength the testing procedure. This is not common practice in clinical settings, however we suggest that it be considered a potential means to limit inconsistency among examiners. At the same time, this would increase the complexity of the test and the time to perform it, which may mitigate the additional attempts to increase the control. Additionally, taking an instantaneous self-report VAS measure of each muscle group to correspond with single MAS scores may shed light on how close examiner- and individual-based assessments truly are.

The authors of the SCI-SET did not develop any quantifiable descriptors for the various scores. This would have enhanced the meaningfulness of degrees of change (i.e. 1 point, 2 points, etc). Additionally, some of the terminology was confusing for our subjects, particularly such vague statements as ‘how have your spasticity symptoms affected your manual wheelchair use’. While we believe this topic is essential, there are a variety of components involved in manual wheelchair use (steering, energy expenditure, fatigue), and as such, subjects were unsure how to respond. We suggest a review of the terms may further improve the effectiveness of this new and insightful tool.

One month follow up values for each of the intervention outcome measures would have enabled us to compare whether the Segway had a lasting effect post-training. We were not able to gather these for all subjects and so they were excluded. Additionally, in hindsight, having the subjects complete the questionnaires prior to testing may have been a more appropriate tactic, as there is a possible bias of immediately completing the intervention which could skew results such as pain and fatigue (we would

expect these to possibly increase immediately after training). However, subjects reported improvements in spite of this time frame.

Standardizing the variety of terrain covered by the Segway would minimize vibration from the surface; however this was not possible, given the training environment (city sidewalks, alleyways and parks). In addition, we wanted to portray an environment that was as applicable to the real-world as possible. However, the examiners endeavored to minimize this effect by maintaining the device on the lowest speed (6kph) while the subjects trained. Depending on their functional capabilities, some subjects also used various strategies of weight transfer when performing tasks like going over obstacles and adjusting to sidewalk camber and curbs. These strategies may also have influenced the outcome measures, should they have reduced the impact felt by the subjects who were able to perform these adjustments. Perhaps keeping a record of these strategies would have been useful.

Now that a possible intervention effect has been established regarding the Segway PT, future work should incorporate a control group or a single subject repeated measures design to eliminate any effect of upright standing. It may also wish to include other populations that experience spasticity to see if the effects we saw would translate to these other groups.

4.4 Implications for Research

Clinical measures like the MAS may provide examiners with valuable information regarding specific muscles involved in spasticity. Introducing methods of standardization (a single examiner, the addition of an electrogoniometer and metronome) may serve to improve the reliability of these measures. Nevertheless, they should be used in combination with other self-report measures such as the SCI-SET which provide

interpretations of the functional impacts of spasticity, and which can account for a beneficial effect of this spasticity. We have provided some evidence to support the responsiveness of the SCI-SET to functional change, however more research is needed to substantiate this. Future work may also discern whether the SCI-SET alone, or in combination with another measure can identify a distinction between upper and lower extremity contributions of spasticity.

The Segway protocol of 30-minutes in duration was an appropriate length, given the variation of ability levels of the subjects who participated in the study. It would be beneficial to investigate a dose effect of a Segway to ask the question "Do longer periods of sessions/more frequent sessions/longer training durations change the results?" This is of interest in light of some subject's predictions that these effects would continue and further improve over a longer time frame. We might also see the changes in sleep, appetite, digestion, and bladder and bowel health similar to those reported in the work of Eng et al. (2001), as these may be affected to a greater degree over the course of time. Additionally, further investigation of these measures after removing the Segway intervention may lend credibility to findings.

4.4.1 Measurement of Spasticity

The value of the Ashworth lies in its' popularity in the literature and the clinic, its' objective nature, and its' relative ease to perform. However, its' clinicometric credibility remains unsound, and in the words of Craven et al. (2009) 'Perpetual use of an inadequate tool because of its familiarity is unacceptable'. New assessment techniques and measurement tools for spasticity should be embraced by the rehabilitation sciences community. Science may demand objective, replicable methods, however what really

matters in the lives of the individuals we are working with are life satisfaction, confidence, comfort, and the like. Arguably the latter issues are of greater significance.

Therefore we would argue that self report measures are of greater assistance in deciphering particular problems related to the condition and how best to improve quality of life for these individuals, and these should have equal, if not greater import in the research. Researchers have a responsibility to educate the public about SCI research and at the same time take the opportunity to learn from individuals living with SCI about what is truly relevant to improving the quality of life. In this way scientists may develop outcome measures that are more relevant to the priorities of the consumers (Anderson 2004).

4.4.2 The Segway

The Segway is a valuable research tool. Combining dynamic mobility and upright posture, it enables researcher to explore a variety of phenomena of interest for individuals with mobility impairments. It has been found to be user-friendly and preferable to other devices within this population (Sawatzky et al., 2007, 2009). It has also demonstrated significant short term effects in reducing spasticity and pain, and improving overall well being in individuals with SCI. Improvements in fatigue and longer-term spasticity were indicated. It is our hope that other researchers will recognize the value of the Segway and will choose to incorporate it into their future investigations. As it is currently it is still a novel, yet socially recognizable tool, this may assist with recruitment.

4.5 Implications for Rehabilitation

4.5.1 Measurement of Spasticity

Treatment goals for individuals with SCI should revolve around attainment of a high quality of life, and in some cases spasticity can positively contribute to an improved physical functioning. Measurement of spasticity must account for these sometimes beneficial contributions. Examiner-based clinical measures are not an appropriate stand-alone tool for measuring spasticity clinically. Therefore, we implore clinicians to include self-report measures such as the SCI-SET in their evaluations to facilitate a greater patient-centered approach.

