VIBRATION, STATIC STANDING, DYNAMIC STANDING, AND SPASTICITY IN
INDIVIDUALS WITH SPINAL CORD INJURY

by

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Abstract

Spasticity is a common consequence of upper motor neuron lesions such as spinal cord injury (SCI). Spasticity is experienced by 65-78% of individuals with SCI. Spasticity management is one of the most important challenges that clinicians and researchers encounter. Physical therapy techniques are the essential component of spasticity management that are used during and after other spasticity management tools. Vibration and standing training are two physiotherapy techniques that might be beneficial to manage spasticity in individuals with SCI. This thesis is divided into two major parts to study how these two physiotherapy techniques are useful to manage spasticity in individuals with SCI. The first study was a systemic review exploring how effective whole body and focal vibration are for spasticity management in individuals with SCI. The second study was a cross-over study that evaluated the difference between the effects of static and dynamic (using the Segway) standing training on spasticity immediately and one hour after the interventions in individuals with SCI. Spasticity was measured by three different outcome measures including self-assessment (visual analog scale), clinical (Modified Ashworth Scale), and electrophysiologic (electromyography) measures. There was evidence to support the decreasing effects of either whole body or focal vibration on spasticity in individuals with SCI. There was no statistically significant difference between the dynamic and static standing interventions for spasticity reduction. We concluded that vibration (focal and whole body) has promising effects for spasticity reduction in individuals with SCI. We also concluded that dynamic standing training has no greater effect on spasticity reduction compared to the static standing training.
Preface

All of the work presented henceforth was conducted in the SCI Mobility Research Laboratory at ICORD, University of British Columbia. All projects and associated methods were approved by the University of British Columbia’s Research Ethics Board [certificate number: H12-02927].

I was responsible for all major areas of concept formation, data collection and analysis, as well as manuscript edits. Dr.Bonita Sawatzky was the supervisory author on this project and was involved throughout the project in concept formation and manuscript composition. Dr. Mark Carpenter and Dr. Heather Finlayson were the supervisory committee on this project. They engaged throughout the project in study design and final data analysis decision making.

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# Table of Contents

**Abstract**........................................................................................................................................... ii  
**Preface**................................................................................................................................................ iii  
**Table of Contents**................................................................................................................................. iv  
**List of Tables**......................................................................................................................................... x  
**List of Figures**......................................................................................................................................... xii  
**List of Abbreviations**........................................................................................................................... xiii  
**Acknowledgements**............................................................................................................................. xv  
**Dedication**............................................................................................................................................... xvi  

## Chapter 1: Introduction .......................................................................................................................... 1  
1.1 Spasticity Definition ......................................................................................................................... 1  
1.2 Spasticity Pathophysiology ............................................................................................................. 2  
  1.2.1 Normal Pathways and Stretch Reflex ...................................................................................... 2  
  1.2.2 Pathophysiology of Spasticity .................................................................................................. 4  
1.3 Spasticity Measurement .................................................................................................................... 6  
  1.3.1 Clinical Spasticity Assessments .............................................................................................. 6  
  1.3.2 Self-report Questionnaire ...................................................................................................... 9  
  1.3.3 Biomechanical, Electrophysiologic, and Neurophysiologic Spasticity Outcome Measures .... 13  
    1.3.3.1 Biomechanical Spasticity Outcome Measures ................................................................. 13  
    1.3.3.2 Electrophysiologic and Neurophysiologic Spasticity Outcome Measures ..................... 14  
1.4 Spasticity and Spinal Cord Injury (SCI) ........................................................................................... 16
Chapter 2: Vibration Scoping Systematic Review

2.1 Introduction

2.2 Methods

2.2.1 Search Strategy

2.2.2 Inclusion/Exclusion Criteria

2.2.3 Data Extraction

2.2.4 Level of Evidence (LoE)

2.3 Results

2.3.1 Population

2.3.2 Effects on Spasticity

2.3.3 Vibration Frequency

2.3.4 Spasticity Measurement

2.3.5 Level of Evidence
3.2.4.3 Electromyography (EMG) ................................................................. 65
3.2.4.4 Ankle and Knee Joints passive range of motion .......................... 65
3.2.5 Instruments ..................................................................................... 66
   3.2.5.1 Goniometers ............................................................................. 66
   3.2.5.2 EMG ....................................................................................... 66
   3.2.5.3 The Segway ........................................................................... 67
3.2.6 Protocol Schematic ......................................................................... 67
   3.2.6.1 Familiarization ....................................................................... 68
   3.2.6.2 Test Preparation ...................................................................... 68
   3.2.6.3 Static Training Session .............................................................. 69
   3.2.6.4 Dynamic Training Session ......................................................... 70
   3.2.6.5 Measurements ......................................................................... 72
3.2.7 Data Analysis .................................................................................. 74
   3.2.7.1 MAS Analysis .......................................................................... 74
   3.2.7.2 Goniometers Analysis ............................................................... 74
   3.2.7.3 EMG Analysis ......................................................................... 76
3.2.8 Statistical Analysis .......................................................................... 80
3.3 Results .............................................................................................. 81
   3.3.1 Population .................................................................................. 81
   3.3.2 Visual Analog Scale (VAS) ............................................................ 83
   3.3.3 Modified Ashworth Scale (MAS) .................................................... 85
   3.3.4 Electromyography of SCI Participants ........................................ 87
     3.3.4.1 Muscle Changes Over Time .................................................... 89
3.3.4.2 Global Effects of Standing Conditions on Muscle Activity Changes........... 92
3.3.5 Able-Bodied EMG .................................................................................. 95
  3.3.5.1 Muscle Changes with Time ............................................................... 95
  3.3.5.2 Global Effects of Standing Conditions on Muscle Activity ............... 96
3.3.6 Movement Velocity ............................................................................... 98
  3.3.6.1 Spinal Cord Injury Participants......................................................... 98
  3.3.6.2 Able-bodied Participants.................................................................. 99
3.4 Discussion................................................................................................. 100
  3.4.1 Self-assessment Spasticity Outcome Measure....................................... 102
  3.4.2 Clinical Spasticity Outcome Measure.................................................... 104
  3.4.3 Electrophysiologic and Neurophysiologic Outcome Measure .............. 106
  3.4.4 Training................................................................................................. 110
  3.4.5 Modified Ashworth Scale Velocity Consistency..................................... 111
3.5 Study Limitations..................................................................................... 112
  3.5.1 Population............................................................................................ 112
  3.5.2 Training................................................................................................. 113
  3.5.3 Spasticity Outcome Measures............................................................... 113
3.6 Conclusion.................................................................................................. 114

Chapter 4: General Discussion .........................................................................115
  4.1 Spasticity and Vibration.......................................................................... 115
  4.2 Spasticity and Standing Training............................................................... 116
  4.3 Future Direction......................................................................................... 117

References ......................................................................................................118
Appendices

Appendix A Systematic Review Acceptance Letter ............................................. 133
Appendix B Search Strategy .................................................................................. 135
Appendix C CEBM Grades of Recommendation .................................................... 136
Appendix D Study Recruitment Poster .................................................................. 137
Appendix E Consent Form ..................................................................................... 138
Appendix F Visual Analog Scale (VAS) ................................................................. 144
Appendix G Intake Form ......................................................................................... 146
Appendix H Visual Analog Scale (VAS) Results ..................................................... 148
List of Tables

Table 1.1 Modified Ashworth Scale ................................................................. 7
Table 1.2 Modified Tardieu Scale ...................................................................... 9
Table 1.3 Penn Spasm Frequency Scale ............................................................ 11
Table 1.4 Modified Penn Spasm Frequency Scale ............................................. 11
Table 1.5 The Five Categories of the ASIA Impairment Scale (AIS) ..................... 17
Table 1.6 Electrical Stimulation Modalities Mechanism .................................... 20
Table 1.7 Antispastic Agents Side Effects .......................................................... 23
Table 2.1 Risk of Bias Assessment ..................................................................... 36
Table 2.2 Study Characteristics ......................................................................... 40
Table 2.3 Whole Body Vibration Study Characteristics ....................................... 42
Table 2.4 Focal Vibration Study Characteristics ................................................ 44
Table 2.5 Vibration Characteristics .................................................................. 50
Table 3.1 Spinal Cord Injury Participants Characteristics .................................... 82
Table 3.2 Able-Bodied Participants Characteristics ............................................. 82
Table 3.3 Visual Analog Scale (VAS) Outcome Measure Results ........................ 83
Table 3.4 Modified Ashworth Scale Outcome Measure Results ........................ 86
Table 3.5. Average MASSum Results ................................................................. 87
Table 3.6 SCI Group Average ΔIEMG Changes for the Primary Muscles ............... 89
Table 3.7 Global SCI Group Ankle Dorsiflexion Average Δ IEMG Changes .......... 92
Table 3.8 Global SCI Group Knee Flexion Average Δ IEMG Changes .................. 93
Table 3.9 Global SCI Group Knee Extension Average Δ IEMG Changes ............... 94
Table 3.10 Able-bodied Group Average Δ IEMG Changes for the Primary Muscles .... 95
Table 3.11 Passive Movement Average Velocity and Range of Spinal Cord Injury Participants 98

Table 3.12 Average velocity Intraseason ICC\textsubscript{2,1} of SCI Participants ........................................ 99

Table 3.13 Average velocity Betweenseason ICC\textsubscript{2,1} of SCI Participants ........................................ 99

Table 3.14 Passive Movement Average Velocity and Range of Able-Bodied.............................. 99
List of Figures

Figure 1.1 Stretch Reflex Arch ................................................. 3
Figure 1.2 Stretch Reflex Component ....................................... 4
Figure 2.1 Flowchart of Study Selection .................................... 38
Figure 3.1 Study Design .......................................................... 64
Figure 3.2 Ankle and Knee Goniometers .................................. 66
Figure 3.3 Static Segway/Static Standing Training........................... 67
Figure 3.4 Participant Preparation Setup .................................... 69
Figure 3.5 Static Standing Training .......................................... 70
Figure 3.6 Dynamic Standing Training ...................................... 71
Figure 3.7 Dynamic Standing Training Procedure ....................... 72
Figure 3.8 Protocol Schematic .................................................. 73
Figure 3.9 Knee Goniometer Processing .................................... 75
Figure 3.10 Ankle Goniometer Processing .................................. 76
Figure 3.11 EMG Processing ..................................................... 78
Figure 3.12 Integrated EMG ..................................................... 79
Figure 3.13 Visual Analog Scale in Individuals with Spinal Cord Injury ...... 84
Figure 3.14 SCI participant Overlapped EMG of three time points ...... 88
Figure 3.15 SCI Group ΔIEMG Changes .................................... 90
List of Abbreviations

AB: Able-bodied
AS: Ashworth Scale
AIS: American Spinal Injury Association (ASIA) Impairment Scale
ASIA: American Spinal Injury Association
ATR: Achilles tendon reflex
BBB: Blood Brain Barrier
CEBM: Centre for Evidence Based Medicine
CNS: Central Nervous System
CP: Cerebral Palsy
EMG: Electromyography
EPSP: Excitatory Postsynaptic Potentials
FES: Functional Electrical Stimulation
FSS: Fatigue Severity Scale
FV: Focal Vibration
H-reflex: Hofmann reflex
GTO: Golgi Tendon Organ
ICC: Interclass Correlation Coefficients
ICF: International Classification of Functioning, Disability and Health
IEMG: Integrated Electromyography
ISO-2631-1: International Standardization Organization 2631-1
LMN: Lower Motor Neuron
MAS: Modified Ashworth Scale
MASSum: Modified Ashworth Scale Sum Score
MS: Multiple Sclerosis
MTS: Modified Tardieu Scale
MCID: Minimal Clinically Important Difference
MVIC: Maximum Voluntary Isometric Contraction
NTSCI: Non-Traumatic Spinal Cord Injury
POQ: Pain Outcome Questionnaire
PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-analysis
PSFS: Penn Spasm Frequency Scale
PAD: Post-activation depression
RCT: Randomized Control Trials
SCI: Spinal Cord Injury
SCI-SET: Spinal Cord Injury Evaluation Tool
SR: Systematic Reviews
TBI: Traumatic Brain Injury
TENS: Transcutaneous Electrical Nerve Stimulation
T-reflex: Tendon reflex
TSCI: Traumatic Spinal Cord Injury
UMN: Upper Motor Neuron
VAS: Visual Analog Scale
WBV: Whole Body Vibration
WPT: Wartenberg Pendulum Test
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Dedication

I dedicate this work to my lovely husband Mehdi who has never left my side. I thank you for being there for me all the time and encouraging me to do my best. You have been my best cheerleader.

I dedicate my thesis work to my lovely family. A special feeling of gratitude to my loving parents, Ali and Fereshteh Sadeghi whose words of encouragement and support helped me throughout my Master program.
Chapter 1: Introduction

Spasticity is one of the most common sources of disability developing after upper motor neuron (UMN) lesions for patients with spinal cord injury (SCI), stroke, multiple sclerosis (MS), cerebral palsy (CP), and traumatic brain injury (TBI) (1–8). Spasticity occurs at a variable rate within these UMN lesions. It has been shown that spasticity affects between 37-78% of individuals with MS (1), 53-78% of individuals with SCI (3,2,5,4), approximately 35% of stroke population (6,7), around 90% of CP population (8) and approximately 50% of those with TBI (9).

1.1 Spasticity Definition

The most common acceptable definition of spasticity was defined by Lance in 1980 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 representing one component of the UMN lesion (10). Although this definition has been accepted widely, there is discrepancy within the literature for the precise definition of spasticity (2,11–16).

There are some literature that include other symptoms such as clonus, hyperactive tendon reflexes, and spasm within the original definition (14–16). Decq in 2003 divided spasticity into three specific subcomponents: (a) intrinsic tonic spasticity, explained as exaggeration of the tonic component of the stretch reflex that is manifested as increased muscle tone, (b) intrinsic phasic spasticity, involving exaggeration of the phasic component of the stretch reflex that is manifested as increased tendon reflexes and clonus, and (c) extrinsic reflexes, defined as increased exteroceptive reflexes such as flexor reflex and pathologic radiation of reflexes between spinal segments demonstrated as increased spinal reflexes over time (17). Burridge et al. in 2005 proposed a broader definition of spasticity to explain how abnormal sensory inputs would affect the motor control by resulting in hyperreflexivity, increased tone over extended periods of rest, or disordered motion during activities such as walking (18).
1.2 **Spasticity Pathophysiology**

Although the pathophysiology of spasticity is not well understood, it has been explained as a sensorimotor interruption in central nervous system (CNS). It is explained as a disruption between sensory input integration and motor responses in CNS (19). Upper motor neuron lesions such as SCI result in loss of descending inhibitory drive from the CNS to the muscles that manifest clinically as abnormal impulses and overactive muscles. This defect makes the motor neurons hyperexcitable to sensory inputs (16,19).

1.2.1 **Normal Pathways and Stretch Reflex**

In normal pathways, the sensory signals enter the posterior root of spinal cord and the motor signals travel back from the CNS to the muscles (19). Some of the sensory inputs go through the grey matter of the spinal cord, whereas some other sensory signals travel to the upper CNS centers such as brainstem and cerebrum. The signals from the upper CNS centers travel back to muscle fibers through different motorneurons including alpha and gamma motorneurons (19). Alpha motorneurons innervate extra and intrafusal muscle fibers of large skeletal muscles, where as gamma motorneurons transmit impulses to intrafusal muscle fibers (19).

Intrafusal muscle fibers are called the muscle spindle fibers that are categorized as primary and secondary muscle spindle endings (19,20). Muscle spindles are located parallel to the extrafusal muscle fibers in large muscles and transmit the length and rate of extrafusal muscle fiber changes to the spinal cord. The primary ending responds to rapid and slow extrafusal fibers length changes, whereas the secondary ending transmits slow extrafusal fibers length changes (19,20). Muscle stretch activates the muscle spindles and increases the firing rate of muscle spindles that results in transmission of sensory impulse to the spinal cord (19,20). The spinal cord sends motor impulses through alpha motor neuron to extrafusal muscle fibers to control muscle tone and contraction. This monosynaptic reflex pathway between 1a afferent (originates from the muscle spindles) and alpha motor neuron is called the monosynaptic stretch reflex (Figure 1.1) (19,21).
The stretch reflex controls the muscle tone mostly by the inhibitory interneurons that usually integrate with the impulses from other parts of nervous system such as the corticospinal tract. The inhibitory inputs from the corticospinal tracts specifically dorsal reticulospinal is the most significant inhibitory mechanism to control the muscle tone (19,20). Spinal cord Golgi tendon organs (GTOs) with the primary role of preventing excessive tension between loose and tense, agonist and antagonist muscles has inhibitory effects on alpha motor neuron (19,20). All these mechanisms together control the muscle tone in a normal peripheral and central nervous system (Figure 1.2).
1.2.2 Pathophysiology of Spasticity

The central and peripheral neural changes, as well as some non-neural factors contribute to spasticity in UMN lesions (21) are described below. Responsible central neural changes are described as decreased inhibitory drives from the central motor pathways such as cortico-reticulospinal and other descending pathways to spinal cord polysynaptic connections (19,22). Several peripheral neural changes that have been described as spasticity contributing factors are listed below. Decreased 1a interneuron

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**Figure 1.2 Stretch Reflex Component**
inhibitory effects and reduction of GTO inputs to 1b inhibitory interneurons cause a disruption in muscle spindles and alpha motor neuron negative feedback. The disrupted negative feedback results in increased muscle tone (19,20,22,23). Non-neural factors include muscle biomechanics and non-muscular factors that lead to increased muscle tone and spasticity (24–26). The muscle biomechanics and non-muscular factors such as soft tissue compliance, joint integrity, muscle fiber atrophy, musculotendinous units, and connective tissues lead to increased tension and reflex sensitivity (26,27). Non-muscular noxious stimuli including urinary tract infection, constipation, ingrown toenails, pressure ulcers and poor fit in brace or wheelchair increase the afferent input of stretch reflex and result in increased muscle tone (28).

The pathophysiology of spasticity is also described based on explanation of Decq in 2003. Intrinsic tonic spasticity associated with increased muscle tone due to a maintained stretch of the central region of muscle fibers and their impaired connection with type Ia and II afferents, inhibitory interneurons, and alpha motor neuron (17). However, maintained passive stretch can result in increased muscle tone and it might be attributed to the hyperexcitability of the stretch reflex tonic component. Hyperexcitability of the tonic component might result from either lower firing rate threshold or increased stretch reflex gain (16,29). Intrinsic phasic spasticity results from tendon hyper-reflexia and clonus which are phasic component of the stretch reflex (17). The exaggerated phasic component of stretch reflex is mostly due to interruption of inhibitory descending pathways from upper CNS centers (13). Tendon hyper-reflexia, an exaggerated muscle response to an externally applied tap of deep tendons, is suggested to be due to reduced pre-synaptic inhibition of group Ia fibers (15). Clonus, involuntary rhythmic muscle contraction, occurs by a sudden rapid stretch of the muscles and recurrent activation of the stretch reflex (12,13,15). Extrinsic spasticity, involuntary muscle spasms, is mostly a response to external factors that influence the muscle activity (13,15,17). Flexor spasms is the most common subdivision of extrinsic spasticity that triggered by the afferent inputs including skin, muscle, subcutaneous tissue and joints (3).

In summary, decreased central inhibitory drives, peripheral neural changes, and non-neural factors result in changes in tonic, phasic component of stretch reflex and extrinsic spasticity. These
pathophysiologic impairments result in different manifestations of spasticity in individuals with UMN lesions.

1.3 Spasticity Measurement

Several methods are used to measure the degree of spasticity that are mostly categorized as self-report questionnaires, biomechanical, neurophysiological, and clinical outcome measures (30,31). Although within the framework of the International Classification of Functioning, Disability and Health (ICF) spasticity outcome measures should be reliable and valid, spasticity outcome measures are employed in isolation of one another and have limited crossover with each other (32,33). For instance, although biomechanical methods such as electrogoniometry and dynamometry are frequently used as spasticity outcome measures in research, they are rarely used in routine clinical settings (14,34). The clinicians and researchers manage spasticity based on the results of spasticity outcome measures which shows the importance of choosing the most accurate spasticity outcome measure (33,35). Spasticity outcome measure methods are described in the next following sections.

1.3.1 Clinical Spasticity Assessments

Clinical spasticity outcome measures are mainly based on the resistance of spastic limb to passive movements which are evaluated by a physician or therapist (34). Clinical measurements mostly involve the examination of a single agonist group of muscles over a specific range of motion either with or without employment of gravity (14,26,36). Although clinical spasticity outcome measures have the benefits of minimal equipment cost and setup, there are some limitations including assessment of only one component of spasticity rather than providing a measure of a person’s overall spasticity (14,26,36).

The most common clinical spasticity assessments are the Ashworth Scale and the Modified Ashworth Scale (14,19,26,37). The Ashworth scale (AS) is a five-point nominal scale based on the subjective clinical assessment of muscle tone (38,39). AS was first used as a clinical classification to assess antispastic effects of the drug, carisoprodol, for patients with multiple sclerosis. Spasticity was quantified based on the reflex activity elicited in muscle groups oppose to the passive movement (38,39).
Modified Ashworth Scale (MAS) is described as a six point clinical measurement by adding a +1 grade to AS that enhance the sensitivity of Ashworth scale (Table 1.1) (38,40).

**Table 1.1 Modified Ashworth Scale**

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No increase in muscle tone</td>
</tr>
<tr>
<td>1</td>
<td>Slight increase in tone with a catch and release or minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion and extension</td>
</tr>
<tr>
<td>+1</td>
<td>Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of motion</td>
</tr>
<tr>
<td>2</td>
<td>More marked increase in muscle tone through most of the range of motion, but the affected part(s) are easily moved</td>
</tr>
<tr>
<td>3</td>
<td>Considerable increase in tone, passive movement difficult</td>
</tr>
<tr>
<td>4</td>
<td>Affected part rigid in flexion or extension</td>
</tr>
</tbody>
</table>

MAS +1 grade was added to Ashworth scale to assess hemiplegic patients spasticity that was often graded at the lower end of scale (38,40). Each grade corresponds to a level of spasticity based on the resistance examiner experiences through the passive range of motion (38). Although AS and MAS are simple spasticity outcome measures that do not need specialized equipment, the examiner should be well trained with the experience of clinical outcome measures (38).

The inter-rater reliability of the AS and MAS is considered adequate for lower limb muscles such as adductors (kappa value=0.61 for AS and 0.62 for MAS) and flexors (kappa value=0.49 for AS) compared to a poor inter-rater reliability for plantar flexor muscles (kappa value=0.21 for AS and 0.20 for MAS) (41,42). Validity of AS and MAS has been partially established and even less so for responsiveness, a significant limitation of clinical spasticity outcome measures (38).

There is a lack of compatibility between AS and MAS correlation with self-assessment and electrophysiologic spasticity outcome measures. The AS correlates poorly with spasticity self-assessment (VAS) but the correlation is higher between MAS and VAS (r=0.44-0.62, P<0.1) (43,44). Sherwood et al. found significant differences between muscle activity (EMG) of lower extremity muscles at higher
level of spasticity reported by MAS versus the lower [0,1] levels (45). None of recorded EMG of those muscles was able to distinguish between low Ashworth categories [0 vs 1] (45). However, Sköld et al. showed an increase of correlation values between EMG modalities (mean, peak, start-peak) of different muscles and corresponding MAS grading of 0-4 compared to 1-4 (46).

The Tardieu Scale has been recently suggested as a suitable and reliable spasticity outcome measure (47). The Tardieu Scale was originally developed by Tardieu et al. in 1954 (48) and later modified by Held and Pierrot-Deseilligny in 1969 (49). Boyd and Graham in 1999 have further modified the Tardieu Scale to an outcome measure called Modified Tardieu Scale (MTS) (50,51).

