DOES FLEXIBILITY TRAINING INFLUENCE THE BIOMECHANICS OF THE SIT-TO-STAND FOR PEOPLE WITH MULTIPLE SCLEROSIS

by

BRADLEY J BOWSER

(Under the Direction of Kathy J. Simpson)

ABSTRACT

For people with multiple sclerosis (MS), compromised neuromuscular control, muscle weakness and balance impairments can reduce their ability to perform activities that require them to rise from a seated position. To date no research has examined how people with MS perform the sit-to-stand (STS) movement. Furthermore, no research has examined the possible influence of flexibility training on the performance of the STS for people with MS. Therefore the purpose of this study was to first determine the biomechanical differences between an MS and non-MS control group for the STS movement and second, to determine whether16 weeks of flexibility training would improve the functional performance and biomechanics exhibited by individuals with MS during the STS. A series of t-tests and repeated measures ANOVAs were used for analysis ($\alpha = 0.05$).

Compared to controls, MS participants exhibited decreased leg extensor strength and increased movement time, maximum trunk flexion, and maximum trunk flexion velocity. Decreased muscle activation of rectus femoris and hamstrings were found for the leg affected more by MS when compared to the leg affected less my MS for both the control and MS groups. No differences for peak magnitudes for joint kinetics were found. However, MS participants did

employ the ‘trunk flexion’ strategy commonly exhibited by other clinical populations with neuromuscular deficits. This strategy is suggested to both reduce joint moment requirements as well as simplify the motor coordination of the sit-to-stand movement.

Following 16 weeks of flexibility training participants increased ROM for knee flexion, ankle dorsiflexion and plantarflexion by 8%, 31%, and 19%, respectively. Additionally, maximum posterior ground reaction force and maximum hip extensor moments occurred approximately 5.5% earlier. No other differences were detected. MS participants were able to increase ROM at the ankle and knee joints and exhibit some biomechanical changes with low-intensity flexibility training.

In summary, ambulatory individuals with relapsing-remitting MS display strength and functional deficits (slower STS movement times) and demonstrate an adaptation that is commonly used to compensate for impaired muscle function, a common symptom of MS. Although no changes to muscle strength and spasticity were detected following 16 weeks of flexibility training, static stretching programs may be beneficial in maintaining current functional levels as well as improve joint ROM in people with MS. To improve flexibility of other joints, increase muscle strength and increase function of the STS movement, that is, movement time, and reduce atypical movement kinematics and kinetics, a more intense flexibility training program may be needed**.**

INDEX WORDS: Sit to stand, Static Stretching, Muscle Strength, Kinematic, Electromyography

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TABLE OF CONTENTS

Page

ACKNOWLEDGMENTS iv

CHAPTER

1 INTRODUCTION 1

Background 1

Premises of the Study 7

Hypotheses 8

Significance 9

Assumptions 10

2 REVIEW OF LITERATURE 11

Multiple Sclerosis 11

Exercise as a treatment 21

Flexibility Training 23

The Sit-to-Stand Movement 27

3 STUDY 1: BIOMECHANICS DURING SIT-TO-STAND OF INDIVIDUALS WITH MULTIPLE SCLEROSIS 41

Abstract 42

Introduction 43

Methods 45

Results 50

Discussion 52

References 62

4 STUDY 2: FLEXIBILITY TRAINING EFFECTS ON PHYSICAL FUNCTION FOR PEOPLE WITH MULTIPLE SCLEROSIS....73
Abstract....74
Introduction....75
Methods....77
Results....82
Discussion....83
References....91
5 SUMMARY AND CONCLUSIONS....102
REFERENCES....105
APPENDIXES
A Kurtzke Expanded Disability Status Scale....115
B Modified Ashworth Spasticity Scale....117
C Participant Information and Health Questionnaire....118
D ANOVA Results of all dependent variables for Study #1....124
E ANOVA Results of all dependent variables for Study #2....128

CHAPTER 1

INTRODUCTION

Background

Multiple sclerosis (MS) is a progressive degenerative disease of the central nervous system (CNS) affecting over 400,000 people in the USA and 2.5 million people worldwide ("Who gets MS?," 2008). Furthermore, each day there are over 200 new cases reported ("Who gets MS?," 2008). Therefore, it is not surprising that Frohman (Frohman, 2003), reported that MS is the most common disabling-nuerological disease of young adults, with the onset of the disease occurring most often during the 2^{nd} - 3^{rd} decades of life (Noseworthy, Lucchinetti, Rodrigues, & Weinshenker, 2000).

MS is thought to be an autoimmune disorder that attacks the myelin, oligodendrocytes and the axons resulting in degeneration and scarring that inhibits nerve conduction resulting in distorted, disrupted, or blocked neural impulses (Frohman, Racke, & Raine, 2006; Smith & McDonald, 2003). Disruption of nerve signals create motor dysfunction which subsequently results in decreased function of the neuromuscular system of the body.

One of the most common neuromuscular deficits found in people with MS is muscle weakness. When compared to a healthy matched control group, individuals with MS were weaker in three of four lower body muscle groups with peak torque being between 16.9% and 25.7% lower for both leg extensors and flexors (Lambert, Archer, & Evans, 2001). Furthermore, in another study, when comparing the cross-sectional area of the muscle, no differences were found between MS and healthy controls, even though those with MS were significantly weaker

(Ng, Miller, Gelinas, & Kent-Braun, 2004). This indicates that muscle weakness in people with MS is most likely due to the inability to effectively use the muscle mass they do have (Rice, Vollmer, & Bigland-Ritchie, 1992).

Along with muscle weakness, muscle spasticity is a common symptom exhibited by those with MS (Barnes, Kent, Semlyen, & McMullen, 2003). Clinically, muscle spasticity can be characterized by decreased power production, force generation, muscle relaxation after a contraction, and increased clumsiness (Kesselring, 2003). In addition to the clinical characteristic of muscle spasticity, it is suggested that lesions on the axons may be inhibiting the supraspinal control of reflex activity and spinal programming causing erratic and uncontrolled movements of the body (Crippa et al., 2004). Subsequently, those who have muscle spasticity have significantly greater levels of disability when compared to those who do not have spasticity (Barnes et al., 2003).

It appears evident that muscle weakness and muscle spasticity will inhibit the ability of MS individuals to perform functional activities. Muscle weakness is likely to reduce the ability to produce the necessary joint moments to produce movement creating mobility limitations. Muscle spasticity is likely to create uncontrolled movements and balance impairments and possibly co-contraction that will only further reduce the ability to produce joint moments. The combination of the two along with many of the other symptoms of MS often lead to mobility limitations, which are reported to be among the greatest challenges and concerns identified by those with MS (M. Finlayson, 2004; Schwid et al., 1997). Unfortunately, an inability to perform functional activities often results in a more sedentary lifestyle. It has been reported that MS individuals are less physically active than healthy matched controls (Kent-Braun et al., 1997). A more sedentary lifestyle potentially increases health problems, as such a lifestyle places people

with MS at greater risk for coronary heart disease, diabetes, and obesity (White & Dressendorfer, 2004). Additionally, a sedentary lifestyle is likely to only increase disuse creating greater muscle weakness.

The consequences of MS influence functional movements involved in several activities of daily life are not clearly understood. Of all the functional activities people perform each day walking is the most studied among people with MS. In association with the decreased walking speed, it is reported that those with MS have shorter stride length, spend more time in double support, and have decreased range of montion (ROM) at the hip, knee, and ankle joint (Benedetti et al., 1999; Chung, Remelius, Van Emmerik, & Kent-Braun, 2008; Crenshaw, Royer, Richards, & Hudson, 2006; Gehlsen et al., 1986; Holden, Gill, & Magliozzi, 1986; Martin et al., 2006; Morris, Cantwell, Vowels, & Dodd, 2002; Rodgers et al., 1999). It is evident for the functional activity of walking people with MS display several limitations.

Another activity of equal importance and considered not only a prerequisite for walking, but also for most functional activities, is the sit-to-stand (STS) (Kralj, Jaeger, & Munih, 1990). The STS is an important element of several movements used in daily life and is likely to be very challenging for MS individuals. It is a movement that encompasses the transition of the body from a stable seated position to a less stable upright posture requiring considerable neuromuscular coordination, balance, and leg extensor strength (Inkster & Eng, 2004; Mak, Levin, Mizrahi, & Hui-Chan, 2003) all of which are commonly compromised in people with MS (Noseworthy et al., 2000).

Although no published research to date has examined the STS movement in people with MS, previous investigations have examined the STS movement in other clinical populations with similar symptoms and/or disabilities as people with MS. The most uniform difference in STS

performance found between these clinical populations and healthy matched controls is a significant increase in overall movement time (MT) in the clinical population (Camargos, Rodrigues-de-Paula-Goulart, & Teixeira-Salmela, 2009; Inkster & Eng, 2004; Mak & Hui-Chan, 2005; Mourey, Grishin, d'Athis, Pozzo, & Stapley, 2000). A slower MT in these clinical populations is most commonly attributed to lower extremity muscle weakness. In particular, leg extensor strength is reported to be the most influential variable affecting an individual's ability to perform the STS movement (Bernardi et al., 2004; Inkster, Eng, MacIntyre, & Stoessl, 2003; Papa & Cappozzo, 2000). Previous research has indicated that increased muscle weakness reduces the ability to produce the required leg extensor moments used in performing the STS movement. Inkster and Eng (2004) found that knee extensor (KE) and hip extensor (HE) moments were up to 20% lower in Parkinson's patients when compared to healthy controls. Subsequently, in order to overcome the inability to produce sufficient KE moments, compensatory movement strategies are often implemented (Doorenbosch, Harlaar, Roebroeck, & Lankhorst, 1994). When comparing a flexed trunk strategy to a non-flexed trunk strategy in an elderly population, Doorenbosch et al. (1994) found the flexed trunk strategy resulted in significant decreases in maximum KE moments when compared to the non-flexed strategy. This may explain why a significant increase in trunk flexion during STS movements has been consistently found in clinical populations with leg extensor weakness (Alexander, Schultz, & Warwick, 1991; Inkster & Eng, 2004; Kerr, White, Barr, & Mollan, 1997; Papa & Cappozzo, 2000; Schultz, Alexander, & Ashton-Miller, 1992). Furthermore, it is suggested that increased trunk flexion also serves to place the body's center of mass (COM) closer to the new base of support increasing stability in those who have balance impairments. Parkinson's patients are

reported to place their body COM up to 17% in a more anterior direction during STS than healthy controls in an effort to increases their stability (Inkster & Eng, 2004).

With no published research examining the STS for people with MS, the biomechanics and movement strategies used by MS individuals is not known. Therefore, the purpose of the first study was to determine the biomechanical differences between an MS and non-MS control group for the STS movement.

In an effort to increase physical activity, improve physical function and ameliorate some of the neuromuscular deficits exhibited by people with MS, several researchers have designed both aerobic and resistance training exercise programs with the benefits of these exercise programs including increased cardiorespiratory fitness (Mostert & Kesselring, 2002; Petajan et al., 1996; Ponichtera-Mulcare, Mathews, Barrett, & al., 1997; Rodgers et al., 1999), increased muscle strength and endurance (DeBolt & McCubbin, 2004; Husted, Pham, Hekking, & Niederman, 1999; Mostert & Kesselring, 2002; White et al., 2004), reduced systemic fatigue (Husted et al., 1999; Mostert & Kesselring, 2002; White et al., 2004), improved mood (Mostert & Kesselring, 2002), and improved functional performance of walking (Gutierrez et al., 2005). However, traditional exercise programs may not be feasible for people with MS who have increased heat sensitivity, excess fatigue, do not have access to exercise equipment, have high levels of MS-related disability, or have orthopedic/other limitations.

Therefore, another type of low intensity exercise requiring less exertion that is cost effective, relatively easy, and that may provide a viable alternative to traditional exercise programs is flexibility training Only two studies exist in which any flexibility training or movements requiring flexibility were part of the physical training for people with MS. Debolt and McCubbin (1997) found that 5-10 minutes of total body flexibility exercise plus resistance

training 3 times a week for 16 weeks resulted in no significant improvements in balance for individuals with MS. However, Husted, et al. (1999) found that an hour of tai chi, which often includes stretching exercises, performed twice a week for 8 weeks increased walking speed and hamstring flexibility.

Engaging in a flexibility-training program involving all of the lower extremity muscles should improve flexibility, reduce spasticity and potentially improve strength (Kokkonen, Nelson, Eldredge, & Winchester, 2007; Zhang et al., 2002). Improvements in these components should then result in enhanced performance outcomes (e.g., time required to complete a movement) of functional activities, specifically rising from a chair. Improved performance is expected for several reasons for the STS following flexibility training. First, KE muscle strength, or maximum KE moment, is related to the ability to rise from a chair without using compensatory actions of other segments, e.g., rapid trunk flexion/extension (Inkster et al., 2003). For healthy populations, stretching programs have resulted in increased leg extension strength (Kokkonen, Nelson, Eldredge, & Winchester, 2007). Furthermore, flexibility training has been reported to reduce muscle spasticity of clinical, non-MS populations (Zhang et al., 2002). For people with MS, flexibility training should have a similar effect on spasticity. It is likely that reducing muscle spasticity will lead to increased stability. Furthermore, reducing muscle spasticity may reduce the amount of co-contractions that may occur during the STS movement which will subsequently increase the ability to produce great joint moments. We surmise that increasing muscle strength and reducing muscle spasticity will improve the functional ability to perform the STS. Therefore, the purpose of the second study was to determine if 16 weeks of flexibility training would improve the functional performance and biomechanics exhibited by individuals with MS during the STS.

Premises of the studies

As mentioned previously, leg extensor strength is the most influential variable affecting an individual's ability to perform the STS movement. We surmise that MS participants will be able to perform the STS movement, but will use an adaptive strategy to compensate for their reported muscle weakness. The strategy commonly used by clinical populations with muscle weakness is a 'flexed trunk' strategy (Alexander et al., 1991; Inkster & Eng, 2004; Kerr et al., 1997; Papa & Cappozzo, 2000; Schultz et al., 1992). Prior to the instant of seatoff, participants who use this strategy display a greater degree of trunk flexion. Following seatoff, the trunk remains in the flexed position until the knees fully extend, at which time they begin to extend their trunk. In healthy populations, the trunk only slightly flexes prior to seat off and knee extension and trunk flexion begin simultaneously at seatoff resulting in one smooth motion.

There are several potential benefits to the flexed trunk strategy for people with MS who have muscle activation deficiencies and leg extensor weakness. First, the flexed trunk strategy is suggested to reduce the KE moment requirement needed for the STS movement. In addition to observing that the flexed trunk strategy resulted in decreased KE moments, Doorenbosch et al., (1994) reported that the decreased KE moment was accompanied by decreased muscle activity of the rectus femoris. Furthermore, as was mentioned before, Inkster et al., (2003) found that individuals with Parkinson's disease who used a flexed trunk strategy displayed decreases in both KE and HE moments while performing the STS.

Second, all else equal, greater trunk flexion displacement causes higher gravitational angular work done to the trunk, which, in turn, results in the higher, anterior-directed linear velocity of the COM of the body. Thus, the line of gravity can be shifted farther forward within the boundaries of the base of support, thereby increasing stability during the post-seatoff

(postSO) phase. Balance impairments are a common symptom and MS and using the flexed trunk strategy is likely create more stability during the STS.

It was previously stated that traditional exercise treatment including aerobic and resistance training programs have reported increased cardiorespiratory fitness, increased strength and endurance, reduced fatigue and improved functional performance for people with MS. Although flexibility training alone has not been used as an intervention program for MS individuals, increases in leg extensor strength and decreases in muscle spasticity are reported to occur in other non-MS populations (Kokkonen et al., 2007; Zhang et al., 2002). For people with MS, flexibility training should have a similar effect on spasticity. With both leg extensor strength and muscle spasticity playing a major role in functional ability, it is likely that improvement made in these areas from flexibility training will also improve STS performance. More specifically, by increasing leg extensor strength through flexibility training it is likely that MS participants will not have to use the flexed trunk strategy to reduce the KE moment requirements for the STS. Furthermore, reducing muscle spasticity will likely result in a more controlled movement which will also reduce the possibility of needing to move the COM of the body in a more anterior direction for increased stability. Decreasing muscle spasticity may also reduce the possibility of co-contractions allowing the agonists to produce greater extensor moments.

Hypotheses

For the first study it was hypothesized that the MS group compared to a control group would demonstrate:

1. Decreased leg extensor strength.
2. Increased STS MT.
3. Decreased KE and HE moments during the STS.

4. Decreased EMG muscle activity of the KE and HE during the STS.
5. Increased trunk flexion displacement and trunk flexion velocity during the STS.
6. Increased anterior displacement of the body's COM during the STS.

For the second study it was hypothesized that following 16 weeks of flexibility training of the lower extremity people with MS would demonstrate:

1. Decreased lower extremity muscle spasticity.
2. Increased ROM at the ankle, knee, and hip.
3. Increased leg extensor strength.
4. Decreased STS MT.
5. Increased KE and HE moments during the STS.
6. Increased EMG muscle activity of the KE and HE during the STS.
7. Decreased trunk flexion displacement and trunk flexion velocity during the STS.
8. Decreased anterior displacement of the body's COM during the STS.

Significance

MS is an extremely debilitating, potentially life-ending disease that occurs relatively early in life (Noseworthy et al., 2000). As MS is a progressive musculoskeletal disease that can lead to increased loss of voluntary muscle control, hence, loss of mobility and movement, it is crucial that we determine how muscle strength and control can be maintained for as long as possible. Due to neuromuscular problems caused by many of the symptoms of MS, such as muscle weakness, fatigue, muscle spasticity, and impaired balance, people with MS are often faced with mobility limitations (Frohman et al., 2006). These limitations are of great concern for people with MS and often lead to a more sedentary lifestyle. A sedentary lifestyle not only

further increases disability it can also introduce secondary illnesses (White & Dressendorfer, 2004).

As a prerequisite to being able to perform gait and other functional activities (Kralj et al., 1990), the ability to complete the STS movement is paramount to increasing mobility. Determining how performers' biomechanics are affected by MS will provide a foundation for developing more successful rehabilitation and/or exercise programs aimed at improving functional capacity and slowing down the rate of disability. Furthermore, traditional exercise programs (aerobic and resistance training) have shown positive outcomes while increasing physical activity. However, traditional exercise programs may not be feasible for people with MS who do not have access to exercise equipment, have increased heat sensitivity, have high levels of MS-related disability or have orthopedic/other limitations. It is therefore imperative to also assess alternative exercise training programs such as flexibility training that may result in increased physical activity as well as improve functional performance of the STS movement.

Assumptions

The first assumption is that, when using the inverse dynamics model for calculating net muscle moments the body is modeled as a set of rigid, linked-segments connected by frictionless joints (Winter, 2004). Second, it is assumed that the values of the estimated segmental moments of inertia, mass magnitudes and center of mass locations are valid. The third assumption is that the healthy, matched control group is an accurate sample of the corresponding typical healthy population.

CHAPTER 2

REVIEW OF LITERATURE

The literature review consists of three major topics broken down into three sections. The first section will be a discussion of what MS is, who it affects, and how it influences functional ability. In addition to describing the pathology and symptoms of MS, the first section will also examine exercise interventions aimed at improving functional performance for those with MS. The second section will define flexibility training and outline the reported benefits of this type of training. The second section will also discuss the implications of implementing flexibility training to improve functional ability for those with MS. The third and final section of this chapter will describe the biomechanics of the STS movement and the mechanics used to perform it. As there is no research to date specifically examining the mechanics of STS being performed by those with MS, a description of the STS performed by those with similar functional limitations will be described.

Multiple Sclerosis

MS is a progressive degenerative disease of the central nervous system (CNS) diagnosed in an estimated 2.5 million people worldwide ("Who gets MS?," 2008). A diagnosis of MS usually occurs between the ages of 25 and 65 years (Martinelli, Rodegher, Moiola, & Comi, 2004) with 2-3 times more women being diagnosed with the disease than men (Pelfrey, Cotleur, Lee, & Rudick, 2002). In addition to the higher prevalence in women, there is a strong genetic predisposition with a probability of diagnosis with MS being 15 times higher when a sibling or parent has already been diagnosed with the disease (Latash, 2008). Even though MS was

officially classified as a disease in 1873 by the medical community ("The History of MS," 2003), neither the cause nor cure for MS is yet known. However, the pathology of the disease has been well documented.