4.5.2 The Segway

The psychological benefits associated with reductions in symptoms associated with SCI (spasticity, pain, fatigue), as well as the sense of accomplishment and personal freedom from standing unassisted may be the most compelling argument for widespread use of the Segway. Standing alone may achieve the physiological benefits, but may not enhance the quality of life to the same degree as standing and unrestricted mobility. Mobility enhances opportunities for social interaction. Use of assistive technology that is almost exclusively associated with disability leads to erroneous assumptions by some members of the general public. However, in addition to greater independence it affords, an individual with SCI using a Segway is indistinguishable from an able-bodied user. Therefore, the Segway minimizes the stigma of ‘disability’. With positive benefits universally reported by the subjects, the dynamic standing program is promising. No harmful effects have been documented in previous work (Sawatzky 2007, 2009) or in the

present study. Whether or not the participants in this study manifested a positive expectancy effect is of minor import when one considers the issue of quality of life. That is, quality of life benefits might be seen as the primary objective rather then an ancillary benefit.

Despite it's relatively low cost and effectiveness in enhancing physiological function and general satisfaction of the user, Segway use has been largely curtailed in the Vancouver area because of it's designation as a non-conforming motor vehicle. As such, it cannot be driven on the sidewalk because it is motorized and it cannot be driven on roadways because its speed is less than 30kph. Therefore its use is limited to private property. Without legislative change in the regional municipality this may impede its widespread acceptance and health advantages accruing to individuals within the SCI community. It is our hope that recognition of the potential benefits of the Segway for the SCI community may assist lobbying of private health care insurance companies to cover the costs associated with the purchase of this device as a mobility aid.

4.6 Conclusions

A correlational analysis of two instruments for the measurement of spasticity demonstrated some agreement, particularly with respect to the upper extremity musculature. However, they appear to measure similar, yet distinct aspects of the patients' spasticity. This information may be helpful for clinicians to compile a more comprehensive picture of spasticity as it affects the individual. While the MAS is quick and offers an objective interpretation, perhaps the SCI-SET better reflects the multifaceted nature of spasticity and how it affects the individual.

The Segway PT® provides definitive short term reductions in clinical and self-report ratings of spasticity, pain and fatigue. There is some evidence to suggest that

these beneficial outcomes may have lasting effects, however further investigations of a longitudinal nature are required to support this notion. The Segway may provide an adjunct to current therapy options for treating spasticity by introducing a stimulus to the system which overrides some underlying mechanisms involved. Additional research is needed to understand these findings.

4.7 References

1. Anderson KD. (2004). Targeting recovery: priorities of the spinal cord-injured Population. Journal of Neurotrauma. 21:1371-1383.

2. Craven BC, Morris AR. (2009). Modified Ashworth scale reliability for measurement of lower extremity spasticity among patients with SCI. Spinal Cord. E-pub ahead of press, Accessed Sept. 29. 1-7

3. Eng JJ, Levins SM, Townson AF, Mah-Jones D, Bremner J, and Huston G. (2001). Use of Prolonged Standing for Individuals with Spinal Cord Injury. Physical Therapy. 81:1392-1399.

4. Hammell KW, Miller WC, Forwell SJ, Forman BE, Jacobsen BA. (2009). Fatigue and spinal cord injury: a qualitative analysis. Spinal Cord. 47:44-49.

5. Lechner HE, Frotzler A, Eser P. Relationship between self- and clinically rated spasticity in spinal cord injury. Archives of Physical Medicine and Rehabilitation. 87:15-19.

6. Pandyan AD, Johnson GR, et al. (1999). A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measure of spasticity. Clinical Rehabilitation. 13:373-383.

7. Sanger TD, Delgado MR, Gaebler-Spira D, Hallet M, Mink JD, and the Task force on Childhood Motor Disorders. (2003). Classification and Definition of Disorders Causing Hypertonia in Childhood. Pediatrics. 111:89-97.

8. Sawatzky B, Denison I, Langrish S, Richardson S, Hiller K, Slobogean B. (2007). The Segway Personal Transporter as an Alternative Mobility Device for People with Disabilities: A Pilot Study. Archives of Physical Medicine and Rehabilitation. 88:1423-1428.

9. Sawatzky B, Denison I, Tawashy A. (2009). The Segway for People with Disabilities: Meeting Clients' Mobility Needs. American Journal of Physical Medicine and Rehabilitation. 88:484-90.

10. Sjölund BH. (2002). Pain and rehabilitation after spinal cord injury: the case of sensory spasticity? Brain Research Reviews. 40(1-3):250-6.

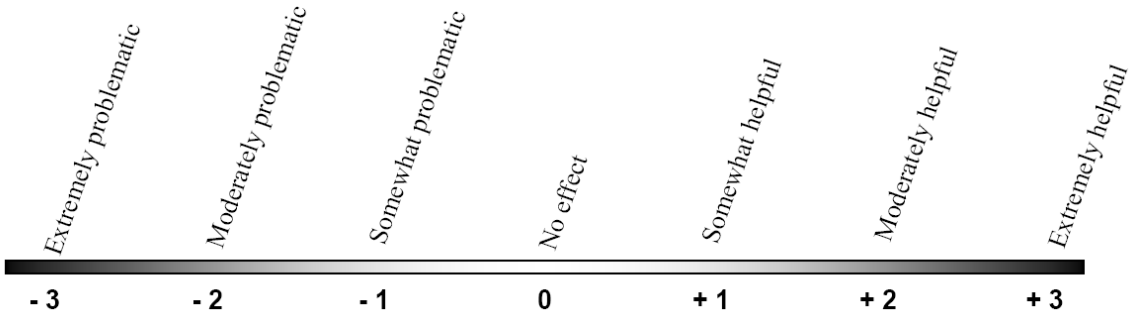
11. Sköld C.(2000). Spasticity in spinal cord injury: self- and clinically rated intrinsic fluctuations and intervention-induced changes. Archives of Physical Medicine and Rehabilitation. 81:144-9.