MTS standardized procedures measure the quality of muscle reaction to passive movements at specific velocities including fast stretch, speed of the limb segment falling under gravity, and slow controlled motion (50). The primary MTS evaluation includes assessment of quality of muscle reaction during the slow controlled motion by using full passive range of motion (R2) grading from 0-4 (Table 1.2) (47,50). The point of spasticity provoked ‘catch’ is done if the quality of muscle reaction is above 2 (47,50). The next step is the fast stretch maneuver during hyperactive stretch reflex and the particular angle at which ‘catch’ occurs is called R1. This secondary evaluation represents the angle of muscle reaction (R1) (47,50,52). The difference between two measures (R2-R1) is dynamic tone component of the muscle (55,58,60). A large difference between full passive range of motion and angle of muscle reaction (R2-R1) represents spasticity and small difference between these two variables represents contracture (47,50,51).
Table 1.2 Modified Tardieu Scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No resistance throughout the course of the passive movement</td>
</tr>
<tr>
<td>1</td>
<td>Slight resistance throughout the course of the passive movement, with no clear catch at precise angle</td>
</tr>
<tr>
<td>2</td>
<td>Clear catch at precise angle, interrupting the passive movement, followed by release</td>
</tr>
<tr>
<td>3</td>
<td>Fatigable clonus (&lt;10 seconds when maintaining pressure) occurring at precise angle</td>
</tr>
<tr>
<td>4</td>
<td>Infatigable clonus (&gt;10 seconds when maintaining pressure) occurring at precise angle</td>
</tr>
</tbody>
</table>

For MTS R2-R1 measures, test-re-test and inter-rater reliability has been reported to be high and low respectively (53). Mehrholz et al. study showed a good test-retest reliability (kappa=0.52-0.87) whereas the inter-rater reliability was poor to moderate (kappa=0.29-0.53) for MTS (53). Ben-Shabat et al. showed moderate to high intra- (kappa=0.52-0.77) and interrater (kappa=0.27-0.53) reliability for lower extremity muscles MTS (54). The MTS might be a reliable spasticity outcome measure for lower extremity muscles of individuals with chronic neurologic disorders (54).

1.3.2 Self-report Questionnaire

Routine clinical spasticity outcome measures are incorporated with personal assessment of spasticity (34). Clinicians rely on the personal assessment of patients to determine their level of spasticity as well as other clinical or non-clinical spasticity outcome measures. The significance of using spasticity self-assessment in the clinical setting initially became popular from various research studies. Different self-report spasticity outcome measures assess the severity, frequency, and impact of spasticity on daily life (34,43). Visual Analog Scale (VAS), Penn Spasm Frequency Scale (PSFS), and Spinal Cord Injury Evaluation Tool (SCI-SET) are mostly used as the self-report spasticity outcome measures (38,55–57).

The Visual Analog Scale (VAS) is a simple graphical designed measurement instrument for subjective characteristics that cannot be directly measured objectively. By using the VAS measurement, the participants indicate a point along a continuous line between two end-points (38). VAS is a simple and quick self-report outcome measure to assess baseline spasticity and after a specific intervention (38). The participants score spasticity from ‘no spasticity’ to ‘the most imaginable spasticity’ (43).
VAS reliability appeared to be high but there is controversy between evidence regarding to the correlation between VAS and clinical outcome measures (34,38,43,58). Sköld in 2000 demonstrated a significant correlation (r=0.44 to 0.62, P<0.001) between VAS and MAS measures (43). Lachner et al. showed a poor (ρ=0.36) and moderate (ρ=0.70) correlation between MAS and self-rating (VAS) of general and present spasticity respectively. The present spasticity was defined as the level of spasticity after an intervention (34). The lower VAS and MAS correlation is suggested to be most likely due to scoring spasticity after a specific activity rather than the general spasticity perception that the participants experienced during their daily life (34). However, VAS might represent a different dimension of spasticity compared to the clinical outcome measures such as MAS (34).

Penn et al. in 1989 defined a five-point ‘Spasm Frequency Scale’ self-report questionnaire (56). The first five-point scale component assesses the frequency of spasms ranging from ‘0=no spasm’ to ‘4=spontaneous spasms occurring more than 10 times per hour’ (Table 1.3) (38,56). Priebe et al. (1996) later modified the five-point Penn Spasm Frequency Scale (PSFS) to a two component self-report questionnaire to provide a more comprehensive understanding of the spasticity status of the participants (35,36). The complementary component of the modified PSFS is a three point scale assessing the severity of spasms ranging from ‘1=mild’ to ‘3=severe’, if the participant indicates spasms through the first five-point scale (Table 1.4) (35,38). The modified PSFS is a simple and appropriate alternative to the clinical spasticity outcome measures with no especial equipment requirement (35,38). The PSFS is only correlated moderately with routine clinical spasticity outcome measures such as MAS, suggesting that the physical examination might evaluate some aspects of spasticity that do not represent entirely what is important to individuals with spasticity (36).
Table 1.3 Penn Spasm Frequency Scale

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>0</td>
<td>No spasm</td>
</tr>
<tr>
<td>1</td>
<td>Mild spasm induced by stimulation</td>
</tr>
<tr>
<td>2</td>
<td>Infrequent full spasm occurring &lt;1/h</td>
</tr>
<tr>
<td>3</td>
<td>Spasms occurring &gt;1/h</td>
</tr>
<tr>
<td>4</td>
<td>Spasms occurring &gt;10/h</td>
</tr>
</tbody>
</table>

Table 1.4 Modified Penn Spasm Frequency Scale

Part 1: Spasm Frequency Scale

<p>| | |</p>
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>0</td>
<td>No spasm</td>
</tr>
<tr>
<td>1</td>
<td>Mild spasm induced by stimulation</td>
</tr>
<tr>
<td>2</td>
<td>Infrequent full spasm occurring &lt;1/h</td>
</tr>
<tr>
<td>3</td>
<td>Spasms occurring &gt;1/h</td>
</tr>
<tr>
<td>4</td>
<td>Spasms occurring &gt;10/h</td>
</tr>
</tbody>
</table>

Part 2: Spasm Severity Scale

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mild</td>
</tr>
<tr>
<td>2</td>
<td>Moderate</td>
</tr>
<tr>
<td>3</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Adams et al. in 2007 developed a new spasticity self-report questionnaire, called Spinal Cord Injury Spasticity Evaluation Tool (SCI-SET), to provide a broad scope evaluation of either ‘helpful’ or ‘harmful‘ effects of spasticity in individuals with spinal cord injury who experience spasticity (55). The SCI-SET is a 35 items, 7 day recall questionnaire that target different aspects of daily life in individuals with SCI (55). Each individual with SCI rates the impact of spasticity on daily life from -3 (extremely problematic) to +3 (extremely helpful) with the option of choosing ‘0’ that means spasticity has no effect on that aspect of life (55).
The SCI-SET questionnaire consists of a wide range of daily living (showering, eating, dressing), emotional health (feeling of embarrassment, being annoyed), independence (control over the body, concern of falling need to ask for help), and social activities (hobbies, recreation, sex life) (55). The instruction includes a statement asking individuals with SCI to recall the previous week and choose the scale from -3 to +3 to demonstrate how spasticity affects their life during the past 7 days. In this spasticity evaluation tool they used a broad definition of spasticity including 1) uncontrolled, involuntary muscle contraction or movement which can be slow, rapid, short or prolonged, 2) involuntary, repetitive, quick muscle movement which can be up and down or side to side, 3) muscle tightness, and 4) what the subject might describe as spasm (55). The total SCI-SET score is calculated by adding all applicable scores together including positive and negative that retain the signs to represent the global impact of spasticity (55). The average score is calculated by dividing the total score by the number of applicable items. Positive and negative scores indicate the beneficial or detrimental effects of spasticity on daily living of individuals with SCI (55). Although SCI-SET is moderately (r=0.66) correlated with PSFS self-report questionnaire, there is not enough evidence yet to show the relationship between SCI-SET and clinical spasticity outcome measures (55).

In summary, self-report spasticity outcome measures are mostly reported with respect to global perception of spasticity, while clinical outcome measures are reported based on each specific muscle group activity provoked by a movement (34,43). Self-report scores might be attributed to other symptoms such as pain as a part of spasticity, while clinicians only consider muscle tone responses and reflexes as spasticity (34,43). Sensory spasticity is described as sensations caused by paresthesia including pain, prickle, tension, and constriction may also be reported as spasticity by self-report questionnaires. Sensory spasticity has no relation with clinical spasticity outcome measures (59,60). This evidence may suggest there is a difference between what is measured subjectively through self-report versus clinical outcome measures.
1.3.3 Biomechanical, Electrophysiologic, and Neurophysiologic Spasticity Outcome Measures

Biomechanical spasticity outcome measures include Wartenberg pendulum test and isokinetic dynamometer (38,53). Electrophysiologic and neurophysiologic spasticity outcome measures are mostly Electromyography (EMG), Hofmann reflex (H-reflex), and Tendon reflex (T-reflex) (46,61).

1.3.3.1 Biomechanical Spasticity Outcome Measures

The Wartenberg Pendulum Test (WPT) was primarily used as a simple and reliable clinical outcome measure for Parkinson lower limb hypertonia (62). It has been changed to a score with specific quantitative measurements for upper motor neuron disorders (63). The pendulum score is calculated during the gravity induced pendulum-like movement of the lower limb and the ratio of the joint angles are measured by goniometers or computerized video motion analysis (64). Spasticity is reported as a reduction of limb swing of WPT (65). The WPT is not used widely in clinical spasticity assessment, probably due to the requirement of specialized equipment and post-test analysis however it could be more applicable as a more reliable research tool (38,66). Although validity and responsiveness of WPT has not yet been well established, there was a significant correlation between AS and the amplitude of first swing of the WPT (P<0.001) (38,66).

An isokinetic dynamometer is used to measure spasticity quantitatively according to Lance definition (67). The joint is sinusoidally oscillated at different constant angular velocities and the generated muscle force during the movement is recorded by isokinetic dynamometer (67). Spasticity is reported as the amount of generated muscle force (67). Kim et al. showed higher isokinetic dynamometer values (e.g. peak torque, work, and threshold angle) in spastic population compared to the normal control group at different angular velocities (67). In that study, the torque threshold angle was suggested as the most precise isokinetic outcome measure for spasticity assessment (67). The peak torque (r=0.557-0.718), torque threshold angle (r=0.454-0.566), and work (r=0.648-0.724) were all significantly correlated with the MAS (67).
1.3.3.2 Electrophysiologic and Neurophysiologic Spasticity Outcome Measures

EMG is the electrophysiologic and H-reflex and T-reflex are the neurophysiologic spasticity outcome measures in individuals with UMN lesions (29,46,61). EMG records the reflexive electrical activity of the testing muscle against stretch to evaluate the muscle spasticity (46). Surface EMG is a reliable and repeatable outcome measure that is conducted by placing the electrodes directly on the skin over the muscles and different analyzing methods are available to evaluate spasticity (46,68). Zupan et al. in 1998 described spastic response as any non-zero EMG activity resulting from passive maneuver without any specific defined cut off of the muscle electrical activity (69). Laessoe et al. defined spasm as EMG activity amplitude of equaling 4 times or more than the baseline amplitude with duration of more than 5 seconds (70). However there is no standard definition for the amplitude and duration of involuntary muscle activity that constitutes a spasm.

The correlation between EMG and clinical spasticity outcome measures is not clear. Kim et al. found no correlation between EMG and MAS (67). Sökld et al. studied the correlation between EMG and MAS during simultaneous EMG recording and MAS assessment of thigh muscles in nine complete tetraplegic individuals (46). Different EMG variables including mean, peak, duration, starting peak were significantly correlated with corresponding MAS assessments, ranging from 0 to 4 (46). The MAS might accurately reflect the movement provoked spasticity in motor-complete tetraplegic individuals (46). Albani et al. in 2010 studied the effects of botulinum toxin (Botox) injection therapy on spasticity using MAS and EMG outcome measures after 30 and 180 days (71). EMG was measured at baseline, during active and passive movements. In that study, the root mean square (RMS) of flexor and extensor carpi radialis muscles EMG signals was decreased significantly at baseline and during passive flexion after 30 and 180 days. The results showed stronger improvements for EMG activity compared to the MAS clinical assessment (71). In summary, there is no precise conclusion regarding to the correlation between EMG and MAS spasticity outcome measures based on the present described literature and further studies are required to clarify how EMG and MAS are similar or different for spasticity assessment.
H-reflex is a quantitative electrical analog of the monosynaptic stretch reflex. Although H-reflex was first described by Paul Hofmann in 1910 (72), it was used as a neurophysiologic measurement to assess the influence of Ia sensory afferent on alpha motor neuron activation in 1940s and early 1950s (73). Between late 1950s and mid 1980s, H-reflex was modified with using conditioning electrical stimulation to show the effects of inhibitory or excitatory pathways on alpha motor neuron (74). Recently, H-reflex has been used to demonstrate the neural input influences from sensory afferents, descending pathways on alpha motor neuron, motorneuron excitability and reflecting presynaptic inhibition of Ia afferent neuron synapses (74,75).

H-reflex is applied by electrical stimulation of peripheral nerve, e.g. tibial nerve in popliteal fossa for soleus muscle, thereby bypassing the effects of gamma motorneuron and muscle spindle discharge (75,76). Soleus muscle is convenient for H-reflex measurement due to large diameter of Ia afferent neuron and small diameter of alpha motor neuron. This considerable different size of these two nerves helps to electrically stimulate Ia afferent neuron selectively by using low-intensity stimulus and stimulate alpha motorneuron by using high-intensity stimulus (75,77). Percutaneous electrical stimulation is delivered to Ia sensory afferent and the sensory nerve action potential travels from stimulus point to the spinal cord motorneuron pool (75,78). Released neurotransmitters in the spinal cord result in excitatory postsynaptic potentials (EPSP) and when the EPSP reaches the excitation threshold, it causes an action potential on alpha motorneuron and its innervating muscle fibers (78). Alpha motorneuron action potential through this pathway is reported as H-wave and the amplitude of H-wave differs depending on excitability of individual alpha motorneuron in the spinal cord motorneuron pool (78). Gradual increase of stimulus intensity in stimulation site results in direct action potential of alpha motorneuron that travels to the muscle fibers and results in direct M-response (M-wave) (75). M-wave amplitude reflects simultaneous activation of alpha motorneuron and the amplitude of M-wave depends on the physiological characteristics of efferent nerve fibers, neuromuscular junction and muscle fibers without any influence from spinal mechanisms (75,79).
H-reflex has been used as a spasticity outcome measure due to its capability of measuring alpha motorneuron excitability, presynaptic inhibition, and reciprocal inhibition disturbances. These variables play an important role in pathophysiology of spasticity (29). Although there is a relation between MAS and H-reflex variables specifically alpha motor neuron excitability in individuals with UMN lesions, there is no significant correlation between these two outcome measures (80–83).

T-reflex is considered the mechanical counterpart of the H-reflex. Tendon tap stimulates muscle spindles as primary sensory receptors and impulses travel to spinal cord through Ia afferent sensory neuron (61,84,85). The Ia afferent sensory neuron synapses with alpha motor neuron in the spinal cord. Alpha motor neuron sends the impulses to the muscle fibers which results in a short quick contraction (61,84,85). Surface EMG is used to record the muscle responses after the tendon taps. These muscle responses generate three different variables; tendon reflex, a silent period, and a subsequent long-loop reflex (86–88). The silent period might be due to Ib inhibitory interneuron activation, activation of mechanoreceptors, or simply the time advancing the action potential occurrence (86–88). Several T-reflex parameters reflect the pathophysiology mechanism underlying UMN lesions (61). For example, T-reflex latency, indicating alpha motor neuron excitability, was found to be shorter in spastic individuals compared to longer (30–40 ms) Achilles tendon latency in normal able-bodied individuals (61). T-reflex amplitude represents the number of motorneurons that are activated by tendon taps. This parameter might be increased in individuals with spasticity (61,89). There is a moderate correlation (r=0.459-0.706) between T-reflex amplitude and clinical spasticity outcome measures by MAS (89).

### 1.4 Spasticity and Spinal Cord Injury (SCI)

SCI is an UMN lesion that can be classified as either a traumatic SCI (e.g. motor vehicle accidents, work-related accident, falls, violet incidences (gun wounds, stabbing), sport and recreational injuries) or a non-traumatic SCI (e.g. tumors, birth defects, and vascular ischemia) (90). Traumatic and non-traumatic SCI can both result in spinal cord dysfunction, with loss of sensory and motor function distal to the level of injury (90–92).
SCI is generally classified based on the sacral sparing definition to ‘complete’ or ‘incomplete’ injuries (93). In complete SCI, both sensory and motor functions are permanently lost in the lowest sacral segments (S4-S5). Whereas incomplete SCI involves partial preservation of sensory and/or motor function below the neurological level that includes the lowest sacral segments (S4-S5) (94,95). The American Spinal Injury Association (ASIA) develops a standardized method of assessing the neurologic functional status of individuals with SCI (93,96). American Spinal Injury Association (ASIA) Impairment Scale (AIS) includes five categories from A to E with different level of preserved sensory or motor function in individuals with SCI (Table 1.5) (93,96). Although AIS provides a precise classification for SCI, SCI can also be classified as quadriplegia and paraplegia (93). Quadriplegia (or tetraplegia) is referred to impairment or loss of motor and/or sensory function in cervical segment of the spinal cord that results in loss of function of four extremities, trunk and pelvic organs (93). Paraplegia is defined as impairment or loss of motor and/or sensory function in spinal segments lower than the cervical spinal segment that are thoracic, lumbar, or sacral segments (93).

Table 1.5 The Five Categories of the ASIA Impairment Scale (AIS)

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A=Complete</td>
<td>No motor or sensory function is preserved in the sacral segments S4-S5</td>
</tr>
<tr>
<td>B=Incomplete</td>
<td>Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5</td>
</tr>
<tr>
<td>C=Incomplete</td>
<td>Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade of less than 3</td>
</tr>
<tr>
<td>D=Incomplete</td>
<td>More function is preserved below the neurological level, and at least half of the key muscles below the neurological level have a muscle grade of 3 or more</td>
</tr>
<tr>
<td>E=Normal</td>
<td>Motor and sensory function are normal</td>
</tr>
</tbody>
</table>

SCI associated hospital cost accounted for $61.6 million CAN dollar in Canada in 2000-2001 (92). In the United States, estimated average annual medical cost of an SCI is $15,000-$30,000 per year with an estimated lifetime cost of $500,000 to more than $3 million USD depending on injury severity.
Incidence and prevalence of SCI, spanning various population groups throughout the world, demonstrate an increasing trend during recent decades (98,100,101). Tetraplegic and complete SCI epidemiology has been increased during the last decade (101). Increasing trend of incidence of tetraplegics and complete lesions are anticipated to have further detrimental effects on society, health care system, treatment, and rehabilitation of patients with SCI (2,98,100,101).

With the long-term neurologic impairment in body organs innervating by injured nerves, an SCI has devastating effects on an individual’s life from a physical, psychological, and socioeconomic point of view as well as an immense effect on society health properties (92,97). Spasticity is one of the many challenges faced by those with an SCI effecting 65-78% of individuals (2).

Symptoms of spasticity including exaggerated tendon reflexes, increased muscle tone, and involuntary muscle spasms are developed gradually after injury (102). There is a period of flaccid muscle paralysis with loss of tendon reflexes below the level of injury lasting between 1-3 days to few weeks after injury (102). Spasticity results in a widespread range of clinical consequences such as pain, fatigue, disfigurement and disability (21). Untreated spasticity causes abnormal posture, movement difficulties, deformities, contractures, more pain and fatigue (103,104). These clinical consequences limit active and passive joint mobility and interfere with activities of daily living (ADLs) such as feeding, dressing, hygiene, bladder and bowel control, and mobility (37,103,104). Spasticity also limits workplace participation and independence living (2,4,5). Some external factors such as medication, pain, urinary tract infection, constipation, fatigue, and mental state might influence and exacerbate spasticity (105).

While spasticity has many negative outcomes, some beneficial outcomes such as improved transfer, weight bearing due to maintained muscle tension, preventing muscle atrophy, improve circulation by increasing venous return, reduce risk of deep venous thrombosis (DVT) and osteoporosis would help individuals with SCI in terms of their ADLs (102,106,107).
1.5 Spasticity Management

Spasticity management is based on the reduction of passive challenges by preventing contractures, pain reduction, facilitating splint wearing, improve positioning, and hygiene as well as increasing the ability to perform useful tasks (12). In general, rehabilitation modalities, pharmacologic intervention and surgery are three main therapeutic techniques for spasticity management (3).

1.5.1 Physical Therapy Techniques

Physical therapy techniques are the essential component of spasticity management as these techniques are useful and beneficial during and after pharmacologic and surgical strategies (3). Passive stretch, passive lengthening, weight-bearing techniques, muscle strengthening, electrical stimulation, vibration, splints and orthoses are the most common physical therapy techniques for spasticity management (102,3). Passive stretch and passive lengthening move the muscles through their full range of motion and maintain the muscles in a prolonged desired holding position. Although spasticity reduction by these techniques lasts only for several hours, they are simple and easy to apply. Spasticity reduction effectiveness by these techniques has not been quantified and the efficacy has not been determined (102). Weight-bearing physical therapy techniques provide a prolonged stretch by using a tilt-table or standing frame (3,108). Spasticity reduction by weight-bearing techniques lasts for a short period of time but may last into the next day (3). Muscle strengthening technique includes improving of voluntary muscle control by progressive addition of muscle resistance. Muscle strengthening physical therapy programs of the lower extremity using stair climbing or treadmill training result in activating different muscle groups that may produce better functional improvement, better functional position, and a greater effect on overall patient performance (102). In general, all these physical therapy techniques reduce spasticity from several hours to a maximum of one day. Although valuable the limitations that explain the importance of using additional spasticity management modalities and interventions to improve management (3,102).
Electrical stimulation physical therapy modalities include several methods such as antagonist muscle stimulation, application of titanic contraction of spastic muscle, transcutaneous electrical nerve stimulation (TENS) and functional electrical stimulation (102). Electrical stimulation therapeutic modality mechanisms are described in Table 1.6.

Table 1.6 Electrical Stimulation Modalities Mechanism

<table>
<thead>
<tr>
<th>Modality</th>
<th>Suggested Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antagonist muscle stimulation</td>
<td>Augmentation of the reciprocal inhibition of the antagonist muscle</td>
</tr>
<tr>
<td>Application of titanic contraction</td>
<td>Repetitive stimulation causes fatigue in muscles</td>
</tr>
<tr>
<td>FES</td>
<td>Strengthening of the antagonist of spastic muscle or augmentation of the reciprocal inhibition of the spastic muscle</td>
</tr>
<tr>
<td>TENS</td>
<td>Stimulation of the large diameter afferent fibers that travel from mechanoreceptors to spinal cord</td>
</tr>
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</table>

A significant concern regarding the benefits of these electrical stimulation modalities for spasticity reduction is that the repetitive nature of these modalities might cause muscle fatigue. Antagonist muscle stimulation antispastic effects on spasticity have similar temporary results to muscle stretching and strengthening lasting from 15 min to 3 hours with an average of 1 hour (102). TENS is an electrical stimulation modality that needs special equipment, time, and well-trained personnel but failed to show definitive and consistent decrease of spasticity after interventions (102). Functional electrical stimulation (FES) is an electrical stimulation modality in which the electrical current is passed through peripheral nerve to the paralyzed muscle. Electrical current results in muscle contraction and the contraction of antagonist muscle helps with spasticity reduction (109). The FES combined programs with cycling resulted in a significant reduction of spasticity muscle resistance in individuals with SCI (109). In summary, electrical stimulation modalities need special equipment, well-trained and experienced
personnel to do the procedures that make electrical stimulation modalities less practical for spasticity management.

Vibration including whole body vibration (WBV) and focal vibration (FV) has been used for spasticity management (70,110–118). Vibration can be applied with different frequencies, magnitudes, and durations to different muscle groups (FV) in different body positions (70,110–118). Although spasticity reduction was reported by WBV and FV, the appropriate vibration frequency, magnitude, and duration are largely unknown. The mechanism by which vibration results in spasticity reduction is still unclear (119).

Splints and orthoses are used as side physical therapy techniques to compensate the paresis of upper and lower extremities, prevent deformities, and might help to reduce pain (102). Splints and orthoses prevent contracture formation in individuals with spasticity and improve the range of motion. The mechanism that splints and orthoses result in spasticity reduction is not clear and it might be due to mechanical effects of these devices on musculoskeletal system (120–122).

In summary, some physical therapy techniques are easy to apply and they do not need special equipment or well-trained and experienced therapist but they are not long lasting. Conversely, complicated techniques result in long lasting spasticity reduction but special equipment and presence of experienced therapist are the essential components of those techniques.