MS pathology and symptoms

MS is characterized by the formation of acute inflammatory, demyelinating lesions typically found on the white matter and affects the myelin, oligodendrocytes, and axons of the CNS (Smith & McDonald, 2003). MS is an autoimmune disorder where autoreactive T-cells attack the myelin sheath and the myelinating cells, the oligodendrocytes. The attacks are often referred to as exacerbations and are accompanied by both inflammation and a demyelination of the axons. The myelin sheath acts as an insulator to the axon much like plastic around a copper wire. When the myelin is destroyed by the T-cells, the insulation of the axon is lost and is replaced by plaque/lesions. Both the lesion itself and the inflammation caused by the lesions inhibit nerve conduction (Frohman et al., 2006) which may result in partial or complete nerve conduction block (Smith & McDonald, 2003). Additionally, because the T-cells also attack the oligodendrocytes, the ability to effectively repair damage caused by the lesions is inhibited.

The resulting symptoms caused by the lesions in the CNS vary greatly among those diagnosed with MS and largely depend on the area of the CNS that is attacked. Lesions on the optic nerve often result in blurred vision, a dull aching of the eye, impaired vision acuity and possibly acute blindness. Unilateral deafness may occur when the olfactory and optic nerves are affected. Demyelination within the brain can cause losses in cognition. When the axons of the brainstem are affected, discoordinated limb movements, balance impairments, muscle weakness, and numbness may result (Latash, 2008).

MS diagnosis

Because the symptoms of MS vary among individuals, it is often difficult to quickly diagnose an individual with the disease. The classical criteria for diagnosing MS requires at least two separate exacerbations in time with the presence of lesions at two necessarily separate sites in the CNS. The assesment can be done completely with clinical tests, but most neurologists prefer to confirm their diagnosis using magnetic resonance imaging (MRI) (McDonald, Fazekas, & Thompson, 2003). The McDonald criteria which "incorporates MRI to demonstrate multiple areas of involvement and also involvement over time with the appearance of new enhancing lesions," has been suggested to aid in earlier diagnosis (Murray, 2006). Earlier diagnoses of MS can lead to earlier decisions on therapy and possible reductions in the severity of symptoms (Murray, 2006).

In order to standardize the technology used and more accurately diagnose MS, a consensus-building effort was conducted among leading MS specialists worldwide (Lublin & Reingold, 1996). Subsequently, the consensus among medical professionals and clinicians categorized MS into 4 disease types (Figure 2.2):

1. Relapsing-remitting MS- Clearly defined disease relapses with full recovery or with sequelae and residual deficit upon recovery. The periods between disease relapses are characterized by a lack of disease progression
2. Primary progressive MS- Disease progression from onset with occasional plateaus and temporary minor improvements permitted. These patients have no acute exacerbations.

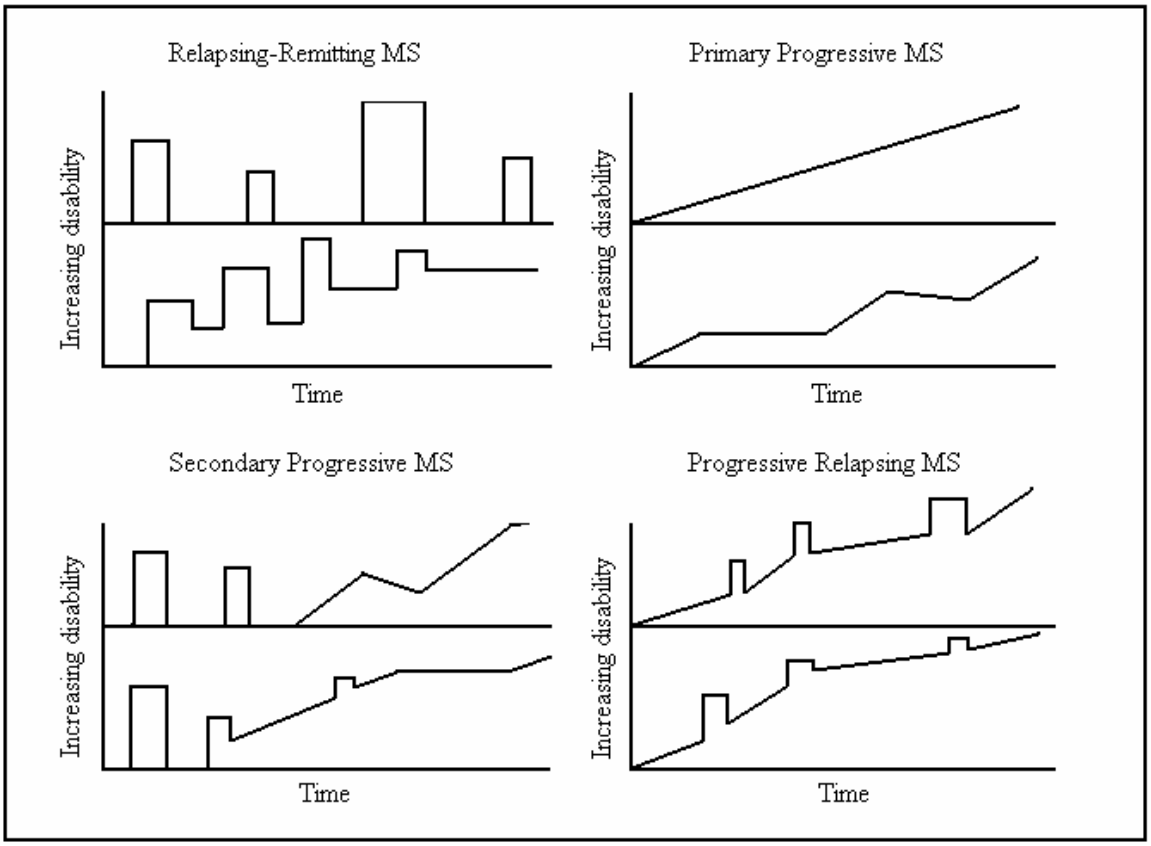

Figure 2.1 The four clinical courses of MS (modified from (Lublin & Stephen, 2003).

3. Secondary progressive MS- Initial relapsing-remitting disease course followed by steady progression of disease, with or without occasional superimposed relapses, minor remissions, and plateaus. Between relapses, steady progression of disability is seen.
4. Progressive relapsing MS- Disease progression from onset, has no clear acute relapses, with or without full recovery during the disease course. The periods between relapses display continuing progression (Lublin & Reingold, 1996; Lublin & Stephen, 2003).

Of the 4 types of MS, 85% of those diagnosed have relapsing-remitting MS, making it the most common. The remaining 15% have one of the two progressive-at-onset forms. Within the first 10 years of being diagnosed, 50% of those with relapsing remitting MS will develop secondary progressive MS (Weinshenker et al., 1989). Understanding the MS type is essential in providing accurate treatment and possible rehabilitation programs for those with MS (Lublin & Stephen, 2003).

Disability in MS

In addition to knowing the type of MS, it is also important to know the level of disability displayed by those with MS. In order to determine the level of disability exhibited by those with MS, John Kurtzke developed the Expanded Disability Status Scale (EDSS) (Kurtzke, 1983). The EDSS is one of the most widely used clinical scales to measure disability (Rudge, 2003). It is a scale that ranges from 0-10 with 0 being defined as no disability and 10 as death due to MS. Furthermore, it can be broken down into two categories a score from 0 to 6 is considered ambulatory and greater than 6 to 10 is non-ambulatory. For a more detailed description of each of the scores and how they can be broken down into other sub-categories see appendix A.

Disability in MS individuals is a direct result of axonal degeneration and the lesions left on the axons as was discussed earlier. Complete or partial nerve conduction block inhibits the neuromuscular system which often compromises the motor functioning of a person. The neuromuscular deficits for those with MS come in the form of muscle weakness, muscle spasticity, fatigue, and balance impairments. With compromised motor function caused by the neuromuscular deficits, simple activities of daily living such as walking and rising from a chair are often difficult for those with MS.

Muscle weakness

Muscle weakness is a hallmark symptom for those with MS (White & Dressendorfer, 2004). When compared to a healthy matched control group, individuals with MS were weaker in three of four lower body muscle groups with peak torque being 16.9%, 25.7%, and 20.8% lower for non-dominant extensors, dominant flexors, and non-dominant flexors respectively (Lambert et al., 2001). Lambert et al. suggests these differences were a result of MS individuals being unable to effectively activate the muscle mass they have. Although they provided no empirical evidence of this suggestion, others have found that those with MS display a large increase in force when a supra-maximal electrical stimulus is imposed during a maximal voluntary isometric contraction (Rice et al., 1992). Additionally, Rice et al. found that the motor unit firing rates in those with MS were significantly reduced ($p < 0.05$) during maximum voluntary isometric contractions when compared to healthy controls. Furthermore, when examining the cross-sectional area of the muscle, no differences were found between MS and healthy controls, even though those with MS were significantly weaker (Ng et al., 2004). However, in another study MS individuals showed 30% less fat free cross-sectional area (CSA) in skeletal muscles. Additionally, type II fibers showed greater atrophy in those with MS, and all fibers displayed a

markedly lower CSA (Kent-Braun et al., 1997). These findings indicate that it is likely a combination of both an individual with MS' inability to effectively use the muscle mass they do have and a decreased CSA, resulting in greater muscle weakness.

Not only has it been found that those with MS have greater muscle weakness than healthy controls, individuals with MS have also displayed decreased power and rate of force development (Chung et al., 2008; Ng et al., 2004). Ng et al. (2004) attributes the decline in the rate of force development to decreased central motor function. A battery of tests was used to determine the central motor function including foot tap speed, rapid voluntary contractions, and a central activation ratio. All of these tests were significantly slower in MS individuals indicating that a decreased central motor function may cause muscle weakness (Ng et al., 2004).

Muscle Spasticity

Along with muscle weakness one of the most common neuromuscular deficits exhibited by those with MS is muscle spasticity (Barnes et al., 2003). Clinically, muscle spasticity can be characterized by decreased power production, force generation, muscle relaxation after a contraction, and increased clumsiness (Kesselring, 2003). In addition to the clinical characteristic of muscle spasticity, it is suggested that lesions on the axons may be inhibiting the supraspinal control of reflex activity and spinal programming causing eratic and uncontrolled movements of the body (Crippa et al., 2004). Subsequently, those who have muscle spasticity have significantly greater levels of disability when compared to those who do not have spasticity (Barnes et al., 2003). It is likely that reducing the voluntary movements of the body will decrease functional ability making it difficult to perform activities of daily living. For people with MS, muscle spasticity is more markedly affected in the legs than the arms (Kesselring, 1997) indicating that activities involving the legs such as walking and rising from a chair may pose the biggest

challenge. A common assessment tool for MS is the Ashworth spasticity scale (Appendix B) (Ashworth, 1964). Although spasticity can change throughout the day and between days, this scale is reported to be reliable and widely used among clinicians (Bohannon & Smith, 1987).

Fatigue

The two most common neuromuscular deficits, muscle weakness and spasticity, are thought to be highly intertwined with the most common symptom of MS which is fatigue. Over 85% of those with MS report feeling fatigued (Vercoulen et al., 1996). Fatigue is defined as an overwhelming sense of being tired, having no energy, and a feeling of exhaustion greater than is normally felt during a given activity (Kesselring & Thompson, 1997). Fatigue felt by those with MS goes beyond the fatigue felt by a healthy population. Typically, healthy individuals feel fatigue following continuous strenuous activity. However, those with MS often suffer from fatigue during the regular course of the day without performing any type of strenuous activities. This is evidenced in the literature that has found significantly greater levels of fatigue in MS patients performing the same tasks as non-MS patients (Chung et al., 2008; Crenshaw et al., 2006; Ng, Miller, & Kent-Braun, 1997). Ng et al. (2004) suggest that there are several factors that may influence fatigue in MS individuals including decreased central motor function and depression. Another factor may be co-activation of agonists and antagonist muscles where the agonist has to fight to overcome the antagonist (Ng et al., 1997). Muscle spasticity is also likely to increase fatigue due to the constant over-activation of tonic stretch reflexes (muscle tone) and exaggerated tendon jerks.

One last explanation for increased fatigue in MS individuals may be a change in skeletal muscle. Research indicates that skeletal muscle fibers in those with MS are altered in the direction of disuse (Kent-Braun et al., 1997). Type II fibers are reported to display greater

atrophy in those with MS. When specifically examining the CSA of the tibialis anterior muscle in MS individuals, the results were similar or worse than the disuse models (Kent-Braun et al., 1997). Additionally, Kent-Braun et al. (1997) found that the MS group was less physically active than the healthy control group. This poses a problem for those with MS. As was mentioned previously in the introduction, a more sedentary lifestyle can lead to secondary illness such as coronary heart disease, diabetes, and obesity which may further exacerbate the problem (White & Dressendorfer, 2004).

Balance

It is likely that individuals with MS who have muscle weakness, muscle spasticity, and increased fatigue will exhibit trepidation over many types of physical activity due to their mobility limitations. These limitations are among the greatest challenges and concerns identified by those with MS (M. Finlayson, 2004; Schwid et al., 1997). It has been reported that over 50% of those diagnosed with MS will experience a fall (M. L. Finlayson, Peterson, & Cho, 2006) with the primary cause being attributed to a loss of balance due to inadequate muscle strength and flexibility (Cattaneo et al., 2002). It is reported that the erratic body movements associated with muscle spasticity can also be attributed to the loss of balance (Soyuer, Mirza, & Erkorkmaz, 2006).

Impaired balance may explain some of the trepidation felt by those with MS concerning participating in physical activity. When using a force plate to measure center of pressure excursion while standing, it was discovered that MS individuals had greater excursions than the control group. These findings indicate that those with MS have less ability to maintain balance (Karst, Venema, Roehrs, & Tyler, 2005). During seated balance tests, MS individuals display greater sway patterns than healthy controls (Lanzetta, Cattaneo, Pellegatta, & Cardini, 2004).

Frzovic et al. (2000) concluded that balance impairments increase the difficulty of functional activities after finding that MS individuals showed significant impairments in single leg stance, self perturbation functional reach test and arm raise tests, step tests, and an external perturbation test.

Functional ability

It appears evident that muscle weakness, muscle spasticity, increased fatigue, and impaired balance will inhibit the ability of MS individuals to perform functional activities. Although no research to date has examined the STS task, many researchers have examined the functional performance of walking. When compared to healthy matched controls, individuals with MS have a slower self-selected walking speed (Gehlsen et al., 1986; Holden et al., 1986; Morris et al., 2002; Rodgers et al., 1999). Considering the EDSS scale examines gait ability as one of the determinants of disability, it seems obvious that those who have a higher EDSS score will be slower. However, research indicates that all levels of the ambulatory category of EDSS (≤ 6) appear to demonstrate slower walking speeds. In three separate studies the average EDSS was 4, 3, and <2, all showing that MS individuals walked slower (Chung et al., 2008; Crenshaw et al., 2006; Martin et al., 2006). In association with the decreased walking speed, it is reported that those with MS have shorter stride length, spend more time in double support, and have decreased ROM at the hip, knee, and ankle joint (Benedetti et al., 1999; Chung et al., 2008; Crenshaw et al., 2006; Gehlsen et al., 1986; Holden et al., 1986; Martin et al., 2006; Morris et al., 2002; Rodgers et al., 1999). Benedetti et al. (1996), who studied minimally impaired MS individuals (EDSS ≤ 2), attributed differences in walking speed, stride length, double support time, and joint ROM to a protective strategy that favors stability and balance at the expense of speed. A decreased ability to walk may be one of the primary factors influencing the possible

trepidation of performing physical activity by those who have MS. Furthermore, the STS is a prerequisite to walking and is one of the most functionally demanding activities of daily living (Kralj et al., 1990; Riley, Schenkman, Mann, & Hodge, 1991). A decreased ability to stand will only increase the likelihood of a sedentary lifestyle.

Exercise as a treatment

Although many pharmacological agents have been shown to improve many of the symptoms of MS (Kita & Goodkin, 2000; Paisley, Beard, Hunn, & Wight, 2002), several symptoms including muscle weakness, muscle spasticity, and fatigue lie within the continuum of adaptive responses to disuse (Kent-Braun et al., 1997). It therefore seems reasonable that using exercise interventions as a rehabilitative tool may enhance physical functioning and promote increased physical activity of those with MS. Literature suggests that exercise interventions improve functional ability and reduce the severity of many symptoms associated with MS and will be discussed hereafter.

Aerobic training

Individuals with MS who engaged in 30 minutes of aerobic training 3 times a week for 24 weeks using hand and leg powered cycle ergometers were able to increase their VO_{2MAX} by 15%. However, these individuals were unable to improve their speed and joint ROM during walking, and actually decreased their walking speed and joint ROM (Rodgers et al., 1999). Other data that implemented aerobic training for an MS group used arm and leg ergometry showed similar results reporting 15 weeks of training improved VO_{2MAX} by 21% compared to only a 5% increase for an MS control group who did not do the aerobic training (Petajan et al., 1996). Although Petajan et al. (1996) did not assess gait, they found improvements in upper extremity strength (+17%) lower extremity strength (+11%), and decreases in depression, anger, and

fatigue that may lead to improved gait patterns. Interestingly, a shorter 4 week aerobic training program with 30 minute sessions 5 times a week also displayed improvements in physical health in MS individuals. These improvements included decreased fatigue (-14%), increased physical activity (+17%), and aerobic threshold (12%) (Mostert & Kesselring, 2002). Although there appear to be few changes improving the ability to perform activities of daily living, many of the health benefits reported by these studies will likely aid in increasing physical activity and reduce the likelihood of developing secondary illnesses associated with a sedentary lifestyle. Increasing physical activity in MS individuals is likely to carry over to improved performance of many functional activities.

Resistance training

In addition to improving general physical health parameters, resistance training has also been shown to improve functional performance in MS individuals. Resistance training 30 minutes, 2 times a week for 8 weeks not only improves knee extension strength (+7.4%), plantar flexion strength (+52%), and reduced self-reported fatigue, but also improved functional performance increasing the number of steps (8.7%) during a 3 minute step test in MS individuals (White et al., 2004). Similar results were found for those with MS when participating in resistance training 3 times a week for 12 weeks. MS individuals demonstrated increased muscle strength in the quads, hamstrings, biceps, and triceps (Kraft, Alquist, & deLateur, 1996b). Additionally, using the same resistance training protocol, MS individuals significantly increased walking velocity, and decreased stair climb time, up and go time, and self reported disability (Kraft, Alquist, & deLateur, 1996a). Furthermore, Gutierrez et al. (2005) reported that 8 weeks of lower-body resistance training resulted in decreased fatigue and increased muscle strength in MS individuals. Functionally, the researchers found significant ($p < 0.05$) increases in % time in

swing phase, increases in step and stride lengths, decreases in % stride time in stance, decreases in double support phases, and decreased duration of the double support phase during walking in MS individuals (Gutierrez et al., 2005). For MS individuals, the benefits gained in strength training not only improve general physical health parameters such as reducing fatigue and strengthening muscle, but also result in improved ability to perform activities of daily living such as walking and climbing stairs.

Flexibility Training

Flexibility is defined as the ability to move a joint or series of joints through a relatively painless range of motion (ROM) (Gleim & McHugh, 1997). There are three main factors that determine flexibility/joint ROM - joint structures, muscle length, and muscle elasticity (Liebesman & Cafarelli, 1994). Although changing the joint structure cannot be manipulated without surgery, the elasticity and length of the muscle can be altered to increase ROM through regular stretching. Figure 2.3 displays the relationship between the muscle length and amount of stretch placed on the muscle. Over time in humans, repeated stretches lasting longer than 10 seconds will not only add sarcomeres to the end of the muscle, which will lengthen the muscle during a stretch, it can also reset the sensitivity of the muscle spindle, shifting the point of rupture further to the right (Figure 2.3), allowing the joint to go through a greater ROM (MacIntosh, Gardiner, & McComas, 2006).