APPENDICES

Appendix I: 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

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: _____
Number of (-) items: _____
Number of (0) items: _____

Negative score: _____
Positive score: _____
Total score: _____
Applicable items (#): _____
Average score: _____

Appendix II: The modified Ashworth Scale Assessment Form

The modified Ashworth Scale Spasticity Assessment

Pre/Post Intervention (circle one)

Subject #: _____ Date: _____

Muscle Groups	Modified Ashworth Left	Right
Elbow Flexors		
Elbow Extensors		
Forearm Pronators		
Wrist Flexors		
Wrist Extensors		
Finger Flexors		
Hip Flexors		
Hip Extensors		
Hip Adductors		
Knee Flexors		
Knee Extensors		
Ankle Dorsiflexors		
Ankle Plantarflexors		

Specific Muscle Groups	Modified Ashworth Left	Right

Examiner: _____ Signature: _____

Modified Ashworth Scale

0 No increase in muscle tone
1 Catch and release, < 50% of ROM
1+ Catch with slight tone, <50% or ROM
2 Moderate tone, part easily moved, >50% of ROM
3 Significant tone, passive ROM difficult
4 Rigid in flexion or extension

Appendix III: The Pain Outcomes Questionnaire- Veterans Affairs

Pain Outcomes Questionnaire – Short Form

Michael E. Clark, Ph.D. and Ronald J. Gironda, Ph.D.
James A. Haley Veterans Affairs Hospital, Tampa, Florida

1.) Enter today's date: ____ / ____ / ____ (MM/DD/YY)

2.) On a scale of 0 to 10, with 0 being no pain at all and 10 being the worst possible pain, how would you rate your pain on the **AVERAGE** during the **LAST WEEK**?

0 1 2 3 4 5 6 7 8 9 10
no pain at all — worst possible pain

3.) Does your pain interfere with your ability to walk?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

4.) Does your pain interfere with your ability to carry/handle everyday objects such as a bag of groceries or books?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

5.) Does your pain interfere with your ability to climb stairs?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

6.) Does your pain require you to use a cane, walker, wheelchair or other devices?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

7.) Does your pain interfere with your ability to bathe yourself?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

8.) Does your pain interfere with your ability to dress yourself?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

9.) Does your pain interfere with your ability to use the bathroom?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

10.) Does your pain interfere with your ability to manage your personal grooming (for example, combing your hair, brushing your teeth, etc.)?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

11.) Does your pain affect your self-esteem or self-worth?

0 1 2 3 4 5 6 7 8 9 10
not at all — all the time

12.) How would you rate your physical activity?

0 1 2 3 4 5 6 7 8 9 10
significant limitation in basic activities — can perform vigorous activities without limitation

13.) How would you rate your overall energy?

0 1 2 3 4 5 6 7 8 9 10
totally worn out — most energy ever

14.) How would you rate your strength and endurance **TODAY**?

0 1 2 3 4 5 6 7 8 9 10
very poor strength and endurance — very high strength and endurance

15.) How would you rate your feelings of depression **TODAY**?

0	1	2	3	4	5	6	7	8	9	10
not depressed at all										extremely depressed

16.) How would you rate your feelings of anxiety **TODAY**?

0	1	2	3	4	5	6	7	8	9	10
not anxious at all										extremely anxious

17.) How much do you worry about re-injuring yourself if you are more active?

0	1	2	3	4	5	6	7	8	9	10
not at all										all the time

18.) How safe do you think it is for you to exercise?

0	1	2	3	4	5	6	7	8	9	10
not safe at all										extremely safe

19.) Do you have problems concentrating on things **TODAY**?

0	1	2	3	4	5	6	7	8	9	10
not at all										all the time

20.) How often do you feel tense?

0	1	2	3	4	5	6	7	8	9	10
not at all										all the time

Appendix IV: The Fatigue Severity Scale

The Fatigue Severity Scale (FSS) is designed to differentiate fatigue from clinical depression, since both share some of the same symptoms. Essentially, the FSS consists of answering a short questionnaire that requires the subject to rate his or her own level of fatigue.

Instructions: The FSS questionnaire contains nine statements that rate the severity of your fatigue symptoms. Read each statement and circle a number from 1 to 7, based on how accurately it reflects your condition during the past week and the extent to which you agree or disagree that the statement applies to you.

- A low value (e.g., 1); indicates strong disagreement with the statement, whereas a high value (e.g., 7); indicates strong agreement.
- It is important that you circle a number (1 to 7); for every question.

FSS Questionnaire

During the past week, I have found that:	**Disagree <----> Agree**						
My motivation is lower when I am fatigued.	1	2	3	4	5	6	7
Exercise brings on my fatigue.	1	2	3	4	5	6	7
I am easily fatigued.	1	2	3	4	5	6	7
Fatigue interferes with my physical functioning.	1	2	3	4	5	6	7
Fatigue causes frequent problems for me.	1	2	3	4	5	6	7
My fatigue prevents sustained physical functioning.	1	2	3	4	5	6	7
Fatigue interferes with carrying out certain duties and responsibilities.	1	2	3	4	5	6	7
Fatigue is among my three most disabling symptoms.	1	2	3	4	5	6	7
Fatigue interferes with my work, family, or social life.	1	2	3	4	5	6	7
	Total Score:						

Scoring: The total score is the sum of all the numbers circled. A total score of 36 or more suggests that the individual may suffer from fatigue.

Appendix V: The Daily Log

Daily Log- Week No. ____

Please note any changes (infections, injuries, stress) you experienced and indicate the day(s) this occurred.

Mon
Tue
Wed
Thurs
Fri
Sat
Sun

Please note any changes to medications you experienced and indicate the day(s) this occurred.

Mon
Tue
Wed
Thurs
Fri
Sat
Sun

Please rank you perceptions of Segway training on your overall daily level of the factors listed below.

1. *Spasticity*

Spasticity is broken down into frequency (please indicate number of times per day that you experience spasms) and intensity (1= no intensity, to 10= the most intense spasm I have ever had).

	Day1	Day 2	Day 3	Day4	Day5	Day6	Day7
Frequency							
Intensity							

2. *Pain*

Pain is broken down into frequency (please indicate times number of times per day that you experience pain) and intensity (1= no intensity, to 10= the most intense pain I have ever had).