### 1.5.2 Pharmacological Agents

Pharmacological agents are considered to manage spasticity by minimizing complications, pain, and functional deficit (12). Most of pharmacological antispastic agents can be divided into three categories as listed below; a) GABAergic agents such as baclofen, gabapentine, and diazepam, b) alpha-2-adernegic agents such as tizanidine and clonidine, c) peripheral acting agents such as dantrolene (3). GABAergic, alpha-2 adernegic, and peripheral acting agents act through gamma-aminobutyric acid (GABA) receptors in CNS, alpha-2 receptors in CNS, and at the neuromuscular level respectively (3).
Intrathecal antispastic agent (baclofen) injection is also a well-tolerated method of spasticity management with minimal side effects compared to oral medications (25,123). Intrathecal injection technique solved the limitation of crossing the blood brain barrier (BBB) (28). In general, the long-term side effects of intrathecal antispastic injection are unknown. Surgical catheter implantation complications include dislodgment, disconnection, migration, catheter kinking, blockage, pump failure, battery depletion, infection, and accidental overdose (3). Oral baclofen side effects are possible while managing spasticity by intrathecal baclofen pump (3).

Phenol and botulinum toxin are injective antispastic agents that are effective for a longer period of time compared to oral antispastic agents (28). Phenol injection provides a temporary peripheral motor nerve block that lasts for weeks to months (124). Temporary nerve block reduces spasticity, prevents limb contracture and gives the muscle a better range of motion (124). Botulonium toxin A (Botox®) injection provides a temporary nerve-ending block at neuromuscular junction and in most cases, is better tolerated compared to phenol injection (28). Botox® is a safe injective antispastic agent with few side effects and rare drug reactions. Although Botox® is considered a well-tolerated and effective antispastic agent, it is expensive and again lasts only temporarily between 2 to 6 months (102).

Anti-spastic agents produce unwanted pharmacologic-induced side effects and a non-targeted release of pharmacologic agents that result in a general suppression of nervous system (3). General side effects of the anti-spastic agents are listed in Table 1.7 (28,55,123).
### Table 1.7 Antispastic Agents Side Effects

<table>
<thead>
<tr>
<th>Anti-spastic Agent</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam (GABAergic)</td>
<td>Sedation, risk of dependence</td>
</tr>
<tr>
<td>Baclofen (GABAergic)</td>
<td>Daytime sedation, dizziness, weakness, fatigue, nausea, lower seizure threshold</td>
</tr>
<tr>
<td>Gabapentine (GABAergic)</td>
<td>Somnolence, dizziness, ataxia, fatigue</td>
</tr>
<tr>
<td>Clonidone (Alpha-2-adrenergic)</td>
<td>Bradycardia, hypotension, depression, dry mouth, sedation, dizziness, constipation</td>
</tr>
<tr>
<td>Tizanidine (Alpha-2-adrenergic)</td>
<td>Dry mouth, sedation, dizziness, mild hypotension, weakness</td>
</tr>
<tr>
<td>Dantrolene (Peripheral acting drug)</td>
<td>Generalized muscle weakness, mild sedation, dizziness, nausea, diarrhea, hepatotoxicity</td>
</tr>
<tr>
<td>Phenol</td>
<td>Chronic dysesthesia, pain, peripheral edema, skin sloughing, wound infection</td>
</tr>
<tr>
<td>Botox</td>
<td>Drug reaction (rare), muscle pain, bruising, transient fever (self-limiting), expensive, tolerance</td>
</tr>
</tbody>
</table>

#### 1.5.3 Surgical Intervention

Surgical interventions for spasticity reduction include peripheral ablative, central ablative and non-ablative procedures (125). Peripheral ablative surgical interventions such as posterior rhizotomy and peripheral neurectomy interrupt afferent or efferent neuron fibers (125). Posterior rhizotomy interrupts the entire posterior nerve root (afferent fibers) of the stretch reflex with a good efficacy for spasticity management (125). The main side effect of posterior rhizotomy is a wide unacceptable sensory loss after the surgery (125). Peripheral neurectomy interrupts the peripheral motor nerve branch and through the surgery the nerve is divided to reduce the muscle tone and overactivity (125). This surgical technique may result in weakness and sensory loss (125). Central ablative procedures such as cordectomy and myelotomy target the CNS directly at the level of spinal cord and specific brain nuclei for spasticity treatment. Non-ablative surgical procedures include peripheral nerve point block or implantation of cerebellar or spinal stimulators (125). Surgical techniques are mostly performed in individuals with CP.
These techniques are usually reserved for complications of spasticity such as contractures and as the last spasticity management option (3,125).

1.6 Standing, Walking, and Mobility in Individuals with SCI

The International Classification of Function (ICF) defines mobility as ‘moving by changing body position or location, transferring from one place to another, by carrying, moving or manipulating objects, by walking, running or climbing, and by using various forms of transportation’(33). Mobility and standing, other than their ambulatory role, might be beneficial for spasticity management (123,126).

1.6.1 Standing and Walking

Supported standing has many proposed effects and potential benefits from early stages of rehabilitation after SCI to late stages of rehabilitation (127). In early stages of rehabilitation, supported standing improves respiratory function, increases arousal and helps for orthostatic hypotension management (128,129). Supported standing can improve posture and prepare the individuals with UMN lesions for gait training by strengthening antigravity muscles and retraining head and upper limb control (127,128,130,131). It provides prolonged weight-bearing stretch to lower extremities that is useful for muscle length and spasticity management, prevention of contracture development and bone mineral density reduction, and improving level of function such as feeding, dressing, hygiene, mobility, bladder and bowel control (37,127).

Upright standing has been used for spasticity management in individuals with UMN lesions. Increased sensory inputs to the sole of the feet during standing might increase the inhibition of stretch reflex, reduce the motor neuron excitability and reduce spasticity (129,132). Standing training might result in spasticity reduction after some training sessions (127,133,134). Baker et al. tested standing training by using standing frames in individuals with Multiple Sclerosis (MS) during a two/three week period (133). Post-standing spasticity outcome measures showed a significant change in ankle dorsiflexion and hip extension range of motion and no significant change in MAS and PSFS (133).
Bakheit et al. performed a 20 min single session isotonic and isokinetic stretch therapeutic interventions in standing and semi-reclined positions in individuals with stroke. The soleus H-reflex was used as the spasticity outcome measure (133). H-reflex variables (latency and H/M ratio) did not show any significant difference between the baseline values and those recorded immediately after the interventions or 24 hour later in either standing or semi-reclined position (134). They suggested the probability of spasticity reduction by mechanisms other than the direct effect on alpha motor neuron (134). Field-Fote et al. examined the effects of standing on spasticity compared to sitting position in individuals with incomplete SCI versus able-bodied (AB) (135). Post-activation depression (PAD) was greater in standing position, although PAD was less in individuals with incomplete SCI (135). This evidence supports the role of different standing training protocols on spasticity reduction. However, this evidence is not strong enough to make a clear evidence-based conclusion for managing spasticity by supported standing.

Spasticity has negative effects on different aspects of walking and mobility in individuals with UMN lesions (136). It can affect walking speed, endurance, and self-report walking impairment as well as balance (136). Spasticity reduction and walking improvement are in a two directional pathway as spasticity reduction can improve walking and walking training result in spasticity reduction (126,137). Nash et al. used a bilateral AFO orthotics as spasticity-reducing tool in individuals with incomplete SCI. The AFO orthotics resulted in increased step length, gait velocity, and reduced the amount of time spent in double limb support (137). Mirbagheri et al. trained individuals with incomplete SCI for 4 weeks with Robotic-Assisted Locomotor (LOKOMAT) to assess how spasticity would change after walking training. Different aspects of spasticity including reflex stiffness and muscular stiffness reduced significantly after the 4-week gait training (126).

Different walking speeds change spinal reflex modulation with a greater effect of faster walking speeds on spasticity reduction. Phadke et al. compared the effects of two different walking speeds on H-reflex modulation in AB and individuals with SCI. Although the position-dependent H-reflex modulation
is impaired in individuals with SCI, the H-reflex modulation was greater at speed of 1.2 m/s compared to speed of 0.6 m/s (138).

It is clear that spasticity reduction improves walking skills but in some individuals spasticity and spasms facilitate walking. Excessive muscle tone keeps the lower extremities straight and provides support for walking with crutches, splints and orthoses. Spasms also assist them in chair to bed transfer (33,139,140). However, the minimal positive effects that spasticity and spasms have on walking are not enough to compensate the negative effects of them on their walking skills (139).

1.6.2 Mobility

Most individuals with SCI are dependent on a variety types of mobility aids at least 10 years after their injury to live independent with a better quality of life (141). The mobility aids vary from simple devices such as crutches, rolling walkers, sliding boards, hand cycles, standing frames, lower extremity braces (ankle foot orthoses) to complicated manufactured electrical scooters, stand-up devices (Segway), manual and electrical wheelchairs (141–143). The mobility device that is needed for each individual with UMN lesion is determined based on the users’ neurologic functional level, physical dimension, goals in life and their surrounding environment (144). Orthoses and splints are useful for mobility of individuals with higher level of function. These aids support the paretic segment of the limb, prevent deformities secondary to contractures, and reduce pain in extremities (102). Orthoses provide the opportunity for individuals with UMN lesion to stand and walk with other mobility devices such as crutches with additional benefits of joint stability and contracture prevention (102,140).

Wheelchairs are the most common mobility devices for individuals with SCI at the present, and most likely will remain the most common for at least the next decade (140). Individuals can use wheelchair for longer distances with less fatigue or any other physical challenges (141,144). There are lots of wheelchair design improvements, material changes, and manufacturing that have resulted in decrease of wheelchair weight and increase in the ease of mobility of the chairs which enables even more ease of use (140). Although wheelchair manufacturing have been improved, SCI wheelchair users
anecdotal report shows spasm triggering while they use their wheelchairs in over a variety of surfaces such as sidewalk bumps or rougher surfaces (145). Whole body vibration (low frequency) transmission is a possible mechanism triggering spasms while wheelchair users wheel over different surfaces. Vibration transmission is higher in wheelchair users traversing specific surfaces such as bevel interlocking concrete surface and poured concrete surface (146). According to the International Standards Organization 2631-1 (ISO 2631-1), the vibration frequency range of 4-12.5 Hz is considered to put human at greatest physical risk especially on lumbar spine and connected nervous system (119,147). However, higher vibration frequencies such as frequency of 50Hz was used to manage spasticity (119). This needs to explore further since the evidence is unclear. First a systematic review is needed to understand what is known based on the available evidence.

Innovations for the able-bodied population have also evolved such as the Segway personal transporter. It was developed in 2001 and introduced as the first self-balancing, electric-powered transportation device that requires no special skill (148). Structurally Segway is a small platform supported by 2 parallel wheels 20 cm above the ground that supports by a standing rider (148). The platform includes a close loop dynamic stability control system that consists of gyroscopes and some other sensors (148). Five gyroscopes called sold-state angular rate sensors control movements and regulate stability of the Segway (148). Forward and backward movements are detected by three of these gyroscopes and the other two gyroscopes are responsible for stability and reliability of the Segway (148). The rider’s balance and upright posture is adjusted by minute adjustments of gyroscopes (148). When the rider leans forward, the Segway moves forward and when the rider leans backward, the Segway moves backward or stops (148). Segway velocity is determined by the angle of either forward or backward leans with higher speeds as a result of greater forward leans and lower speeds as a result of backward leans (148). The inventor of the Segway, Dean kamen, suggested that this device was not designed as a mobility aid for individuals with different types of disability. However, there are many personal accounts
posted online by a group of Segway users with different types of mobility disabilities. They indicated how fast and easily they integrated into this novel mobility-related activity (149).

Sawatzky et al. studied the correlation between functional outcome measures such as strength, range of motion, and balance with the ability to use the Segway in a wide range of disabilities, so the therapists may have a mechanism to determine who can use the Segway. This study evaluated the functional ability level that is needed to operate the Segway easily and safely (143). The results of that study revealed no correlation between functional scores and the Segway skills as all the participants operated the Segway safely and effectively. They concluded the Segway is a useful mobility device in individuals with a wide range of different level of functional abilities (143). The same researchers also compared how well the Segway met personal goals to the client’s current mobility device. They used the Wheelchair Outcome Measure (Whom) to determine the clients’ goal (150). They found a significant difference in Whom score between participants’ current mobility device and the Segway for clients specific goals. It shows that the Segway met the clients’ goal better than their own mobility device. However, there was no significant difference between the indoor obstacle courses (142).

During these studies, some of the study participants also reported a reduction in their level of spasticity. This raised the question of whether there might be some benefits for those with SCI to use the Segway to manage their spasticity (143,151). Boutilier et al. examined the immediate and long-term effects of a dynamic standing training program using the Segway on spasticity, pain and fatigue (151). The dynamic standing training included three sessions of 30 min simple tasks such as navigating around, negotiating around obstacles with increasing level of difficulty. The level of spasticity was measured by a clinical outcome measure (MAS) and a self-report questionnaire (SCI-SET). Pain and fatigue were evaluated by Pain Outcome Questionnaire (POQ-VA) and Fatigue Severity Scale (FSS). The results of this study showed a short-term reduction in spasticity and long-term reduction of pain and fatigue over a month with three training sessions (151). Since the dynamic standing training with the Segway personal transporter has showed spasticity reduction in individuals with SCI and the evidence of decreasing
spasticity by using static standing training, this raised the question to know what is responsible mechanism for spasticity reduction. Was the reduction due to riding the Segway, or simply the act of standing on the Segway?

1.7 Summary

The literature review shows that individuals with SCI deal with considerable array of spasticity complications that is a challenge for patients and clinicians to manage. The pharmacologic side effects and invasive surgical procedures make the management of spasticity difficult and magnify the role of physical therapy techniques as a management tool. Among physical therapy techniques, vibration therapy including WBV and FV is a simple technique that can be applied by simple equipment without any specific training. Although this technique is on the list of spasticity management physical therapy techniques, there is no specific guideline with defined beneficial vibration frequencies for spasticity management. Supported standing is another physical therapy technique that might be useful to manage spasticity. The Segway PT transporter is an alternative mobility device that allows the individuals to ride it in a standing position. The dynamic standing on the Segway showed spasticity reduction. It is unknown how the Segway results in spasticity reduction in individuals with spasticity. There might be some factors other than standing that would result in spasticity reduction after the Segway dynamic standing training.

1.8 Purpose

The primary purpose of this thesis was to systematically review the effects of vibration physical therapy technique on spasticity in individuals with SCI to provide a better understanding of beneficial vibration frequencies either by WBV or FV application. The Secondary purpose of this study was to examine how spasticity management is different between dynamic standing training on the Segway and the static standing training.
1.9 **Research Objectives**

This research has two main objectives:

1. To systematically review and evaluate the existing evidence that has examined the effects of either WBV or FV on spasticity in individuals with SCI (Chapter 2).

2. Examine the difference of two (static and dynamic) standing training effects on spasticity in individuals with SCI and to evaluate how these interventions affect spasticity and muscle activity using self-assessment (VAS), clinical (MAS), and electrophysiologic (EMG) outcome measures (Chapter 3).
Chapter 2: Vibration Scoping Systematic Review

The objective of this systematic review was to evaluate how whole body vibration (WBV) or focal vibration (FV) would change spasticity in individuals with spinal cord injury (SCI). A search was conducted of MEDLINE, EMBASE, CINAHL, and PsycINFO electronic databases. A hand search was conducted of the bibliographies of relevant articles and journals to the research question. The inclusion criteria were ≥ 3 individuals, ≥ 17 years old, with SCI who experience spasticity, WBV or FV application. The evidence level of all 10 identified studies (195 SCI subjects) was low based on Centre for Evidence Based Medicine level of evidence. The WBV (n=1) and FV (n=9) were applied to assess the effects of vibration on different measures of spasticity in individuals with SCI. FV application resulted in a short-term spasticity reduction lasting for a maximum of 24 hrs. Neurophysiologic measures showed H-reflex inhibition in individuals with SCI after FV application. WBV resulted in a decrease of spasticity lasting for 6-8 days after the last vibration session. WBV and FV might decrease spasticity for a short period of time but no evidence-based recommendation can be drawn from the literature to guide rehabilitation medicine clinicians to manage spasticity with vibration application.

Key Words: Muscle Spasticity, Vibration, Spinal Cord Injury, Spasm

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1 The electronic version of this paper was published in American Journal of Physical Medicine and Rehabilitation on April 16th ahead of print (PMID: 24743464). Sadeghi M, Sawatzky JB. The Effects of Vibration on Spasticity in Individuals with Spinal Cord Injury: A scoping Systematic Review. The acceptance letter of this systematic review is available in Appendix A.
2.1 Introduction

Spasticity, defined by Lance as “a velocity-dependent increase in muscle tone with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex” (10). Although Lance’s definition has been accepted widely, there is discrepancy within the literature for the precise definition of spasticity (2,13–16). While spasticity is described specifically as an increased muscle tone, there are some other symptoms such as clonus, hyperactive tendon reflexes, and spasms that are included within the original definition by Lance (2,10,14–16). Spasticity is a result of hyperexcitable stretch reflex in spinal cord but spasms are involuntary muscle contractions due to hyperexcitable or disinhibited spinal reflexes such as flexor withdrawal reflexes (13).

Individuals with spinal cord injury (SCI) experience significant challenges with spasticity during their daily routine (2). It is estimated that 67-78% of the individuals with SCI experience spasticity at rehabilitation hospital discharge and follow-up (2). Spasticity can degrade quality of life by causing pain and fatigue, contributing to the development of contractures, pressure ulcers, infection, negative self-image and may interfere with a wheelchair user’s seating, transfers and wheeling (2,3).

Spasticity management in individuals with SCI involves a wide range of approaches including antispastic pharmaceuticals given orally, through injections, or intrathecally. Medications produce a non-targeted release of pharmacological agents which result in a general suppression of neuronal activity in population who already suffer from reduced voluntary drive (3). Additionally, these medications have unwanted pharmacologic-induced side effects. Potential side effects vary between medications but may include muscle weakness, sedation, drowsiness, dizziness, ataxia, hallucination, depression, hypotension, liver toxicity, and possible addiction (3). Botulinum toxin (Botox A) is an injectable neurotoxin that blocks the acetylcholine transfer across the neuromuscular junction causing a weakness in that muscle. However due to the eventual sprouting of new nervelets, the effect of Botox is not permanent (3,152,153). A surgical technique known as a rhizotomy is an invasive approach with surgical risks along with
significant weakness as a result of the muscle nerves being cut and usually reserves for spasticity complications such as contracture (154).

The least invasive method involves physical therapy techniques and is often considered adjuvant essentials in the management of spasticity as they are often used to complement the pharmacological and surgical strategies (107,155). The techniques include a wide range of treatments such as positioning, muscle strengthening, stretching, weight-bearing techniques, electrical stimulation, vibration, cold/heat application, splinting and orthoses (3,156). The physical therapy techniques are intended to maintain the muscle length, prevent contractures, and change mechanical properties of musculoskeletal system, and plastic changes within the central nervous system (3). Both whole body vibration (WBV) and focal vibration (FV) have been used for spasticity management in individuals with SCI (70,110–112).

In normal spinal pathways, prolonged muscle tendon vibration (30 sec-15 min) results in decreased resting discharge rate of the sensory receptors (muscle spindles) and decreased short-latency of stretch reflex of lower extremity muscles (157–159). These mechanisms are mainly cause lower excitatory sensory inputs by Ia afferent neurons to the spinal cord and consequently lower motor units activity during muscle contraction (157–159). Although it is unknown how vibration either WBV or FV would change muscle spasticity, lower Ia afferent neuron discharge might be the decisive responsible mechanism.

While there is some literature to support the use of either WBV or FV for spasticity management in individuals with SCI, it has still not fully understood, nor adopted into clinical practice guidelines for spasticity management in SCI. For example, the appropriate frequency range, magnitude, or durations are largely unknown. In addition to the variables described above, understanding the difference between WBV and FV is important since typically WBV devices provide a significantly greater power since the motors are larger and the amplitudes are greater than through handheld devices that are smaller. There are considerably more variances in how handheld FV than WBV is administered. With FV, it can vary on the muscle depending upon location relative to musculotendinous junction, as well as force applied in holding
it on a specific limb. For WBV, typically one can stand (knees typically flexed or sit in a chair or wheelchair). These are all issues that need to be addressed in studying vibration effects on spasticity.

Theoretically, there may be a vibration frequency that might play an important role in for therapeutic goals. However, there may also be a danger to exposing individuals to some vibration frequencies. Based on International Standards Organization (ISO) 2631-1 the vibration range of frequencies between 4-12.5 Hz is considered to put human at greatest physical risk mainly on lumbar spine and connected nervous system (147). Before guidelines can be established or suggest more research in this area, we need a systematic review of what is the current knowledge on the use of vibration (WBV or FV) and how it should be used in the SCI population.

For this systematic review our primary question was what was the effect on spasticity and spasms after WBV or FV application in those with SCI? However, to put the results into context, we also needed to know what was the frequency range of WBV or FV used in this population, how was the vibration applied to individuals with SCI and what were the measurement tools used to evaluate spasticity and spasms?
2.2 Methods

2.2.1 Search Strategy

The Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) checklist and guideline was used to develop this review (160). However, the meta-analysis checklist items were not applicable. The following electronic databases were searched; MEDLINE, EMBASE, CINAHL, and PsycINFO since 1946, 1974, 1977, and 1887 until Jan 3\textsuperscript{rd} 2013 respectively. No data restrictions were applied when the databases were searched. Peer-reviewed articles were identified using the keywords vibration, whole body vibration, spasticity, spasm, and spinal cord injury. Search terms were adjusted for each database (Appendix B). Additional adjusted search terms were spastic paraplegia and spastic paresis. The bibliographies of relevant studies and review articles were searched. A hand search of potential journals was undertaken for articles relevant to the research question regarding to research key words. The corresponding authors of five of the papers included in the study were contacted by email to identify any additional studies related to the study purpose. Email address of an additional five corresponding authors were not available. The title and abstracts of all identified papers were reviewed by both authors. Articles not related to the objectives were excluded. The studies reporting the spastic muscles changes in individuals with SCI after either WBV or FV were reviewed in full papers. Assessing the Risk of Bias of Individual Studies in Systematic Reviews of Health Care Interventions used to assess the risk of bias for each of the reviewed papers (Table 2.1) (161).
Table 2.1 Risk of Bias Assessment

<table>
<thead>
<tr>
<th>Author</th>
<th>Selection bias/Confounding</th>
<th>Performance bias</th>
<th>Attrition bias</th>
<th>Detection bias</th>
<th>Reporting bias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alaca et al. (111)</td>
<td>Low risk</td>
<td>Low risk</td>
<td>n/a</td>
<td>Unclear</td>
<td>Low risk</td>
</tr>
<tr>
<td>Ashby et al. (113)</td>
<td>Medium risk</td>
<td>Low risk</td>
<td>n/a</td>
<td>Low risk</td>
<td>Low risk</td>
</tr>
<tr>
<td>Butler et al. (116)</td>
<td>Low risk</td>
<td>Low risk</td>
<td>n/a</td>
<td>Low risk</td>
<td>Low risk</td>
</tr>
<tr>
<td>Calancie et al. (117)</td>
<td>Medium risk</td>
<td>Low risk</td>
<td>n/a</td>
<td>Low risk</td>
<td>Low risk</td>
</tr>
<tr>
<td>Laessoe et al. (119)</td>
<td>Medium risk</td>
<td>Low risk</td>
<td>Low risk</td>
<td>Unclear</td>
<td>Low risk</td>
</tr>
<tr>
<td>Murillo et al. (112)</td>
<td>Low risk</td>
<td>Low risk</td>
<td>n/a</td>
<td>Medium risk</td>
<td>Low risk</td>
</tr>
<tr>
<td>Ness et al. (110)</td>
<td>Low risk</td>
<td>Low risk</td>
<td>Low risk</td>
<td>Low risk</td>
<td>Low risk</td>
</tr>
<tr>
<td>Perez et al. (115)</td>
<td>Medium risk</td>
<td>Low risk</td>
<td>n/a</td>
<td>Low risk</td>
<td>Low risk</td>
</tr>
<tr>
<td>Taylor et al. (114)</td>
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<td>Low risk</td>
<td>Unclear</td>
<td>Low risk</td>
<td>Low risk</td>
</tr>
<tr>
<td>Hilgevoord et al. (118)</td>
<td>Medium risk</td>
<td>Low risk</td>
<td>n/a</td>
<td>Low risk</td>
<td>Low risk</td>
</tr>
</tbody>
</table>

Abbreviations: n/a, not applicable.