There are three main types of stretching used in flexibility training. The first, and possibly the most widely known, is static stretching which is characterized by moving gradually to the maximum joint ROM for a given joint or series of joints, then holding it there between 10-60 seconds. Static stretching can be performed either passively (someone moving the body segments for you) or actively (moving the segments using gravity, pulling on other body

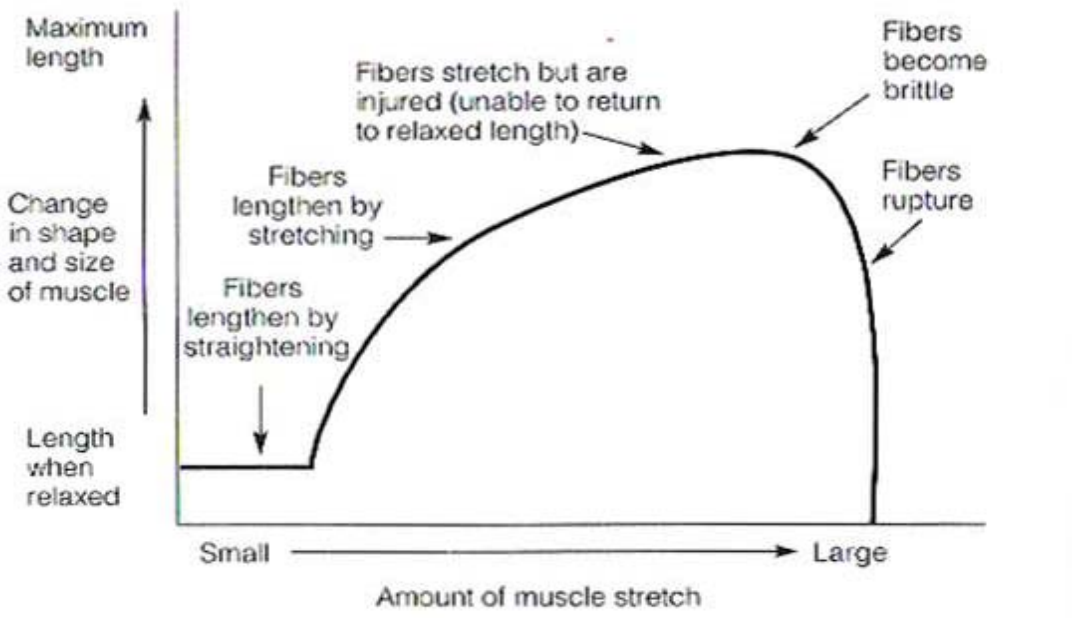

Figure 2.2. The relationship between muscle length and the amount of muscle stretch

segments, activating the un-stretched muscles placing the body into the desired position, or any combination of the three) (Gleim & McHugh, 1997). The second type of stretching is ballistic stretching, which is a dynamic muscle stretch that implements a bouncing motion to reach and often go slightly go beyond the ROM reached during a static stretch (Liebesman & Cafarelli, 1994). The third type of stretching is called proprioceptive neuromuscular facilitation (PNF). There are several different types of PNF stretching, but the general idea is to combine a static stretch with muscle contractions of the stretched muscle (Liebesman & Cafarelli, 1994). These stretches can also be performed passively or actively and usually are held for 10-60 seconds. All three types of stretching have been reported to increase ROM.(Bacurau et al., 2009; Feland, Myrer, Schulthies, Fellingham, & Measom, 2001; Ferber, Osternig, & Gravelle, 2002; Kokkonen et al., 2007; Woolstenhulme, Griffiths, Woolstenhulme, & Parcell, 2006; Yuktasir & Kaya, 2009; Zhang et al., 2002).

Traditionally the purpose of flexibility training has been to improve ROM, but there are other reported benefits of stretching. In addition to increasing hamstring flexibility by 21.3%, 20 minutes of static stretching of the hamstring muscles 5 days a week for 3 weeks is reported to increase eccentric peak torque at 60°/s and 120°/s by 8.5% and 13.5% respectively during isokinetic knee flexion exercises (Worrell, Smith, & Winegardner, 1994). Peak concentric torque at 120°/s also increased 11.2% during isokinetic knee flexion (Worrell et al., 1994). More recently 40 minutes of static stretching of the lower extremity muscles 3 times a week for 10 weeks has shown significant increases in combined lower extremity flexibility (18.1%), power performance (standing long jump 2.3%; vertical jump 6.7%; 20 meter sprint 1.3%), knee flexion strength (15.3%), knee extension strength (32.4%), knee flexion endurance (30.4%), and knee extension endurance (28.5%) for healthy college age participants when compared to healthy

college age participants who performed no stretches during the same time period (Kokkonen et al., 2007).

Another major finding that could potentially benefit MS patients is that static stretching significantly reduces muscle spasticity in neurologically impaired patients (Zhang et al., 2002). It is likely that the reported stiffness (Foran, Steinman, Barash, Chambers, & Lieber, 2005) and hyper excitability (Kita & Goodkin, 2000) of a spastic muscle is reduced as a result of the "resetting" of the sensitivity of the muscle spindle from repeated static stretches. Furthermore, reducing the hyper excitability and stiffness of a muscle may in turn result in a less spastic muscle.

A few studies examined the influence of flexibility training on improving functional performance and reducing symptom severity in MS individuals. One study that combined 30 minutes of home based resistance training accompanied with 5-10 minutes of whole body stretches performed 3 times a week for 8 weeks found leg extensor power increased, but balance was unchanged for those with MS (DeBolt & McCubbin, 2004). Tai chi instruction, which incorporates improving flexibility into its training, is reported to increase walking speed and hamstring flexibility in MS individuals (Husted et al., 1999). However, both of these studies did not directly examine the influence of flexibility training making it difficult to ascertain whether the stretching was responsible for the improvements.

Although research examining the affects of flexibility training on an MS population is limited, it is likely that the reported benefits of stretching in other populations, which include increased ROM, increased muscle strength and endurance, and decreased muscle spasticity, will also benefit those with MS who have compromised deficiencies in all of these areas. Furthermore, these benefits may increase the ability of MS individuals to perform activities of

daily living which can in turn increase their activity levels, subsequently reducing the likelihood of obtaining a secondary illness associated with a sedentary lifestyle. Additionally, flexibility training is a relatively low intensity exercises that can be done in the privacy of the home. It is possible that MS individuals with higher disability may be able to experience improvements through stretching that will later permit them to participate in more traditional exercise programs such as resistance training (Kokkonen et al., 2007).

The protocol used in the current study is was a supervised flexibility training program (White & Dressendorfer, 2004) that was based on the American College of Sports Medicine guidelines for musculoskeletal flexibility (*ACSM's Guidlines for Exercise Testing and Prescription*, 2000). Active static stretches of the hip abductors, adductors, flexors and extensors, knee flexors and extensors, and ankle plantar- and dorsiflexors were performed 3 times each for 30s. The volume and intensity of a static stretching program designed to create strength improvements, reduce muscle spasticity is unknown in an MS population. In order to err on the side of caution, the length of the program, frequency of visits, number and types of stretches, and how long the stretches were held were similar to or greater than other flexibility programs that have reported improvements in joint ROM, muscle spasticity, and muscle strength {Feland, 2001 #63; Kokkonen, 2007 #44; Zhang, 2002 #43}

The Sit-to-Stand Movement

The sit-to-stand movement (STS) is a self-perturbing task (Mak et al., 2003) that transitions from a large stable base of support, comprising of the area encompassed by the feet and the chair, to a much smaller less stable base of support, comprising of solely the area encompassed by the feet (Vander Linden, Brunt, & McCulloch, 1994). Not only is the movement one of the most mechanically demanding activities of daily living (Riley et al., 1991), greater

than both walking and stair ascent (Berger, Crossett, Jacobs, & Rubash, 1998), it is a prerequisite to other movements (Kralj et al., 1990). In addition to requiring neuromuscular coordination (Pai & Rogers, 1991a; Ramsey, Miszko, & Horvat, 2004), lower extremity muscle strength (Bernardi et al., 2004; Bohannon, 2007; Eriksrud & Bohannon, 2003; Lord, Murray, Chapman, Munro, & Tiedemann, 2002; Papa & Cappozzo, 2000; Sibella, Galli, Romei, Montesano, & Crivellini, 2003; Wretenberg & Arborelius, 1994), and balance (Mak et al., 2003), it is a skill used to determine the functional level of person (Janssen, Bussmann, & Stam, 2002) and an important goal in improving patient mobility (Doorenbosch et al., 1994). MS patients have deficiencies in muscle function and balance that may affect their ability to easily rise from a seated position, but there is no research to date that has performed a biomechanical analysis on how MS patients perform the STS. A biomechanical analysis of the STS of people with MS may provide specific mechanisms for possible deficiencies in performing the STS movement. By focusing on the specific mechanisms clinicians can more accurately prescribe treatments or interventions aimed at improving these problem areas. The following areas of discussion are aimed at providing an outline that describes the STS movement. These areas include the kinematics and kinetics of the STS movement, variables influencing performance mechanics of the STS, STS performance in populations that have similar disabilities as MS patients, and the influence of exercise training on STS performance. This outline will provide a context in which we can compare MS patients' STS performance to STS execution in other populations and examine how flexibility training may affect the STS performance of individuals with MS.

Phase definitions

The phases of the STS vary depending on the study, but most divide the movement up into either 4, 3, or 2 phases all determined by temporal parameters. When examining the

kinematics and kinetics of segmental and whole body center of mass data, Riley et al. (1991) proposed dividing the STS into four phases, flexion momentum (phase 1), momentum transfer (phase 2), vertical extension (phase 3), and stabilization (phase 4). The flexion momentum phase begins with the initial movement of the body from the original seated position and ends when the legs lose contact with the seat (SO). During this phase, the trunk and knees flex while the COM moves forward and slightly downward. The momentum transfer phase begins at SO and ends at maximum ankle dorsi-flexion. During this phase, the COM shifts from predominantly horizontal movement to predominantly vertical motion with the trunk and knees shifting from flexion to extension. The vertical extension phase begins at max ankle dorsi-flexion and ends when the patient has reached their normal erect posture. Full trunk and knee extension occurs during this phase. The stabilization phase begins when the body reaches erect posture and is comprised of the postural sway of the participant (Riley et al., 1991).

Depending on the strategy used to perform the STS and the placement of the foot, the timing of peak ankle dorsi-flexion could vary greatly. Another method proposed by Roebroeck et al. (1994) avoids this problem by defining the phases using the whole body COM horizontal and vertical velocities. They divide the STS movement into three phases that are similar to the first three phases proposed by Rylee et al. (1991). Phase 1 (acceleration phase) begins with initiation of movement and ends when horizontal velocity of the COM reaches its maximum. Phase 2 (transition phase) begins at maximum horizontal COM velocity and ends at maximum vertical COM velocity. Phase 3 (deceleration phase) begins when the vertical velocity of COM reaches its maximum and ends at the end of the movement (Roebroeck, Doorenbosch, Harlaar, Jacobs, & Lankhorst, 1994).

Another method commonly used by researchers further simplifies the STS movement into two phases using the instant of SO as the critical event dividing the two phases. Phase 1 begins with movement initiation and ends at SO. Phase 2 begins at SO and ends at the end of the STS movement. These phases are commonly called pre and post SO and represent the horizontal and vertical motions of the STS movement respectively (Alexander et al., 1991; Doorenbosch et al., 1994; Inkster & Eng, 2004; Inkster et al., 2003; Mak et al., 2003; Schultz et al., 1992). One major benefit of dividing the movement into two phases with SO as the critical event is its clinical relevance. It is difficult to clinically determine the phase definitions when more than two are present. Clinicians can easily determine the instant of seat-off without any expensive equipment and can therefore clinically assess movement before and after SO which can subsequently aid them in providing treatments or interventions to improve the STS performance in clinical populations. One of the aims of the current study is to determine the clinical relevance of flexibility training on STS performance for people with MS. It was therefore determined that the STS movement would be divided into two phases with SO as the critical event dividing the two phases.

Segmental and joint contributions

The STS is a complex movement that begins as a horizontal movement and transitions into a vertical movement. Different biomechanical models of the STS movement attribute to the horizontal and vertical movements of the STS to segment angular motions referred to as segmental contributions (Yu et al., 2000). Ae et al. (1980) developed a four segment model of the STS comprising the foot, shank, thigh, and head-arm-trunk (HAT) segments. Using this model, Pai and Rodgers (1991a) found that the HAT segment was the major contributor to maximum horizontal momentum of the entire body COM while the thigh segment was the major

contributor to maximum vertical momentum of the entire body COM during STS. In a different study using the same model, Pai and Rodgers (1991b) examined slow, natural, and fast STS movements. As the speed of the STS increased progressively, the hip and ankle torques remained relatively constant while the knee torque increased significantly ($p < 0.05$). These results indicate that the thigh segment was not only primarily responsible for both the magnitude of vertical momentum of the entire body COM, but also is a major determinant in the speed of the STS movement (Pai & Rogers, 1991b).

Another study that examined segmental contributions on the linear momentum components found the HAT segment to be the major contributor for both the maximum horizontal and vertical momentum of the entire body COM during STS (Riley et al., 1991). Riley et al. suggest that the HAT segment contributions on vertical momentum are due to the transfer of horizontal momentum to vertical momentum. Differences between the results of Pai and Rogers (1991a) and Riley et al. (1991) may be due to the different samples used and/or strategies used by the participants. Additionally, differences may be attributed to differing calculation methods.

Yu et al. (2000) used a different approach to analyzing the linear momentum of the STS by creating a model that describes the entire body linear velocity vector as a function of hip, knee, and ankle joint velocities. Yu et al. (2000) suggest that examining joint angles is more meaningful from a clinical rehabilitation perspective. They found that all three joint angles significantly ($p < 0.001$) contribute to both horizontal and vertical velocity of the COM, with the knee joint contributing the most in the horizontal direction and the hip contributing the most in the vertical direction. However, the knee joint contribution to horizontal velocity only played a role toward the end of the movement. When eliminating the knee joint contribution at the

beginning of the STS movement, the horizontal velocity during that portion of the movement was unchanged. When hip and ankle contributions were eliminated, forward horizontal velocity was 50% lower, indicating that without the hip and ankle contributions at the beginning of the movement, the participant may not be able to produce sufficient forward horizontal velocity to perform the STS movement. When examining the vertical COM velocity without the hip angular velocity contribution, the total vertical COM velocity was 65% lower than when the hip angular velocity is included. Similarly, but with less impact, is a 35% drop when knee angular velocity contribution is not included in the calculation of the total vertical COM velocity. No drop in total vertical COM velocity occurred when leaving out the ankle angular velocity contribution, further indicating that it is the hip and ankle joint velocities that are imperative to providing sufficient vertical velocity of the COM of the body during the STS movement.

Variables influencing STS mechanics

It has been suggested by many researchers that lower extremity strength is the most influential variable affecting an individual's ability to stand (Bernardi et al., 2004; Inkster et al., 2003; Papa & Cappozzo, 2000). Research confirms this suggestion indicating that lower extremity strength is a significant predictor of STS success ($p = 0.006$, $r = .64$) (Schenkman, Hughes, Samsa, & Studenski, 1996). Of the lower extremity muscles, the KE play the greatest role with 72% of the concentric force during STS coming from the KE (Wretenberg & Arborelius, 1994) and KE torque also significantly predicting STS success ($p < 0.05$, $r = .68$) (Eriksrud & Bohannon, 2003).

In order to more accurately assess the joint moment contribution of the lower extremity and determine the minimum joint moments required to perform the STS movement, Yoshioko et al. (2007) generated over 5 million possible STS movements using 85 sets of STS kinematic

data. Of these 5 million possible STS movements only 160,000 were feasible without falling. They found that the hip and knee joints contributed to a majority of the moment required to raise the body from a seated position with some successful trials having a peak ankle moment of zero. Yoshioko et al. also found that hip and knee moments were inversely related and nearly always summed to 1.53 n*m/kg. They suggested that people automatically perform the STS in a way that optimizes their moment development abilities, the HAT segment playing the greatest role in joint moment variability.

The moment variability created by the HAT segment is likely due to the degree of trunk flexion angle and magnitude of trunk flexion velocity when performing the STS movement. When examining differences between a flexed and a non-flexed hip strategy for STS, the flexed hip strategy resulted in significantly ($p < 0.05$) higher HE moments and lower KE moments (Doorenbosch et al., 1994). It was also reported in this same study that the flexed strategy resulted in greater EMG activation of the HE muscles and less activation of the KE muscles. They suggest that a more flexed hip STS strategy is used by those with KE weakness. Those people who have KE weakness often display increased trunk angular velocity in an effort to maximize horizontal momentum that can subsequently be transferred into vertical momentum allowing lower KE moments (Kerr et al., 1997; Papa & Cappozzo, 2000).

The experimental set-up and methodology of how the participant is required to perform the task also influences the mechanics of the STS movement. Placement of the feet, chair height, and whether or not the participant is permitted to use their hands during the movement all have a major influence on the mechanics and ability to perform STS movements (Janssen et al., 2002). Sibella et al. (2003) reported that obese individuals place their feet in a more posterior position prior to performing the STS in order to decrease trunk flexion and HE moments. They also

determined that a more posterior foot placement increased the KE moments during STS. After finding that foot position significantly influences overall rise time in stroke patients, Camargos et al. (2009) suggested that controlling for foot position is imperative. When examining the influence of chair height during the STS, Schenkman et al. (1996) found lower extremity strength was significantly correlated to chair height during STS ($p = 0.006$, $r = -0.64$). This indicates that lower chair heights require greater lower extremity strength and KE and HE moments. Other studies reported similar results finding that using higher chair heights resulted in up to 60 and 50% reductions in KE and HE moments respectively (Janssen et al., 2002). Additionally, when relative chair height (relative to the shank length) is lower during STS it is also reported that EMG activation of the muscles is higher (Yamada & Demura, 2004). It is evident that chair height plays a major role in STS mechanics. If not controlled, this variable can cause confounding results. Finally the use of hands is reported to reduce the KE and HE moments up to 50% during STS (Arborelius, Wretenberg, & Lindberg, 1992; Burdett, Habasevich, Pisciotta, & Simon, 1985; Janssen et al., 2002; Seedhom & Terayama, 1976). It is evident that foot position, chair height, and the use of hands all play a role in STS mechanics and need to be considered when designing a study. In an effort to reduce the confounding effects of different chair heights, foot positions, and the use of the hands, all participants were positioned in a standardized position for all STS trials. Participants were positioned at a seat height that created a 90 degree angle at the knee and ankle joints. Furthermore, participants folded their arms across their chest with their feet placed shoulder width apart. During the movement participants were required to keep their arms crossed and not shift their feet. Although this position may not be how a person would stand-up in a normal situation, it does eliminate most of the confounding effects associated with seat height, foot position, and the use of hands.

STS performance in clinical populations

With no known studies examining the biomechanics of the STS movement for people with MS, this section will describe the biomechanics of the STS in clinical populations that exhibit similar symptoms to those with MS. These symptoms include muscle weakness, muscle spasticity, and balance impairments. Among the clinical populations that exhibit these symptoms are the elderly, stroke patients, and patients with Parkinson's disease. Due to all three of these populations demonstrating similar biomechanics during the STS, their STS performance will be described as a group and only individually when describing specific studies.

The most common trend among these clinical populations with similar symptoms as those with MS is a significant increase in rise time when performing the STS task (Camargos et al., 2009; Cheng et al., 1998; Inkster & Eng, 2004; Inkster et al., 2003; Kerr et al., 1997; Mak & Hui-Chan, 2005; Mourey et al., 2000). More specifically, it is reported that the average time for a healthy population to stand is around 1.63-1.87 (0.28) seconds (Baer & Ashburn, 1995; Doorenbosch et al., 1994). However, those with Parkinson's disease take significantly ($p < 0.001$) longer with an average time of 2.86(0.77) seconds (Mak & Hui-Chan, 2005). Stroke patients took almost twice as long to stand as a healthy matched control group (Cheng et al., 1998). Although the difference between an elderly and young population was not as different as stroke and Parkinson's patients, it is reported that elderly individuals take approximately a half second longer to stand than younger individuals (Mourey et al., 2000).