	Day1	Day 2	Day 3	Day4	Day5	Day6	Day7
Frequency							
Intensity							

3. *Fatigue*

Fatigue should be ranked as 1=I have no fatigue, to 10= I am the most fatigued I have ever been

	Day1	Day 2	Day 3	Day4	Day5	Day6	Day7
Fatigue							

4. *Relaxation/Sleep*

Sleep values should relate to the previous evening and should be ranked as 1=I had the best sleep I've ever had, to 10= I had the worst sleep I've ever had

	Day1	Day 2	Day 3	Day4	Day5	Day6	Day7
Sleep							

5. *Digestion*

Digestion should be ranked as 1=I am hungry/digesting very well, to 10= I have no appetite/Am digesting very poorly

	Day1	Day 2	Day 3	Day4	Day5	Day6	Day7
Appetite							
Digestion							

6. Bladder/Bowel Health

Bladder and Bowel health should be ranked as 1= no problems with voiding/excreting, to 10= I am unable to void/excrete properly.

	Day1	Day 2	Day 3	Day4	Day5	Day6	Day7
Bladder							
Bowel							

7. Overall Wellness

Wellness should be ranked as 1=I feel the best I have ever felt, to 10= I feel the worst I have ever felt.

	Day1	Day 2	Day 3	Day4	Day5	Day6	Day7
Wellness							

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Appendix VI: Letter of Invitation

The role of the Segway in managing pain and spasticity in Individuals with Spinal Cord Injuries: A Pilot Study.

Principal Investigator: Dr. Bonita Sawatzky, PhD
Department of Orthopaedics, University of British Columbia

Co-Investigators: Grace Boutilier, BKinH, MSc (candidate)
Ian Dennison, PT
Dr. Heather Finlayson, MD
Dr. Richard Beauchamp, MD
Chris Grant, MD (candidate)

Letter of invitation

Dear Sir/Madam,

We are writing this letter to invite you to participate in a spinal cord research study. You are a potential subject for this study because you are an individual with a spinal cord injury.

The study will take place at the ICORD Research Centre and GF Strong Rehabilitation Centre during the spring of 2009. You will receive one-on-one training with a physical therapist and a researcher using a Segway self-balancing motorized device, for the course of one month.

As part of the study, Dr. Bonnie Sawatzky and her colleagues will be investigating the impact of the Segway on objective and perceived levels of spasticity, as well as any reported discrepancies in pain, fatigue, sleep and diet.

If you have any questions or think you might like to participate, please contact Dr. Bonita Sawatzky at 604.675.8806 (bsawatzky@cw.bc.ca) or her research assistant, Grace at boutilier@icord.org.

Sincerely,
Dr. Bonita Sawatzky

Appendix VII: Consent Form

The role of the Segway in managing pain and spasticity in Individuals with Spinal Cord Injuries: A Pilot Study.

Principal Investigator: Dr. Bonita Sawatzky, PhD
Department of Orthopaedics, University of British Columbia

Co-Investigators: Grace Boutilier, BKinH, MSc (candidate)
Ian Dennison, PT
Dr. Heather Finlayson, MD
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Primary Contact: Grace Boutilier
ICORD Research Facility
604.675.8815

Sponsor: Natural Sciences and Engineering Research Council of Canada

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1. THE INVITATION TO PARTICIPATE

You are being invited to take part in this research study because you have a spinal cord injury (SCI). We are looking for those who have sustained an injury at least one year prior to this study. You are able to stand independently or with braces, and able to walk a few steps, either independently or with assistance, and experience spasticity in one or more muscle groups.

2. YOUR PARTICIPATION IS VOLUNTARY

Your participation is entirely voluntary, so it is up to you to decide whether or not to take part in this study. Before you decide, it is important for you to understand what the research involves. This consent form will tell you about the study, why the research is being done, what will happen to you during the study and the possible benefits, risks and discomforts.

If you wish to participate, you will be asked to sign this form. If you decide to take part in this study, you are still free to withdraw at any time and without giving any reasons for your decision.

If you do not wish to participate, you do not have to provide any reason for your decision not to participate, nor will you lose the benefit of any medical care to which you are entitled or is presently receiving. Please take time to read the following information carefully and to discuss it with your family, friends, and therapist before you decide.

3. WHAT IS THE PURPOSE OF THE STUDY?

Previous research done by several of the current investigators has examined whether people with disabilities can benefit from using a Segway Personal Transporter as an alternative mobility device. It was found that the Segway was easy to use for most and it allowed them to get to places and do things they would not be typically be able to do. During this study we also found some positive effects on spasticity for some participants.

The aim of this research study is to investigate whether a rehabilitation program using a Segway can provide physiologic benefits such as reductions in spasticity, pain and fatigue, in individuals with incomplete spinal cord injuries (iSCIs). The research will also attempt to explore whether these potential benefits are short or long term in nature.

4. WHO CAN PARTICIPATE IN THE STUDY?

You are eligible to participate in this study if you:

1. Are aged between 19 and 65 years
2. Have a spinal cord injury
3. Have a history of spasticity in one of more muscle groups for at least one month prior to the study
4. Are able to understand and follow instructions given in English
5. Are able to come ICORD or GF Strong for the 12 sessions over a month period.

5. WHAT DOES THE STUDY INVOLVE?

Overview of the study
The study will take place at the Blusson Pavilion in the ICORD Research Centre, 818 West 10th Ave., Vancouver, BC or at the GF Strong Rehabilitation Centre 4255 Laurel St. Vancouver, BC, whichever is more convenient to you.

Specific Procedures

Preparation:
Before participating in the study, you will need to sign this consent form.

Familiarization

During the first session you will first be asked to complete several questionnaires pertaining to your current levels of spasticity (periodic, involuntary muscle contractions), pain and fatigue, respectively. You will not be required to answer any questions you feel uncomfortable answering. Your spasticity will also be manually assessed by a physician. This will take about 30 minutes. Subsequently, you will undergo a one-on-one introductory training session with our research staff and a physical therapist, in which you will be shown how to perform some simple tasks (getting on/off, going forwards/backwards, turning, etc). You will be assisted on and off the Segway, and will be taught the tasks previously demonstrated. The remainder of the session will be spent practicing easy steering and turning. This session will last approximately 30 minutes. Prior to your departure you will be instructed on filling out a daily log relating to your personal overall ratings of spasticity, pain, fatigue, as well as quality of relaxation, sleep, appetite, and wellness using a 10-point scale.