2.2.2 Inclusion/Exclusion Criteria

To be included in the review, the paper must have included ≥3 participants, ≥17 years old participants, with chronic SCI who had spasticity for at least 4 months after their injury (stable spasticity). Due to the diverse nature of definition for spasticity used in clinical and rehabilitation settings, the following spasticity definition were included: velocity dependent hypertonia, significant resistance to passive movement, involuntary EMG muscles activity, Modified Ashworth Scale (MAS) ≥1, spasm frequency scale grade 1, and abnormal H-reflex activity. Studies with and without spasticity medication management were included in the review. Studies that specifically applied either WBV or FV were included. The studies that did not report a specific frequency of vibration were excluded from the review.
2.2.3 Data Extraction

All information related to the research question including the methods of applying vibration, spasticity outcome measures, and study results, were extracted from each of the reviewed papers. Authors extracted the data from each study together. The extracted data comprised of study type, level of evidence, number of participants, age, level of injury, American Spinal Injury Association (ASIA) Impairment Scale (AIS) score, complete/incomplete, tetraplegia/paraplegia, medication, vibration type and frequency.

2.2.4 Level of Evidence (LoE)

The Centre for Evidence Based Medicine (CEBM) level of evidence was used to determine the level of evidence for each of the reviewed papers. The level of evidence was determined based on the type of study including systematic reviews (SR), randomized control trials (RCT), cohort studies, case-controls, and case-series. The CEBM categorizes the level of evidence as 1 (a to c), 2 (a to c), 3 (a and b), 4, and 5 based on the type of studies described above (162). The studies are then categorized from grade A to D based on their level of evidence. Grade A is consistent with level 1 (a to c) studies and Grade B is consistent with either level 2 (a to c) or 3 (a and b) studies. Grade C and D studies are consistent with level 4 and 5 studies respectively (Appendix C). Each study was graded by two authors using CEBM levels (1, 2, 3, and 4) and grades (A, B, C, and D). Disagreements over the evidence level and grade of each study were resolved through discussion between two authors.

2.3 Results

In total, 109 papers were found by the primary electronic and hand research. The search retrieved a total of 64 articles after duplications were removed. After reviewing the titles and abstracts, 40 articles were excluded with 24 full text papers were assessed by the authors to assess for eligibility based on the inclusion criteria. After review of the 24 full text papers, 10 met the inclusion criteria while 14 papers were excluded. The hand search uncovered one study (118) while electronic search identified the remaining nine studies (70,110–116,118). The study selection process is described in Figure 2.1.
Figure 2.1 Flowchart of Study Selection

Records identified through electronic databases (n=105); MEDLINE (n=48), CINAHL (n=11), EMBASE (n=39), PsychINFO (n=7)

Number of articles after duplications removed (n=64)

Study titles (n=64)

Title excluded (n=15): Case studies, not related to objectives (Worked on blood pressure, pharmacological agents, genito-sexual dysfunction)

Abstract reviewed (n=49)

Abstract excluded (n=25): case studies, congress presentation, not related to objectives (Genito-sexual dysfunction, brain control)

Articles reviewed (n=24)

Articles excluded (n=14): population, stimulation other than vibration, without defined vibration frequency

Articles included in the review (n=10)
2.3.1 Population

Collectively, the studies involved 195 individuals with chronic SCI. Six studies included an able-bodied control population totaling 87 participants (114-116,117,119,120). Two studies included all three groups: acute SCI, chronic SCI, and able-bodied population (115,119). However, the results of the individuals with acute SCI were not reported in this review due to lack of spasticity in this group. Six studies reported the AIS of the SCI participants including A, B, C, and D levels (70,110–112,115,116). The level of injury (ranging from C2 to L1) and being complete or incomplete SCI were reported in seven (72,112–115,118,119) and six (72,112,113,115,116,119) studies respectively. Six studies reported the spasticity medication taken by the participants (70,110,112,116–118). In one study the spasticity medication were washed out 12 hours before the study (112). Participants were not taking any medication in one study (118) and in four studies the participants maintained their previous anti-spasticity medication regime during the study (70,110,116,117). The characteristics of the studies are described in Table 2.2.
Table 2.2 Study Characteristics

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>N</th>
<th>N (control)</th>
<th>Age (mean±SD), Range</th>
<th>Level of injury</th>
<th>AIS</th>
<th>Medication</th>
<th>Type of study</th>
<th>LoE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alaca et al.</td>
<td>2005</td>
<td>10</td>
<td>n/a</td>
<td>27.8±3.7</td>
<td>C8-T12</td>
<td>A</td>
<td>n/a</td>
<td>Case series</td>
<td>4</td>
</tr>
<tr>
<td>Ashby et al.</td>
<td>1974</td>
<td>7</td>
<td>6</td>
<td>21-57</td>
<td>C4-T7</td>
<td>n/a</td>
<td>n/a</td>
<td>Case-control</td>
<td>3b</td>
</tr>
<tr>
<td>Butler et al.</td>
<td>2006</td>
<td>8</td>
<td>n/a</td>
<td>37±3</td>
<td>C4-T6</td>
<td>A, B</td>
<td>2 baclofen</td>
<td>Case series</td>
<td>4</td>
</tr>
<tr>
<td>Calancie et al.</td>
<td>1993</td>
<td>22</td>
<td>16</td>
<td>26.2 ±2.3</td>
<td>C4-T10</td>
<td>n/a</td>
<td>Baclofen/diazepam</td>
<td>Case-control</td>
<td>3b</td>
</tr>
<tr>
<td>Laessoe et al.</td>
<td>2004</td>
<td>9</td>
<td>n/a</td>
<td>27-67</td>
<td>C2-T8</td>
<td>A, C,D</td>
<td>Yes (n=8)</td>
<td>Cross-over</td>
<td>2b</td>
</tr>
<tr>
<td>Murillo et al.</td>
<td>2011</td>
<td>19</td>
<td>9</td>
<td>36.0 ±10.6</td>
<td>C3-T12</td>
<td>C, D</td>
<td>Yes (n=7)</td>
<td>Case-control</td>
<td>3b</td>
</tr>
<tr>
<td>Ness et al.</td>
<td>2009</td>
<td>16</td>
<td>n/a</td>
<td>28-65</td>
<td>C3-T7</td>
<td>C, D</td>
<td>Yes (n= 7)</td>
<td>Prospective experimental study</td>
<td>2c</td>
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<tr>
<td>Perez et al.</td>
<td>2004</td>
<td>14</td>
<td>5</td>
<td>42.8±10.2</td>
<td>n/a</td>
<td>C, D</td>
<td>n/a</td>
<td>Case- control</td>
<td>3b</td>
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<tr>
<td>Taylor et al.</td>
<td>1984</td>
<td>57</td>
<td>21</td>
<td>17-77</td>
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<td>n/a</td>
<td>Case-control</td>
<td>3b</td>
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<tr>
<td>Hilgevoord et al.</td>
<td>1996</td>
<td>33</td>
<td>30</td>
<td>18-68</td>
<td>n/a</td>
<td>n/a</td>
<td>No medication</td>
<td>Case-control</td>
<td>3b</td>
</tr>
</tbody>
</table>

Abbreviations: n, number; AIS, American Spinal Injury Association (ASIA) Impairment Score; LoE, Level of evidence; n/a, not applicable.
2.3.2 Effects on Spasticity

*Whole Body Vibration*

Only one study (Table 2.3) measured spasticity after using WBV in incomplete SCI (110). The pendulum test was used to measure quadriceps muscle spasticity after WBV (3/wk x 4 weeks). The first swing excursion and number of oscillation were the two components of the pendulum test used to evaluate spasticity. The study reported a significant decrease in spasticity in all but one week of WBV training. There was a statistically significant (p=0.005) increase in first swing excursion from initial to final test with no significant change in oscillation number (p=0.195) in the one-month period. The similarities between the first swing excursion values measured 1-4 days and 6-8 days after the last vibration sessions suggested that the effects of WBV intervention persisted for at least 6-8 days (110).
<table>
<thead>
<tr>
<th>Author</th>
<th>Intervention</th>
<th>Testing</th>
<th>Outcome measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ness et al.[110]</td>
<td>3-day/week for 4 weeks Each session: 4* 45-sec bouts/ 1 min seated rest, standing on platform with the knees slightly flexed (30 degree from full extension)</td>
<td>Within 5 min after vibration (immediate), Approximately 15 min later (delayed post-WBV), last-testing: 8 days after last vibration session</td>
<td>Pendulum test; FSE&lt;sup&gt;a&lt;/sup&gt;, OSC&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1. Initial to last testing: increase in FSE (p=0.005); No significant change in OSC.  2. Significant weekly with-in session immediate and delayed post-WBV FSE changes for week 1, 2, and 4.  3.Final test; 13 participants tested within 1-4 days and 3 participants tested 6-8 days (mean changes 12.05±4.04 and 11.47±18.31 respectively)</td>
</tr>
</tbody>
</table>

<sup>a</sup>FSE, First swing excursion (angle at which the swinging leg first reversed direction from flexion to extension indicating the point in the knee range of motion at which a reflex contraction of the quadriceps caused the knee to extend. An increase in FSE was interpreted as a decrease in spasticity);  
<sup>b</sup>OSC, number of oscillation
Focal Vibration

Nine articles (Table 2.4) reported the effects of FV on clinical and neurophysiologic spasticity measures. Three studies reported clinical spasticity reductions before and after applying FV (70,111,112). Two studies applied FV by penile vibration stimulation (PVS) and found a short term decrease in spasticity for a maximum of 3-6 hours after applying vibration (70,111). Murillo et al. found a significant decrease in spasticity after applying FV to the rectus femoris muscle (112).

The other six papers, the main outcome was spasticity neurophysiologic measure changes after FV application. Butler et al. (116) displayed inconsistent EMG muscle activity and spasticity changes after applying FV. Although most of the EMG recordings after FV application showed a reduction in muscle activation, some of the trials showed increased muscle activity. Exposure to FV decreased EMG activity in seven participants, but evoked spasm in one of the participants (116). Five of the studies that reported the spasticity neurophysiologic measure changes had able-bodied control group. Changes in H-reflex in individuals with SCI who experienced spasticity, measured as H-reflex vibration/H-reflex control demonstrated less reduction compared to control groups in four of the studies included in the review (113,114,117,118). Ashby et al. study revealed suppression in T-reflex after vibration with an increase in tension of the force applied by tendon hammer in two SCI participants (113). The Achilles tendon reflex (ATR) suppression was greater in that study compared to H-reflex suppression (113). Perez et al. reported a significant increase in strength of Ia inhibition for 5 minutes after vibration in the spastic population with no significant change in presynaptic inhibition measured by H-reflex (115).
<table>
<thead>
<tr>
<th>Author</th>
<th>Intervention</th>
<th>Testing</th>
<th>Outcome measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alaca et al. (111)</td>
<td>3 min PVS followed by 1 min rest intervals until ejaculation or maximum of six sessions</td>
<td>Baseline, 3, 6, 24, and 48 hrs after PVS</td>
<td>MAS, frequency spasm severity, painful spasms, plantar stimulation response, DTR, clonus, effect on function</td>
<td>MAS significantly decreased in 3 hr (p = 0.001) and 6 hr (p = 0.03) after PVS. Lower MAS after 24 and 48 hrs but not significant. No significant difference for other measurements.</td>
</tr>
<tr>
<td>Ashby et al. (113,a)</td>
<td>Lay prone with fixed angle at 90 degree. Vibration applied to Achilles tendon to evoke TVR. EMG recorded from SOL</td>
<td>Baseline, after applying vibration</td>
<td>H-reflex, T-reflex, TVR</td>
<td>1. H-reflex: vibration less effective to suppress H-reflex in established spasticity group compared to control and acute SCI group. 2. TVR: absent in established SCI group. 3. ATR: suppression was greater than H-reflex suppression.</td>
</tr>
</tbody>
</table>
### Table 2.4. Focal Vibration Study Characteristics

<table>
<thead>
<tr>
<th>Study</th>
<th>Intervention Details</th>
<th>Condition Types</th>
<th>EMG Measurements</th>
<th>Results</th>
</tr>
</thead>
</table>
| Bulter et al. (116)    | Stimulate SPN nerve to evoke muscle spasm (5 pulses of 300Hz), 30 sec for PAD recovery | 1. Conditioned (with vibration)  
2. Unconditioned | EMG: 10 pairs (20 trials) from TA, MG, LG, SOL muscles  
H-reflex<sup>b</sup>: 10 pairs (20 trials) | 1. EMG: 66% of trials decreased, 22% abolished, 28% increased, 6% no response, group mean peak EMG decreased between 36-45%, 7 participants with decreased EMG and 1 participant had spasm after vibration.  
2. H-reflex: amplitude not significantly reduced after vibration except in 2 participants. |
| Calancie et al. (117)<sup>a</sup> | Semi-reclined position, vibration applied to Achilles tendon, EMG recorded from SOL | Baseline, after applying vibration | H-reflex, T-reflex                                                                                   | 1. Hvib/Hnvib ratio: significantly higher than control (P<0.05) and acute SCI (P< 0.01); showed less inhibition.  
2. T-reflex: changes after vibration were not reported in the paper. |
### Table 2.4. Focal Vibration Study Characteristics

<table>
<thead>
<tr>
<th>Study</th>
<th>Intervention</th>
<th>Measurements</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laessoe et al.(^{(70)})</td>
<td>24 hr of EMG from QF and TA bilaterally, followed by either PVS or no treatment subsequently and 24 hr EMG recording. After at least 1 week repeated protocol.</td>
<td>EMG recording 24 hr before, 24 hr after PVS (48 hr in total). MAS and PSFS 24 hrs before, after PVS and 24 hrs later</td>
<td>EMG, MAS, PSFS</td>
</tr>
<tr>
<td>Murillo et al.(^{(112)})</td>
<td>Vibration applied to RF</td>
<td>1. Baseline 2. After vibration stimulation</td>
<td>MAS, ROM measured with Modified Tardieu scale, Clonus, H-reflex, T-reflex</td>
</tr>
<tr>
<td>Study</td>
<td>Position and Setup</td>
<td>Baseline Measurement (before), immediately after (time 0), and at 5, 10, 15, and 20 min</td>
<td>H-reflex: Ia inhibition $^a$, Presynaptic inhibition $^e$</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Perez et al. $^{(115)}$</td>
<td>Rest sitting position, TA tendon vibrated for 3 min. Vibrator pressed onto the tendon 5 cm above the ankle</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 2.4. Focal Vibration Study Characteristics

<table>
<thead>
<tr>
<th>Hillgevoord et al. (^{(118)})</th>
<th>Semi-recline chair. EMG recorded from SOL muscle</th>
<th>Measurement with and without vibration</th>
<th>H-reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>1. Max H-response vibratory suppression: significantly less in SCI group ((P&lt;0.001)). 2. Maximal H-response: evoked at higher stimulus intensity than without vibration. 3. Average level to which vibration inhibited the H-reflex: 20% for controls, 50% for SCI group.</td>
</tr>
</tbody>
</table>

Abbreviations: PVS, Penile vibration stimulation; MAS, Modified Ashworth Scale; DTR, Deep tendon reflex; TVR, Tonic vibration reflex; EMG, Electromyography; SOL, Soleus; ATR, Achilles tendon reflex; SPN, Superficial proneal nerve; PAD, Post activation decrease; TA, Tibialis anterior; MG, Medial gastrocnemius; LG, Lateral gastrocnemius; QF, Quadriceps femoris; PSFS, Penn spasm frequency scale; RF, Rectus femoris; SCI, Spinal cord injury.

\(^{a}\) Data for control group and acute SCI is not reported [results from chronic SCI group after vibration is reported]; \(^{b}\) H-reflex (H and M wave onset latency and peak to peak amplitude, Hmax, Mmax); \(^{c}\) results for control group and comparing complete and incomplete groups are not reported; \(^{d}\) 2 ms to 3 ms to evoke Ia inhibition; \(^{e}\) 10 ms and 15 ms to evoke presynaptic inhibition.
2.3.3 Vibration Frequency

The type and frequencies of vibration varied considerably between studies as well as the application point to the person. The vibration was applied as WBV (110) or FV (70,111–118) with different frequencies however each study used only one specific frequency. The frequency range was 50-110 Hz with amplitude range of 1-4 mm. FV was applied with a frequency of 50 Hz (112), 60 Hz (113–115), 80 Hz (116), 100 Hz (70,111,118), 110 Hz (117). The WBV frequency was 50 Hz (110). The FV was applied to different points on the body including the Achilles tendon (113,114,116–118), the penis (111,70), the tibialis anterior tendon (115) and the rectus femoris muscle (112). The testing was conducted in sitting, semi-reclined or lying supine positions while the vibration was applied. Vibration characteristics are described in Table 2.5.

2.3.4 Spasticity Measurement

Spasticity was measured by different outcome measurements in the included studies. One study used the Penn Spasm Frequency Scale self-assessment (70). The clinical measurements used in different studies including MAS (70,111,112), MTS (112), pendulum test (110), spasm frequency (111), spasm severity (111), painful spasm (111), plantar stimulus response (111), deep tendon reflex (111), clonus (111,112), and effects on function (18). The neurophysiologic measurements were most common and these included EMG (70,116), H-reflex (112–118), and T-reflex. Three studies (110,115,118) measured spasticity with only one outcome measure and the other seven studies (70,111–114,116,117) used more than one outcome measure to report the changes after applying vibration. Two studies used the clinical and neurophysiologic measurements at the same time to assess the effects of vibration on spasticity (70,112).
Table 2.5 Vibration Characteristics

<table>
<thead>
<tr>
<th>Author</th>
<th>WBV/FV</th>
<th>Frequency/Amplitude</th>
<th>FV application</th>
<th>Position</th>
<th>Muscles testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alaca et al. (111)</td>
<td>FV</td>
<td>100 Hz, 2.5 mm</td>
<td>PVS</td>
<td>Supine</td>
<td>Knee&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Ashby et al. (113)</td>
<td>FV</td>
<td>60 Hz, 3 mm</td>
<td>Achilles tendon</td>
<td>Lying prone</td>
<td>SOL</td>
</tr>
<tr>
<td>Butler et al. (116)</td>
<td>FV</td>
<td>80 Hz</td>
<td>Achilles tendon</td>
<td>Sitting</td>
<td>SOL, MG, LG, TA</td>
</tr>
<tr>
<td>Calancie et al. (117)</td>
<td>FV</td>
<td>110 Hz, 2.2 mm</td>
<td>Achilles tendon</td>
<td>Semi-reclined</td>
<td>SOL</td>
</tr>
<tr>
<td>Laessoe et al. (70)</td>
<td>FV</td>
<td>100 Hz, 3 mm</td>
<td>PVS</td>
<td>n/a</td>
<td>QF, TA</td>
</tr>
<tr>
<td>Murillo et al. (112)</td>
<td>FV</td>
<td>50 Hz</td>
<td>RF</td>
<td>Sitting</td>
<td>SOL, knee</td>
</tr>
<tr>
<td>Ness et al. (110)</td>
<td>WBV</td>
<td>50 Hz, 2-4 mm</td>
<td>n/a</td>
<td>Stood with knee flexed</td>
<td>QF</td>
</tr>
<tr>
<td>Perez et al. (115)</td>
<td>FV</td>
<td>60Hz</td>
<td>TA tendon</td>
<td>Sitting</td>
<td>SOL</td>
</tr>
<tr>
<td>Taylor et al. (114)</td>
<td>FV</td>
<td>60Hz, 1.5mm</td>
<td>Achilles tendon</td>
<td>Lay prone</td>
<td>SOL</td>
</tr>
<tr>
<td>Hilgevoord et al. (118)</td>
<td>FV</td>
<td>1mm/100Hz</td>
<td>Achilles tendon</td>
<td>Semi-recline</td>
<td>SOL</td>
</tr>
</tbody>
</table>

Abbreviations: WBV, Whole body vibration; FV, Focal vibration; PVS, Penile vibratory stimulation; SOL, Soleus; MG, Medial gastrocnemius; LG, Lateral gastrocnemius; TA, Tibialis anterior; QF, Quadriceps femoris; RF, Rectus femoris; n/a, not applicable. <sup>a</sup>Knee: Both flexion and extension.
2.3.5 Level of Evidence

The level of evidence of studies included in the review differed from 2b to 4 based on CEBM. Six studies were case-control studies with the level of evidence of 3b (112–115,117,118). Two studies were case series with the level of evidence of 4 (111,116). The other two studies were cross-over and a prospective experimental study with the level of evidence of 2b (70) and 2c (110) respectively. Based on CEBM grades of recommendation, eight of the reviewed studies were categorized as grade B and the other two reviewed studies were categorized as grade C.

2.4 Discussion

The main purpose of this review was to explore what is the current state of knowledge regarding the effects of WBV and FV on spasticity in individuals with SCI. Although there may be some encouraging results linking WBV and FV to improved spasticity from the existing evidence, this evidence is still relatively, weak due to limited number of studies and lack of high quality clinical studies.

2.4.1 Vibration Frequency

This review showed the significant breadth of vibration frequencies and duration were applied to individuals with SCI. Frequencies ranged from 50 to 100 Hz, from 30 to 60 sec. Although what seems a relatively wide range, the studies all showed some reduction of spasticity in individuals with SCI. Since vibration frequency range and duration might be factors that play a role in either FV or WBV application to manage spasticity, vibration research will need to undergo various stages of development, focusing on factors such as frequency level and frequency duration or dose. Similar to drug trials, dose response trials are essential to isolate what is effective and what may be ineffective or possibly spasticity inducing.

2.4.2 Whole Body Vibration

The 50 Hz frequency used in WBV exposures demonstrated a decrease in spasticity through a 1-month training in individuals with SCI, lasting 6-8 days after the last training session (110). These results were consistent with the WBV application studies for able-bodied as well as individuals with CP and stroke (163–165). Ahlborg et al. evaluated the effects of 8 week (24 sessions) WBV training on spasticity,
muscle strength, and motor performance in adults with CP and they found a reduction in knee extensors spasticity without any significant changes in other muscle groups after the 8 week training (165). Chan et al. applied a single session of WBV with a magnitude of 12 Hz and 4 mm amplitude in stroke patients with spasticity (164). This randomized control trial showed a significant decrease in spasticity measured by MAS, self-report visual analog scale and the H-reflex measurement (Hmax/Mmax ratio) (164). Pang et al. completed a randomized control trial study with WBV stimulation (20-30 Hz) and exercise training during an eight-week period (15 min x 3 days/ week) and showed a significant reduction of spasticity measured by MAS in a stroke population. In their study, the spasticity reduction showed a decreasing trend during the eight-week training as well as a significant reduction after one month in the group who got vibration and exercise compared to the control group who just performed the exercise (163). Although the mechanism of spasticity might be different among different upper motor neuron disorders, the results of studies on CP and stroke participants offer a change in spasticity by WBV application.

Despite the evidence, although limited, in using vibration to help manage spasticity in individuals with SCI and other upper motor neuron disorders, there is still concern about the appropriate frequency to use. The International Standardization Organization 2631-1 (ISO-2631-1) shows that WBV exposures of 4-12.5 Hz may be subjecting individuals to health hazards (147). In this frequency range, it is not clear if vibration either WBV or FV would trigger spasticity or spasm in individuals with SCI.

All the above WBV studies, except one, used frequencies higher than the ISO-2631-1 WBV health hazard range (147,164–166). Messenberg et al. created a vibration platform that could vibrate individuals with SCI in a sitting position at frequency ranges from 8 to 100Hz. With their sample of two participants they found they could provide sufficient vibrations to induce spasticity (145). Some frequencies resulted in a decrease in muscle activities and spasticity while others exacerbated spasticity. However, this was only a pilot study to show a proposed methodology (145). More work with a larger sample size is needed to understand range of frequencies and their impacts. This methodology may be a
good way to apply various frequencies to individuals with SCI and examine the muscle responses in a systematic fashion.

2.4.3 Focal Vibration

Nine studies used the FV with a frequency range of 50-110 Hz to evaluate the effects of focal vibration on spasticity (70,111–118). Two studies which used PVS, found a short-term decrease in spasticity, using EMG and MAS, lasting for up to 6 hours (70,111). Laessos et al. described two mechanisms including release of a humoral factor with a general muscle relaxant effect and the activation of pudendal afferent nerve generated by PVS procedure (72). Pudendal afferent nerve activation might result in changes of inhibitory spinal pathways including increased presynaptic inhibition, increased reciprocal inhibition, and decreased Ib interneuron excitation (70). These changes in spinal cord inhibitory pathways result in spasticity reduction after PVS application (70). Halstead et al. also proposed two mechanisms for the antispastic effect of rectal-probe electroejaculation. The periprostatic area nerve supply is rich and the stimulation is close to spinal cord. The stimulation provoke a humoral agent release (166). These mechanisms are proposed for PVS and rectal-probe electroejaculation which cannot be generalized as the FV mechanism for spasticity and spasms reduction. However, Sonksen et al. used PVS to assist ejaculation in individuals with SCI which resulted in abdominal muscle contraction and leg spasms (167). Inducing abdominal muscle contraction and leg spasms by PVS application (167) support the probability of spasms triggering by FV application in individuals with SCI who experience spasticity thus consistent with the other studies that showed spasticity and spasms reduction after PVS.