One explanation for the increased rise time in clinical populations is the inability to produce sufficient ground reaction force (GRF) and joint moment as quickly as controls. Cheng et al. (2003) reported that controls took a third the amount of time to produce max vertical GRF when compared to stroke patients. Furthermore, Mak et al. (2003) reported that controls were

able to produce maximum HE moments approximately fourfold faster than those with Parkinson's disease. In addition to slower moment and force development, clinical populations also produce less maximum joint moments when compared to controls. Inkster et al. (2003) reported in one study that controls were able to produce greater KE and HE moments than those with Parkinson's disease during the STS. In a later study, Inkster and Eng (2004) found that KE moments were 20% lower in Parkinson's patients with no differences being found between HE moments when compared to healthy controls. The differences in these two studies may be due to samples or possibly the strategies used by the samples.

Clinical populations who have lower extremity weakness generally use compensatory strategies in order to successfully perform the STS movement (Doorenbosch et al., 1994). When comparing a flexed trunk strategy to a non-flexed trunk stategy, Doorenbosch et al. (1994) found the flexed trunk strategy resulted in significant increases in maximum HE moments and significant decreases in maximum KE moments when compared to the non-flexed strategy. This may explain why a significant increase in trunk flexion has been consistently found in the clinical populations discussed (Alexander et al., 1991; Inkster & Eng, 2004; Kerr et al., 1997; Papa & Cappozzo, 2000; Schultz et al., 1992). Most of the authors of these articles have suggested that increased trunk flexion also serves to place the body's COM closer to the new base of support, increasing stability to those who have balance impairments. It is reported that Parkinson's patients place their body COM up to 17% in a more anterior direction during STS than healthy controls which likely increases their stability (Inkster & Eng, 2004). This may further explain why rise time is greater in clinical populations. Increased trunk inclination requires further to raise the body to an erect posture (Papa & Cappozzo, 2000).

Differences in trunk flexion velocity have been reported to be significantly different between clinical populations and healthy controls. One study found that the elderly had significantly ($p < 0.05$) greater trunk velocity than controls (Papa & Cappozzo, 2000). It is suggested that increasing trunk flexion velocity is used to increase horizontal momentum to subsequently transfer to vertical momentum, thus reducing the KE moment requirement for the STS movement (Berger, Riley, Mann, & Hodge, 1988; Bernardi et al., 2004; Kerr, White, Barr, & Mollan, 1994; Kerr et al., 1997; Pai & Rogers, 1991b; Papa & Cappozzo, 2000). However, Kerr et al. (1997) found that elderly women displayed less trunk flexion velocity than young healthy females. They suggested that while increasing velocity may aid in producing greater vertical momentum, elderly women prefer slower movements to ensure greater stability.

Finally, when analyzing muscle activity, Ramsey et al. (2004) found no differences in mRMS or median frequency EMG between those with Parkinson's disease and healthy matched controls during STS movements. They attribute their lack of differences to the high variability in EMG for the Parkinson's patients. However, Doorenbosch et al. (1994) found that using an increased trunk flexion strategy significantly ($p < 0.05$) increased the hamstring muscle activity while reducing the rectus femoris muscle activity, possibly further explaining why clinical populations with muscle weakness of the KE exhibit increased trunk flexion.

The biomechanical differences found between clinical populations and healthy controls are likely due to muscle weakness, muscle spasticity, and balance impairments commonly found in clinical populations. Although a biomechanical analysis of the STS in MS individuals has not been reported to date, it is likely that those with MS will exhibit many of the same differences as the elderly, stroke patients, and those with Parkinson's disease.

Influence of exercise training on STS

As discussed previously, lower extremity strength has been reported to be the biggest predictor of the ability to perform the STS movement (Bernardi et al., 2004; Eriksrud & Bohannon, 2003; Inkster et al., 2003; Papa & Cappozzo, 2000; Schenkman et al., 1996; Wretenberg & Arborelius, 1994). Subsequently, several studies have examined the influence of exercise training of the lower extremities on STS performance in clinical populations (Bernardi et al., 2004; Fisher et al., 2008; Freiberger, Menz, Abu-Omar, & Rutten, 2007; Kraft et al., 1996a; Krebs, Scarborough, & McGibbon, 2007; Monger, Carr, & Fowler, 2002; Schlicht, Camaione, & Owen, 2001; Schot, Knutzen, Poole, & Mrotek, 2003; Taylor, Dodd, & Larkin, 2004). Several of these studies did in fact report significant increases in lower extremity strength, but improvements to STS performance are somewhat split.

When comparing 6 weeks of functional exercise training involving agility drills, regular walking, and repeated chair rising, to a traditional strength training program involving resistance training in a weight room, no differences in strength or chair rise kinematics were found between training protocols in an elderly sample (Krebs et al., 2007). However, the elderly in the functional training and traditional strength training groups did significantly ($p < 0.05$) increase in knee extension strength. Unfortunately, increasing knee extension strength did not transfer to improving functional performance of the STS with no changes for either group in STS time or the amount of trunk flexion during the movement (Krebs et al., 2007). Similarly, it is reported that 8 weeks of total body resistance training 3 times a week resulted in significant ($p < 0.05$) strength gains in older adults (Schot et al., 2003). Although a 7% decrease was found in rise time no significant difference was found. However, following the 8 week training program the older adults displayed increased vertical and horizontal velocities and trunk flexion. Schot et al. (2003)

suggest that the increased trunk flexion may have been a reason for not finding significantly faster rise times, but that this strategy indicates improved STS ability and was chosen to increase stability when horizontal and vertical velocities increased.

Both Schot et al. (2003) and Krebs et al. (2007) examined participants who performed the STS at a self-selected speed. However, other exercise intervention studies that reported increased strength and improvements in STS performance, examined participants who performed the STS as fast as possible either once or repeated times. In addition to improving maximum voluntary contractions of the KE, Bernardi et al. (2004) reported that a 40 day rehabilitation program involving proprioceptive gymnastics, resistance training, and walking 2km/day will decrease STS time in elderly patients when performed at maximum speed. In other clinical populations, 10 weeks of strength training increased lower extremity strength and decreased 3 repetition STS time in those with cerebral palsy (Taylor et al., 2004). Similarly, three weeks of home based exercise training improved strength and decreased 10 repetition STS time in stroke patients (Monger et al., 2002). Although no other studies have examined an MS population performing the STS at self-selected speed or at maximal speed, Kraft et al. (2006) did assess MS individuals' ability to perform the up and go agility test following 12 weeks of strength training. They found that the time for up and go agility test, which involves standing up walking 3 meters and returning to the chair and sitting down, decreased by 17% in ambulatory MS patients (Kraft et al., 1996a).

Although research has shown a decrease in time for the up and go and the STS tasks performed at maximal speeds in these clinical populations, time differences alone will not give us the full picture or explain why clinical populations are able to perform the task more quickly following an exercise intervention. In the case of the repeated sit to stands, decreases in time may

be solely a result of increased endurance rather than any mechanical difference. The up and go test time differences may be associated with improvements in standing, walking, turning, or sitting or any combination of the four. It is difficult to determine which part of the up and go test is responsible for the decreased time reported. In order to determine more accurately the causes of the proposed improvements in the STS movement following an exercise intervention, it is imperative to do a complete biomechanical analysis of the STS movement. Additionally, earlier sections of this literature review have indicated that flexibility training may improve the functional performance of STS movements. Therefore, with no other research reporting the possible benefits of using flexibility training as an intervention, it is equally as important to examine the influence of flexibility training on STS performance. Improving strength through flexibility training may also improve the STS performance.

CHAPTER 3

SIT-TO-STAND BIOMECHANICS OF INDIVIDUALS WITH MULTIPLE SCLEROSIS[1]

[1] Bowser, B., O'Rourke, S., Brown, C., Simpson, K., White, L. To be submitted to *Journal of Biomechanics.*

Abstract

For people with multiple sclerosis (MS), the ability to perform activities that require rising from a seated position is reduced due to compromised neuromuscular control, muscle weakness and balance impairments. To understand the adaptations individuals with MS use to perform the sit-to-stand, the purpose of this study was to determine the biomechanical differences between people with and without MS. Twenty-one ambulatory individuals (18 females, 3 males), with relapsing-remitting MS and 12 healthy, matched-controls (10 females, 2 males), performed 5 sit-to-stand trials. One-way (Group), for leg extensor strength, and 2 × 2 mixed-model, measures ANOVAs, (Group × Phase [RM]) for movement time, and (Group × Leg [RM]) for sagittal plane kinematics and kinetics, and electromyography of lower extremity muscles were used to determine differences. Compared to controls, MS participants exhibited decreased leg extensor strength and increased movement time, maximum trunk flexion, and maximum trunk flexion velocity. Decreased muscle activation of rectus femoris and hamstrings were found for the affected- compared to the less-affected leg for both groups. Potentially, due to the lack of group differences in muscle activation, no differences for peak magnitudes for joint kinetics were found. However, MS participants did employ the 'trunk flexion' strategy commonly exhibited by similar clinical populations with neuromuscular deficits. This strategy is suggested to both reduce joint moment requirements as well as simplify the motor coordination of the sit-to-stand movement. In summary, ambulatory individuals with relapsing-remitting MS display strength and functional deficits (slower) and use an adaptive strategy that is commonly used by clinical populations with lower extremity muscle weakness to compensate for impaired muscle function, a common symptom of MS.

Key Words**:** TRUNK FLEXION, LOWER EXTREMITY, JOINT MOMENT, ELECTROMYOGRAPHY, DISABILITY, KINEMATICS, KINETICS, STANDING UP

Introduction

Multiple sclerosis (MS) is a progressive, degenerative disease of the central nervous system thought to be an autoimmune disorder that attacks the myelin, oligodendrocytes and the axons (Noseworthy et al., 2000). Degeneration and scarring on the axons inhibit nerve conduction, resulting in distorted, disrupted, or blocked neural impulses (Frohman, Racke, & Raine, 2006; Smith & McDonald, 2003). Consequently, neuromuscular deficits in the form of muscle weakness, muscle spasticity, fatigue, and balance impairments occur that result movement limitations. Therefore, functional activities that require considerable neuromuscular coordination, balance, and leg extensor strength (Riley et al., 1991) are challenging for people with MS.

The sit-to-stand (STS) is likely one of the most demanding of such daily activities. The most influential variable affecting an individual's ability to perform the STS movement is the amount of leg extensor joint moment that can be generated (Bernardi et al., 2004; Inkster et al., 2003; Papa & Cappozzo, 2000). Unfortunately, when compared to healthy controls, people with MS are reported to have decreased leg strength and less ability to effectively use the muscle mass that they do have (Lambert, Archer, & Evans, 2001; Ng, Miller, & Kent-Braun, 1997; Rice, Vollmer, & Bigland-Ritchie, 1992). However, it is not known how these factors affect the kinetics or kinematic strategies used by those with MS when rising to a stand.

Clinical populations with lower extremity weakness are reported to use compensatory movement strategies (Doorenbosch et al., 1994); particularly the 'trunk-flexion' movement strategy (Alexander, Schultz, & Warwick, 1991; Inkster & Eng, 2004; Kerr et al., 1997; Papa &

Cappozzo, 2000; Schultz, Alexander, & Ashton-Miller, 1992). During the pre-seatoff phase of this strategy, participants flex the trunk much further than during a typical STS. While rising after seatoff, the participant maintains a flexed trunk until the knees are extended, then trunk extension occurs. In contrast, during a typical STS, the trunk only slightly flexes before seat-off and as seat-off occurs, the trunk and knees immediately extend simultaneously in one smooth motion (Doorenbosch et al., 1994).

As strength and muscle activation deficits are problematic for individuals with MS, we surmised that this population also would utilize the trunk-flexion strategy, as it has several potential advantages compared to the typical movement strategy. First, using greater trunk flexion displacement may allow the performer a means to reduce the knee extensor (KE) moments. For a group of elderly individuals who were asked to perform both strategies, Doorenbosch et al., (1994) observed that the flexed trunk strategy resulted in decreased knee extensor (KE) moments, accompanied by decreased muscle activity of the rectus femoris. Furthermore, Inkster et al., (2003) found that individuals with Parkinson's disease who used a flexed trunk strategy displayed decreases in both KE and HE moments while performing the STS. We therefore hypothesized that MS individuals would display decreased KE and HE moments, as decreased activation of KE and HE muscles would occur.

Second, all else equal, greater trunk flexion displacement causes higher gravitational angular work done to the trunk, which, in turn, results in higher, anterior-directed linear velocity of the COM of the body. Thus, the line of gravity can be shifted farther forward within the boundary and closer to the center of the base of support, thereby increasing stability during the post-seatoff phase. Inkster and Eng (2004) surmised that Parkinson's patients, compared to controls, place the location of the body's COM up to 17% farther in the anterior direction to

increase stability. As balance deficiencies are commonly exhibited by people with MS (Frzovic, Morris, & Vowels, 2000; Karst et al., 2005; Lanzetta et al., 2004; Soyuer, Mirza, & Erkorkmaz, 2006), we hypothesized similar outcomes for MS individuals.

As a prerequisite to being able to perform a variety of functional activities (Kralj, Jaeger, & Munih, 1990), the ability to complete the STS movement is paramount. Determining how performers' biomechanics are affected by MS will provide a foundation for developing more successful rehabilitation and/or exercise programs aimed at improving functional capacity and slowing down the rate of disability. Therefore, the purpose of this study was to determine the biomechanical differences between an MS and non-MS control group for the STS movement.

Methods

Participants

Twenty-one individuals with MS and 12 healthy controls (CON) similar in age, height, and mass to the MS group, were recruited from the university, surrounding communities, MS support groups, and neurology clinics. Descriptive statistics of participants is found in Table 3.1. All potential participants provided written informed consent as approved by our institutional human subjects review board. A medical history questionnaire (Appendix C) was filled out by each participant to determine if participation criteria were met. For individuals with MS, only those who were physician diagnosed with relapsing-remitting MS and an expanded disability status score (EDSS) of < 6.5 were included in the study. It was acceptable for participants to use MS-modifying drugs interferon beta 1α and 1β. Excluded was any potential participant who was pregnant, had orthopedic limitations of the lower extremity or trunk, had used prednisone or other steroids for an MS flareup during the previous three months or did not receive physician's

clearance to participate. All CON participants were healthy, with no known visual, vestibular or neurological disorders.

Experimental procedures

All data were collected during 3 separate data collection sessions. During each of the first two sessions, lower extremity muscle strength was tested using a one-repetition maximum (1RM) protocol (Barnard et al., 1999) on a Cybex® leg press machine (Cybex International Inc., Medway, MA). The STS data collection session occurred during the third visit. After a brief description of the procedures for the session, the participant's age, height, and mass were recorded. The skin was then cleaned and prepared for the placement of Ag-Ag Cl disposable monitoring electrodes (Biopac System Inc., Goleta, CA, 3.49 cm diameter) following the method of Cram and Kasman (1998). A 16-channel MYOPAC® surface electromyography (EMG) system (Run Technologies, Inc., Mission Viejo, CA.) was used to collect the electrical activity (1000 Hz, CMRR = 10,000:1) of muscles of both legs: tibialis anterior (TA); gastrocnemius (GAS); soleus (SOL); vastus lateralis (VL); rectus femoris (RF); and semimembranosis and biceps femoris, collectively called the hamstrings (HAM). A reference ground electrode was placed on the tibial tuberosity of the right leg. The EMG signals were captured and amplified via the data pack that the participant wore on the trunk, then transmitted through an attached fiber optic cable to the receiver (MPRD-101 Receiver/Decoder®, Run Technologies, Mission Viejo, CA) then to the computer (16-bit ADC).

Once the electrodes were placed on the participant, EMG was collected during sub-maximal isometric tests of the knee flexors, knee extensors, ankle plantarflexors, and ankle dorsiflexors using a 108 N weight. To test the knee extensors, the participant was in a seated position, with the leg fully extended and the weight hanging on the distal end of the shank. For

the knee flexors, the isometric tests were performed while standing on one leg with the other leg flexed at the knee 90° and the femur perpendicular to the ground. The weight was placed at the distal end of the shank of the flexed leg. For isometric testing of the dorsiflexors, participants sat upright with both shanks hanging over the side of a plinth and the ankle in a neutral position. The weight used for the sub-maximal test was placed on the heads of the metatarsals. For the plantarflexors, the participant lay prone on a plinth, with the non-test leg flat on the plinth and the test leg flexed at the knee so that the shank was perpendicular to the plinth. The ankle was slightly plantarflexed at ≈ 5°. The weight was placed over the heads of the metatarsals. For all sub-maximal isometric tests, the participant held the position for approximately 5 seconds. The same researcher aligned the limbs in the proper position using a goniometer for all participants.

Flock of Birds™ sensors (Ascension Technologies Corp. Burlington, VT) were placed on the foot, shank, and upper leg of both legs and the sacrum (Brown et al., 2008) as well as the sternum. Joint locations were digitized in the electromagnetic field during natural stance. An extended range transmitter collected sensor position data at (100 Hz).

For the STS task, the participant started from a sitting position on a box whose height was adjusted so that the knee and ankle joints were at approximately 90°. The arms were folded across the chest; the lower extremities were aligned parallel to the sagittal plane, with each foot on one of two force platforms (Model #4060-NC, Bertec Co., Columbus, OH) which were used to collect (1000 Hz) ground reaction force signals (GRF) in the vertical (VGRF) and anterior-posterior (AP-GRF) directions. Without shifting the feet, the participant rose from the chair at a self-selected speed, stood still for 5 s, then returned to the seated position. Following a minimum of two practice trials, each participant performed 5 STS trials. Trials in which the feet shifted,

arms were not folded across the chest the entire time, or participant was unable to completely stand were considered unsuccessful and not used as one of the five successful trials.

The STS began when the horizontal center of mass (COM) velocity was > 0 $m \cdot s^{-1}$ and continued to increase; the movement ended the first instant when the body's vertical COM stopped displacing upwards (Yu et al., 2000). The seatoff event, the instant when the value of the anterior GRF attained its greatest value (Kralj et al., 1990), was used to divide the STS into two phases: pre- and post-seatoff.

It is common to have one leg affected by MS more severely than the other, often resulting in greater muscle weakness in this leg. To account for this, the participants were asked to identify their more affected limb. If the participant did not feel one leg was more affected than the other, they were asked which of their legs was weaker. For comparison purposes, CON participants also were asked which of their legs was weaker. The weaker and stronger legs of CON participants corresponded to the affected (A-leg) and less-affected (LA-leg) legs of the MS group, respectively.

Data Reduction

All 1RM strength data were scaled to body mass. The spatial locations of the Flock of Birds sensors were reconstructed and sagittal plane kinematic and kinetic quantities were calculated using *The MotionMonitor* ™ software (v. 7). Following ISB guidelines, Euler angles (Euler, Z Y′ X′′) were calculated and adjusted relative to natural standing with sagittal plane motion occurring around the third rotation. The trunk inclination angle was calculated relative to the positive y axis. The kinematic quantities generated included: horizontal displacements of the COM, trunk flexion angles and velocities, and angular displacements and velocities of the joints of both lower extremities. Anthropometric data based on the modified-Zatziorsky equations

(Zatziorsky et al., 2002) and an inverse dynamics model by Gagnon and Gagnon (1992), were used to calculate hip, knee, and ankle joint moments (JM). GRF data were synchronized to the kinematic data to produce JM. Movement time (MT), maximum magnitudes and relative times (RT) (% of the entire movement) to these maxima for kinematic and kinetic variables were calculated in Matlab® (v. 7.0). Kinematic and GRF data were filtered using 4th-order, recursive low-pass Butterworth filters with cut-off frequencies of 5 and 20 Hz, respectively (Mak et al., 2003). All GRF and joint moment data were scaled to body weight.

Raw EMG signals were bandpass (5-178 Hz) and notch filtered (60 Hz and 100 Hz; 4 Hz notch width) using *The MotionMonitor* ™ software to remove electromagnetic and electrical interference based on a frequency spectrum analysis. For each muscle and STS trial, the mean of all root-mean square values (T = 50 ms) displayed during the STS was generated (mRMS) (Cram & Kasman, 1998) and scaled by dividing out the mRMS collected during the corresponding sub-maximal isometric test.