Day 1:
Should you choose to continue in the study after the familiarization session, you will return two days later to begin training sessions. Again, you will be asked to fill out spasticity, pain and fatigue questionnaires. The co-investigator will review the basic Segway proficiencies learned at the familiarization session, followed by instruction and undertaking of more complex activities, including travelling up and down ramps. To make the sessions fun and interesting we will take you on a variety of routes which will be done both indoors and outdoors. Outdoor tasks may include traversing small bumps and uneven terrain. This session will again last approximately 30 minutes.

Follow up:
These testing days will occur on a regular basis as is convenient for the researcher and yourself, 3 days per week for the course of 1 month. You will undergo a muscle spasticity assessment by a physician, along with the questionnaires, pre and post Segway training during your 6th session, and again at your last visit (12th session) at the end of one month.

6. WHAT ARE MY RESPONSIBILITIES?

Your responsibilities in participating in this study include attending a familiarization

session (approximately one hour), in addition to the Segway training sessions 3 times per week for (approximately 30 minutes each), and fill out the questionnaire forms at the scheduled visits (approximately 10 minutes). You are also asked to complete the daily log for the entirety of the one-month time period (approximately 5 minutes). The total time commitment for your participation in this study should not exceed 10 hours.

7. WHAT ARE THE POSSIBLE HARMS AND SIDE EFFECTS OF PARTICIPATING?

It is unlikely that there will be any harm from participating in this study. You will be carefully instructed prior to performing any tasks on the Segway, and will only be asked to perform those which you are comfortable doing. You will never be required to move into an uncomfortable position or do anything that causes pain. To ensure your safety, you will be provided with protective equipment (helmet, padding) and will be well supervised by spotters (including a physical therapist) whilst using the Segway. During our previous two studies with the Segway, we did not experience any injury to a study subject, however there is a small risk of falling off the device. If you do feel uncomfortable at any point or simply wish to quit, you may stop participating without it affecting your care or other commitments to ICORD related research.

8. WILL MY TAKING PART IN THIS STUDY BE KEPT CONFIDENTIAL?

Your confidentiality will be respected. No information that discloses your identity will be released or published without your specific consent to the disclosure. Any records which identify you by name or initials will be kept in a locked filing cabinet and will not be allowed to leave the Investigators' offices. Computerized data files will be password protected. Subjects will not be identified by name in any reports of the completed study.

9. WHOM DO I CONTACT IF I HAVE QUESTIONS ABOUT THE STUDY DURING MY PARTICIPATION?

If you have any questions or desire further information about this study before or during participation, you can contact Grace Boutilier at 604.875.8815.

10. WHO DO I CONTACT IF I HAVE QUESTIONS ABOUT MY RIGHTS AS A RESEARCH SUBJECT?

If you have any concerns about your treatment or rights as a research subject, you may contact the Research Subject Information Line in the UBC Office of Research Services at 604-822-8598.

11. CONSENT

Your participation in this study is entirely voluntary and you may refuse to participate or withdraw from the study at any time without penalty or loss of benefits to which you are otherwise entitled. Your future medical care will not be affected. Signing this consent form in no way limits your legal rights against the sponsor, investigators, or anyone else.

Your signature below indicates that you have received a signed and dated copy of this consent form for your own records.

Your signature indicates that you consent to your participation in this study.

_____ _____

Subject's Signature Date

_____

Printed Name of the Subject signing above.

_____ _____

Witness's signature Date

_____

Printed Name of the Witness signing above.

_____ _____

Investigator's Signature Date

_____

Printed Name of the Investigator signing above.

Appendix VIII: Poster Advertisement

Are you an individual with a **Spinal Cord Injury** (1year) between the ages of 19 and 65 years? Do you experience **spasms, pain** and **fatigue** on a regular basis?

Researchers at GF Strong Rehabilitation Center and ICORD invite you to participate in a research study involving the Segway Personal Transporter.

We are investigating the impact of a 30 day Segway program (1 hour, 3 days/ week) on spasticity, pain and fatigue in individuals with spinal cord injuries, and whether these potential benefits are short or long term in nature.

Location: Blusson Pavilion, GF Strong Rehab Centre

If you are interested in participating or would like some more information please contact **Grace** at: **boutilier@icord.org** or by phone **(604) 675-8815.**

Segway Study – Grace: 675--8815 boutilier@icord.org
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The University of British Columbia
Office of Research Services
Clinical Research Ethics Board –
Room 210, 828 West 10th Avenue,
Vancouver, BC V5Z 1L8

Appendix IX: Clinical Research Ethics Board Approval

ETHICS CERTIFICATE OF FULL BOARD APPROVAL

PRINCIPAL INVESTIGATOR:	INSTITUTION / DEPARTMENT:	UBC CREB NUMBER:
Bonita Sawatzky	UBC/Medicine, Faculty of/Orthopaedics	H08-02482

INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT:

Institution	Site
Vancouver Coastal Health (VCHRI/VCHA)	GF Strong Rehabilitation Centre

Other locations where the research will be conducted:
ICORD Blusson Pavilion building Rehab Lab GF Strong SCI Gym

CO-INVESTIGATOR(S):
I. Denison
Richard D. Beauchamp
Heather Finlayson

SPONSORING AGENCIES:
- Natural Sciences and Engineering Research Council of Canada (NSERC) - "Wheelchair vibration: origin, implications, and reduction for manual wheelchairs"

PROJECT TITLE:
The role of the Segway in managing pain and spasticity in Individuals with Spinal Cord Injuries: A Pilot Study.

THE CURRENT UBC CREB APPROVAL FOR THIS STUDY EXPIRES: December 9, 2009

The full UBC Clinical Research Ethics Board has reviewed the above described research project, including associated documentation noted below, and finds the research project acceptable on ethical grounds for research involving human subjects and hereby grants approval.