FV application has been used to manage spasticity in other upper motor neuron disorders such as stroke as well as SCI. Caliandro et al. applied FV (100Hz, 0.2-0.5 mm) to muscles of the upper extremities over 3 consecutive days to assess the effects of vibration on stroke patients with spasticity (168). Spasticity outcome measures by MAS and visual analog scale showed no difference through 3 different measurements immediately, one week and one month. The Wolf Motor Function Test score showed a significant difference of spasticity over time (168). Noma et al. applied focal vibration (91 Hz,
1mm) to the upper limbs of stroke patients to assess the effects of focal vibration on spasticity. F-wave and MAS measurements found the application of direct vibration significantly decreased spasticity for at least 30 minutes (169).

Focal vibration was seen to be less effective to suppress H-reflex in individuals with established spasticity compared to those in the able-bodied population (113,114,117,118). Perez et al. studied the effects of mechanical stimulation by vibration (60 Hz) on reciprocal inhibition in individuals with chronic SCI (115). Reciprocal inhibition is necessary between agonist and antagonist muscle pairs to perform smooth movement alteration during motor activities (170,171). Reciprocal inhibition is modulated by a number of polysynaptic pathways including Ia inhibition and presynaptic pathways (115,172–174). Vibration induction increased the Ia inhibition (2-3 ms latency) significantly to inhibit the antagonist muscle after agonist muscle activation. The presynaptic inhibition (10-25 ms latency) did not change significantly in individuals with chronic SCI (115). The increase of Ia inhibition would play a role in muscle relaxation and spasticity reduction by vibration intervention in individuals with SCI.

2.4.4 Spasticity Measurement

Alaca et al. showed that spasticity outcome measurements other than MAS did not change significantly after PVS (111). This raises the ongoing question as to what is the best method for assessing spasticity in the SCI population. Quantifying a precise spasticity outcome measure is challenging and complex for researchers and clinicians. Although there are some correlations among spasticity outcome measures, the lack of strong correlation among self-assessment, clinical, and neurophysiologic spasticity outcome measures makes it difficult to find a valid and reliable measurement tool to reveal the degree of spasticity (38,175). The included studies in the review chose a variety of outcome measures to assess spasticity that were not consistent among the studies (70,110–118). The majority of studies only included neurophysiologic measures which reduces clinical validity of the findings (113–118). Isokinetic dynamometers measure spasticity quantitatively by measuring the resistance of a joint while it is sinusoidally oscillated at different constant angular movement (67). The amount of force that is generated
by the muscles is reported as spasticity. Although this measurement is a simple quantitative outcome measure (67), it was not used as spasticity outcome measure in any of the included paper in the review. It has been recommended in future research to use a combination of the different outcome measures including the self-assessment scales, clinical, biomechanical, and neurophysiologic to evaluate the degree of spasticity in individuals with different upper motor neuron disorders including individuals with SCI (175).

2.4.5 Quality of Papers

As mentioned earlier the quality of studies in this topic is relatively poor for clinical intervention based studies. The best evidence was two prospective studies, one being a cross over design (70,110). Sample size in these studies is small as typical in many rehabilitation studies investigating SCI. So although from a CEBM analysis, these are not great quality, they may be on par with most of SCI literature, which is often small sample size with a relatively heterogeneous population. Thus, we should not discard the results of these studies too quickly. There does seem to be some evidence that WBV and FV may have some impact on spasticity. This evidence definitely suggests larger prospective and multi-centre studies need to be performed.

2.4.6 What We Know and Future Direction of Research?

According to the results of the studies in this review, most of the individuals with SCI experienced a short-term reduction of spasticity for a short period of time when FV (50-100 Hz and 2.5-3 mm) was applied. The FV with a specific range might be helpful for individuals with SCI who experience spasticity. The impact of this research could potentially lead to individuals with SCI purchasing an appropriately adjusted vibrator with a defined vibration frequency and intensity range to use for their daily life to manage their spasticity.

WBV may have an effect on spasticity however it is still unclear what frequencies would be more beneficial. More work using a WBV platform that can apply a wide frequency range would help to demonstrate the relationship between a variety of frequencies and the impact on spasticity with greater
accuracy. Future RCTs with larger sample size, using a wide range of frequencies, repetitive WBV and FV exposure, along with valid and reliable spasticity outcome measurements would allow researchers to tease out better what are the advantages and disadvantages to vibration exposure for those with SCI who experienced spasticity.

Finally, in future research we need to address some of functional changes that may result from using either form of vibration. What are the impacts of vibration on the ability to transfer, dress or even walk in some individuals? For example, in 2009, Ness and Field-Note (176) studied the use of vibration for 4 weeks (3 times/week) and its effect on walking speed. They found that vibration increased walking speed by 0.062 m/s (p<0.001). This improvement corresponds to result from gait training protocols. Seven of the seventeen participants were on medication for spasticity although the authors did not specifically study spasticity. They suggested that consistent use of afferent input improves the motor output of the control mechanisms that are impaired after an SCI. Are motor control mechanisms responsible, too, for spasticity and muscle spasm changes in vibration application? Future research should probably not only examine muscle activity changes by either WBV or FV application but also include walking or other exercises to vibration trainings.
2.5 Conclusion

In conclusion, this unique review of how vibration, both whole body and focal affect spasticity in individuals with SCI provides some limited support for use of WBV and FV to manage spasticity in individuals with SCI. The support is limited to relatively low quality papers that indicates the need for further studies with larger sample sizes, wider frequency ranges from low to high, apply vibration with other exercise trainings and randomized control trial designs to provide stronger evidence for vibration effects on spasticity. Having a better understanding of the role of vibration in individuals with SCI for managing spasms will lead to innovations in therapy as well as changes to assistive technology that may potentially decrease the use of anti-spasmodic medications that often have significant side effects. However, this review clearly shows that the current knowledge is limited. Thus we need to encourage clinicians and researchers to perform RCT studies on vibration in order to provide clinicians and patients with a more clear direction on the use of vibration to manage spasticity for those with SCI.

2.6 Acknowledgment

This research was partially funded by Natural Sciences and Engineering Research Council of Canada (NSERC).
Chapter 3: Static and Dynamic Standing and Spasticity Study

3.1 Introduction

SCI prevalence was estimated to be 85,556 persons including 51% traumatic SCI (TSCI) and 49% non-traumatic SCI (NTSCI) in Canada in 2010 (177). SCI was associated with many costs such as total hospital costs of $61.1 million CAN dollars in Canada in 2000 and 2001. The annual medical cost of SCI was $15,000-$30,000 CAN dollars per year per patient in 2000 and 2001 (92,97,98). The estimated lifetime cost of SCI ranges from $500,000 to more than $3 million USD in the United States (99). Spasticity is one of the most common sources of disability developing after SCI.

3.1.1 Spasticity and SCI

Spasticity is a common long-term consequence of SCI that is experienced by 65-78% of individuals with SCI (2). Spasticity can result in clinical consequences such as pain, fatigue, disfigurement and disability which can interfere with activities of daily living. Spasticity also decreases the quality of life and limits independency of individuals with SCI (4,5,21,37,103,104). Spasticity is defined as a velocity dependent increased muscle tone (tonic stretch reflex) with exaggerated tendon jerks (phasic component of stretch reflex), clonus, and spasms (increased extrinsic reflexes) (10,16,17,21).

Although the pathophysiology of spasticity is not completely understood, at least a few different mechanisms are responsible for spasticity in individuals with SCI (21). Decreased inhibitory drives from descending central motor pathways mostly from cortico-reticulospinal, alteration of LMN activity due to decreased Ia interneuron inhibitory effect, decreased Golgi tendon organ inputs to Ib inhibitory interneuron, and some non-neural factors are partially responsible for pathophysiology of spasticity (19,24,26,172).

3.1.2 Spasticity Management and SCI

There is no universal and successful guideline for spasticity management in individuals with SCI (106). Spasticity is usually managed by three different modalities including medications, physical
therapy, and surgical interventions (3). Unwanted pharmacologic-induced side effects and non-targeted release of pharmacologic agents with a general suppression of nervous system of anti-spastic agents provide some limitation for anti-spastic medication usage (3). Surgical techniques are invasive and are usually reserved for complications of spasticity such as contractures and as the last spasticity treatment option (3,125). Physical therapy techniques are an essential component of spasticity management and have a beneficial role before, during and after pharmacologic and surgical strategies (3). Passive stretch, passive lengthening, weight-bearing techniques, muscle strengthening, electrical stimulation, vibration, supported standing, splints and orthoses are the most common physical therapy techniques that are used to manage spasticity (102,3).

Supported standing is a weight-bearing physical therapy technique with many proposed effects and potential benefits from early stages of rehabilitation after SCI to late stages of rehabilitation (127). Supported standing, with potential benefits on different stages of therapy after injury, would improve posture, strengthen antigravity muscles, prepare muscles for walking training, retrain head and upper limb control, prevent contracture development and reduce spasticity (127,128,130,131). Upright standing and sensory inputs to the sole of the feet during standing may increase the inhibition of stretch reflex, reduce the motor neuron excitability and subsequently reduce spasticity (129,132).

3.1.3 Segway and Spasticity

The Segway personal transporter was introduced as the first self-balancing, electric-powered transportation device that requires no special skills (148). Structurally the Segway is a small platform with 2 parallel wheels and a close loop dynamic stability control system. The stability control system consists of gyroscopes and other sensors (148). Gyroscopes are responsible for detecting forward and backward movements, and ensuring the stability and reliability of the Segway (148). When the rider leans forward, the Segway moves forward and when the rider leans backward, the Segway moves backward or stops (148). The Segway’s velocity is determined by the angle that a person leans forward and backward. Leaning forward results in a greater velocity and leaning backward results in a lower velocity (148).
Individuals with different functional level such as strength, range of motion, and balance could easily and safely drive the Segway (143).

The Segway is a mobility device that has the potential to change spasticity in individuals with SCI (151). The long-term (one-month) dynamic standing training with the Segway showed a short-term reduction in spasticity measured by individual self-report and clinical spasticity outcome measures (151). The evidence suggests that both still standing and dynamic standing could reduce spasticity. However, it is not clear if dynamic standing such as Segway training would reduce spasticity more than still standing training. Although the primary spasticity reduction mechanism might be standing on the Segway, there might be some extra responsible mechanisms for spasticity reduction by this type of training. For this study we examined the effects of two different standing training (dynamic and static) on spasticity to explore which type of training could reduce spasticity more.
3.1.4 Research Questions

Research Question 1:

A. Does muscle activity and spasticity measured by electrophysiologic (EMG) outcome measures respond differently to the dynamic standing training (Segway) versus the static standing training over time in individuals with SCI?

B. Does muscle activity and spasticity measured by self-report (VAS) and clinical (MAS) outcome measures respond differently to the dynamic standing training (Segway) versus the static standing training over time in individuals with SCI?

Research Question 2:

Does muscle activity measured by EMG change after dynamic standing training (Segway) and static standing training in AB individuals?

3.1.5 Hypothesis

Hypothesis 1:

The hypothesis of this study was that spasticity reduction as measured by self-report (VAS), clinical (MAS) and electrophysiologic (EMG) would be significantly higher over time for the dynamic standing training compared to the static standing training.

Hypothesis 2:

We also hypothesized that there is no significant reduction in muscle activity (measured by EMG) of AB individuals after either the static or dynamic standing training.
3.2 Methods

3.2.1 Subject Recruitment

A total of 20 individuals including 10 SCI and 10 AB were recruited to participate in this study through study posters (Appendix D). The study posters were posted at the International Collaboration On Repair Discoveries (ICORD, Vancouver, BC, Canada) and the G.F. Strong Rehabilitation Center (G.F. Strong Rehabilitation Center, Vancouver, BC, Canada). This was a pilot feasibility study to determine how individuals with SCI could do the training and to develop a methodology to find the best way to analyze the EMG results. To establish the sample size in our study, we based it on previous studies with similar standing training which typically included between 8-15 participants (129,133,135,151,186,178). We included the AB in the study to determine the muscle responses in that group. We recruited the convenience sample size of 20 (10 in each group) for this study.

To be considered for participation in the study, SCI and AB participants had to have the following inclusion Criteria:

A) AB participants:

- Between the ages of 18 and 65 years
- Had no chronic lower and upper neurologic disorders based on their health history (self-report)

B) SCI participants:

- Between the ages of 18 and 65 years
- Spinal cord injury more than 1 year
- Spasticity of lower extremities (MAS ≥ 1) for at least 1 month before participating in the study
- Have the ability to rise from sitting to standing with no more than moderate assistance from one person or using long leg braces
• Without the experience of autonomic dysreflexia based on their history (experience symptoms such as high blood pressure, pounding headache, profuse sweating, lightheadedness, dizziness, and confusion)
• Without clinical situations such as acute urinary tract infection and pressure ulcers

3.2.2 Ethical Approval

Ethical approval was obtained from the University of British Columbia’s Ethical Review Board before the beginning of the study. The Ethical Review Board approval ID for this research study is H12-02927. Each participant received a detailed oral explanation of study’s background, procedure, and possible side effects. The participants read and signed the consent form (Appendix E).

3.2.3 Study Design

This research was a cross-over designed efficacy study (Figure 3.1). We included SCI participants (n=10) with a SCI history of more than one year who experienced spasticity. We also included AB participants (n=10) with no chronic lower and upper neurologic disorders which was determined based on their medical history (self-report).

Cross-over Design

All participants (AB and SCI) were randomly allocated to either the static or dynamic standing training sessions. Spasticity reduction by weight-bearing techniques such as supported standing can last for a short period of time but has been reported to last into the next day (3). We chose a one-week washout period to attempt to eliminate any carry over effect from the previous training. After at least one week, each participant participated in the other standing training session.

Efficacy Study

This study was an efficacy study that was designed to evaluate the difference between dynamic and static standing training effects on spasticity. We tested the static and dynamic standing training on the Segway under ideal and controlled circumstances in a laboratory setting and included a safety harness for precaution (179).
3.2.4 Outcome Measures

Spasticity was measured through the following three different outcome measures:

Visual Analogue Scale (VAS), Modified Ashworth Scale (MAS), and Electromyography (EMG).

Spasticity was measured before, immediately after, and one hour after the interventions.

3.2.4.1 Visual Analogue Scale (VAS)

VAS is a self-report questionnaire that was used in the study to assess spasticity in four different muscle groups. VAS is a simple 10 cm line and the participants reported the spasticity level in each muscle group ranging from ‘0= no spasticity’ to ‘10= the most imaginable spasticity’ (38). Participants
reported their level of spasticity in the four different muscle groups; the quadriceps, hamstrings, adductors, and calf muscles (Appendix F).

3.2.4.2 Modified Ashworth Scale (MAS)

MAS is the most common clinical spasticity outcome measure and is a six-point scale (0, 1, +1, 2, 3, 4). The scale is reported based on the subjective clinical assessment of muscle tone. Each grade corresponds to a level of spasticity and depends on where in the passive range of motion the examiner experiences the resistance (Table 1.1). MAS was used to report the level of spasticity of the three different muscles by knee flexion (rectus femoris), knee extension (biceps femoris), and ankle dorsiflexion (medial gastrocnemius). MASSum was used to report the global clinical measured spasticity. Self-report of spasticity is usually reported globally thus MASSum may be useful to have a clinical global understanding of spasticity. In a previous study by Boutilier et al., MASSum was used to report the spasticity changes after standing training on the Segway (151). In this study, the MASSum of each time point is equal to the sum of MAS of the ankle dorsiflexion, knee flexion, and knee extension.

3.2.4.3 Electromyography (EMG)

Surface EMG is a reliable and repeatable method that is used to measure the reflective electrical activity of the muscles (46,68). EMG was used to measure the electrophysiologic activity of the rectus femoris, biceps femoris, adductors, and medial gastrocnemius muscles.

3.2.4.4 Ankle and Knee Joints Passive Range of Motion

The onset and offset of the passive movements were determined by using ankle and knee goniometers. The joints’ angles were used to define the speed and velocity of the passive movements. The average velocity of the movements was further analyzed to assess the consistency of the passive movements. A reliability analysis was used to assess the consistency of the movements.
3.2.5 Instruments

3.2.5.1 Goniometers

Biometrics goniometers (Twin Axis Goniometers SG Series, Biometrics Ltd, UK) were used to measure the range of motion of the ankle (SG110/A) and knee (SG150) joints. The accuracy and repeatability of the goniometers were ±2° and 1° over a range of ±90° (180). The sample rate of recorded data was 1KHz. Two goniometers were available in the lab; the single axis and the twin axis. The twin axis was capable of measuring the ankle joint range of motion. The single axis was capable of measuring both knee and hip joint range of motion. We chose to use the single axis goniometer to measure knee flexion and knee extension range of motion. However, the hip range of motion would have been useful to measure. The ankle and knee joint goniometers are shown in Figure 3.2.

![Figure 3.2 Ankle and Knee Goniometers](image)

3.2.5.2 EMG

The electrical activity of the muscles was recorded by a 16 channels Bagnoli™ Desktop EMG system (Delsys Inc, MA, US). The extension of the Bagnoli TM system was used to measure the goniometers data. The Bagnoli™ system EMG bandwidth was 20-450 Hz±10%. The EMG was recorded
at a frequency of 1KHz. Delsys surface EMG single differential model sensors were used for data collection.

### 3.2.5.3 The Segway

The Segway PT i2 (Segway Inc., US) was used for both the static and dynamic standing training. The Segway in SCI Mobility Research Lab was modified by adding two sidebars to the Segway. This modification was done to provide more safety for the SCI participants. For the static standing training, we put 4 blocks under the Segway platform and fixed the Segway on the ground (Figure 3.3).

![Figure 3.3 Static Segway/ Static Standing Training](image)

### 3.2.6 Protocol Schematic

The study took place at the SCI Mobility Research Lab at ICORD. The participants came to ICORD three times for this study. The sessions were divided into familiarization and 2 testing sessions. Each participant was in two different training sessions and had the static and dynamic standing in two different days. Participants were randomly assigned to participate in either the static or dynamic standing training session as described in study design section (3.2.3). A minimum one week wash-out period was incorporated between the standing training periods to attempt to eliminate any carry-over effect between
the first and second training sessions. Muscle spasticity might change during the day, thus we asked the participants to come in at the same time for both training session. The participation sessions are described below.

3.2.6.1 Familiarization

I performed the primary familiarization session for all of the participants. First, I evaluated the participant’s lower extremity spasticity level. The participants with a spasticity level of $\geq 1$ remained in the study and finished the familiarization session. The primary interview was done to complete the intake form (Appendix G). All participants filled out the intake form which asked for the individuals demographic information such as age, gender, year of injury, injury level, AIS, complete/incomplete SCI, mobility device, and antispastic medication. AIS was determined for each SCI participant based on their medical history (self-report). The demographic information of the AB participants was limited to their age and gender. I explained the consent form step by step to the participants and answered their questions before the participants signed the consent form. In this session, each participant had a 10 min Segway familiarization session to be prepared for the dynamic training session. The participants were asked to remain on their regular medication and exercise regimes during the study time. We excluded the participants if at the time of the study they had medical conditions such as acute urinary tract infection and pressure ulcers.

3.2.6.2 Test Preparation

All participants were asked to empty their bladder before each training session in order to reduce the possibility of a full bladder causing spasms triggering. The examiner prepared the participants for the measurements. The participants were asked about which leg they felt had more spasticity and spasms. The AB participants were asked to indicate their dominant leg. Four muscle sites on the rectus femoris, biceps femoris, adductors, and medial gastrocnemius were shaved. We shaved the muscle sites on the leg they felt more spasticity for SCI participants and on the dominant leg of AB participants. The skin was cleaned by isopropyl alcohol and EMG sensors were attached on the skin. The knee and ankle goniometers were
attached on the knee and ankle of the same leg as it is shown in Figure 3.4. The EMG ground electrode was attached to the other leg medial malleolus. The whole setup is shown in Figure 3.4.

![Participant Preparation Setup](image)

**Figure 3.4 Participant Preparation Setup**

### 3.2.6.3 Static Training Session

The static training session lasted for 20 minutes based on the standing training durations used in previous studies (131,134,181). The SCI participants evaluated their lower extremity muscle groups’ level of spasticity by VAS before starting the training session. The MAS and EMG outcome measures were done before the training session as well. The Segway was fixed as described in 3.2.5.3 before the participants came into the lab. The safety harness in the lab was used to ensure the SCI participants safety during the testing session (Figure 3.5). The safety harness was used for all the participants in order to keep the testing situation consistent. Participants stood on the Segway for 20 minutes by themselves while wearing the safety harness. After 20 minutes of standing training, the participants rested for 5 minutes in a lying position. All outcome measures were repeated similar to the pre-training measures. The participants remained in a lying position for 1 hour and the outcome measures were repeated after one hour. During
that one-hour rest the participants could read a book or talk with the research team but they were not allowed to eat or do any stimulating activities such as playing games on a media device.

Figure 3.5 Static Standing Training

3.2.6.4 Dynamic Training Session

The dynamic training session lasted for 20 minutes. There is no precise training period for dynamic standing training but we decided to choose 20 minutes period similar to the static standing training. The 20 min training session was close to a previous study done with the Segway (151). The SCI participants evaluated their lower extremity muscle groups’ spasticity level by VAS before starting the training session. The MAS and EMG outcome measures were done before the training session. There was a defined pathway in the SCI mobility research lab that was used for the dynamic training session. The
ceiling-track mounted safety harness in the lab was used to ensure the SCI participants safety during the dynamic training session (Figure 3.6). The safety harness was used for all the participants in order to keep the testing situation consistent. The participants stood on the Segway by themselves with the protection of the safety harness. The 20 minutes training session was divided into 4x5 minutes training as describe in Figure 3.7. After 20 minutes of dynamic standing training, the participants rested for 5 minutes in a supine lying position. All outcome measures were repeated similar to the pre-training session. The participants remained in a lying position for 1 hour and the outcome measures were repeated after one hour. During that one-hour rest the participants could read a book or talk with the research team but they were not allowed to eat or do any stimulating activities such as playing games on a media device.

Figure 3.6 Dynamic Standing Training
3.2.6.5 Measurements

The measurements are described step by step below:

1. Participants evaluated their level of spasticity by VAS self-assessment before either the static or dynamic training.

2. The EMG during the passive movements: the participants remained in supine position as the examiner applied a passive dorsiflexion movement to assess the gastrocnemius muscle resistance (MAS) (182) and the EMG was recorded at the same time during the movement. Participants were asked to roll over to a prone position. The rectus femoris and biceps femoris muscles were tested in a prone position with the ankles fell beyond the end of the plinth (183). The EMG was measured during the passive knee movement from the maximum possible extension to the maximum possible flexion and then back to maximum possible extension again in one motion.
3. The participants rested for 5 minutes after either the static or dynamic standing training. The measurements were repeated with the same order described in number 1 and 2 after a 5 min rest.

4. All measurements were repeated after one hour as described in number 1 and 2.

5. The AB participants’ EMG was recorded during the ankle and knee passive movements with the same order as described for SCI participants.

The protocol schematic is shown in Fig 3.8.
3.2.7 Data Analysis

A custom interactive program was written in MatLab (MathWorks Inc. Natick, MA) to process the goniometers and the EMG data offline.

3.2.7.1 MAS Analysis

To make the MAS categories convenient for statistical analysis because of the category of +1, the scale was changed from (0-4) to (0-5). The reported scales in Table 3.4 are the original reports of the study (0-4). The MAS scores of passive movements were assessed independently and globally. To obtain a global MAS score for each participant, the scores of three passive movements were summed to get a Modified Ashworth Scale Sum Score (MASSum) (44,151).

3.2.7.2 Goniometers Analysis

The knee and ankle goniometers data was converted from voltage to angles. The regression model was used for the goniometers calibration and the calculated equations are listed below:

- Ankle goniometer (degree) = 93* Average voltage (V) + (-233)
- Knee goniometer (degree) = 93* Average voltage (V) + (-234)

The goniometer data was filtered using a low pass filter at 6 Hz. Each goniometer was set to zero before each movement e.g. when the ankle was held in maximum plantar flexion to start the movement, the ankle goniometer was set to zero. To calculate the angular speed (deg/s) of each movement, we used the ankle and knee joints range of motion data. The angular speed of 5 deg/s was used as the threshold to define the start and stop of the ankle and knee passive movements. The knee goniometer processing figure shows the raw goniometer data, as converted to the angle and the angular speed (Fig 3.9). The ankle goniometer figure represents the angle and the angular speed of the ankle movement (Fig 3.10).