Statistical analysis

The mean of the 5 trials for each variable was used for analysis for each participant. All statistical analyses were conducted using SPSS (v.16.0, SPSS Inc., Chicago, IL). Differences between groups for trunk kinematics and 1RM on leg press were analyzed using one-way ANOVA. In order to determine group differences for MT and phase times, a 2 × 2 mixed-model ANOVA (Group × Phase[RM]) was conducted. A 2 × 2 mixed-model ANOVA (Group × Leg[RM]) was used to assess differences between groups and legs for the remainder of the variables. Significant interactions ($p < 0.05$) were followed by Bonferroni post hoc analysis with an accepted family-wise error rate of $\alpha = 0.05$. Effect size was reported using eta squared ($\eta 2$).

Results[2]

From the one-way (Group) ANOVA, the groups were not significantly different in age, height, mass, or body mass index (BMI) ($F(1,31) < 0.67$ for all variables), but the MS group demonstrated over 26 $N \cdot kg^{-1}$ less lower extremity strength ($F(1,31) = 8.29$, $p = 0.007$, $\eta^2 = 0.21$) (Table 3.1). For kinematics, as displayed in Figure 3.1, the mean MT for the STS movement was 17% longer for the MS compared to the CON group ($F(1,31) = 5.89$, $p = 0.02$, $\eta^2 = 0.16$), due to longer post-seatoff phase time ($t(32) = 3.89$, $p = 0.001$, $\eta^2 = 0.25$). The RT to seatoff occurred 6% earlier in the MS group compared to the CON group (mean±SD: CON= 42.1±3.5%MT, MS= 36.2±7.0%MT; $p < 0.01$, $\eta^2 = 0.20$) although actual times to seatoff varied by only 0.01 s (MS 0.74±0.20; CON 0.75±0.12, $p = .94$).

Results of the ankle and knee kinematics are reported in Table 3.2. Among the variables of interest, for the ankle joint, a significant (Group x Leg) interaction was revealed for maximum dorsiflexion angle of the ankle ($F(1,31) = 8.83$, $p = 0.006$, $\eta^2 = 0.22$). No differences of interest were revealed during post hoc comparisons. A Group main effect at the knee joint revealed that the MS group displayed lower maximum knee extension velocity than the CON group ($F(1,31) = 4.75$, $p = 0.037$, $\eta^2 = 0.13$). Figure 3.2 displays representative participant curves for trunk inclination angle and velocity. Qualitatively, it is evident that trunk extension begins immediately prior to seatoff for the CON group, whereas trunk extension begins after seatoff for the MS group. A Group main effect showed that the MS group rotated their trunk forward 11° further and attained a maximum trunk flexion velocity that was $20° \cdot s^{-1}$ faster than the CON group ($F(1,31) = 7.34$, $p = 0.011$, $\eta^2 = 0.19$ and $F(1,31) = 5.54$, $p = 0.025$, $\eta^2 = 0.15$,

[2] For dissertation: ANOVA summary tables for all variables can be found in appendix D.

respectively). No differences were found between groups for maximum trunk extension velocity ($p = 0.42$) or maximum anterior COM displacement ($p = 0.86$).

Due to inconsistencies in timing of the ankle joint moments and powers a qualitative analysis is given. A spectrum of ankle joint moment and joint power patterns displayed by participants within both groups and are shown using representative curves of three participants (PP1-3) in Figure 3.3. The following commonalities were shown among the majority of the participants for the ankle joint moment and joint power: Prior to seatoff, all participants displayed a peak concentric dorsiflexor joint moment that occurred between 25% and 30% MT with negligible joint power until around 40%MT. Following this initial peak the ankle joint moment then steadily decreased and became a concentric plantarflexor moment followed by an eccentric plantarflexor moment. Following seatoff the power curves appear to be bimodal.

Qualitatively, the major differences among the exhibited ankle joint moment and joint power patterns were not only the magnitudes, but also the timing of these events and number of fluctuations (i.e., displayed unique local maxima/minima). Whereas PP1 displayed a steady decline in the plantarflexor joint moment from 30% MT to approximately 80% MT, PP2 and PP3 displayed increased oscillations of the plantarflexor moments. Additionally, PP3 displays a greater number of oscillations during plantarflexor joint moment generation than PP2. No peak magnitudes of ankle joint moments displayed any significant differences. Interparticipant variation for ankle joint power patterns indicate that while PP1 and PP2 have the two distinct positive power peaks representing an eccentric plantarflexor joint moments, the interval separating the peaks is much less distinguishable for PP3. PP3 also appears to have a considerably greater negative power magnitude indicating a concentric plantarflexor joint moment.

Group means and standard deviations for knee and hip joint moments and powers are reported in Table 3.2 and representative curves for the MS and CON groups are illustrated in Figure 3.4. Although there were no significant magnitude differences for moments or powers of either joint, the MS group displayed the maximum HE and KE moment magnitudes and the maximum positive knee power 5-6 %MT earlier than the CON group; main effect (Group): $F(1,31) = 5.87$, $p = 0.02$, $\eta^2 = 0.16$; $F(1,31) = 6.94$, $p = 0.01$, $\eta^2 = 0.18$; $F(1,31) = 5.50$, $p = 0.03$, $\eta^2 = 0.15$, respectively.

Table 3.2 shows the outcomes for the GRF variables. No group differences for maximum magnitudes or RT of these magnitudes were found for GRFs. A significant Leg effect ($F(1,31) = 4.67$, $p = 0.04$, $\eta^2 = 0.13$), however, indicated that the maximum anterior magnitude of the AP-GRF of the A-leg occurred relatively earlier than the LA-leg within the MS group.

For the electromyographic results shown in Figure 3.5, the RF displayed a significant Group x Leg interaction, $F(1,31) = 10.24$, $p = 0.003$, $\eta^2 = 0.25$). The A-leg of the MS group displayed approximately 25% and 40% less mRMS, compared, respectively, to the MS LA-leg ($t(32) = 2.90$, $p = 0.009$, $\eta^2 = 0.21$) and the A-leg of the CON group ($t(32) = 2.85$, $p = 0.008$, $\eta^2 = 0.21$). The HAM muscles displayed a significant Leg difference, with the A-leg displaying less mRMS the LA-leg, $F(1,31) = 4.165$, $p = 0.05$, $\eta^2 = 0.12$). No other EMG differences were found.

Discussion

Leg extensor weakness of the MS group was surmised to reduce function and affect the kinematic strategies and decrease the magnitudes of the lower extremity joint kinetics displayed during the STS. For function, as all individuals in our MS group were able to complete the STS task several times, all displayed the functional ability to perform movements that involve raising the body to a standing position. However, the MS group exhibited one overall functional deficit

(Janssen, Bussmann, & Stam, 2002), as it took 0.3 s, or approximately 17 % longer for the MS compared to the CON group to stand. This finding is congruent with previous STS literature comparing other clinical populations to equivalent control groups such as the elderly (Alexander et al., 1991; Mourey et al., 2000), those with Parkinson's disease (Mak & Hui-Chan, 2005), and stroke patients (Cheng et al., 1998; Richards et al., 2003). It appears, however, that the MS participants in this study, who were required to have relatively low disability status, took less time to stand than these other clinical populations. Compared to their respective CON group, those with Parkinson's disease or who have had a stroke, respectively, took almost one second longer (Mak & Hui-Chan, 2005) or double the time to rise (Cheng et al., 1998). Our CON group value for MT (1.76 ± 0.22 s) was similar to previously published research (1.67s -1.83 s) (Baer & Ashburn, 1995; Doorenbosch et al., 1994).

The increased MT of the MS group compared to the CON group was a result of the increased time spent in the post-seatoff rather than the pre-seatoff phase (see Figure 3.1). Interestingly, as the predominant movements in the post-seatoff phase were trunk and leg extension, there are several possible explanations for the increased time in the post-seatoff phase that will be discussed later.

Strength, EMG, and joint kinetics

As predicted, our MS group had less leg extensor strength when compared to that of the CON group. Our mean difference of 25% also is consistent with previously-reported muscle strength deficits for individuals with MS that range from 25-35% (Lambert et al., 2001; Ng et al., 2004). It is possible that the strength deficits detected for MS populations may be due, in part, to disruptions in motor neuron activation. A reduced ability to fully activate the muscle mass has been suggested for individuals with MS (Rice et al., 1992). Rice et al. (1992) demonstrated that,

when an electrical stimulus was applied directly to the surface of the skin during maximal effort isometric actions, a greater increase in muscle activation of the quadriceps muscles was displayed by MS compared to the non-MS group.

Additionally, muscle weakness found in the MS group of this study may possibly be attributed to a decrease in muscle cross-sectional area. Kent-Braun and Ng (1997) reported that MS individuals have 30% less fat-free, cross-sectional area (CSA) in skeletal muscles and greater atrophy of type II fibers compared to healthy controls. Unfortunately, as CSA was not recorded for this study we cannot prove this was true for our sample.

As we had anticipated that increased muscle weakness and possible movement strategy differences between the MS and CON groups would occur, we had hypothesized that the MS group would display decreased mRMS EMG activity of the KE and HE muscles. As surmised, for the A-leg, the MS group showed lower RF mRMS than the CON group. Moreover, consistent with this finding is that, within the MS-group, the A-leg had lower mRMS values than the LA-leg. No differences were found for the VL of either leg between groups. These findings are similar to previous literature that has found decreases in EMG activity of the RF, but not the VL during the STS movement (Doorenbosch et al., 1994). Doorenbosch et al. (1994) suggest that the biarticular RF muscle plays a different role during the STS than the monoarticular VL muscle. Whereas the RF aids in both flexion of the trunk and extension of the knee, the VL directly influences only knee joint kinetics. Furthermore, the position of the pelvic girdle during this movement may play a role as to why the RF displayed decreased EMG activity. The tilt of the pelvis during STS can vary across participants and groups which would subsequently change the length and possibly the amount of muscle activation that would occur. We did not directly

measure the length of the muscle during the activation and therefore cannot definitively determine that pelvic angle influenced the EMG outcomes.

In order to ascertain possible differences associated with one leg being affected more greatly by MS than the other leg, in addition to group differences, differences between legs were also assessed. For the hip extensor muscles (HAM), the A-leg displayed approximately 35% less muscle activation than the LA-leg. It is possible that the difference in EMG activation of the HAM may be a result of the A-leg having greater neurological impairments than the LA-leg. However, this cannot be directly assessed with muscle activation alone. Furthermore, the intralimb difference may have been influenced by having separated the CON group limbs into sham affected (weaker) and less affected (stronger) legs based on the participants' self-perception of their weaker/stronger legs.

One possible explanation for the limited number of EMG differences between groups may be muscle spasticity. Increased muscle tone due to muscle spasticity may have increased the amount of EMG activity found in the lower extremity muscles in the MS group. Although muscle spasticity is a common symptom among people with MS, it is difficult to determine how large of a role muscle spasticity may have played in the limited number of EMG differences found.

It was thought that neuromuscular deficits commonly associated with MS would be reflected by EMG. Furthermore, it was hypothesized that the peak KE and HE moments would be lower for the MS group compared to the CON group. Although it cannot be proven with these data, the lack of between-group differences for EMG activity may partially account for the paucity of findings for maximum magnitudes of the HE and KE joint moments. Although muscle activation of the RF in the A-leg was less in the MS compared to the CON groups, there are

several other muscles that can contribute to the net muscle moment at the knee joint that may have compensated for the RF decreased activity. However, as EMG for these muscles was not collected, this remains unknown.

Other reasons for no differences being detected in maximum magnitude of the HE and KE joint moments may be the level and type of disability found in the MS group. It has been reported that Parkinson's patients display decreased KE and HE moments during the STS movement (Inkster et al., 2003). However, as MT is positively associated with levels of disability (Janssen et al., 2002), the longer MT of those Parkinson's compared to our MS participants suggests that those individuals had greater levels of disability. Hence, it may be possible that joint moment adaptations are only exhibited by individuals who may have higher levels of disability than those of our MS group or may be specific to the neuromuscular disorder. However, with no direct comparisons between disability levels or types of the MS group in this study and the Parkinson's patients in the study by Inkster et al., the influence of disability level and type on joint moment magnitudes cannot be definitively determined.

Group differences were displayed for the RT of the peak magnitudes of knee joint moments and knee powers (Table 3.2; Figure 3.4). Relative to the entire movement these events occurred earlier in the MS compared to the CON group. Qualitatively, the RTs of these events occurred 6% MT earlier in MS compared to CON which is approximately the same difference between groups for seatoff RT. This indicates that relative to the timing of seatoff, peak joint moments and knee powers occur at similar times between groups. Furthermore, the MS group displayed increased trunk flexion velocity which may help explain why these peaks occurred relatively earlier in the MS group.

Movement Strategies

It was hypothesized that to compensate for the inability to produce sufficient KE joint moments, the MS group would implement a compensatory movement strategy. Increased trunk flexion displacement and trunk flexion velocity were displayed by the MS group, confirming that the trunk-flexion strategy was adopted. In association with this strategy is a distinct sequence of trunk flexion, knee extension, then trunk extension into an upright position. This creates a 'trunk forward' during pre-seatoff, then 'pelvis up' that causes the body to leave the chair, followed by 'trunk back' motions to finish raising the body into an upright position. This strategy has been reported to be used by the elderly who have KE weakness and poor motor coordination (Papa & Cappozzo, 2000). This is qualitatively different from the strategy typically displayed by healthy controls. Soon after the trunk begins to flex during pre-seatoff, the knees begin to extend, followed shortly after by trunk extension creating a fluid movement with less forward and back motion.

Using the trunk flexion strategy is evidenced (Figure 3.2) by the MS participants waiting until after seatoff to begin extending the trunk, whereas trunk extension begins at seatoff for the CON group. The increased time in the postseatoff phase for the MS group is likely due not only to the MS group continuing to flex the trunk after seatoff, but also to increased trunk flexion requiring further to raise the body to an erect posture (Papa & Cappozzo, 2000). Additionally, the MS group displayed decreased maximum knee extension velocity, further explaining why the entire upward movement for the MS group took longer. The timing and magnitude of maximum trunk flexion also corresponds to two different strategies.

There are several possible explanations for this movement strategy. First, as surmised, the strategy may reflect compensations for leg extensor weakness. By having a more flexed trunk at

the beginning of trunk extension, trunk extensor muscles can generate more trunk extension work, thereby increasing the upwards momentum of a massive part of the body. Consequently, this should reduce the amount of body mass that must be raised via knee extensors (Figure 3.6). However, as group differences for HE or KE joint moments were not detected, the validity of this explanation is unknown.

Second, this strategy may increase stability during the transition from sitting to standing, a period of low stability. Inkster et al., (2004) and Papa and Capozzo (2000) have suggested that increased trunk flexion are used by clinical populations to bring the body's COM closer to, if not over, the new base of support prior to SO in an effort to increase stability (Inkster & Eng, 2004; Papa & Cappozzo, 2000). However we found no differences in total anterior displacement of the COM of the body.

Third, performing the movements of trunk flexion, knee extension and trunk extension sequentially, rather than more simultaneously, may make it easier to coordinate the muscle actions and control the movements of the body (Papa & Cappozzo, 2000). This may be effective for MS individuals, as they can have reduced ability to control neural activation of CNS regions controlling movement, posture and/or other related regions (Frohman et al., 2006). More specifically with muscle spasticity often creating eratic and uncontrolled movements (Crippa et al., 2004) a more sequential movement may be preferred.

Previous research has indicated that increased trunk flexion velocity is used to increase the horizontal momentum of the body which is subsequently transferred to vertical momentum. The increase in vertical momentum in turn decreases the KE requirement for the STS movement (Berger, Riley, Mann, & Hodge, 1988; Bernardi et al., 2004; Kerr, White, Barr, & Mollan, 1994; Kerr et al., 1997; Pai & Rogers, 1991; Papa & Cappozzo, 2000). However, increasing trunk

flexion velocity to increase the horizontal momentum of the trunk is more likely used to aid in pulling the entire COM of the body forward right at seatoff. By doing so, it simultaneously moves the proximal end of the thigh segment forward, as well. Hence, the thigh begins to extend about the knee joint with more angular momentum. As this action occurs at the initiation of knee extension, when the greatest magnitude of KE moment for the STS is required, the KE moment needed to get the movement started can be decreased.

Although people with MS in this study have increased muscle weakness when compared to healthy controls, they were still able to perform the STS movement most likely by implementing the flexed trunk strategy. Although this strategy is widely used by clinical populations with muscle weakness, it may increase the risk of lower back injuries as a greater moment requirement may be placed on the trunk extensors (Doorenbosch et al, 1994). It may be beneficial for clinicians to implement resistance training programs that will increase leg extensor strength in an effort to increase STS ability. Furthermore, teaching alternative strategies such as foot placement, and using the hands to push off may also be beneficial.

Limitations

It is possible that participants at the lower compared to the higher end of the disability score spectrum of our study may have displayed different biomechanics, thus also implementing different movement strategies. Our participants' EDSS scores ranged from 0 indicating no visible disability to 6 which is one step below requiring assistive devices for walking. Thompson, et al. (2007), however, found that although MT was associated with EDSS score, no other kinematic differences were discovered among their eight participants with a similar EDSS range. However, his small sample size may have precluded detecting differences. Other limitations include the unstable nature of the MS disease. Each participant with MS has unique set of

symptoms that often vary from day to day. Among these symptoms are the level of fatigue, heat sensitivity, muscle spasticity, and psychological well being. In an effort to reduce the impact of some of these limitations ample rest between STS trials were given and room temperature was adjusted to accommodate participants.

Conclusion

Individuals with relapsing-remitting MS with mild to moderate levels of disability (EDSS scores: < 6.5) displayed less leg extensor strength than healthy individuals. MS individuals exhibited the same trunk flexion strategy used by other clinical populations with lower extremity weakness when performing the STS movement. In addition, the trunk and lower extremities move more sequentially than is typical and may partially explain the increased MT and other kinematic differences of the MS group. The RF of the A-leg was the only muscle that displayed less mRMS than that of the CON group, and no differences for peak joint moment magnitudes but timing differences were detected. Therefore, it is unclear whether abnormal signal transduction or decreased leg extensor strength affected the joint moments. However, the earlier times to peak moments in the MS group may suggest the increased trunk velocity aided in initiating knee extension, but not enough to observe changes in moment magnitudes. Furthermore, it appears that although the MS participants in this study took longer to rise than healthy controls, they displayed faster movement times than other clinical populations. As MS participants were reported to have decreased leg extensor strength and the KE are the major agonists used to extend the body upwards, implementing training programs to improve lower extremity and trunk strength may be beneficial (White & Dressendorfer, 2004; White et al., 2004).

Acknowledgements

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Table 3.1. Participant group descriptives and 1 repetition maximum (1RM) for leg press data (Mean ± SD)

Group	Age (yrs)	Ht (cm)	Mass(kg)	BMI	1RM(N/kg)
MS					
Female (n = 18)	43.1 ± 12.5	164.4 ± 4.9	77.4 ± 19.7	28.5 ± 6.1	70.3 ± 19.9
Male (n = 3)	51.3 ± 1.5	177.3 ± 7.7	89.1 ± 21.0	28.2 ± 5.2	98.8 ± 42.6
Group (n = 21)	44.3 ± 11.9	166.2 ± 7.8	79.1 ± 19.8	28.4 ± 5.9	74.4 ± 25.0*
CON					
Female (n = 10)	41.3 ± 12.2	163.6 ± 7.0	69.0 ± 14.8	25.6 ± 3.6	96.7 ± 27.2
Male (n = 2)	50.0 ± 8.5	175.2 ± 1.6	100.1 ± 24.3	32.7 ± 8.5	122.6 ± 3.4
Group (n = 12)	42.8 ± 11.8	165.5 ± 7.8	72.4 ± 19.5	26.8 ± 5.0	101.0 ± 26.6*

* Significantly different between groups ($p < 0.05$).

Table 3.2. Means ± SD of Maximum joint displacement (°), joint velocities (°/s); GRFs for vertical (VGRF) and posterior (PGRF) (N/BW), joint moments (Nm/BW) and powers (W/BW), and relative times (RT) to these maxima (% MT) of the affected (A-leg) and less affected leg (LA-leg) for both the MS and CON groups.