REB FULL BOARD MEETING REVIEW DATE: December 9, 2008	

DOCUMENTS INCLUDED IN THIS APPROVAL:			DATE DOCUMENTS APPROVED:
Document Name	**Version**	**Date**	
Protocol:			
Research Protocol	2	November 1, 2008	
Consent Forms:			
Consent Form	5	January 1, 2009	**January 19, 2009**
Advertisements:			
Segway Poster	3	December 1, 2008	
Questionnaire, Questionnaire Cover Letter, Tests:			

Clinical History	3	December 1, 2008	
Eligibility Screen	2	December 1, 2008	
Modified Ashworth Scale	2	October 1, 2008	
Daily Log	1	October 1, 2008	
Pain Outcomes Questionnaire	3	December 1, 2008	
The Fatigue Severity Scale	2	October 1, 2008	
Spasticity Frequency Scale	2	October 1, 2008	
Letter of Initial Contact:			
Letter of Invitation	3	December 1, 2008	

CERTIFICATION:
In respect of clinical trials:
1. The membership of this Research Ethics Board complies with the membership requirements for Research Ethics Boards defined in Division 5 of the Food and Drug Regulations.
2. The Research Ethics Board carries out its functions in a manner consistent with Good Clinical Practices.
3. This Research Ethics Board has reviewed and approved the clinical trial protocol and informed consent form for the trial which is to be conducted by the qualified investigator named above at the specified clinical trial site. This approval and the views of this Research Ethics Board have been documented in writing.

The documentation included for the above-named project has been reviewed by the UBC CREB, and the research study, as presented in the documentation, was found to be acceptable on ethical grounds for research involving human subjects and was approved by the UBC CREB.

Approval of the Clinical Research Ethics Board by:

Dr. Gail Bellward, Chair

ETHICS CERTIFICATE OF EXPEDITED APPROVAL: AMENDMENT

PRINCIPAL INVESTIGATOR:	**DEPARTMENT:**	**UBC CREB NUMBER:**
Bonita Sawatzky	UBC/Medicine, Faculty of/Orthopaedics	H08-02482

INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT:

Institution	Site
Vancouver Coastal Health (VCHRI/VCHA)	GF Strong Rehabilitation Centre

Other locations where the research will be conducted:

ICORD Blusson Pavilion building Rehab Lab GF Strong SCI Gym

CO-INVESTIGATOR(S):

Christopher Grant
I. Denison
Richard D. Beauchamp
Heather Finlayson

SPONSORING AGENCIES:

- Natural Sciences and Engineering Research Council of Canada (NSERC) - "Wheelchair vibration: origin, implications, and reduction for manual wheelchairs"

PROJECT TITLE:

The role of the Segway in managing pain and spasticity in Individuals with Spinal Cord Injuries: A Pilot Study.

AMENDMENT(S):

Document Name	Version	Date
Questionnaire, Questionnaire Cover Letter, Tests:		
Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET)	1	February 1, 2009

AMENDMENT APPROVAL DATE:
February 16, 2009

CERTIFICATION:

In respect of clinical trials:

1. The membership of this Research Ethics Board complies with the membership requirements for Research Ethics Boards defined in Division 5 of the Food and Drug Regulations.
2. The Research Ethics Board carries out its functions in a manner consistent with Good Clinical Practices.
3. This Research Ethics Board has reviewed and approved the clinical trial protocol and informed consent form for the trial which is to be conducted by the qualified investigator named above at the specified clinical trial site. This approval and the views of this Research Ethics Board have been documented in writing.

The amendment(s) for the above-named project has been reviewed by the Chair of the University of British Columbia Clinical Research Ethics Board and the accompanying documentation was found to be acceptable on ethical grounds for research involving human subjects.

Approval of the Clinical Research Ethics Board by :

Dr. Stephen Hoption Cann, Associate Chair

Appendix X: Clinical History Review

Subject ID: Date:

Medications:

Age:

Gender:

Level of Spinal Cord Injury:

Complete or incomplete:

Date of SCI:

History of pressure ulcer(s):

History of autonomic dysreflexia:

History of cardiovascular disorder:

History of pulmonary disorder:

Other relevant medical history:

PHYSICAL EXAM:

BP: HR:

CVS:

Resp:

Appendix XI: Eligibility Criteria

Subject No: _____

Age: _____

ASIA Level of Injury (circle one)

A B C D E

Year of Injury: _____

My current mobility aid(s) include:

I experience spasticity in the muscle groups listed below:

I experience pain in the areas listed below:

Please indicate any anti-spastic or pain relief medications you are currently on.

Appendix XII: Subject Demographic Information

Subject	Sex	Age	Injury Level	ASIA	Year(s) since Injury
1	M	48	C5-6	C/D	24
2	M	35	T11-12	A	7
3	M	52	C5	C	7
4	M	33	C5-6	C	15
5	M	41	T5	B	6
6	M	54	C6	D	29
7	F	54	C5	C	4
8	M	36	T6	C/D	18
9	F	61	C5	D	1

Baclofen (oral and intrathecal), and botox were the most frequently prescribed spasticity medications. Most subjects used a combination of mobility devices, with the most often cited being a manual wheelchair paired with a cane or crutches for standing/walking short distances. All except one (S7) stood on a daily basis.

Subject	Daily Meds	Current Activities	Mobility Aids
1	Flouexetine	Walking	Cane, L AFO
2	Baclofen, botox (rectus fem 1mo prior), Novotrimnol, Vesicare	Brace walking	Forearm crutches, HKAFOs, manual chair
3	GABAp, Baclofen (oral) Nortripaline	Walking, gym	Cane
4	Baclofen (oral)	Gym, yoga, streching	Manual chair, forearm crutches
5	Baclofen (intrathecal), Pariet, Citalopram	Walking	Walker, HKAFOs, manual chair
6	Baclofen (oral), Diazepam	Walking	Cane
7	Botox (pectoralis~2 mos prev)	Standing frame	Power chair, walker
8	NSAIDS	Gym, WC training	Manual chair, L AFO, forearm crutches
9	Baclofen (oral), vitamins	Walking, stretching	Manual chair, walker

Appendix XIII: Data

MAS_SCI-SET Correlations

Muscle	Test	S1	S2	S3	S4	S5	S6	S7	S8
Up Ex	T1	1	0	1	0	1	2	0	1.5
	T2	1	0	1	0	1.5	2	0	1
	T3	1.5	0	1	0	1	1.5	0	1
Lwr Ex	T1	1	1	0	3	2	2	1	1.5
	T2	2	0	1.5	1	2	2	0	2
	T3	1.5	1	0	0	2	1.5	1	1.5