Due to the velocity dependent nature of spasticity, only the average velocity of the trials within the average velocity of all the trials ±2 SD were included for further analysis. The average velocities over time (before, immediately after, one hour later) were analyzed to show the consistency of the passive movements.
Figure 3.9 Knee Goniometer Processing. Raw knee angle data (v) (A), knee angle (degree) (B), angular speed (deg/sec) (C) during knee flexion and extension of a SCI participant. The first half represents knee flexion and the second half represents knee extension. Dotted black line represents the 5 deg/sec threshold to determine the start and stop of the movements.
Figure 3.10 Ankle Goniometer Processing. Ankle angle (deg) (A) and angular speed (deg/sec) (B) of the ankle dorsiflexion/plantarflexion of a SCI participant.

3.2.7.3 EMG Analysis

As described above the EMG was recorded at a frequency of 1KHz. The Bagnoli™ system EMG bandwidth filter was 20-450 Hz±10%. The recorded EMG was further analyzed by first removing the offset. Then, the EMG was rectified and a notch filter (60 Hz) was applied to the EMG files. The rectified notch filtered EMG was low pass filtered at 10 Hz. Figure 3.11 shows the EMG processing for one of the SCI participants. The challenge with normalizing the EMG files in individuals with SCI is the probability of a weak maximum voluntary isometric contraction (MVIC) and the recorded MVIC is not representative of the involuntary muscle activity in response to the stretch reflex (184). The EMG signals
were not scaled to MVIC however to help with standardization we assessed a standard length of time for each movement. For example, the integrated EMG (IEMG) of ankle dorsiflexion was calculated from the start of the passive movement (angular speed of 5 degree/sec) to 1 sec after the start of the movement (Figure 3.12.A). The ankle EMG processing duration was chosen based on the average duration (SCI: 0.92±0.49 sec, AB: 0.88±0.73 sec) of ankle dorsiflexion movement from the start to the stop point. The IEMG of knee flexion and extension was calculated from the start of the passive movement to 2 sec after the start point (Figure 3.12.B). The knee EMG processing duration was chosen based on the average duration of passive knee flexion (SCI: 2.13±0.82 sec, AB: 1.75±0.65 sec) and knee extension (SCI: 1.94±0.60 sec, AB: 1.75±0.65 sec) from the start to the stop point. The IEMG of the baseline EMG (same duration for each movement) was calculated for each movement. The baseline IEMG was subtracted from the IEMG during the movement and that value was used for further analysis.
Figure 3.11 EMG Processing. Offset removal (A), rectified and notch filter (60Hz) EMG (B), low pass filtered EMG (10Hz) (C).
Figure 3.12 Integrated EMG. Ankle dorsiflexion integrated EMG (A), Knee flexion and extension integrated EMG (B) based on the goniometer start point of the passive movements.
The IEMG changes (Δ) were calculated as the difference of the IEMG before and immediately after the standing training. This formula was used to calculate the changes from before to one hour after the standing training.

\[ \Delta \text{IEMG (B-A)} = \text{IEMG before} - \text{IEMG after} \]

\[ \Delta \text{IEMG (B-H)} = \text{IEMG before} - \text{IEMG one hour later} \]

To compare the static and dynamic standing condition effects on spasticity and muscle activity changes, we used the \( \Delta \text{IEMG (B-A) and (B-H)} \). To determine the muscle activity and spasticity changes over time (before, after, and one hour later), we analyzed the actual IEMG values of the three time points (before, immediately after, and one hour later).

### 3.2.8 Statistical Analysis

The analysis of variance (ANOVA) was used to analyze the velocity, EMG and VAS. A 2x3 (condition x time) repeated measures of ANOVA was used for velocity and VAS analysis. A 2x3 (condition x time) repeated measures of ANOVA was used for actual IEMG analysis. To compare actual values of IEMG, we had a limitation for interpreting significant main effect for the standing conditions due to the potential confounding effect of having recorded the standing conditions on different days. The main effect of time remains relatively unaffected by any differences between the EMG recorded on different days therefore the analysis of actual IEMG was used to determine the main effect of time (before, after, one hour later). A 2x2 repeated measures of ANOVA was used for IEMG changes (\( \Delta \) IEMG) analysis to determine the difference between dynamic and static standing training effects on spasticity.

The MAS is a nonparametric spasticity outcome measure and the related-samples Friedman two-way analysis of variance by ranks statistical analysis method was used to analyze the MAS scores. This method is similar to parametric ANOVA statistical analysis for nonparametric repeated measures that were tested three or more times. To analyze the MASSum scores, we used the Friedman analysis of
variance ANOVA. Although MASSum has been used as an outcome measure in previous studies (44,151), the ordinal nature of MAS score may contravene the statistical assumptions by this analysis.

To ensure the movements performed during the MAS were consistent, the velocities of the movements were analyzed for intra-session and between-session reliability. The intra-session and between-session reliability were calculated using Intraclass Correlation Coefficients (ICCs): ICC (2,1) (single measures) (185). It was based on a 2-way (random effects) repeated measures analysis of the variance model with agreement. Although there are many ways to interpret ICC values, we chose Munro’s classification of reliability coefficients: 0.26 to 0.49 reflects low correlation; 0.50 to 0.69 reflects moderate correlation; 0.70 to 0.89 reflects high correlation; and 0.90 to 1.00 indicates very high correlation (186). All statistical computations were completed by SPSS (PASW Statistics18, 2009). Statistical significance was evaluated at an alpha level of 0.05.

3.3 Results

3.3.1 Population

Ten individuals with SCI (nine males and one female) with the level of injury between C3- T6 and ten AB individuals (nine males and one female) without any recognized neurologic disorder volunteered to participate in the study. The mean±SD age of the SCI and AB groups was 40.4±11.15 years and 39.9±10.9 years respectively. The specific characteristics of SCI and AB group are summarized in Table 3.1 and 3.2 respectively.
### Table 3.1 Spinal Cord Injury Participants Characteristics

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age (Year)</th>
<th>Gender</th>
<th>Year of Injury</th>
<th>Injury Level</th>
<th>AIS</th>
<th>Complete/Incomplete Injury</th>
<th>Antispastic</th>
<th>Mobility</th>
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<tbody>
<tr>
<td>1</td>
<td>42</td>
<td>M</td>
<td>2009</td>
<td>C4-C5</td>
<td>D</td>
<td>Incomplete</td>
<td>No</td>
<td>Walk</td>
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<tr>
<td>2</td>
<td>42</td>
<td>M</td>
<td>1987</td>
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<td>No</td>
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<tr>
<td>3</td>
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<td>M</td>
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<td>4</td>
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<td>2002</td>
<td>T5-T6</td>
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<td>No</td>
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<tr>
<td>5</td>
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<td>M</td>
<td>2003</td>
<td>C5-C6</td>
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<td>Power Wheelchair</td>
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<tr>
<td>6</td>
<td>47</td>
<td>M</td>
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<td>Baclofen/Gabapentine</td>
<td>Walk</td>
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<td>55</td>
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<td>2011</td>
<td>C5-C7</td>
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<td>Incomplete</td>
<td>Baclofen</td>
<td>Manual Wheelchair</td>
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<td>M</td>
<td>1993</td>
<td>C7</td>
<td>C</td>
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<td>No</td>
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<td>9</td>
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<td>C4-C5</td>
<td>D</td>
<td>Incomplete</td>
<td>Nabilone</td>
<td>Walk</td>
</tr>
<tr>
<td>10</td>
<td>29</td>
<td>F</td>
<td>2008</td>
<td>C4-C5</td>
<td>D</td>
<td>Incomplete</td>
<td>No</td>
<td>Walk</td>
</tr>
</tbody>
</table>

Abbreviation: M, Male; F, Female.

### Table 3.2 Able-Bodied Participants Characteristics

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age (Year)</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
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<td>1</td>
<td>30</td>
<td>M</td>
</tr>
<tr>
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<td>M</td>
</tr>
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<td>51</td>
<td>M</td>
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<tr>
<td>10</td>
<td>48</td>
<td>M</td>
</tr>
</tbody>
</table>

Abbreviation: M, Male; F, Female.
3.3.2 Visual Analog Scale (VAS)

The VAS measures of ten SCI participants are reported in Appendix H. The mean and standard deviation (SD) of each tested muscle group is reported in Table 3.3. Figure 3.13 shows the results of the VAS scores. There was a decreasing trend from before to immediately after and from before to one hour after both training but not to a statistically significant level. There was no significant interaction effect between the static and dynamic standing training over time (before, immediately after, and one hour later) for the quadriceps (p=0.10), hamstrings (p=0.07), adductors (p=0.41) and calf (p=0.15) muscle groups. There was no significant standing training (dynamic and static) main effect for the quadriceps (p=0.73), hamstrings (p=0.14), adductors (p=0.31), and calf (p=0.41) muscle groups. There was no significant main effect of time (before, after, one hour later) for the quadriceps (p=0.05), hamstrings (p=0.06), and calf (p=0.16) muscle groups. There was a significant main effect of time for adductors (p=0.04). We did not conduct a post-hoc test due to the lack of interaction effect for the adductors muscle group.

Table 3.3 Visual Analog Scale (VAS) Outcome Measure Results.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Dynamic Standing</th>
<th>Static Standing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Quadriceps</td>
<td>38.2±19.3</td>
<td>30.2±18.7</td>
</tr>
<tr>
<td>Hamstrings</td>
<td>43.6±20.8</td>
<td>36.3±23.6</td>
</tr>
<tr>
<td>Adductors</td>
<td>32.6±22.6</td>
<td>30.6±18.6</td>
</tr>
<tr>
<td>Calf muscle</td>
<td>39.1±30.3</td>
<td>32.5±20.9</td>
</tr>
</tbody>
</table>

The mean±SD is reported for each time point. The results are in millimeter (mm).
Figure 3.13 Visual Analog Scale in Individuals with Spinal Cord Injury. Quadriceps (A), Hamstrings (B), Calf muscle (C), and Adductors (D).
3.3.3 Modified Ashworth Scale (MAS)

MAS

The MAS scores of all the participants are reported in Table 3.4. There was no significant interaction effect between the static and dynamic standing training MAS scores over time (before, immediately after, and one hour later) for ankle dorsiflexion ($p=0.57$), knee flexion ($p=0.24$), and knee extension ($p=0.60$). Although not statistically significant, the average MAS scores of all passive movements were decreased over time from before to immediately after and from before to one hour later for both the dynamic and static standing training.

MASSUM

The average of MASSum scores of dynamic and static standing training overtime are reported in Table 3.5. There was no significant interaction effect between the static and dynamic standing training MASSum measures over time (before, immediately after, and one hour later) ($p=0.07$).
Table 3.4 Modified Ashworth Scale Outcome Measure Results

<table>
<thead>
<tr>
<th>Participant</th>
<th>Dynamic Standing</th>
<th>Static Standing</th>
</tr>
</thead>
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<td>1 1+ 1+ 1 1+ 1</td>
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<td>2</td>
<td>1 1+ 1 1+ 2 1</td>
<td>1 1+ 1 1+ 2 1+</td>
</tr>
<tr>
<td>3</td>
<td>1 1 1 0 0 1</td>
<td>1 1+ 1+ 1+ 1 1</td>
</tr>
<tr>
<td>4</td>
<td>2 2 1+ 1+ 0 0</td>
<td>1+ 2 1 1 1+ 0</td>
</tr>
<tr>
<td>5</td>
<td>2 1+ 1+ 1+ 1 1</td>
<td>1 2 1 1 2 1 1</td>
</tr>
<tr>
<td>6</td>
<td>1+ 2 1+ 1 1+ 1+</td>
<td>1 1 1 1 1 1</td>
</tr>
<tr>
<td>7</td>
<td>1+ 1+ 1+ 2 1 1+</td>
<td>1 1 1+ 1 1+ 2</td>
</tr>
<tr>
<td>8</td>
<td>2 2 2 3 2 1+</td>
<td>2 2 1 1+ 2 1</td>
</tr>
<tr>
<td>9</td>
<td>2 1 1+ 2 0 0</td>
<td>2 1+ 1+ 2 0 0</td>
</tr>
<tr>
<td>10</td>
<td>2 2 1 1+ 2 1</td>
<td>2 2 1 1+ 2 1</td>
</tr>
</tbody>
</table>

Abbreviations: D, Dorsiflexion; K.F, Knee Flexion; K.E, Knee Extension.
3.3.4 Electromyography of SCI Participants

To illustrate what the EMG looked like for a SCI participant over time, Figure 3.14 shows the EMG of the rectus femoris, biceps femoris, and gastrocnemius muscles during knee flexion, knee extension, and ankle dorsiflexion respectively.

### Table 3.5. Average MASSum Results

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Time</th>
<th>Before</th>
<th>After</th>
<th>One hour later</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dynamic</td>
<td></td>
<td>9.2</td>
<td>7.5</td>
<td>7.9</td>
</tr>
<tr>
<td>Static</td>
<td></td>
<td>8.7</td>
<td>7.7</td>
<td>7.7</td>
</tr>
</tbody>
</table>
Figure 3.14 SCI Participant Overlapped EMG of Three Time Points. Rectus femoris EMG (knee flexion) (A) Biceps femoris EMG (knee extension) (B) Gastrocnemius EMG (ankle dorsiflexion) for a SCI participant before, after and one hour later of dynamic standing training.
3.3.4.1 Muscle Changes Over Time

To report the difference between static and dynamic standing we chose to report the change (ΔIEMG) from before the training to immediately after (B-A) as one outcome and from before to one hour later (B-H) as the second. For this part, the interaction effect represents the interaction between the static and dynamic standing training and the time (B-A and B-H). The main effect of standing represents the effect of static and dynamic standing training on ΔIEMG. The main effect of time represents the effect of time (B-A and B-H) on ΔIEMG. The changes for dynamic and static training for the primary muscles (gastrocnemius, biceps femoris and rectus femoris) are reported in Table 3.6 and Figure 3.15.

To report the effect of time on EMG, the actual IEMG values were assessed over time (before, immediately after, one hour later). For this part, the interaction effect represents the interaction between the static and dynamic standing training and the time (before, after, one hour later). The main effect of standing represents the effect of the static and dynamic standing training on actual IEMG. Due to the limitation for interpreting significant main effect of standing conditions, we did not report the main effect of standing on the actual IEMG. The main effect of time represents how the IEMG changed over time (before, after, one hour later).

Table 3.6 SCI Group Average ΔIEMG Changes for the Primary Muscles

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Average Δ IEMG (B-A) /Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-A) /Static (µv.sec)</th>
<th>Average Δ IEMG (B-H) /Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-H) /Static (µv.sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrocnemius</td>
<td>0.93 ± 1.14</td>
<td>0.52 ± 1.22</td>
<td>1.12 ± 1.26</td>
<td>0.50 ± 1.00</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>0.46 ± 1.46</td>
<td>0.30 ± 2.14</td>
<td>-0.50 ± 2.48</td>
<td>0.50 ± 1.56</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>1.28 ± 2.12</td>
<td>0.40 ± 1.52</td>
<td>0.82 ± 1.94</td>
<td>0.10 ± 1.94</td>
</tr>
</tbody>
</table>
Figure 3.15 SCI Group ΔIEMG Changes. Gastrocnemius (A), Rectus femoris (B), Biceps femoris (C).
**Ankle Dorsiflexion: Gastrocnemius**

\( \Delta \text{IEMG} \)

There was no significant interaction effect (p=0.28), standing main effect (p=0.42), and main effect of time (p=0.30) for the gastrocnemius muscle.

\text{IEMG} 

There was no significant interaction effect (p=0.86) but there was a *significant main effect of time* (p=0.001) for the gastrocnemius muscle. We did not conduct a post-hoc test due to the lack of interaction effect between time and conditions for this muscle.

**Knee Flexion: Rectus Femoris**

\( \Delta \text{IEMG} \)

There was no significant interaction effect (p=0.26), standing main effect (p=0.25), and main effect of time (p=0.16) for the rectus femoris muscle.

\text{IEMG} 

There was no significant interaction effect (p=0.26) and main effect of time (p=0.56) for the rectus femoris muscle.

**Knee Extension: Biceps Femoris**

\( \Delta \text{IEMG} \)

There was no significant interaction effect (p=0.84), standing main effect (p=0.22), and main effect of time (p=0.30) for the biceps femoris muscle.

\text{IEMG} 

There was no significant interaction effect (p=0.44) and main effect of time (p=0.19) for the biceps femoris muscle.
3.3.4.2 Global Effects of Standing Conditions on Muscle Activity Changes

Ankle Dorsiflexion

Δ IEMG

Average Δ IEMG changes of three muscles other than the gastrocnemius for the ankle dorsiflexion are shown in Table 3.7. There was no significant interaction effect for the biceps femoris (p=0.46), rectus femoris (p=0.08), and adductor (p=0.64) muscles. There was no significant standing main effect for the biceps femoris (p=0.55), rectus femoris (p=0.21), and adductor (p=0.66) muscles. There was no significant main effect of time for the biceps femoris (p=0.44), rectus femoris (p=0.12), and adductor (p=0.57) muscles.

Table 3.7 Global SCI Group Ankle Dorsiflexion Average Δ IEMG Changes

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Average Δ IEMG (B-A) /Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-A) /Static (µv.sec)</th>
<th>Average Δ IEMG (B-H) /Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-H) /Static (µv.sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectus femoris</td>
<td>0.12 ± 0.14</td>
<td>-0.001± 0.09</td>
<td>0.02± 0.15</td>
<td>0.02± 0.08</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>0.13± 1.10</td>
<td>0.04± 0.52</td>
<td>0.40± 1.22</td>
<td>0.01± 0.57</td>
</tr>
<tr>
<td>Adductors</td>
<td>0.19± 0.14</td>
<td>0.13± 0.16</td>
<td>0.21± 0.16</td>
<td>0.09±0.73</td>
</tr>
</tbody>
</table>

IEMG

There was no significant interaction effect for the biceps femoris (p=0.66), rectus femoris (p=0.08), and adductor (p=0.73) muscles. There was no significant main effect of time for the biceps femoris (p=0.27), rectus femoris (p=0.08), and adductor (p=0.59) muscles.
Knee Flexion

Δ IEMG

Average Δ IEMG changes of three muscles other than the rectus femoris for the knee flexion are shown in Table 3.8. There was no significant interaction effect for the biceps femoris (p=0.43), gastrocnemius (p=0.33), and adductor (p=0.23) muscles. There was no significant standing main effect for the biceps femoris (p=0.66), gastrocnemius (p=0.91), and adductor (p=0.69) muscles. There was no significant main effect of time for the biceps femoris (p=0.13), gastrocnemius (p=0.69), and adductor (p=0.17) muscles.

Table 3.8 Global SCI Group Knee Flexion Average Δ IEMG Changes

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Average Δ IEMG (B-A)/Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-A)/Static (µv.sec)</th>
<th>Average Δ IEMG (B-H)/Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-H)/Static (µv.sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrocnemius</td>
<td>0.04 ± 0.34</td>
<td>0.14 ± 0.46</td>
<td>0.16 ± 0.30</td>
<td>0.08 ± 0.20</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>0.64 ± 3.60</td>
<td>0.64 ± 2.00</td>
<td>1.52 ± 2.32</td>
<td>0.46 ± 0.13</td>
</tr>
<tr>
<td>Adductors</td>
<td>0.02 ± 0.36</td>
<td>-0.08 ± 0.22</td>
<td>0.04 ± 0.34</td>
<td>0.08 ± 0.16</td>
</tr>
</tbody>
</table>

IEMG

There was no significant interaction effect for the biceps femoris (p=0.59), gastrocnemius (p=0.51), and adductor (p=0.43) muscles. There was no significant main effect of time for the gastrocnemius (p=0.26) and adductor (p=0.51) muscles. There was a significant main effect of time for the biceps femoris (p=0.04) but we did not conduct a post-hoc test due to the lack of interaction effect between time and conditions for this muscle.
Knee Extension

Δ IEMG

Average Δ IEMG changes of three muscles other than the biceps femoris for the knee extension are shown in Table 3.9. There was no significant interaction effect for the rectus femoris (p=0.40), gastrocnemius (p=0.70), and adductor (p=0.77) muscles. There was no significant standing main effect for the rectus femoris (p=0.96), gastrocnemius (p=0.83), and adductor (p=0.39) muscles. There was no significant main effect of time for the rectus femoris (p=0.46), gastrocnemius (p=0.07), and adductor (p=0.87) muscles.

Table 3.9 Global SCI Group Knee Extension Average Δ IEMG Changes

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Average Δ IEMG (B-A) /Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-A) /Static (µv.sec)</th>
<th>Average Δ IEMG (B-H) /Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-H) /Static (µv.sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrocnemius</td>
<td>0.36 ± 0.46</td>
<td>0.32± 0.84</td>
<td>0.46±0.72</td>
<td>0.40± 1.04</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>0.10 ± 0.16</td>
<td>0.20± 0.90</td>
<td>0.16±0.44</td>
<td>0.20± 0.92</td>
</tr>
<tr>
<td>Adductors</td>
<td>0.06± 0.22</td>
<td>-0.008±0.10</td>
<td>0.04±0.32</td>
<td>0.016±0.14</td>
</tr>
</tbody>
</table>

IEMG

There was no significant interaction effect for the rectus femoris (p=0.69), gastrocnemius (p=0.82), and adductor (p=0.71) muscles. There was no significant main effect of time for the rectus femoris (p=0.28) and adductor (p=0.75) muscles. There was a significant main effect of time for the gastrocnemius (p=0.01) but we did not conduct a post-hoc test due to the lack of interaction effect between time and conditions for this muscle.
3.3.5 Able-Bodied EMG

The AB participants EMG was analyzed similarly to the SCI participants.

3.3.5.1 Muscle Changes with Time

The IEMG changes for the static and dynamic training for the primary muscles (gastrocnemius, biceps femoris and rectus femoris) are reported in Table 3.10. The Δ IEMG scales were smaller than the SCI participants for the primary muscles.

Table 3.10 Able-bodied Group Average Δ IEMG Changes for the Primary Muscles

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Average Δ IEMG (B-A) /Dynamic (µv.sec)</th>
<th>Average Δ IEMG (B-A) /Static (µv.sec)</th>
<th>Average Δ IEMG (B-H) /Dynamic (µv/sec)</th>
<th>Average Δ IEMG (B-H) /Static (µv/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrocnemius</td>
<td>0.01± 0.11</td>
<td>0.03± 0.10</td>
<td>0.05±0.28</td>
<td>0.03± 0.08</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>0.002± 0.26</td>
<td>0.1 ±0.14</td>
<td>0.08 ±0.2</td>
<td>0.01 ±0.26</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>0.26± 0.82</td>
<td>0.08±0.52</td>
<td>0.80±1.44</td>
<td>0.02±0.14</td>
</tr>
</tbody>
</table>

Ankle Dorsiflexion: Gastrocnemius

Δ IEMG

There was no significant interaction effect (p=0.71), standing main effect (p=0.98), and main effect of time (p=0.63) for the gastrocnemius muscle.

IEMG

There was no significant interaction effect (p=0.86) and main effect of time (p=0.36) for the gastrocnemius muscle.
**Knee Flexion: Rectus Femoris**

Δ IEMG

There was no significant interaction effect (p=0.19), standing main effect (p=0.87), and main effect of time (p=0.95) for the rectus femoris muscle.

IEMG

There was no significant interaction effect (p=0.32) and main effect of time (p=0.39) for the rectus femoris muscle.

**Knee Extension: Biceps Femoris**

Δ IEMG

There was no significant interaction effect (p=0.46), standing main effect (p=0.55), and main effect of time (p=0.44) for the biceps femoris muscle.

IEMG

There was no significant interaction effect (p=0.51) and main effect of time (p=0.28) for the biceps femoris muscle.

3.3.5.2 **Global Effects of Standing Conditions on Muscle Activity**

**Ankle Dorsiflexion**

Δ IEMG

There was no significant interaction effect for the biceps femoris (p=0.14), rectus femoris (p=0.99), and adductor (p=0.95) muscles. There was no significant standing main effect for the biceps femoris (p=0.69), rectus femoris (p=0.73), and adductor (p=0.42) muscles. There was no significant main effect of time for the biceps femoris (p=0.70), rectus femoris (p=0.68), and adductor (p=0.29) muscles.
IEMG

There was no significant interaction effect for the biceps femoris (p=0.43), rectus femoris (p=0.92), and adductor (p=0.41) muscles. There was no significant main effect of time for the biceps femoris (p=0.23), rectus femoris (p=0.42), and adductor (p=0.43) muscles.