	A-leg		LA-leg	
	MS	**CON**	**MS**	**CON**
Kinematics				
† Dorsiflexion displ.	20 ± 6	22 ± 6	21 ± 6	19 ± 6
Dorsiflexion velocity	44 ± 10	43 ± 10	46 ± 11	42 ± 11
* Knee extension velocity	116 ± 27	138 ± 29	118 ± 25	137 ± 30
Joint Moments				
PF moment	-0.03 ± 0.01	-0.03 ± 0.01	-0.03 ± 0.02	-0.02 ± 0.01
DF moment	0.01 ± 0.01	0.01 ± 0.004	0.01 ± 0.01	0.01 ± 0.004
KE moment	0.09 ± 0.02	0.09 ± 0.01	0.09 ± 0.02	0.09 ± 0.02
* KE moment RT	40.5 ± 7.1	46.6 ± 4.6	40.9 ± 7.3	46.7 ± 4.5
HE moment	- 0.06 ± 0.02	-0.07 ± 0.01	-0.07 ± 0.03	-0.07 ± 0.01
* HE moment RT	38.0 ± 6.3	42.9 ± 4.7	37.8 ± 7.2	43.3 ± 4.7
Joint Powers				
Positive ankle power	0.67 ± 0.39	0.91 ± 0.37	0.73 ± 0.51	0.72 ± 0.36
* Negative ankle power	-0.45 ± 0.42	-0.27 ± 0.13	-0.53 ± 0.60	-0.22 ± 0.08
Positive knee power	5.61 ± 1.85	6.34 ± 1.67	5.92 ± 2.38	7.28 ± 2.42
* Positive knee power RT	57.7 ± 9.0	64.3 ± 5.7	57.3 ± 10.0	64.6 ± 6.5
Positive hip power	3.05 ± 1.59	3.95 ± 1.05	3.74 ± 1.58	3.74 ± 0.95
Positive hip power RT	53.5 ± 6.4	55.3 ± 5.4	52.4 ± 5.4	55.6 ± 5.7
GRF				
VGRF	0.57 ± 0.14	0.60 ± 0.02	0.59 ± 0.14	0.61 ± 0.04
VGRF RT	54.7 ± 33.4	58.8 ± 26.8	63.8 ± 33.2	50.5 ± 10.5
PGRF	0.02 ± 0.01	0.02 ± 0.01	0.02 ± 0.02	0.02 ± 0.01
ŧ PGRF RT	46.7 ± 25.3	33.2 ± 9.2	57.2 ± 42.3	53.9 ± 41.4

* Significant group main effect $p < 0.05$.

ŧ Significant leg main effect $p < 0.05$.

† Significant (Group × Leg) interaction $p < 0.05$.

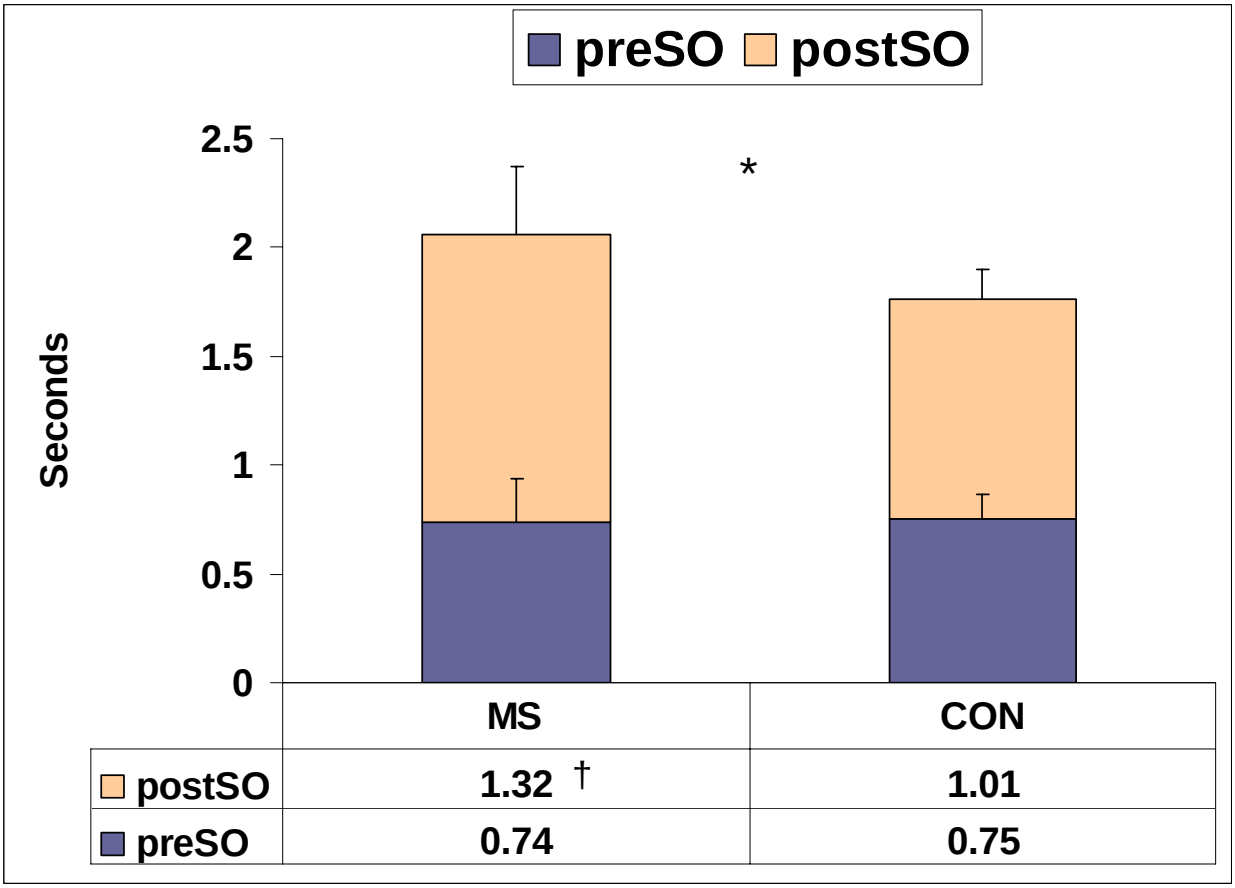

Figure 3.1. Overall rise (MT) and phase times (s) for the STS movement for multiple sclerosis (MS) and control (CON) groups. * indicates group difference for MT ($p < 0.05$); † indicates group differences for post-seatoff phase ($p < 0.01$).

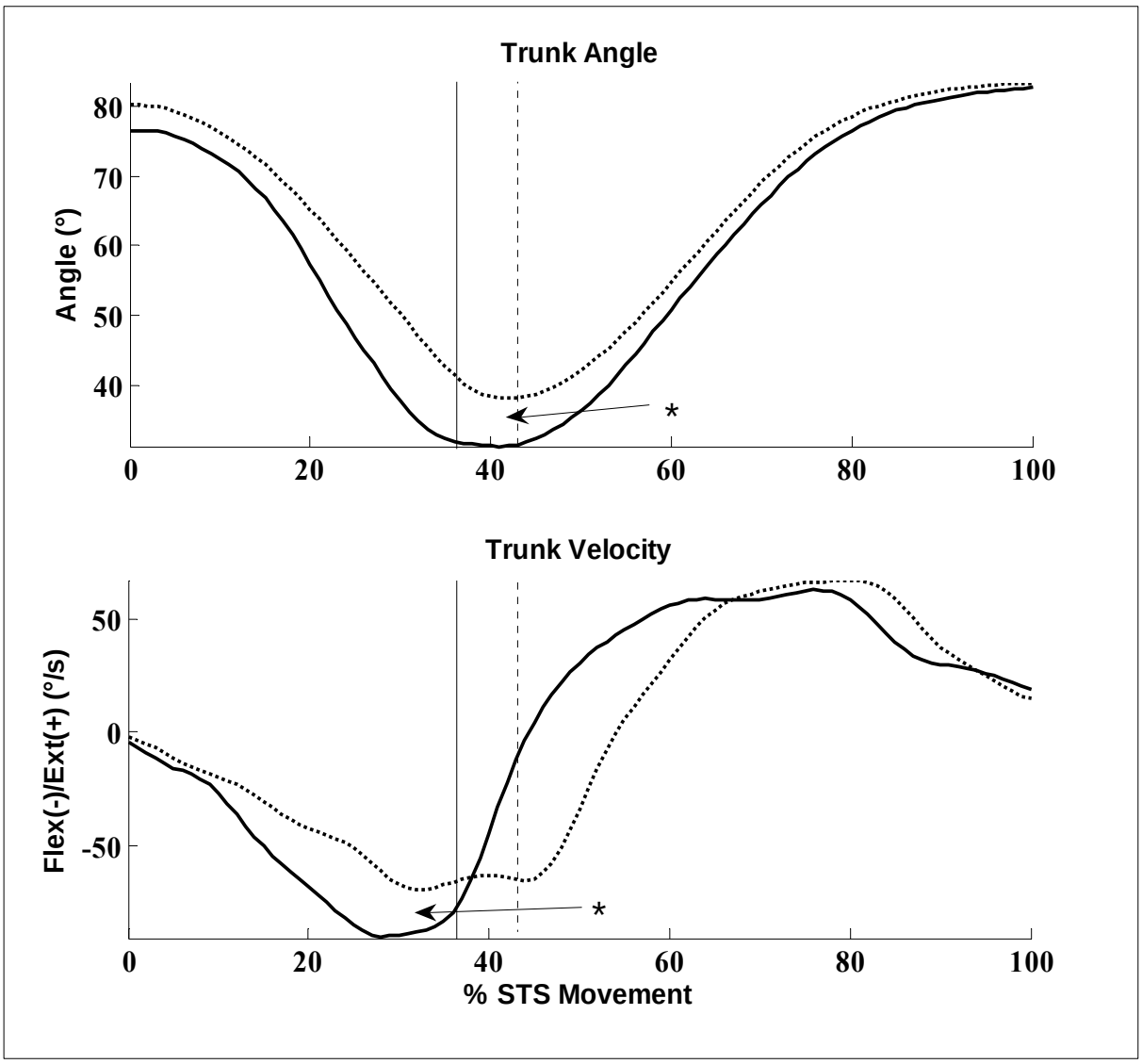

Figure 3.2. Trunk displacement and velocity displayed during the STS for a representative participant of the MS (solid line) and the CON (dotted line) groups with corresponding vertical lines representing seatoff during the STS. * indicates significant difference between groups for maximum magnitudes ($p < 0.05$).

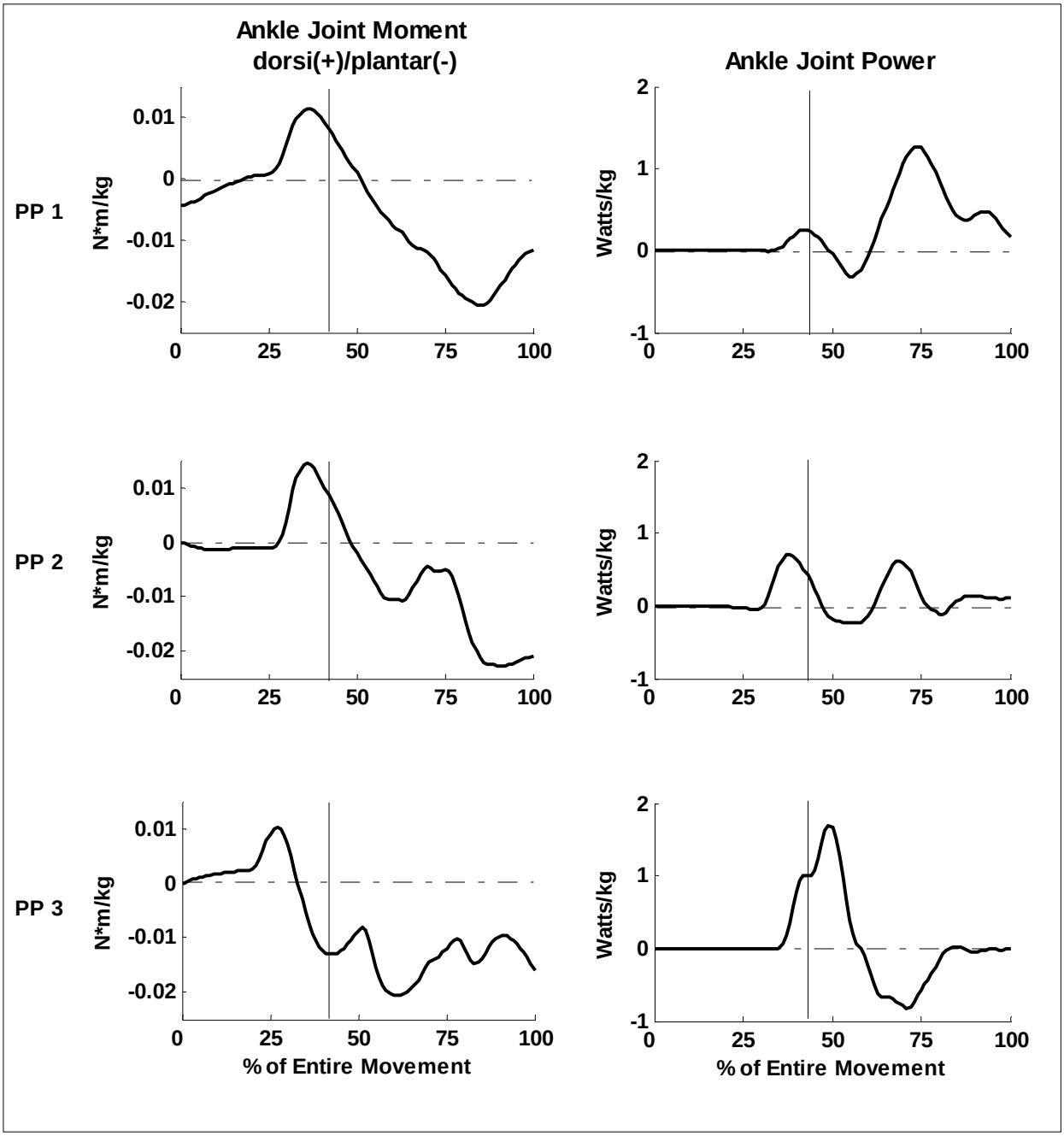

Figure 3.3. Three different and distinct patterns (PP1, PP2, PP3) displayed by participants in both CON and MS groups for sagittal ankle joint moment and power. The solid vertical line represents the moment of seatoff. PP1 and PP3 represent the range of patterns with PP2's patterns within the middle of the range.

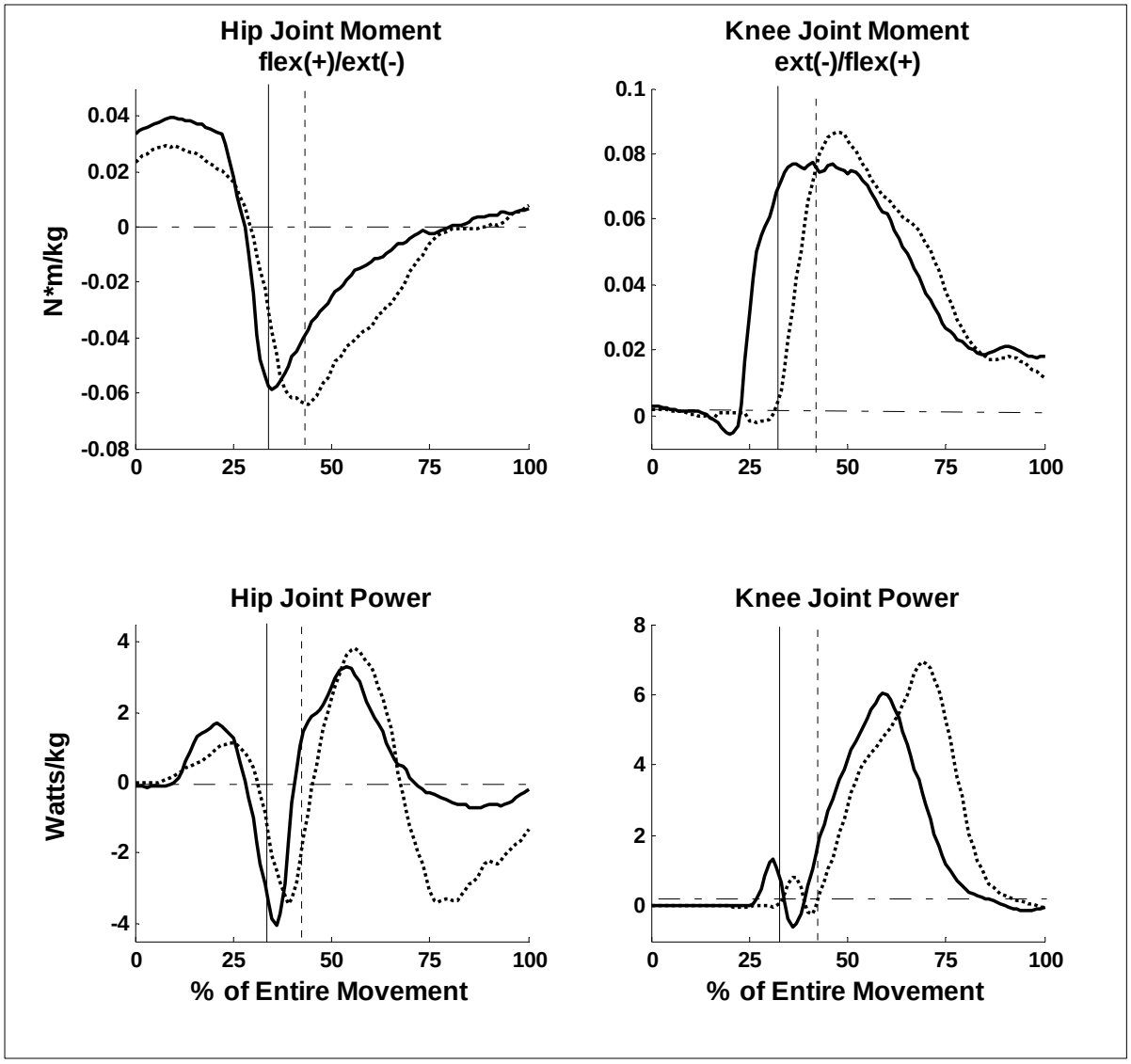

Figure 3.4. Participant joint moment and power curves representing the MS (solid) and CON (dotted) groups with corresponding vertical lines representing the instant of seatoff. Only peaks post seatoff were analyzed for the hip.