Pre-Post MAS Scores

Subject	Test	M1_PRE	M1_POST	M2_PRE	M2_POST	M3_PRE	M3_POST
S1	T1	3	1	3	1	2	1
	T2	3	2	3	2	1	0
	T3	3	1.5	2	1.5	1	0
S2	T1	1.5	1	1.5	1	3	2
	T2	1.5	0	1.5	0	1.5	1
	T3	1.5	1	1.5	0	1.5	1
S3	T1	2	0	3	2	1	1
	T2	2	1.5	2	2	1	0
	T3	0	0	2	1	1.5	1
S4	T1	1.5	0	0	0	3	3
	T2	1	0	1.5	0	2	1
	T3	0	0	1	0	1	0
S5	T1	2	2	2	1.5	1.5	1
	T2	2	2	1	1	1.5	1
	T3	3	2	2	1	2	1.5
S6	T1	3	2	2	2	2	2
	T2	3	2	2	1.5	2	1.5
	T3	3	1.5	2	1.5	2	1.5
S7	T1	1.5	1	2	1.5	3	1.5
	T2	1	0	1.5	0	1.5	1.5
	T3	1.5	1	1.5	1	2	1.5
S8	T1	2	1.5	1.5	1.5	3	2
	T2	3	2	1	0	3	2
	T3	2	1.5	2	1.5	3	2

T1PRE_T3PRE MAS Scores

	M1		M2		M3	
Subject	**T1_PRE**	**T3_PRE**	**T1_PRE**	**T3_PRE**	**T1_PRE**	**T3_PRE**
S1	3	2	3	1	2	1
S2	1.5	3	1.5	1.5	3	1.5
S3	2	1	2	1	1	1.5
S4	1.5	3	0	2	3	1
S5	2	1.5	2	1.5	1.5	2
S6	3	2	2	2	2	2
S7	1.5	3	2	1.5	3	2
S8	2	3	1.5	3	3	3

SCI-SET Scores

Test	**S1**	**S2**	**S3**	**S4**	**S5**	**S6**	**S7**	**S8**	**MEAN**
1	-1.000	-0.559	-0.429	-0.364	-1.222	-3.000	-0.364	-0.346	-0.910
2	-0.743	-0.314	-0.800	-0.030	-0.333	-2.304	-0.303	-0.172	-0.625
3	-0.514	-0.206	-1.314	0.000	-0.281	-2.038	-0.182	0.000	-0.567

POQ-VA Scores

	Test	**S1**	**S2**	**S3**	**S4**	**S5**	**S6**	**S7**	**S8**	**MEAN**
PTOT	1	67	12	70	14	45	68	26	40	43
	2	44	6	69	13	40	93	29	33	41
	3	46	6	60	7	42	52	23	27	33
Pain	1	4	1	3	0	3	5	3	5	3
Intensity	2	2	3	4	0	4	7	3	5	4
	3	1	0	2	0	3	5	2	5	2
Mobility	1	24	0	24	0	15	8	14	0	11
	2	9	0	27	0	11	14	19	3	10
	3	5	0	19	0	12	11	16	4	8
ADLS	1	6	0	3	0	0	0	0	0	1
	2	0	0	8	0	0	14	0	0	3
	3	1	0	4	0	0	0	0	0	1
Vitality	1	20	2	20	7	17	24	5	20	14
	2	20	4	20	8	16	23	8	21	15
	3	27	6	15	5	17	20	7	15	14
Negative	1	15	1	13	4	9	20	6	5	9
Affect	2	11	0	5	5	10	32	2	4	9

	3	8	0	13	2	12	11	2	3	6
Fear	1	2	3	10	0	4	16	1	10	6
	2	3	2	9	0	3	10	0	0	3
	3	5	6	9	0	1	10	1	0	4

Fatigue Severity Scores

Test	S1	S2	S3	S4	S5	S6	S7	S8	MEAN
1	6.1	3	4	3.6	6.4	3.3	3.4	3.3	4.1375
2	5.4	2.9	4.9	2.8	6.6	3.4	3.2	4.1	4.1625
3	5.3	2.3	4.7	3.4	6.2	2.7	1.9	3.3	3.725

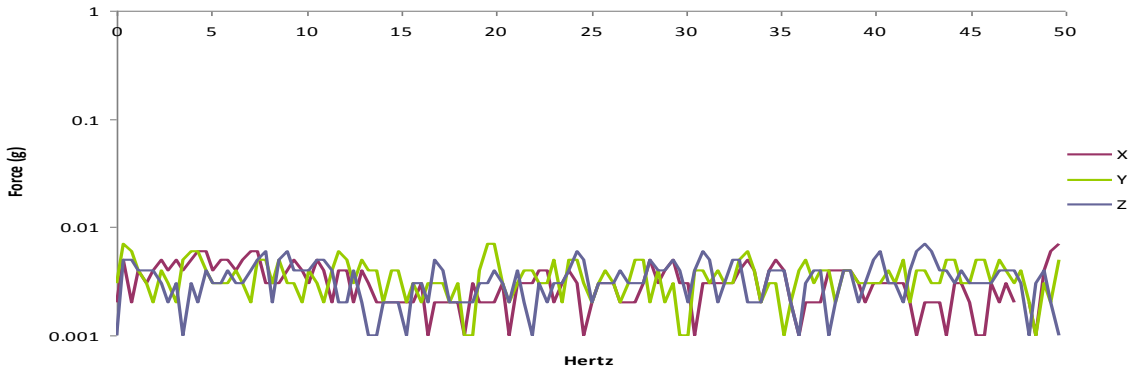
Daily Log Scores

	Test	S1	S2	S3	S4	S5	S6	S7	S8	MEAN
Spasticity										
Frequency	1	1	15	10	6	1	10	11	7	8
	2	1	12	10	6	2	10	12	7	8
	3	1	11	10	4	1	5	11	7	6
Intensity	1	1	4	10	3	2	7	3	5	4
	2	1	3	9	4	2	6	3	5	4
	3	1	2	10	3	2	5	3	5	4
Pain										
Frequency	1	1	6	6	0	10	10	10	10	7
	2	1	6	3	0	10	10	10	7	6
	3	1	0	2	0	10	5	10	7	6
Intensity	1	1	2	5	0	3	7	3	5	3
	2	1	3	3	0	2	6	3	3	3
	3	1	1	1	0	2	5	1	3	2
Fatigue	1	1	4	5	4	3	9	5	5	5
	2	2	4	5	5	3	6	5	5	4
	3	2	2	5	2	4	5	3	5	4
Sleep	1	2	5	7	5	2	7	5	5	5
	2	2	4	8	4	3	7	4	5	5
	3	2	2	9	2	3	5	3	5	4
Appetite	1	2	1	1	2	1	5	3	1	2
	2	2	1	1	9	3	5	2	1	3
	3	2	1	1	1	1	5	2	1	2