Knee Flexion

Δ IEMG

There was no significant interaction effect for the biceps femoris (p=0.72), gastrocnemius (p=0.55), and adductor (p=0.51) muscles. There was no significant standing main effect for the biceps femoris (p=0.91), gastrocnemius (p=0.63), and adductor (p=0.72) muscles. There was no significant main effect of time for biceps femoris (p=0.37), gastrocnemius (p=0.44), and adductor (p=0.37) muscles.

IEMG

There was no significant interaction effect for the biceps femoris (p=0.91), gastrocnemius (p=0.65), and adductor (p=0.70) muscles. There was no significant main effect of time for the biceps femoris (p=0.48), gastrocnemius (p=0.45) and adductor (p=0.55) muscles.

Knee Extension

Δ IEMG

There was no significant interaction effect for the rectus femoris (p=0.99), gastrocnemius (p=0.81), and adductor (p=0.20) muscles. There was no significant standing main effect for the rectus femoris (p=0.39), gastrocnemius (p=0.90), and adductor (p=0.74) muscles. There was no significant main effect of time for the rectus femoris (p=0.27), gastrocnemius (p=0.36), and adductor (p=0.19) muscles.
There was no significant interaction effect for the rectus femoris (p=0.79), gastrocnemius (p=0.95), and adductor (p=0.34) muscles. There was no significant main effect of time for the rectus femoris (p=0.33), gastrocnemius (p=0.19), and adductor (p=0.29) muscles.

### 3.3.6 Movement Velocity

#### 3.3.6.1 Spinal Cord Injury Participants

The knee flexion, extension, and ankle dorsiflexion average velocity mean± SD and the range are reported in Table 3.11. There was no significant interaction effect between the static and dynamic standing training over time (before, immediately after, one hour later) for the average velocity of knee flexion (p=0.88), knee extension (p=0.84), and ankle dorsiflexion (p=0.32).

ICC values for the intra-session and between-session reliability are shown in Table 3.12 and 3.13 respectively. The intra-session ICC2,1 was high for the ankle dorsiflexion and knee extension and very high for the knee flexion. The between-session ICC2,1 was moderate for the ankle dorsiflexion, knee extension, and knee flexion.

<table>
<thead>
<tr>
<th>Movement</th>
<th>Knee Flexion</th>
<th>Knee Extension</th>
<th>Ankle Dorsiflexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average velocity</td>
<td>58.75±13.85</td>
<td>67.31±12.43</td>
<td>30.44±10.64</td>
</tr>
<tr>
<td>(deg/sec)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range (deg/sec)</td>
<td>29.96-94.02</td>
<td>35.80-96.27</td>
<td>12.99-52.99</td>
</tr>
</tbody>
</table>

Table 3.11 Passive Movement Average Velocity and Range of Spinal Cord Injury Participants
Table 3.12 Average Velocity Intra-session ICC$^2_{2,1}$ of SCI Participants

<table>
<thead>
<tr>
<th>Passive Movement</th>
<th>ICC$^2_{2,1}$</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle dorsiflexion (dynamic)</td>
<td>0.78</td>
<td>0.000</td>
</tr>
<tr>
<td>Ankle dorsiflexion (static)</td>
<td>0.75</td>
<td>0.000</td>
</tr>
<tr>
<td>Knee flexion (dynamic)</td>
<td>0.90</td>
<td>0.000</td>
</tr>
<tr>
<td>Knee flexion (static)</td>
<td>0.90</td>
<td>0.000</td>
</tr>
<tr>
<td>Knee extension (dynamic)</td>
<td>0.83</td>
<td>0.000</td>
</tr>
<tr>
<td>Knee extension (static)</td>
<td>0.88</td>
<td>0.000</td>
</tr>
</tbody>
</table>

Table 3.13 Average Velocity Between-session ICC$^2_{2,1}$ of SCI Participants

<table>
<thead>
<tr>
<th>Passive Movement</th>
<th>ICC$^2_{2,1}$</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle dorsiflexion</td>
<td>0.54</td>
<td>0.000</td>
</tr>
<tr>
<td>Knee flexion</td>
<td>0.54</td>
<td>0.000</td>
</tr>
<tr>
<td>Knee extension</td>
<td>0.58</td>
<td>0.000</td>
</tr>
</tbody>
</table>

3.3.6.2 Able-bodied Participants

As EMG was recorded with the same method between SCI and AB participants, the average velocity mean±SD and range of the knee flexion, knee extension, and ankle dorsiflexion are reported in Table 3.14.

Table 3.14 Passive Movement Average Velocity and Range of Able-Bodied

<table>
<thead>
<tr>
<th>Movement</th>
<th>Knee Flexion</th>
<th>Knee Extension</th>
<th>Ankle Dorsiflexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average velocity</td>
<td>72.91±8.60</td>
<td>71.86±11.80</td>
<td>41.61±13.55</td>
</tr>
<tr>
<td>Range</td>
<td>50.21-89.38</td>
<td>42.43-95.87</td>
<td>20.93-83.04</td>
</tr>
</tbody>
</table>
3.4 Discussion

The main purpose of this study was to explore how two different standing conditions, dynamic (on the Segway) and static standing, are effective to decrease spasticity in individuals with SCI. The secondary purpose of this study was to explore the AB participants’ muscle activity changes after standing training.

There was a significant main effect of time for the adductor muscle group VAS measures without any significant interaction effect between the conditions and the time. We also observed a statistically significant main effect of time for the actual values of IEMG of the gastrocnemius (during ankle dorsiflexion and knee extension) and the biceps femoris (during knee flexion). These findings represent the effect of standing on these muscles over time (before, after, one hour later). However, the results of this study did not support the presence of additional spasticity reduction after the dynamic standing on the Segway compared to the static standing. There was no significant difference between the dynamic and static standing training in AB participants’ muscle activity.

Although the results of the present study has no evidence to support more spasticity reduction after dynamic standing compared to the standing alone, both interventions have been shown to reduce spasticity in individuals with SCI (11,131,135,151,187). There is no conclusion based on the present evidence that can explain the primary responsible mechanism for spasticity reduction after standing training. Individuals with SCI generally showed hyperexcitability of the spinal motor neuron pool, mostly due to the lack of inhibitory descending pathways from upper parts of the CNS to the spinal cord (196,197). The reduction of spasticity after standing training was suggested to be due to the inhibitory neural inputs that generated from standing posture (192). Kawashima et al. proposed the role of load on somatosensory inputs that might result in spasticity reduction after standing interventions (198). Non-neural mechanisms such as visco-elastic property changes of spastic muscles and the load on the vertebral column and lower limb joints may assist spasticity reduction after standing interventions (134,192,
Bulter et al. and Ibrahim et al. suggested that the stretch reflex modulation is mostly due to the motor activity rather than load or passive stretch (199,200). Consequently, Bakheit et al. argued that motor activity and voluntary muscle activities would be more effective to change muscle spasticity rather than stretch reflex approaches (134). CNS plasticity has been proposed as one of the possible responsible mechanisms for spasticity management. Trimble and Koceja showed that repeated standing training enhanced the CNS plasticity (201) and it is comprehensible from the present study that one standing training session is not sufficient to induce any neural plasticity. However, this neural plasticity idea may be supported by the importance of voluntary muscle activities for spasticity management after SCI (134). The sustained corticospinal pathways such as vestibulospinal pathway with postural adjustment generation may also be a responsible mechanism for spasticity reduction after standing training (151,199,200). However due to the lack of statistically significant difference between the standing interventions, we could not suggest any of these proposed mechanisms to be more active after dynamic standing training compared to the static standing training.

This study was an efficacy study under ideal and controlled circumstances in a laboratory setting and included a safety harness for precaution (179). In a previous study on the Segway, participants underwent a community setting training such as navigating around the indoor gymnasium and outdoor experiences (151). The community setting training may provide more postural adjustment (vestibulospinal pathway activation) and motor activation compared to the setting with the safety harness. This may be a possible explanation of why we could not find a difference between the standing interventions.

Dhindsa et al. showed that the whole leg blood flow was reduced in individuals with SCI who experienced a greater spasticity score but were of similar age, time-post SCI, and AIS (202). Although the whole leg blood flow is lower with greater spasticity, standing may change lower extremity blood flow and consequently reduce spasticity. A new unpublished study by Raczak et al. showed an incidental increase in lower extremity blood flow after WBV training (203). They showed spasticity reduction in the
participants with clinical symptoms of lower extremity increased blood flow (203). Their finding supports the probability of increased blood flow and spasticity reduction. However, we did not assess the clinical symptoms of lower extremities blood circulation.

We observed increased muscle activity in some of the participants after standing training. Fall threat may have affected the participants and that would increase their level of excitement, fear, and anxiety while they were in standing position. The postural threat with the probability of falling, may change the reflex modulation in AB population (204). For example, Horslen et al. used an elevated surface and measured T-reflex and H-reflex changes after the fall threat. The T-reflex measurement showed a significant increase when the AB participants were standing on an elevated surface or expecting a balance perturbation without any changes in H-reflex measurements (204). The increased T-reflex amplitude as a result of postural threat with the probability of fall was explained to be the result of an increase in muscle spindles sensitivity or increased in sympathetic drive (204). Increased muscle activity after the standing training in some participants might be a result of excitement, fear, or anxiety that increased spinal reflexes.

Additionally, this study was a feasibility study to develop a methodology to analyze the EMG in individuals with SCI. Due to the limitation of measuring the MVIC in individuals with SCI, we developed a standard method to be able to compare the muscle activity changes after standing interventions. To add the subjective and clinical spasticity changes, we also used VAS and MAS outcome measures. The measurement techniques are discussed in the following sections.

3.4.1 Self-assessment Spasticity Outcome Measure

Although there was a decreasing trend for both static and dynamic standing training, there was no statistically significant difference between these two interventions. There was a main effect of time for adductor muscle group VAS measures. However, this effect was only observed in adductor muscle group. The self-assessment spasticity outcome measures typically report spasticity with respect to the global perception of spasticity (34,46). However, we asked the participants to report their level of spasticity in
four specific lower extremity muscle groups. Spasticity reduction measured by self-assessments has been reported after dynamic and static standing training (133,151,187). Boutilier et al. showed an improvement but not statistically significant change of SCI-SET scores over a one-month (x3/week) dynamic training program (using the Segway) from the beginning to the last training session in individuals with SCI (151). Baker et al. studied the effects of a standing frame over a three-week (30 min each) training in individuals with Multiple Sclerosis (MS) mixed with five exercise training during the day apart from the standing. Their results showed a reduction trend but no significant change of spasticity measured by PSFS (133). The findings of the present study were consistent with these studies. There was an improvement after the static and dynamic standing training, but not to a statistically significant level.

The question of the study was to determine how spasticity reduction is different after static and dynamic standing training. Spasticity evaluation by self-assessment measures does not support any difference between dynamic and static standing. Adams and Hicks compared the effects of tilt-table still standing and body-weight support treadmill training on spasticity. Spasticity was assessed by two self-assessments: SCI-SET and PSFS (187). The self-assessments showed a decrease in spasticity from the baseline measurements to post-activity measurements after multiple sessions with more reductions after the body-weight support treadmill training (187). Although they found no statistical difference between the still standing and body-weight support treadmill training, the treadmill training resulted in more spasticity reduction compared to still standing (187). They showed no significant difference between still standing and treadmill training effects on spasticity that is consistent with the findings of the present study. However, the treadmill training is mostly dependent on walking which is different from dynamic standing training on the Segway.

The minimal clinically important difference (MCID) can be explained as the smallest difference in the scores which the participants identify as beneficial for that specific score. For most of the measures that are used in a clinical setting little attempt has been done to define the specific MCID for that measure (188). The MCID for the VAS pain scores was 9mm (189). Although the MCID was defined for the pain
VAS scores, it may be similar for the VAS spasticity measures. The quadriceps, hamstrings, and calf muscles VAS scores dropped more than 9 mm one hour after the dynamic training that may define a MCID after the dynamic training. However, the MCID should be determined specifically for spasticity VAS scores.

The importance of this study was the non-global spasticity assessment as compared to previous studies. The participants judged four different lower extremity muscle groups. Although it was hard for the study participants to imagine their muscles’ spasticity level separately, it might provide a better understanding of how each muscle group spasticity level changed after standing interventions. It is considered that spasticity self-assessment measures are the best predictor of spasticity impacts on an individual’s daily life (14,187). However, different subjective perception of participants is an ongoing challenge for researchers and clinicians. The question is how scientists and clinicians can rely on self-assessment outcome measures. Thus, it is essential to evaluate spasticity more accurately and precisely by self-assessment outcome measures. A precise, clear, and simple definition of spasticity and muscle tone that is understandable for study participants might be essential to make these outcome measures more useful for research and clinical reasons. Self-assessment by the study’s participants could not provide any additional supportive evidence to show a better spasticity management after the dynamic standing training compared to the static standing training.

### 3.4.2 Clinical Spasticity Outcome Measure

While the MAS has been criticized for emphasizing on one aspect (muscle tone) of spasticity, it still remains the most common clinical spasticity outcome measure that is used by clinicians and scientists (26). Therefore, this clinical spasticity outcome measure was included in the present study. While the evidence indicates that MAS reliability is adequate for the lower extremity (41,42), there are still concerns about the reliability of MAS. Inter-rater reliability is defined as the degree of agreement between the raters (41,42). In this study, one clinician did all the MAS tests and had no concerns in terms of the inter-rater reliability.
Although MAS was reduced over time after both standing interventions, there was no statistically significant difference between the static and dynamic standing training MAS scores. The MASSum scores, representing the global spasticity change, were not statistically different between the static and dynamic standing training.

Both static and dynamic standing training has been shown to reduce spasticity in individuals with different UMN disorders (11, 151, 187). Bohannon examined the effects of tilt table static standing in a case study and assessed the spasticity changes by clinical outcome measures (MAS and pendulum testing). Tilt-table standing changed spasticity and resulted in spasms reduction that lasted until the following morning (11). The results of that study support the effects of still standing on spasticity reduction (11). Adams and Hicks studied the effects of a single session of still standing and body weight support treadmill training on spasticity. They showed a decrease after a single session of body weight support treadmill training setting for sum of MAS (hip flexors, hip extensors, adductors, knee flexors, and knee extensors) without any change during multiple sessions (187). They showed extensor spasms and clonus reduction after a single-session of dynamic body weight support treadmill training setting and tilt-table standing (187). However, extensor spasms and clonus represent extrinsic and phasic spasticity respectively that are different manifestations of spasticity from the tonic spasticity measured in the present study. Boutilier et al. showed that dynamic standing training on the Segway resulted in an immediate significant lower extremity sum of the MAS (MASSum) reduction (151). While clinical spasticity reduction has been associated with standing frame, tilt table, and dynamic standing using the Segway, there was no study that assessed the difference between the effects of static and dynamic standing training on spasticity. However, we found no statistically significant difference between the dynamic and static standing training.

Although MASSum has been used as a representative of global clinical spasticity change, it is not definitely ascertainable if MASSum could reflect the level of spasticity similar to MAS. MAS is an ordinal outcome measure where the order matters but not the difference between values. For example, the
increment between MAS scores of 0 and 1 may not be the same as that between 1 and 1+. However, it has been used as an outcome measure to assess spasticity after standing training (151,187). Due to the ordinal nature of MAS, MASSum may not be a suitable clinical outcome measure to show the global spasticity change after an intervention.

In this study, the MAS was measured for ankle dorsiflexion (gastrocnemius), knee flexion (rectus femoris), and knee extension (biceps femoris) regardless of the muscles in which the participants experienced the greatest spasticity. Boutilier et al. asked the participants to select their top three muscle groups in which they had the greatest spasticity (151). Different subjective perception of spasticity should be considered in the methodology they used in their study. To eliminate the effects of different subjective perception of spasticity, the muscle groups with the greatest spasticity level can be selected based on the baseline MAS examination before the actual study measures. For example, the examiner reports the MAS scores of the lower extremity muscle groups and then selects the top 3 or 4 muscles of each participant with the greatest MAS scores.

Odeen and Knutsson used a quantitative spasticity outcome measure which was similar to subjective MAS outcome measure. They assessed spasticity changes after standing training with feet in dorsiflexion/plantarflexion positions. They used a strain gauge to calculate the calf muscle resistance quantitatively pre and post two different standing training (190). The resistance was measured during fast displacement similar to MAS assessment. They showed a significant reduction at a fast speed in dorsiflexion and plantarflexion positions (190) that supported the effects of standing training on spasticity reduction measured by a clinical quantitative technique. In summary, the clinical spasticity outcome measure could not provide any evidence to show more spasticity reduction after dynamic standing training compared to the static standing training.

3.4.3 Electrophysiologic and Neurophysiologic Outcome Measure

The muscles we measured, using a passive movement technique did not show statistically significant change of EMG activity between the dynamic and static standing training over time. However,
the gastrocnemius and biceps femoris muscle activity dropped more after the dynamic standing training compared to the static standing training during ankle dorsiflexion and knee extension respectively but not to a statistically significant level. The rectus femoris muscle activity reduced more immediately after the dynamic standing training compared to the static standing during knee flexion but not to a statistically significant level. The rectus femoris muscle activity increased to a level higher than its pre training level one hour post dynamic training. We observed significant changes in main effect of time for actual IEMG values of the gastrocnemius (during ankle dorsiflexion and knee extension) and the biceps femoris (during knee flexion) which represent the effect of standing on these muscles. Due to the lack of interaction effect between time and the conditions, we could not apply more statistical analysis on the IEMG of these muscles. These results only represent the effect of standing on the level of activity of these muscles over time without the potential to definitely define which standing training had more effect on spasticity. However, the time effect was only limited to these three trials.

The ankle and knee passive movements stretch the muscle spindles and activate the stretch reflex arc. The stretch reflex activation during the passive movements results in muscle activity that can be recorded by EMG (1,17,21). The passive movements stretch the related muscle spindles but the movement might trigger afferent inputs such as skin, subcutaneous tissue, tendon, and joints (3,13,15,17). Thus, the muscle activity was evaluated in stretched muscles and globally in the lower extremities.

The global activity of the muscles was reported to evaluate how the activation of afferent inputs could activate other lower extremity muscles. For example, the adductor muscle group stretch reflex is originally activated during hip abduction movement (191) but activity of this muscle group was assessed to realize how this muscle group responded globally during ankle and knee passive movements. The global activity of the muscles was much lower than the activity of the stretched muscles. There was no significant difference between dynamic and static standing training global muscle activity. Low global muscle activity specifically adductors explains the non-prominent role of afferent inputs except the muscle spindles in this study settings.
It has been shown that standing training was associated with spasticity reduction compared to non-standing positions (135,188). Field-Fote et al. studied the effects of a single session standing in frame setting in individuals with motor incomplete SCI and measured the pre and post training soleus H-reflex post activation depression (PAD) (135). Standing in frame setting produced a greater PAD (18% less PAD) versus the sitting position (135). Kawashima et al. compared a single session of sitting and standing in frame setting in individuals with complete SCI (192). The spasticity measured by soleus H-reflex amplitude and H/M ratio displayed a significant lower H/M ratio and maximum H-reflex amplitude in the standing training compared to the sitting training (192). These two studies suggested the stretch reflex excitability reduction and consequently spasticity reduction after standing training in individuals with SCI. Dynamic training with locomotor-training paradigm resulted in a significantly more depressed H-reflexes (H/M ratio) during over ground walking (training paradigm) versus still standing (193). To our knowledge this study was the first study that compared spasticity changes after dynamic and static standing training in individuals with SCI. The findings of this study could not provide enough evidence to support more spasticity reduction after the dynamic standing training compared to the static standing training.

Although Field-Fote et al. and Kawashima et al. measured spasticity changes by H-reflex and revealed changes after standing training in frame setting (135,192), other studies found no difference in the H-reflex measurements after standing training (134,187). Adams and Hicks assessed the effects of body weight support treadmill training and tilt-table training on spasticity. They revealed no change in H/M ratio post-activity after one or multiple sessions (187). Bakheit et al. examined two semi-reclined standing interventions (isotonic and isokinetic ankle stretch) effects on spasticity in stroke population (134). The Hmax/Mmax ratio reduced from pre to post training for the isotonic and isokinetic standing training that did not reach the significance (134). The H-reflex latency did not change from pre to post training for isotonic and isokinetic standing training in the same stroke population (134). Isokinetic ankle stretch exercise applied passive movement to the joint but it is not comparable with dynamic standing
training that has whole body activation and incorporation of many lower extremity muscles. The lower response of the H-reflex in individuals with SCI who experience spasticity indicates the impairment of the reflex modulation in that population (135).

Study participants may respond differently to physical therapy techniques. For example, in our study participants number two, nine, and ten gastrocnemius muscle activity showed a lot of spasticity reduction during ankle dorsiflexion whereas for participant number eight, the gastrocnemius muscle activity actually increased after the dynamic standing training. Different muscles might respond differently to training based on their role in standing position and lower extremity stabilization. For example, participant number two and ten with a great gastrocnemius muscle activity reduction during ankle dorsiflexion showed increased rectus femoris muscle activity during knee flexion after the dynamic standing training. This type of inconsistency was seen in another study where Butler et al. reported spasticity changes in individuals with SCI after a non-standing intervention. They reported spasticity reduction in 66% of the trials and increased muscle activity in 28% of the trials with no change or abolished activity in 28% of the trials measured by EMG (116). Non-consistent muscle activity responses to different spasticity management interventions might be due to muscle size, blood circulation, different types of muscle fibers, and the fatigability of the muscles.

This study was a feasibility study to figure out how to analyze the EMG and be able to compare the involuntary increased muscle tone in individuals with SCI. Zupan et al. described spastic response as any non-zero EMG activity resulting from passive maneuver without any specific defined cut off of the muscle electrical activity (69). Laessoe et al. defined spasm as EMG activity amplitude of equaling 4 times or more than the baseline amplitude with duration of more than 5 seconds (70). Although these definitions may be satisfactory to detect spasms and the presence of spasticity in individuals with UMN disorders, it is not possible to detect muscle tone changes after different interventions. We standardized the EMG analysis and defined an acceptable method to assess EMG changes after interventions.
However, the proposed standardization method may be reasonable to evaluate spasticity changes pre and post interventions but still is not adequate when comparing absolute EMG values between interventions.

The AB muscle activity measured by EMG revealed a smaller muscle activity scale and smaller change after the static and dynamic standing training. The spinal stretch reflex pathway is intact in AB individuals (37) which results in less response to the muscle spindle stretch during the passive movements. Minor changes of muscle activity might be present in AB participants due to the effects of the training sessions on the residual muscle tone and the passive partial contraction of the muscles. This residual muscle tone results in the normal muscle resistance to passive stretch during the rest and maintains the posture in AB individuals (194). The other mechanism that may be responsible for the muscle activity changes in AB individuals is the neural adaptation through three different movements over time (75,195). The examiner applied passive movements through the range of motion and the AB participants were asked not to activate their muscles during the whole movement. There might be a lower sensory input due to neural adaptation from before to after the training sessions (75,195). The probability of neural adaptation becomes less prominent by observing an increase in muscle activity in some cases over time. However, muscle activity measured by EMG could not differentiate among the proposed mechanisms for muscle activity changes in AB individuals.

3.4.4 Training

There is no guideline that includes a defined standing training duration for spasticity management. Prolonged standing may result in fatigue which can trigger spasms and spasticity. Training durations were between 20-45 minutes in most of the studies included in the systematic review by Newman and Barker (127). This study had training sessions of 20 minutes in order to reduce the probability of fatigue and subsequently control the effect of fatigue on spasticity. There is no study to show how training duration would change spasticity without resulting in pain or fatigue. Defining an appropriate training duration in individuals who experience spasticity may play a key role in future research and clinical spasticity management interventions.
3.4.5 Modified Ashworth Scale Velocity Consistency

The MAS clinical spasticity outcome measure reliability and validity is one of the main concerns for spasticity measurement. The level of spasticity is reported as the amount of resistance felt during the passive displacement of a limb by an examiner. The reported scale can be different from one examiner to the other examiner (205). Craven et al. found a fair to almost perfect intra-rater reliability (0.2 <kappa>1.0), poor-to-moderate (kappa <0.6) inter-rater reliability of MAS for lower extremity muscles in individuals with SCI (206). The intra-rater reliability differed between two raters (examiners) but the inter-session reliability for a single rater was fair to good (206). The MAS reliability might be independent from measuring by the same examiner or an experienced examiner. Mutlu et al. explained that the inter-rater and intra-rater reliability of the MAS is mostly related to muscle and joint characteristics (207). However, in this study the MAS was assessed entirely by a single examiner thus reducing any biases due to multiple examiners.