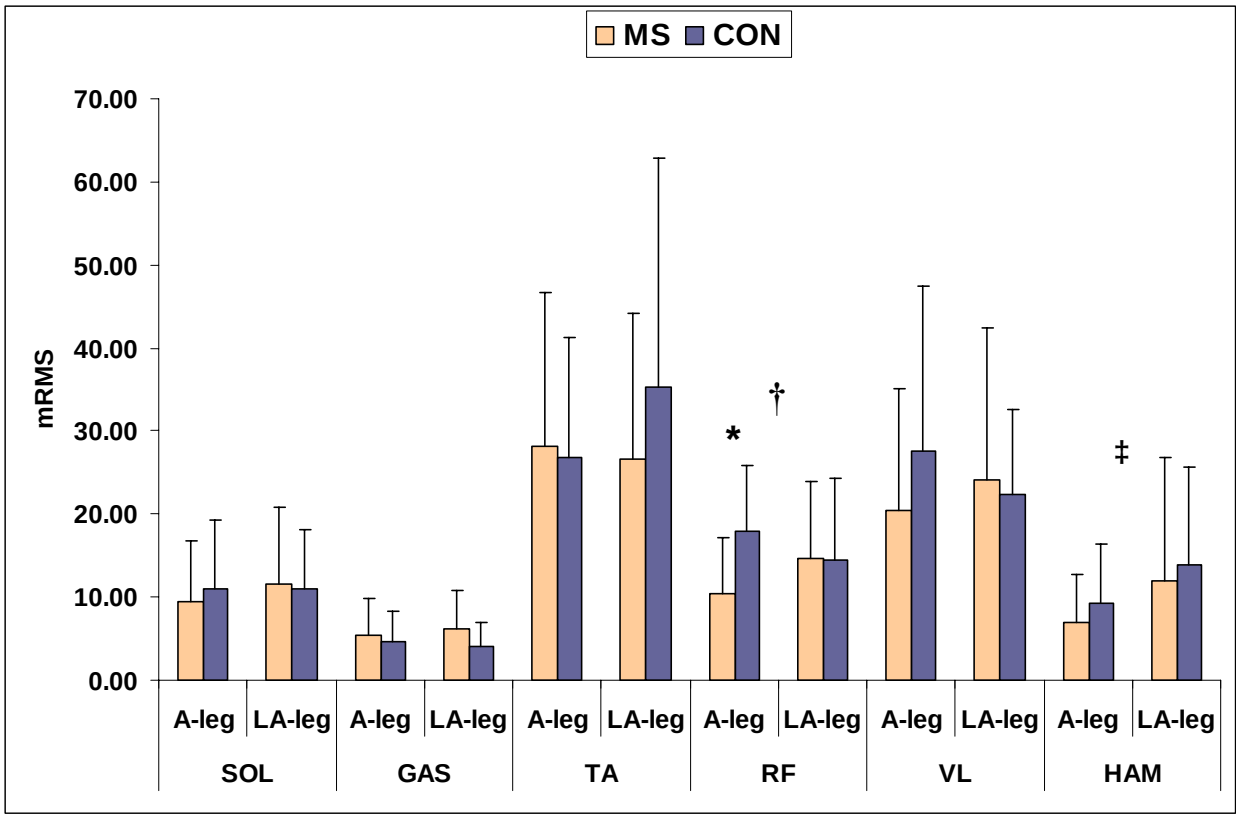

Figure 3.5. Scaled mRMS of the affected (A-leg) and less affected (LA-leg) legs of both groups: soleus (SOL), gastrocnemius (GAS), tibialis anterior (TA), rectus femoris (RF), vastus lateralis (VL) and the combined hamstrings (HAM) in the A-leg and LA-leg. Significant differences: † between Legs of MS group $p < 0.01$; * between groups for the A-leg $p < 0.01$. ‡ between Legs, main effect $p < 0.05$.

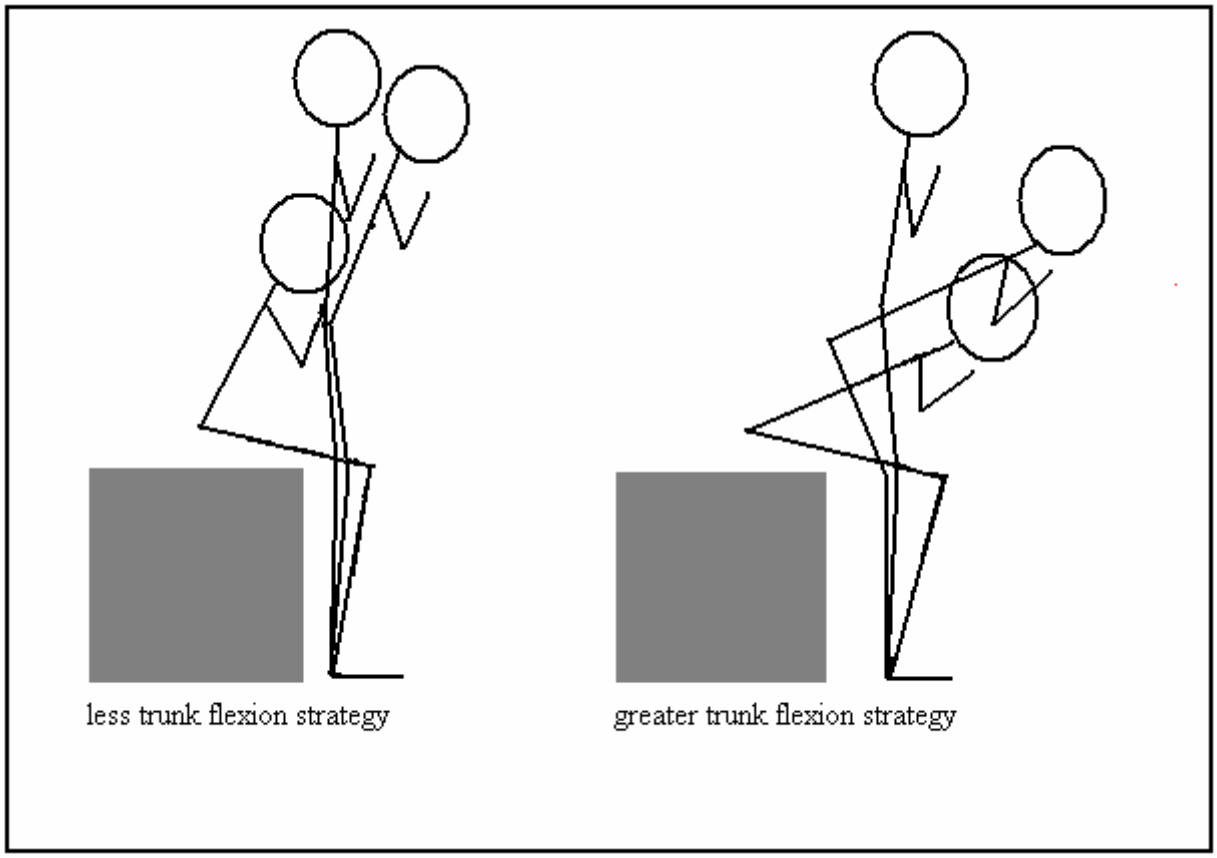

Figure 3.6. Two movement strategies used to perform the STS movement by healthy and clinical populations with lower extremity muscle weakness.

CHAPTER 4

EFFECTS OF FLEXIBILITY TRAINING ON THE BIOMECHANICS OF SIT TO STAND OF PEOPLE WITH MULTIPLE SCLEROSIS[3]

[3] Bowser, B., Larson, R., Simpson, K., and White. L. To be submitted to *Medicine and Science in Sports and Exercise.*

Abstract

Evidence exists that static stretching programs may enhance not only flexibility, but increase strength and reduce spasticity. However, it is not known if individuals with multiple sclerosis (MS) would display these same outcomes after engaging in a flexibility training regimen. Thus, the purpose of this study was to determine if 16 weeks of flexibility training would decrease muscle spasticity; increase leg strength; and during the sit-to-stand (STS) movement, increase lower extremity joint moments, reduce compensatory kinematics, and, consequently, improve the functional performance of people with MS. Nine ambulatory individuals diagnosed with relapsing-remitting MS and an expanded disability status score of ≤ 6 engaged in a 16-week flexibility training program that included three 30 s static stretches of the lower extremity muscles performed 3 times a week. Pre- and post-tests of leg extensor strength, range of motion (ROM), spasticity and STS biomechanics (kinematics, ground reaction forces, joint kinetics, and electromyographic signals from selected lower extremity muscles) were compared using paired sample t-tests and a series of 2 × 2 repeated measures ANOVAs (Pre-Post × Phase) and (Pre-Post × Leg). Post-intervention, participants increased ROM for knee flexion, ankle dorsiflexion and plantarflexion by 17%, 47%, and 20%, respectively. Additionally, maximum posterior ground reaction force and maximum hip extensor moments occurred approximately 5.5% earlier. No other differences were detected. Although no changes in strength and spasticity were found, MS participants were able to increase ROM at the ankle and knee joints, indicating that flexibility training may be beneficial in maintaining muscle function and increasing joint ROM.

Key Words: SIT TO STAND, STATIC STRETCHING, MUSCLE STRENGTH, ELECTROMYOGRAPHY, DISABILITY

Introduction

Multiple sclerosis (MS) is the most common disabling neurological disease of young people, according to Frohman (2003), with the onset of the disease occurring during the 2nd and 3rd decades of life (Noseworthy et al., 2000). MS is a progressive degenerative disease of the central nervous system that, at present, is thought to be an autoimmune disorder that attacks the myelin sheaths, oligodendrocytes, and axons of neurons (Noseworthy et al., 2000; Smith & McDonald, 2003). Nerve conduction, therefore, is disrupted, and, consequently, produces deleterious affects on all major systems of the body. Related to producing voluntary movements, muscle weakness and spasticity, fatigue, and balance impairments occur that lead to functional limitations (Latash, 2008). In turn, such limitations contribute to a more sedentary lifestyle and a downward spiral of reduced strength, increased fatigue, balance impairments, etc. that further encourage inactivity. Hence, sedentary-related illnesses, such as obesity and coronary heart disease, are more likely to occur, thereby further diminishing the individual's health and quality of life (White & Dressendorfer, 2004).

In an effort to improve physical function and ameliorate some of the effects of MS, several researchers have designed both aerobic and resistance training exercise programs. The benefits of these exercise programs for people with MS include: increased cardiorespiratory fitness (Mostert & Kesselring, 2002; Petajan et al., 1996; Ponichtera-Mulcare et al., 1997; Rodgers et al., 1999), increased muscle strength and endurance (DeBolt & McCubbin, 2004; Husted et al., 1999; Mostert & Kesselring, 2002; White et al., 2004), reduced systemic fatigue (Husted et al., 1999; Mostert & Kesselring, 2002), improved mood (Mostert & Kesselring, 2002), and improved functional performance of activities of daily living (ADL)(Gutierrez et al., 2005). Traditional exercise programs, however, may not be feasible for people with MS who

have high levels of MS-related disability; are experiencing MS-related symptoms, such as increased heat sensitivity and fatigability; have orthopedic/other limitations; or do not have access to exercise equipment.

Therefore, as a low-intensity, cost-effective and relatively easy form of exercise, flexibility training may be a viable alternative to traditional exercise (see figure 4.1). No known studies have examined the influence of flexibility training for people with MS without combining flexibility with other exercises such as resistance training (DeBolt & McCubbin, 2004; Husted et al., 1999). Furthermore, little research exists demonstrating that flexibility training improves functional performance for MS and non-MS individuals.

However, the research that has been conducted on flexibility training has found that for healthy populations, flexibility training increased muscle strength and endurance (Kokkonen et al., 2007). Additionally individuals with other types of neurological disorders have exhibited reduced muscle spasticity due to flexibility training (Zhang et al., 2002). It was anticipated, therefore, that such training would have similar benefits for people with MS that could improve functional activities of daily living.

One of the most common ADL that require considerable muscle strength of the lower extremities is rising from a seated position (Riley et al, 1991). We observed that, compared to healthy controls, people with MS, demonstrated functional limitations while performing the STS movement in the form of increased movement time (MT), (Bowser et al., 2009). Furthermore, the MS group compensated for decreased lower extremity strength by using adaptive movement strategies. We therefore predicted that lower extremity flexibility training would alter the biomechanics displayed during the STS movement to that more closely resembling a healthy control group. More specifically, we hypothesized that the training would cause decreased trunk

flexion and trunk flexion velocity, increased knee extensor (KE) and decreased hip extensor (HE) joint moments, increased electromyographic (EMG) activity of the KE muscles, and decreased EMG of the HE muscles. Therefore, to assess our predictions, the purpose of this study was to determine if 16 weeks of flexibility training would improve the functional performance and biomechanics exhibited by individuals with MS during the STS movement.

Methods

Participants

Eleven volunteers with MS were recruited for this study from the local MS support group meetings and neurology clinics. All individuals provided written informed consent as approved by our institution's human subjects review board prior to participating in the study. All potential participants filled out a medical history questionnaire to determine if they met the following criteria: a) had relapsing-remitting MS, b) an expanded disability status score (EDSS) of < 6.5 (Kurtzke, 1983) as diagnosed by their neurologist; c) had no other orthopedic or other limitations unrelated to MS; and d) had not used prednisone or other steroids for an exacerbations during the previous 3 mo. Participants using immunomodulatory agents and/or spasticity medications during the study were included if a consistent regimen had been established prior to pre-testing.

Experimental Design and Test Sessions

A pre-post treatment design was used. For each participant, pretest measures were obtained during the first four weeks, followed by 16 wk of flexibility training and then 2 wks of post-intervention testing. For pre-testing, an initial visit consisted of filling out the health questionnaire to determine eligibility, an explanation of the study, and scheduling of the subsequent sessions. For each of the following, two separate visits with a minimum of 2 days separation were used to obtain double baseline measures: a) lower extremity muscle strength,

and b) lower extremity spasticity and flexibility. A separate additional session was used to assess STS biomechanics.

For the intervention, participants attended 60-90 min. training sessions at the same time of day 3 times a week with a minimum of one day rest between each session. For the post-testing, during the last two weeks of the experimental period, all of the initial measurements were re-evaluated using the baseline protocols.

Tasks and Measurements

Leg extensor strength of the lower body was assessed using a Cybex® leg press machine (Cybex International Inc. Medway, MA) following the one-repetition max (1RM) protocol used by Barnard et al. (1999). Isokinetic knee extension strength was assessed on right and left legs separately using a Kin-Com® isokinetic dynamometer (Kin-Com®, Chattanooga Group, Inc., Hixson, TN) at 60°/s and 180°/s. Following a warm-up involving 2-5 sub-maximal isokinetic knee extension actions, two maximal effort trials were performed at each predetermined speed, with five minutes of rest between trials.

Spasticity and sagittal plane range of motion (ROM) were assessed for each leg. Spasticity was measured using the Ashworth spasticity scale (Ashworth, 1964; Bohannon & Smith, 1987), while procedures for obtaining ROM measures for ankle, hip, and knee flexion and extension followed the method by Etnyre and Abraham (1986). The researcher who performed all spasticity and ROM measurements had a mean intra-rater reliability ICC = 0.96 across all ROM measurements.

For the biomechanical testing of the STS movement, after anthropometric data (Zatsiorsky, 2002) were obtained, the skin was prepared for EMG electrode placement by lightly abrading and cleaning the skin. Bipolar Ag-Ag CL disposable electrodes (Biopac System Inc.,

Goleta, CA, 3.49 cm diameter) were then placed at an inter-electrode distance of 2 cm on the tibialis anterior (TA), gastrocnemius (GAS), soleus (SOL), vastus lateralis (VL), rectus femoris (RF), and hamstrings (HAM) bilaterally following the method described by Cram and Kasman (1998). The ground electrode was placed on the tibial tuberosity of the right leg. During pre-testing, the location of the center of each electrode was measured based from bony landmarks (see figure 4.2). These measurements were used to place the electrodes at the same anatomical locations at the post-test. Raw signals were amplified/conditioned in the datapack worn on the participant's back, transmitted via optic fiber cable to the receiver and then sent into the computer for later analysis using the Myopac® EMG system (Run Technologies, Inc., Mission Viejo, CA; 1000 Hz, 16-bit ADC, CMRR = 10,000:1).

The participant held a weight (108 N) hung at the distal end of the leg or foot during several different static positions of each limb to obtain isometric EMG of all instrumented muscles (details in Bowser, et al., 2009). These data were used to later scale the EMG signals obtained for the STS trials.

To capture the spatial locations of both lower extremities during the STS task, Flock of Birds™ position sensors (Ascension Technologies Corp. Burlington, VT) were placed bi-laterally on the feet, shanks, thighs, sacrum and sternum as previously described by Bowser et al. (2009). During natural standing with the body aligned as close to neutral as possible, joint center locations were then identified within the electromagnetic field. *The MotionMonitor*™ (Innovative Sports Training, Chicago, IL) electromagnetic tracking system was used to capture 3D kinematic data at 100 Hz during the STS movement. Following ISB guidelines,Euler angles (Euler, Z Y′ X′′) were calculated and adjusted relative to natural standing with sagittal plane

movement occurring around the third rotation. The trunk inclination angle was calculated relative to the positive y axis.

The sitting surface was adjusted so that the lower extremities were aligned at joint angles of approximately 90° in the sagittal plane, (Janssen, Bussmann, & Stam, 2002), with each foot on one of two Bertec® force platforms (Model #4060-NC: 1000 Hz). Participants raised the body to a standing position at a self-selected speed, with the arms folded across the chest and without realigning the feet during the movement, then stood quietly for 5 s. All participants were given a minimum of two practice trials before five experimental trials of the STS were performed. Additionally, as MS can affect one leg to a greater degree than the other leg, prior to pre-test data collection, each participant was asked to identify which leg was more affected by MS.

During the 16- wk training period, participants engaged in a supervised flexibility training program (White & Dressendorfer, 2004) that was based on the American College of Sports Medicine guidelines for musculoskeletal flexibility (*ACSM's Guidlines for Exercise Testing and Prescription*, 2000). Active, static stretches of the hip abductor, adductor, flexor and extensor; knee flexor and extensor; and ankle plantar- and dorsiflexor muscles and soft tissues were performed 3 times each for 30s.

Data Reduction

For the strength measures (scaled to body mass), the peak torques for isokinetic strength data and the maximum weight lifted for leg press tests were selected for analysis. From the two baseline measures obtained, the second session values for the strength data and the average of the two baseline measures for ROM and spasticity were used to compare with the corresponding measure obtained at post-testing.

The starting and ending points of the STS movement were defined by center of mass (COM) parameters. The instant the horizontal COM velocity was > 0 $m \cdot s^{-1}$ and continued to increase was the start point whereas; the end of the movement was the first instant when the body's vertical COM stopped displacing upwards (Yu et al., 2000). The seat off was defined as the instant when the value of the anterior GRF attained its greatest value (Kralj et al., 1990).

The raw EMG signals were bandpass- (5-178Hz) and notch- filtered (60 and 100 Hz; 4Hz notch width) based on a frequency spectrum analysis. Using a Matlab® (v. 7.0) program, the mean of the root-mean square (mRMS) values (T = 50 ms) for each muscle during the entire STS movement was calculated (Cram & Kasman,1998) and scaled to the mRMS of sub-maximal isometric contractions of each of the associated muscle groups and used for comparisons.

Fourth-order, recursive low-pass Butterworth filters with cut-off frequencies of 5 and 20 Hz respectively (Mak et al,, 2003), were used for kinematic and GRF data. Spatial locations of the Flock of Birds™ position sensors (Ascension Technologies Corp. Burlington, VT) were reconstructed and kinematic and kinetic quantities were generated using *The MotionMonitor*™ software (v. 7). To calculate joint moments all joints of lower extremities, the modified Zatziorsky et al.'s equations (2002) that are gender-specific were used to generate the anthropometric-dependent quantities needed in the inverse-dynamics model of Gagnon and Gagnon (1992). All GRF and joint moment data were scaled to body weight.

Statistical analysis

Variables for statistical analysis (laboratory program, Matlab® v.7.0) included: total movement time (MT), maximum and minimum magnitudes, and relative times (RT) to these maxima/minima for kinematic and kinetic variables. The mean of the 5 trials for each participant for each variable were used for comparisons. Using SPSS (v. 16.0) (SPSS Inc., Chicago, IL)

paired-sample t-tests were used to compare pre- and posttest values on trunk kinematics, COM displacement, 1RM strength, and relative timing of seatoff. A series of 2 × 2 repeated measures ANOVAs (Pre-Post × Phase[RM]) for MT and (Pre-Post × Leg[RM]) for kinetic, EMG and the remaining kinematic variables were used to assess Pre-Post as well as Phase and Leg differences. Leg differences were tested to determine if the affected limb (A-leg) displayed different biomechanics than the less-affected limb (LA-leg). For the 2 x 2 ANOVAs, significant interactions ($p < 0.05$) were followed by post hoc comparisons implementing a modified-Bonferoni adjustment with an accepted family-wise error rate of $\alpha = 0.05$. Hochberg's modified adjustment orders the p-values then "rejects all hypotheses with smaller or equal *p*-values to that of any one found less than its critical value (Hochberg, 1988)." Effect size calculations using eta squared (η^2) were calculated for all comparisons.

Results[4]

Physical characteristics are reported for the 9 participants who completed the entire study in Table 4.1. Compliance ranged from 71% to 95% with an average compliance of 87%. The other two participants dropped out due to schedule conflicts. No Pre-Post differences were found for the participants' physical characteristics.

Changes for joint ROM are reported in Table 4.2. A significant Pre-Post main effect was found for knee flexion ($F(1,8) = 10.84$, $p = 0.013$, $\eta^2=0.61$) and ankle dorsiflexion ROM ($F(1,8) = 6.03$, $p = 0.04$, $\eta^2 = 0.46$) indicating increased joint ROM. A significant Pre-Post × Leg interaction was found for plantarflexion ($F(1,8) = 5.89$, $p = 0.046$, $\eta^2 = 0.46$) with post hoc analysis indicating that both legs displayed increases in plantar flexion ROM between 8-11° (A-leg $p < 0.05$, $\eta^2 =0.5$ and LA-leg $p < 0.02$, $\eta^2 =0.57$). No other joint ROM differences were found.