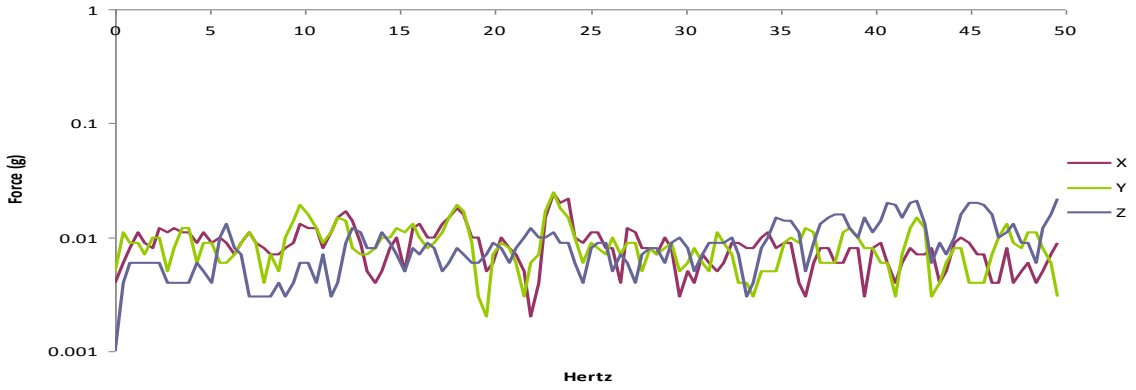
Digestion	1	2	1	1	2	1	5	3	1	2
	2	2	1	1	10	1	5	2	1	3
	3	2	1	1	1	1	5	2	1	2
Bladder	1	1	1	3	1	1	5	1	1	2
	2	2	1	3	1	1	5	1	1	2
	3	1	1	2	1	1	5	1	1	2
Bowel	1	1	1	1	2	1	5	1	1	2
	2	2	1	1	1	1	5	1	1	2
	3	1	1	1	1	1	5	1	1	2
Overall	1	4	4	5	4	3	7	4	5	5
Wellbeing	2	4	4	5	3	2	7	3	5	4
	3	2	2	4	2	2	5	3	5	3

Segway Vibration Data

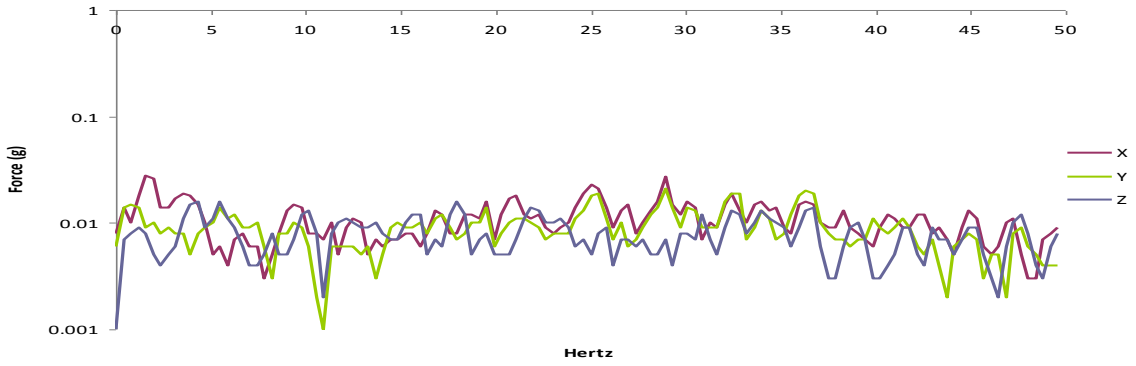
i2 Vibrations Stationary on brick surface



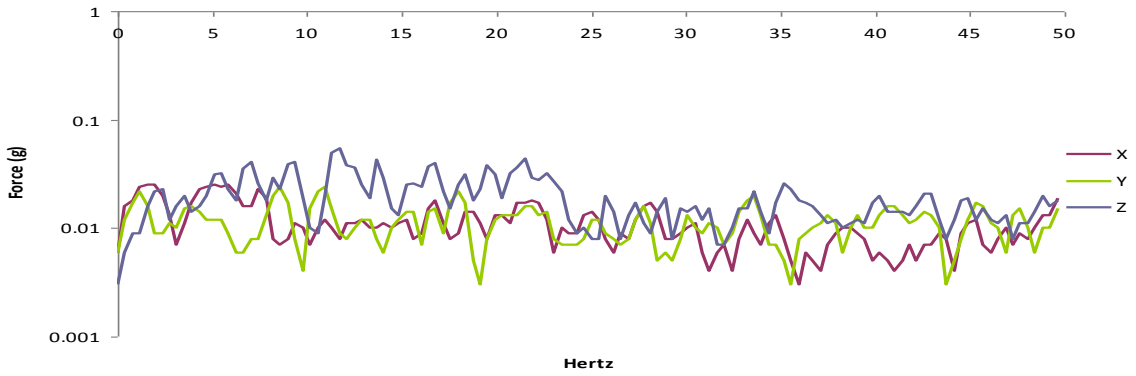
i2 Vibrations 2.5mph on brick surface



i2 Vibrations 5mph on brick surface



i2 Vibrations 10mph on asphalt surface



i2 Vibrations 10mph on rough concrete surface