Due to the velocity dependent nature of spasticity, velocity of passive movement is an important aspect to show the consistency of the movements between trials. In this study, analysis of the angular velocity showed no statistical difference over time in individuals with SCI. The observed ICC_{2,1} values of average velocity showed excellent intra-session reliability for ankle dorsiflexion and knee flexion/extension. The ICC_{2,1} of average velocity showed moderate between-session reliability for ankle dorsiflexion and knee flexion/extension. The recent published paper in 2013 by Bar-On et al. measured the gastrocnemius and medial hamstring muscles MAS. They recorded the joint positions during different velocities (low and high) to calculate the angular velocity of the movements (208). They reported the Interclass Correlation Coefficients (ICC) of the maximum velocity of ankle dorsiflexion and knee extension during the passive movements for between (B) and within-sessions (W). Ankle dorsiflexion and knee extension maximum velocity ICC\_B/ICC\_W were 0.68/0.96 and 0.84/0.92 respectively (208). In this study, the reliability measures showed a moderate to excellent (high and very high) intra-session and between-session reliability of MAS scores. However, the average velocity of passive movements was
lower in SCI individuals who had a higher measured MAS score regardless of ankle or knee joint movements. The previous studies showed that MAS score may be dependent on the muscle and joint characteristics and not the examiner experience (207). These characteristics may have an impact on the velocity of the passive movements but not to a level that can affect the reliability of MAS measures.

3.5 Study Limitations

3.5.1 Population

Similar to the other rehabilitation clinical research, this research suffered from a low sample size. A larger sample size would provide a greater confidence to know if there was a significant difference between the static and dynamic standing training. Statistical power refers to the probability that one study can find a predefined clinical significance (209). The statistical power of 0.8 or higher is usually defined a satisfactory level of power in scientific studies, corresponding to an 80% chance of a real effect in that study (209). The present study was an underpowered study to determine a significant difference between two standing interventions over time. This was a pilot study to develop a methodology to analyze the EMG in individuals with SCI who experience spasticity. The power of this study for EMG measures of the primary stretched muscles was equal to 0.1. To get the power of 0.8 ($\alpha$ error probability= 0.05, effect size= 0.25), we should include the sample size of 28 in future studies.

The level of injury ranged from C3 to T6 in the present study. It is unknown whether the level of injury could change the effect of physical therapy techniques in individuals with SCI. As reported in the study discussion participants with different level of injury responded differently to the standing training. However due to the limited number of participants, we could not conclude how the level of injury affected the findings of this study.

Spasticity is variable across days due to different factors such as the time of the day, physical activity level, fatigue, and medical conditions (e.g. urinary tract infection). The study did not control for the participants’ physical activity while they participated in the study. Spasticity reduction might be dependent on their daily routine physical activity level. The timeline of the study limited the ability to
control for this parameter and to control for participants with the same level of physical activity. However, we asked them to come in at the same time for both visits and refrain from changing their routines.

### 3.5.2 Training

This study was limited due to the limited number of training sessions. Each participant was in either static or dynamic standing training once. As described in discussion the repeated training sessions could add the CNS plasticity and change the level of excitement.

### 3.5.3 Spasticity Outcome Measures

One of the main limitations of this study was EMG analysis in individuals with SCI. Due to impaired neural pathways, it is impossible to apply the MVIC method for EMG analysis. There is controversy about how to measure the MVIC in individuals with SCI. Barry et al. measured MVIC in that population to assess the selective effects of Baclofen on use-dependent modulation of GABA receptors in individuals with SCI (210). Although MVIC was measured in that study, the rational reported in chapter 3 explained this matter better and why it is inappropriate to measure MVIC in individuals with SCI.

To complement the IEMG analysis it would have been helpful to include H-reflex or T-reflex measures. Lack of any neurophysiologic measures such as H-reflex or T-reflex was a limitation. These additional neurophysiologic outcome measures would have provided a better understanding of how standing training could change spasticity. H-reflex could add valuable quantitative assessment of stretch reflex modulation in the spinal cord (78,79). T-reflex could add a better understanding of phasic component of stretch reflex and muscle spindle response to tension on a tendon (204). However, we limited the spasticity outcome measures to VAS, MAS, and EMG to reduce the probability of fatigue and accordingly spasticity in individuals with SCI.

We had only two goniometers in the lab. The twin axis goniometer was only capable of ankle joint range of motion measurement. The single axis goniometer was used for knee flexion and knee
extension range of motion measurement. We did not measure the hip abduction range of motion thus we could not assess the adductors muscle activity during hip abduction.

The last limitation of study was the lack of control over no voluntary muscle activation in AB individuals. Participants were asked not to contract their muscles actively during the passive movements. However, there was no way to identify voluntary muscle activation during passive movements. We could not differentiate between active and reflexive muscle activity in AB population if there was any activity during the passive movements. To reduce the probability of the voluntary muscle activity, we could apply sham trials to control the anticipatory voluntary muscle activity during the passive movements. Sham trials reduce the perception of the participants of having the actual testing procedure (211).

3.6 Conclusion

In conclusion, the dynamic standing training revealed no more beneficial effects for spasticity reduction versus the static standing training measured by three different outcome measures; self-assessment (VAS), clinical (MAS), and electrophysiologic (EMG). There was no significant muscle activity change in the AB participants. Although we concluded that there was no significant difference between the dynamic and static standing training for spasticity reduction, we believe that more research is needed to provide more evidence to show how dynamic and static standing are different for spasticity management.
Chapter 4: General Discussion

The main purpose of this thesis was to evaluate how two different spasticity physical therapy techniques (vibration and standing training) are effective for spasticity management in individuals with SCI. Physical therapy techniques are the least invasive spasticity management modalities (107,155). They are usually considered as adjuvant essentials in the management of spasticity and are often used to complement pharmacological and surgical strategies (107,155). Vibration and standing training (static and dynamic) are modalities that are simple to apply that could be two effective techniques to manage spasticity. Thus it is important to study the effects of these modalities and the evidence to support them in individuals with SCI.

4.1 Spasticity and Vibration

Vibration techniques such as whole body vibration (WBV) and focal vibration (FV), have been used for spasticity management in individuals with SCI (70,110–112). The systematic review of the studies that used either FV or WBV revealed some but relatively low evidence support for the beneficial effects of vibration on spasticity in individuals with SCI. The vibration frequencies that were used in the reviewed papers support the use of mid range frequencies (at least 50 Hz) of vibration for spasticity management. These are not in the frequency range that might be harmful for humans (70,110,111,113–116). Based on International Standards Organization (ISO) 2631-1 the vibration range of frequencies between 4-12.5 Hz is considered harmful to nervous system (147). Different mechanisms have been proposed as responsible mechanisms for spasticity reduction after focal vibration application. The release of a humoral factor with a general muscle relaxant effect, the activation of afferent nerve generated by vibration procedure, and changes of inhibitory spinal pathways such as increased presynaptic inhibition, and increased reciprocal inhibition have been proposed as the probable responsible mechanism (72).

Although the level of evidence is relatively low for the reviewed papers, most of the studies showed a short-term spasticity reduction by using either focal or whole body vibration in the range of 50-100Hz in individuals with SCI. Future randomized control studies with a move from one phase to the next
can develop a safe and evidence base guideline for the use of whole/focal body vibration for spasticity management.

4.2 Spasticity and Standing Training

Supported standing has been used from early stages of rehabilitation after SCI to late stages of rehabilitation (127). Supported static standing has been shown to improve posture, strengthen antigravity muscles, retrain head and upper limb control, prevent contracture development and reduce spasticity (127,128,130,131). The sensory input changes to the sole of the feet during standing might increase the inhibition of stretch reflex, reduce the motor neuron excitability and reduce spasticity (129,132).

Although we found no difference between the dynamic and static standing training for spasticity reduction after one training session, there are some evidence that showed spasticity reduction after either static or dynamic standing (133,151,183,186). The primary mechanism for spasticity reduction after standing training is not completely understood. The reduction of spasticity after standing training was discussed to be due to the inhibitory neural inputs to muscle activity, load on somatosensory inputs, sustained corticospinal pathways such as vestibulospinal pathways, the load on vertebral column and lower limb joints, and cutaneous information, visco-elastic property changes of spastic muscles, motor activity, and CNS plasticity (134,181,187,188,189,190). However, it is essential to provide more evidence to support more spasticity reduction after dynamic standing compared to the static standing.
4.3 Future Direction

Standing Training

Future randomized control trials with larger sample sizes and multiple sessions of standing training would direct us to a better understanding of spasticity changes after either static or dynamic standing training. The lack of neurophysiologic outcome measures was one of the limitations of the study. H-reflex and T-reflex might be helpful to find out the changes of stretch reflex excitement and a better understanding of muscle spindles roles in spasticity management by static and dynamic standing training. H-reflex and T-reflex do not have the challenge of MVIC measurement and the maximum activity is induced by electrical and mechanical stimulation.

Vibration

Future randomized control trials with larger sample sizes, wide range of FV/WBV will provide a better understanding of effective vibration frequencies that can be used for spasticity management. The best focal vibration application site on the muscles or tendons is still unclear. Future studies with the focus on how focal vibration application on different sites could change muscle spasticity are essential to help with better spasticity management.

Standing and Vibration

The effects of both vibration and dynamic standing on systemic body compositions such as blood circulation to the muscles and muscle oxygenation should be studied more to uncover the unknown mechanisms. We also need to study the functional changes after these two proposed physical therapy techniques in individuals with SCI.
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Appendices

Appendix A  Systematic Review Acceptance Letter

11/27/2013
AJ13441
Sadeghi, Mahsa/Sawatzky, Bonita
The Effects of Vibration on Spasticity in Individuals with Spinal Cord Injury: A Scoping Systematic Review

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Sincerely,
The Editors

Bradley R. Johns, Managing Editor
American Journal of Physical Medicine & Rehabilitation
bjohns@physiatry.org
www.physiatry.org
www.AJPMR.com
Appendix B  Search Strategy

MEDLINE and EMBASE
1  Vibration/ or whole body vibration/
2  vibration*.mp.
3  1 or 2
4  Spasticity/ or spastic paraplegia/ or spastic paresis/
5  (spastic* or spasm*).mp.
6  4 or 5
7  3 and 6
8  Spinal cord injury/
9  (spinal cord injur* or SCI).mp.
10  8 or 9
11  7 and 10

CINAHL
1  (MH “Vibration”)
2  Vibration*
3  1 or 2
4  (MH “Muscle Spasticity”)
5  (Mh “Spasm”)
6  Spasm* or spastic*
7  4 or 5 or 6
8  3 and 7
9  (MH “Spinal Cord Injuries+”)
10  Spinal cord injur* or SCI
11  9 or 10
12  8 and 11

PSYCINFO
1  DE “Vibration”
2  Vibration*
3  Spasm* or spastic*
4  1 or 2
5  3 and 4
6  DE “Spinal Cord Injuries”
7  Spinal cord injur* or SCI
8  6 or 7
9  5 and 8
## Appendix C  CEBM Grades of Recommendation

<table>
<thead>
<tr>
<th>Grade</th>
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<tr>
<td>A</td>
<td>Consistent level 1 studies</td>
</tr>
<tr>
<td>B</td>
<td>Consistent level 2 or 3 studies or extrapolations from level 1 studies</td>
</tr>
<tr>
<td>C</td>
<td>Level 4 studies or extrapolation from level 2 or 3 studies</td>
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<tr>
<td>D</td>
<td>Level 5 evidence or troublingly inconsistent or inconclusive studies of any level</td>
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</tbody>
</table>

 keystroke

"Extrapolations" are where data is used in a situation that has potentially clinically important differences than the original study situation.
Appendix D  Study Recruitment Poster

Does your spasticity decrease while driving The Segway?

"Muscle activity and reflex changes after a dynamic standing condition compared to a static standing condition in spinal cord injury individuals."

Research involves coming to ICORD Blusson Spinal Cord Centre for 2 testing session lasting a maximum of two and half hours each.

We will measure how your clinical spasticity measurement outcome, your muscle activity, and your reflexes will change after a dynamic or static standing condition on the Segway.

You may be eligible to participate if:
You are 18-65 years of age
You have had a spinal cord injury for at least one year
You have spasticity in your lower extremities
OR
You are an able-bodied person without any lower extremities/neurologic disorders

If you are interested in participating or would like more information, Please contact:

Dr. Mahsa Sadeghi: luna.sadeghi@gmail.com or
Dr. Bonita Sawatzky: 604-675-8806 / bonitas@mail.ubc
Appendix E  Consent Form

Participant information and Consent Form

Muscle activity and reflexes changes after a dynamic standing condition compared to a static standing condition in Spinal cord injury individuals

Principal Investigator: Bonita Sawatzky, Department of Orthopaedics/ICORD, Blusson Spinal Cord Centre, email: bonitas@mail.ubc.ca, Tel: 604.675.8806

Sponsor: International Collaboration on Repair Discoveries (ICORD)

1. BACKGROUND

Spasticity is a common consequence of spinal cord injury (SCI) that can have beneficial and adverse effects on individual’s daily life. Spasticity is a condition in which there is an abnormal increase in muscle tone or stiffness of muscle. It has shown that 65-78% of individuals with spinal cord injury (SCI) experience spasticity in their daily life. The adverse effects interfere with activities of daily living, limiting workplace participation, and independence living. Spasticity management is a challenge for clinicians regarding to pharmacologic side effects, short-term effects of physiotherapy methods and invasive surgical procedures.

The Segway Personal Transporter is a novel, practical tool that requires minimal functional ability to operate, and is appropriate for use in individuals with disabilities. Previous studies suggested some benefits on spasticity, fatigue, and pain by using the Segway in individuals with SCI. Some other studies showed spasticity decrease in SCI individuals by using standing training programs.

Our goal is to understand how the muscle activity, reflexes, clinical, and self-assessment spasticity measurements will change after dynamic (riding the Segway) and static standing (standing on a stationary Segway). By studying people with SCI as well as able-bodied individuals we hope to understand how the Segway affects the muscles during these activities.

2. THE INVITATION TO PARTICIPATE

We are inviting males and females between the ages of 18-65yrs old and are in one of the two study group:

1. You are invited if you have spinal cord injury and experience spasticity in your lower extremity muscles.
2. You are invited if you are able-bodied (no disability) to be part of a control group.

3. 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 as well as 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.

You may decide at any time during the study that you may not want to continue. You do not have to provide any reason for withdrawing from the study at any time. However, if data had been collected prior to your withdrawal, it may be used if it contributes to the study.

4. WHAT IS THE PURPOSE OF THE STUDY?

The purpose of this study is to determine the changes in muscle activities and reflexes as well as clinical spasticity measurement changes before and after (immediate and one hour later) a dynamic standing condition compared to a static standing condition in spinal cord injury (SCI) and able-bodied populations.

5. WHO CAN PARTICIPATE IN THE STUDY?

You are eligible to participate in this study if you:
1. Are between 18 and 65 years of age
2. Have a spinal cord injury/disease (>1 year)
3. Have spasticity in your lower extremities at least one month before participating in the study
4. Have the ability to rise from sitting to standing with no more than moderate assistance from one person or using long leg braces
5. Without any autonomic dysreflexia

OR

1. Are between 18 and 65 years of age
2. Have no chronic lower and upper extremities neurologic disorders.
6. WHAT DOES THE STUDY INVOLVE?

The study will take place at the Blusson Pavilion in the ICORD Research Centre, 818 West 10th Ave., Vancouver, BC.

The required time for participation during 2 visits lasts a maximum of 2.5 hours for first session and 2 hours for second session for a total of 4.5 hours in total. The study procedures will be explained for you in basic terms. Any questions regarding the study will be answered before consent is obtained. You will also be asked to provide written consent prior to participation in the study.

All subjects will be made familiar with the Segway and other equipment. The consent form will be reviewed with you and must be signed prior to the beginning of the study. Once the signed consent form is received, forms will be completed regarding information about yourself and your spasticity experience information. During each session you will be shaved for Electromyography (EMG) recordings on your muscles. We will shave a small patch on each of four muscles to put the EMG electrodes on your muscles. The either do the static standing protocol or dynamic standing conditions. These will be randomized for the two days. You will have a 50/50 chance of starting with the dynamic or static standing protocol. On the second visit you will do the other.

Measurements:
Measurements will be taken before, immediately after and one hour later following one of the standing conditions, dynamic or static.

1. We will ask and record your experience of spasticity for muscles in your legs.
2. The skin will be cleaned and EMG stickers will be put on your leg muscles. We will record muscle activity for 30 sec at rest in while you lie on your back.
3. We will then measure, clinically, your muscle tone during a passive motion for knee, hip, and ankle. This is called the Modified Ashworth Score. While this is being done, we will record muscle activity using EMG.
4. The tendon or "T"-reflex will be measured of your Achilles tendon (the muscle on the back of your ankle) with you lying on your stomach. A special reflex hammer will be used to apply a consistent tap and an EMG recording of your muscle will be done of your muscle.

Conditions:
1. Dynamic standing condition:

We will use the ceiling mounted safety harness in the lab as a support to ensure you are safe while you are standing on the Segway. The safety harness is on a special track in the ceiling that allows you to move about the lab in approximately a 10x10 m space. Once you are on the Segway you will learn some simple tasks such as going forward/backwards and turning to become familiar with its use. You will drive the Segway for 20min.
2. **Static standing**

The Segway will be mounted to a firm base so it cannot move. We will use the safety harness in the lab as a support to ensure safety while you are standing on the Segway. You will stand on the Segway for 20 min.

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

We do not anticipate any harm can come to you during the study. The safety harness in the lab will make sure you cannot fall off the Segway. You may experience some redness after the EMG stickers are removed. This will typically disappear in an hour or so. We have completed three studies using the Segway and had no falls or injuries during any of these studies.

8. **ARE THERE ANY BENEFITS TO ME FOR PARTICIPATING?**

Based on previously published research some people with spasticity experience a decrease in spasms for a while after standing still or using a segway. You may or may not have some decrease in spasticity.

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

Your confidentiality will be respected. However, research records and health or other source records identifying you may be inspected in the presence of the Investigator or his or her designate by representatives of Health Canada and UBC Ethic Board for the purpose of monitoring the research. No information or records that disclose your identity will be published without your consent, nor will any information or records that disclose your identity be removed or released without your consent unless required by law.

You will be assigned a unique study number as a subject in this study. Only this number will be used on any research-related information collected about you during the course of this study, so that your identity [i.e. your name or any other information that could identify you] as a subject in this study will be kept confidential. Information that contains your identity will remain only with the Principle Investigator and/or designate. The list that matches your name to the unique study number that is used on your research-related information will not be removed or released without your consent unless required by law.

Your rights to privacy are legally protected by federal and provincial laws that require safeguards to insure that your privacy is respected and also give you the right of access to the information about you that has been provided to the sponsor and, if need be, an opportunity to correct any errors in this information. Further details about these laws are available on request to your study doctor.
Signing this consent form in no way limits your legal rights against the sponsor, investigators, or anyone else, and you do not release the study doctors or participating institutions from their legal and professional responsibilities.

10. 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 Bonita Sawatzky, 604.675.8806.

11. 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, toll free: 1-877-822-8598, email: RSIL@ors.ubc.ca
“Muscle activity and reflexes changes after a dynamic standing condition compared to a static standing condition in Spinal cord injury individuals”

CHECK LIST

- I have read and understood the subject information and consent form.
- I have had sufficient time to consider the information provided and to ask for advice if necessary.
- I have had the opportunity to ask questions and have had satisfactory responses to my questions.
- I understand that all of the information collected will be kept confidential and that the result will only be used for scientific objectives.
- I understand that my participation in this study is voluntary and that I am completely free to refuse to participate or to withdraw from this study at any time without changing in any way the quality of care that I receive.
- I understand that I am not waiving any of my legal rights as a result of signing this consent form.
- I understand that there is no guarantee that this study will provide any benefits to me.
- I have read this form and I freely consent to participate in this study.
- I have been told that I will receive a dated and signed copy of this form.

SUBJECT 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.

______________________________  ________________________
Principle Investigator’s Signature  Date

Printed Name of Principle Investigator.

* I would like a copy of the report from this research  yes _____  no  ________

* I would like to be contacted about further projects  yes _____  no  ________

icord from cells to community: solutions for spinal cord injury  6
818 West 10th Avenue, Vancouver BC V5Z 1M9  •  www.icord.org

Version 1
December 17, 2012

Page 6 of 7

143
Appendix F  Visual Analog Scale (VAS)

Visual Analog Scale (VAS)
Subject ID: ___________ Date: ___________

Dynamic Standing Condition
Place an "X" on the scale to estimate your level of spasticity for each muscle.
Based on your experiences at each time, how much spasticity do you experience:

1. Before the dynamic standing condition

1.a) Quadriceps
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
1.b) Adductors
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
1.c) Hamstrings
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
1.d) Calf muscles
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]

Version 1
December 4, 2012

2. Immediately after the dynamic standing condition

2.a) Quadriceps
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
2.b) Adductors
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
2.c) Hamstrings
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
2.d) Calf muscles
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]

Version 1
December 4, 2012

3. One hour after the dynamic standing condition

3.a) Quadriceps
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
3.b) Adductors
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
3.c) Hamstrings
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]
3.d) Calf muscles
No spasticity [ ] ________________________________________________________________________
The most imaginable spasticity [ ]

Version 1
December 4, 2012
Visual Analog Scale (VAS)

Subject ID ___________________________ Date ____________

**Static Standing Condition**

Place an "X" on the scale to estimate your level of spasticity for each muscle.

Based on your experiences at each time, how much spasticity do you experience:

1. Before the Static standing condition

1.a) Quadriceps
No spasticity [ ] The most imaginable spasticity [ ]

1.b) Adductors
No spasticity [ ] The most imaginable spasticity [ ]

1.c) Hamstrings
No spasticity [ ] The most imaginable spasticity [ ]

1.d) Calf muscles
No spasticity [ ] The most imaginable spasticity [ ]

Visual Analog Scale (VAS)

Subject ID ___________________________ Date ____________

2. Immediately after the Static standing condition

2.a) Quadriceps
No spasticity [ ] The most imaginable spasticity [ ]

2.b) Adductors
No spasticity [ ] The most imaginable spasticity [ ]

2.c) Hamstrings
No spasticity [ ] The most imaginable spasticity [ ]

2.d) Calf muscles
No spasticity [ ] The most imaginable spasticity [ ]

Visual Analog Scale (VAS)

Subject ID ___________________________ Date ____________

3. One hour after the Static standing condition

3.a) Quadriceps
No spasticity [ ] The most imaginable spasticity [ ]

3.b) Adductors
No spasticity [ ] The most imaginable spasticity [ ]

3.c) Hamstrings
No spasticity [ ] The most imaginable spasticity [ ]

3.d) Calf muscles
No spasticity [ ] The most imaginable spasticity [ ]
Appendix G  Intake Form

INTAKE FORM
ID_________________ Date_________________

For each question, fill in the blank or answer the question

Personal characteristics
1. Gender _______Male ________Female
2. Age:____________________

Only Spinal Cord injury individuals (Q 3-14)
3. Year of injury (spinal cord injury individuals):____________________
4. Age at time of injury (spinal cord injury individuals):_____________
5. What is your injury level: __________
6. ASIA Score: A_______B_______C______D________
7. Are you a complete or incomplete spinal cord injury?
   _______Complete _______Incomplete
8. Can you lift your legs against gravity? _______Yes _______No
9. Can you walk without braces, other assistive devices, or people?
   _______Yes _______No
10. Did you have spasticity during the last month?
    _______Yes _______No
11. Do you experience autonomic dysreflexia? _______Yes _______No
12. Do you use any antispastic medication? _______Yes _______No
13. If the answer is ‘yes’ to Q12:
    Please indentify your medication_______________________
14. What is your current mobility device?
    _______Manual wheelchair _______Power wheelchair

December 4, 2012  Page 1 of 2
Version 1
INTAKE FORM

ID_________________  Date_________________

Both Spinal Cord injury and able-bodied individuals (Q 15-16)

15. Have you ever driven the Segway before? ________Yes   ________No

16. If your answer is yes to Q14:

   15. How many times have you driven the Segway?
### Appendix H  Visual Analog Scale (VAS) Results

<table>
<thead>
<tr>
<th>Participants</th>
<th>Dynamic Standing</th>
<th></th>
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<tr>
<td></td>
<td>Before</td>
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<td>Before</td>
<td>After</td>
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<td>Q</td>
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</tbody>
</table>

Abbreviation: Q, Quadriceps; H, Hamstrings; A, Adductors; C, calf Muscle. All the numbers reported are the percentage out of 100%.