[4] For dissertation: ANOVA summary tables for all variables can be found in appendix E.

For lower extremity spasticity, no Pre-Post or Leg differences were detected (mean±SD; Pre 2.58±1.08, Post 2.92±0.67, $p > .18$). The strength measures are reported in Table 4.3. No Pre-Post differences where found for 1RM leg press ($p = 0.397$) or maximum isokinetic KE torque produced at 60°/s or 180°/s ($t(8) < p > 0.29$ and >0.34, respectively) ($F(1,8) < 1.03$ for all strength variables.

The STS kinematic and kinetic variables are reported and shown in Table 4.4 and Figure 4.3, respectively. Only the RT to maximum posterior GRF and RT to maximum HE moment displayed significant Pre-Post differences. The RT of peak posterior GRF occurred approximately 6% later following training ($F(1,8) = 6.20$, $p = 0.03$, $\eta^2 = 0.44$). Congruent with the peak posterior GRF finding, the maximum HE moment occurred at an average of 5% MT later ($F(1,8) = 6.84$, $p = 0.03$, $\eta 2 = 0.46$). No other kinematic or kinetic differences were found. Furthermore, no main effects or interactions for EMG were discovered (Figure 4.3). The statistical power for these variables ranged from 0.05 to 0.43.

Discussion

Traditionally, the purpose of flexibility training is to increase the ROM at particular joints. We also had surmised that flexibility training could reduce spasticity and potentially, increase muscle strength. In turn, we predicted that greater joint moments and power could be generated during the STS, thereby also allowing the person to stand using a more typical kinematic STS strategy. In general, we found that flexibility did improve with training, and a few kinematic and times to kinetic variables also changed, suggesting that some behaviorally meaningful changes were detected. However, many of our predictions were not supported.

As MS is a progressive disease that affects individuals differentially, we believe that certain characteristics of MS may be of central importance in explaining the lack of more

statistically-significant outcomes. One typical feature of MS is that symptoms such as spasticity or fatigue often vary from day to day (Starsky et al., 2005). In addition, among individuals, the regions in the CNS, and thus, the specific muscles of the body affected by MS vary considerably among individuals (Frohman, Racke, & Raine, 2006). Thus, the mere fact that the MS group did not display decreased joint ROM and muscle strength, nor increased muscle spasticity over the period of 16 weeks may indicate that flexibility training may, at the least, be beneficial in maintaining functional ability.

It was encouraging that increased ROM for knee flexion, and ankle plantarflexion and dorsiflexion occurred after our MS participants performed 30 s. static stretches 3 times a week for 16 weeks. The length of the program, frequency of visits, number and types of stretches, and how long the stretches were held are similar or greater than other flexibility programs that have reported improvements in joint ROM (Feland et al., 2001; Kokkonen et al., 2007; Roberts & Wilson, 1999; Zhang et al., 2002). It is likely that the lack of statistical difference for knee extension ROM was due primarily to the participants' pre-test ROM values being already close to the mechanical limits of knee extension.

Reasons for a lack of improved ROM for hip flexion/extension are less clear. Although qualitatively the hip ROM appeared to increase, the increase was not great enough, or the within-group variation too sizeable, to detect statistical differences. This is reflected by the insufficient statistical power, ranging from 0.07 to 0.27. Lack of statistical significance may also be attributed to the mechanical limitations imposed by the segment tissues (e.g., increased adiposity) of the thigh and pelvis. Additionally, to increase hip ROM to a statistically meaningful degree may have required that stretches be performed at higher intensities. Although performances of the stretches were monitored visually by a researcher, the intensity of each

stretch may have varied, as the participants were responsible for selecting how far to move the limb. Furthermore, as spasticity did not decrease with training, co-contractions occurring during training sessions may have occurred which may have limited the joint position attained, subsequently limiting the stretch overload. Moreover, immunomodulatory agents and/or spasticity medications taken by participants in this study may have limited their ability to improve joint ROM at the hip.

No changes in spasticity were found using the Ashworth scale. Zhang et al. (2002) reported that non-MS neurological patients increased ROM following static stretching, and decreased in muscle spasticity. One potential reason for the different spasticity outcomes between this and Zhang et al.'s study may be the type of static stretch. In our study participants performed active static stretches whereas Zhang et al. (2002) had the participants doing passive stretches of known intensity. Furthermore, flexibility training targets not only the muscles themselves, but also the musculotendinous structures as well as joint capsule and ligamentous structures making it difficult to accurately assess the exact mechanism responsible for increases in joint ROM and no detectable differences in muscle spasticity.

Another possible explanation could be that the pre-test levels of spasticity exhibited by the participants in the study by Zhang et al. (2002) were much higher than the relatively low levels of spasticity reported for our MS participants. A muscle with high muscle tone often has exaggerated reflexes (Barnes et al., 2003). A static stretch in essence is reported to 'reset' the sensitivity of muscle spindles which can subsequently reduce the exaggerated reflex (Ferber et al., 2002). For this reason, it is likely that higher degrees of muscle spasticity may have greater degrees of change or improvement from static stretching. Additionally, participants in this study were permitted to take antispasmodic drugs in order to reduce their spasticity. Therefore,

participants who used these drugs may have limited their capability to further reduce their level of spasticity following flexibility training. For individuals with MS the level of spasticity can vary from day to day (Starsky et al., 2005). Furthermore, the Ashworth spasticity scale may not be the most accurate measure based on the use qualitative observations for scoring. Thus, it is difficult to test spasticity changes during MS intervention studies.

In addition to the hypothesis that flexibility training would decrease muscle spasticity, we had hypothesized that increased leg extensor strength could be a secondary benefit of flexibility training for people with MS. Strength gains following several days of performing static stretches have been reported for nonMS individuals (Kokkonen et al., 2007; Worrell, Smith, & Winegardner, 1994). It is suggested that one of the mechanisms responsible for strength gain following flexibility training is myoblast proliferation that, subsequently, leads to muscle hypertrophy (Day et al, 1997). Additionally, it is reported that in animal models prolonged static stretches ranging from 40 minutes to several days resulted in increases in both serial sarcomeres and cross-sectional area of muscles (Dix & Eisenberg, 1990; Stauber et al., 1994). However, no detectable strength gains were found in the MS participants of this study. Although unspecified, many of the stretches in the Kokkonen et al. (2007) study were performed passively, that is, an external source of force was used to move a given limb into a stretched position. It is difficult to pinpoint the exact mechanism responsible for the increases in strength when participating in flexibility training. However, studies that have reported strength gains from static stretching required static stretches that were either held statically for long periods of time (over 40 minutes) or were placed in ROM positions that can only be attained passively. Passive static stretches may provide a stronger stimulus that may possibly lead to strength gains.

While it is likely that an increase in ROM does not directly cause strength gains there appears to be a positive correlation. Worrel et al., (1994) reported that two weeks of passive static stretches resulted in a 21.3% increase in hamstring ROM and was accompanied by an 11% increase in maximum concentric isokinetic torque (180°/s). Furthermore, Kokkonen et al. (2007) reported that, for healthy college-aged participants, a series of active and passive15 s. stretches 3 times a week for 10 weeks resulted in an 18% increase in lower extremity flexibility accompanied by a 32% increase in leg extensor strength. In the current study, MS participants displayed ROM gains similar to those reported by Kokkonen et al. (2007) at the ankle joint: plantarflexion and dorsiflexion increased by 31% and 19%, respectively. However, significant, but lower gains for knee flexion ROM (8%) were displayed. The muscles stretched during a knee flexion ROM assessment (knee extensors) showed the smallest improvement in ROM among the muscle groups in this study that displayed significant increases in ROM. Smaller increases in ROM of the knee extensor muscles (major muscles used to raise the body during STS) may further indicate that the intensity of the stretches may have been great enough to increase joint ROM at certain joints, but not enough to induce strength gains.

Although it cannot be proven in this study, it is possible that individuals with relapsing-remitting MS may respond differently to flexibility training compared to healthy, young adults. Additionally, other symptoms associated with MS e.g. muscle spasticity, balance impairments etc, may mask existing strength gains or may make it more difficult to obtain the strength gains reported in other populations.

Finally, we hypothesized that lower extremity flexibility training would alter the biomechanics displayed during the STS movement to that more closely resembling a healthy control group. We surmised that flexibility training would increase muscle strength, decrease

muscle spasticity, and decrease EMG activation. With no changes detected for muscle strength and muscle spasticity with training, and no training influence on muscle activation of selected leg muscles, it was not surprising that increased magnitudes of lower extremity moments were not detected. Consequently, functional performance (MT) and STS movement strategies also did not display training-induced differences.

The only Pre-Post biomechanical changes that were exhibited were decreased trunk extension velocity and decreased RTs to the peak magnitudes of the anterior-directed GRF and HE moment. At this time, without other significant kinematic or kinetic outcomes that may underlie or explain why these outcomes occurred, it is unclear whether these differences are behaviorally meaningful.

Although the group outcomes did not display as many significant improvements as predicted, the majority of participants reported anecdotally that they felt stronger. They commented that they were able to perform other activities, such as being able to lift their bodies out of the bathtub or stand up to wash dishes without loss of balance, for the first time in several years. They also reported that they felt that they could perform these activities safely. Hence, engaging in this flexibility training may have improved the performers' confidence. Future investigations may also find that some individuals do, in fact, improve strength with this program, while others do not.

Likely, the primary limitations potentially precluding detecting greater number of significant biomechanical findings was high group variability. High variability may be due to the disability levels (EDSS) in our study displaying a range from 0 to 6. Functionally, the difference between a 0 and a 6 on the EDSS is the difference of fully ambulatory individual to an individual who is barely able to walk without an assistive device. Those with higher disability may have

better outcomes from flexibility training, but this prediction was not testable without a larger sample size. Furthermore, high intra-group variability of kinematics and kinetics exhibited during the STS may further be explained by MS affecting each person in a unique manner.

An associated potential limitation of the study was the inter-day rater reliability when assessing both joint ROM and the spasticity using the Ashworth scale. To minimize this limitation the same researcher performed all ROM testing and displayed a high average intra-rater reliability (ICC = 0.96). Furthermore, all STS protocols were administered consistently by the same researchers who used the pre-test measurements to locate the post-test spatial locations of the EMG electrodes. One final limitation may have been the MS-related medications our participants were taking. These medications may have affected the participants differently during the pre-and post sessions. However, this limitation was minimized by requiring that potential participants on medications be enrolled in the study only if a consistent regimen had been established prior to and maintained during the study. Furthermore, it would have been unethical and the findings ungeneralizable to actual daily life to ask individuals not to take their previously prescribed medications during the study.

In conclusion, significant improvements in joint ROM were found at the knee and ankle joints. However, neither strength, spasticity nor meaningful STS biomechanical differences were discovered. It is likely that the differential effects MS has on individuals increased the group variability such that reduced statistical power occurred. However, it is also possible that to induce these predicted outcomes, a more intense stretching protocol involving passive, rather than active stretching tasks may be needed. At the least, it appears that not only can ROM and function be improved by participating in a flexibility program, but strength is maintained, which is significant for a population with a progressive, degenerative disease.

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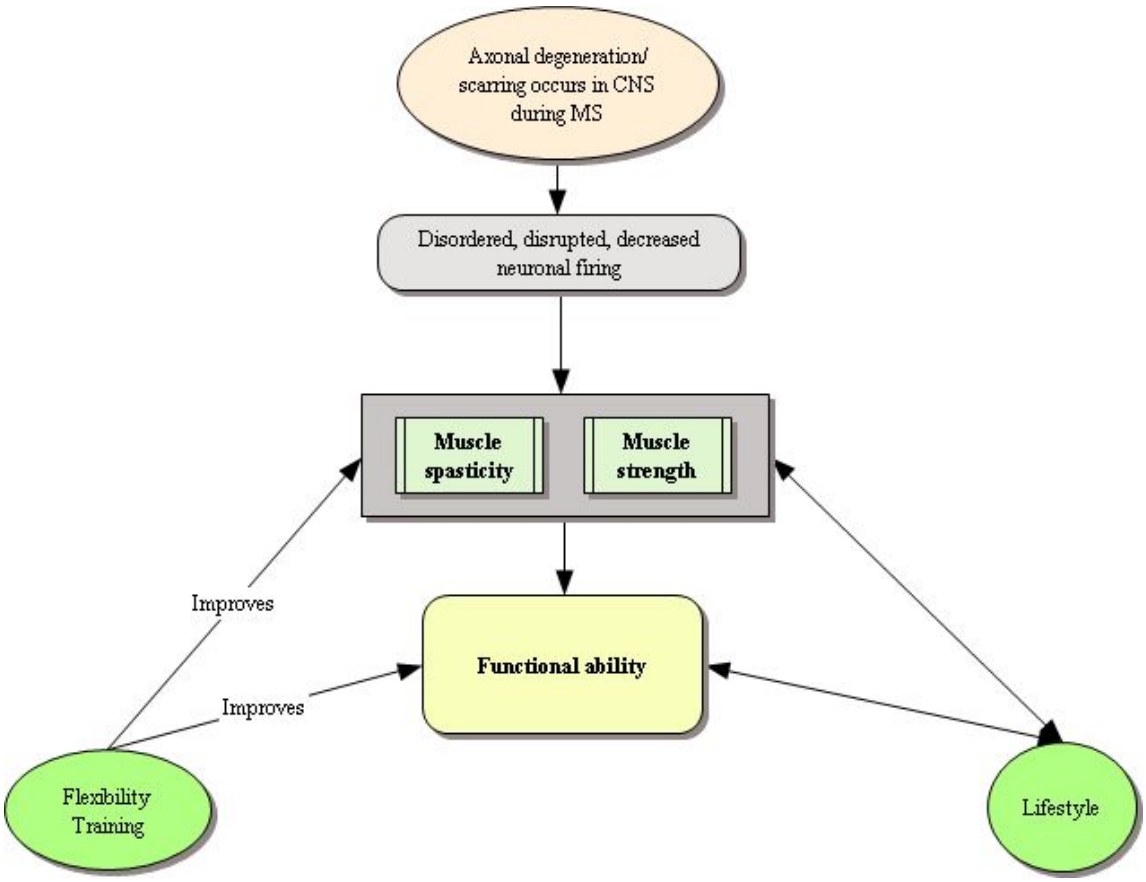

Figure 4.1. Flow chart of how Flexibility training influences other MS-related variables. Courtesy of Kathy Simpson and Bradley Bowser.

Figure 4.2. Spatial locations of the center of each electrode were measured during the pre-test and used to determine post-test placement.

Table 4.1. Participant descriptive data (means ± SD).

			Mass (kg)		BMI	
Group	**Age(years)**	**Height(cm)**	**Pre**	**Post**	**Pre**	**Post**
Male (*n* = 2)	51.5 ± 2.1	175.0 ± 9.2	77.2 ± 5.2	76.4 ± 8.6	25.2 ± 1.0	24.9 ± 0.2
Female (*n* = 7)	45.8 ± 14.3	166.4 ± 4.8	84.0 ± 22.1	82.6 ± 24.5	30.1 ± 6.8	29.6 ± 7.8
Group (*n* = 9)	47.1 ± 12.6	68.3 ± 6.5	82.5 ± 19.5	81.2 ± 21.6	29.0 ± 6.3	28.6 ± 7.0

Table 4.2. ROM Means ± SD (degrees) for Pre- and Post-test measures of the affected and less-affected leg .

	Affected Leg		Less Affected-Leg	
	Pre	**Post**	**Pre**	**Post**
* Knee flexion	129 ± 12	140 ± 9	130 ± 17	142 ± 9
Knee extension	1 ± 2	1 ± 1	1 ± 2	2 ± 1
* Dorsiflexion	10 ± 5	13 ± 6	9 ± 5	15 ± 5
† Plantarflexion	45 ± 17	54 ± 8	42 ± 15	57 ± 7
Hip extension	7 ± 6	13 ± 4	8 ± 5	15 ± 4
Hip flexion	116 ± 14	120 ± 13	116 ± 14	124 ± 10

* significant Pre-Post main effect $p < 0.05$.

† significant Pre-Post × Leg interaction $p < 0.05$.

Table 4.3. Pre- and post –test strength means ± SD for 1 repetition maximum (1RM) leg press ($N \cdot kg^{-1}$) and isokinetic knee extension torque of the affected and less-affected leg ($N \cdot m \cdot kg^{-1}$).

Strength Measure	**Pre**		**Post**	
1RM leg press	71.6 ± 30.3		75.3 ± 23.4	
	Affected Leg		**Less-Affected Leg**	
	Pre	**Post**	**Pre**	**Post**
Knee extension 60°/s	1.05 ± 0.5	1.02 ± 0.4	1.09 ± 0.5	1.00 ± 0.4
Knee extension 180°/s	0.75 ± 0.2	0.75 ± 0.3	0.72 ± 0.2	0.64 ± 0.2

Table 4.4. Maxima for joint angles (°), joint velocities (°/s), GRF (N/BW), joint moments (Nm/BW), joint powers (Watts/BW) and relative time (RT) to these maximums (% of entire STS movement) of the affect leg (A-leg) and less-affected leg (LA-leg) before (Pre) and after (Post) 16 weeks of flexibility training (Mean ± SD).

	A-leg		LA-leg	
Variable	**Pre**	**Post**	**Pre**	**Post**
GRF				
Maximum VGRF	0.60 ± 0.04	0.59 ± 0.04	0.62 ± 0.05	0.61 ± 0.04
VGRF RT	52.9 ± 18.0	52.1 ± 21.7	71.1 ± 41.0	75.6 ± 31.9
Maximum PGRF	0.01 ± 0.01	0.019 ± 0.02	0.014 ± 0.02	0.018 ± 0.01
* PGRF RT	36.1 ± 6.9	41.1 ± 6.9	34.9 ± 8.0	40.3 ± 5.6
Joint Moments				
PF moment	-0.03 ± 0.01	-0.04 ± 0.02	-0.04 ± 0.02	-0.03 ± 0.01
DF moment	0.01 ± 0.01	0.01 ± 0.01	0.01 ± 0.01	0.01 ± 0.01
KE moment	0.09 ± 0.02	0.09 ± 0.02	0.09 ± 0.03	0.09 ± 0.02
KE moment RT	38.6 ± 7.1	42.2 ± 3.6	38.1 ± 5.5	41.0 ± 3.3
HE moment	-0.05 ± 0.02	-0.07 ± 0.03	-0.07 ± 0.03	-0.06 ± 0.02
* HE moment RT	36.1 ± 6.9	41.1 ± 6.9	34.9 ± 8.0	40.3 ± 5.6
Joint Powers				
Positive ankle power	0.60 ± 0.4	0.84 ± 0.5	0.76 ± 0.7	0.88 ± 0.4
Negative ankle power	-0.49 ± 0.4	-0.97 ± 1.1	-0.79 ± 0.8	-0.68 ± 0.4
Positive knee power	5.53 ± 1.4	5.39 ± 1.7	5.83 ± 3.1	6.07 ± 1.7
Positive knee power RT	53.7 ± 10.1	54.8 ± 10.9	53.6 ± 8.2	58.0 ± 6.9
Positive hip power	2.75 ± 1.5	3.89 ± 1.9	3.87 ± 1.7	3.65 ± 1.6
Positive hip power RT	52.4 ± 7.2	55.8 ± 10.7	50.7 ± 6.5	57.3 ± 7.1
Kinematics				
Maximum Dorsiflexion	19 ± 7	18 ± 9	22 ± 7	22 ± 5
Dorsiflexion velocity	44 ± 9	42 ± 6	45 ± 12	44 ± 9
Plantarflexion velocity	-33 ± 10	-34 ± 13	-36 ± 9	-39 ± 11
Knee velocity	109 ± 18	117 ± 26	116 ± 19	120 ± 25
	Pre		**Post**	
Trunk flexion displacement	36 ± 17		37 ± 15	
Trunk flexion velocity	- 98 ± 34		- 93 ± 31	
*Trunk extension velocity	73 ± 17		80 ± 20	

* significant Pre-Post main effect $p < 0.05$.

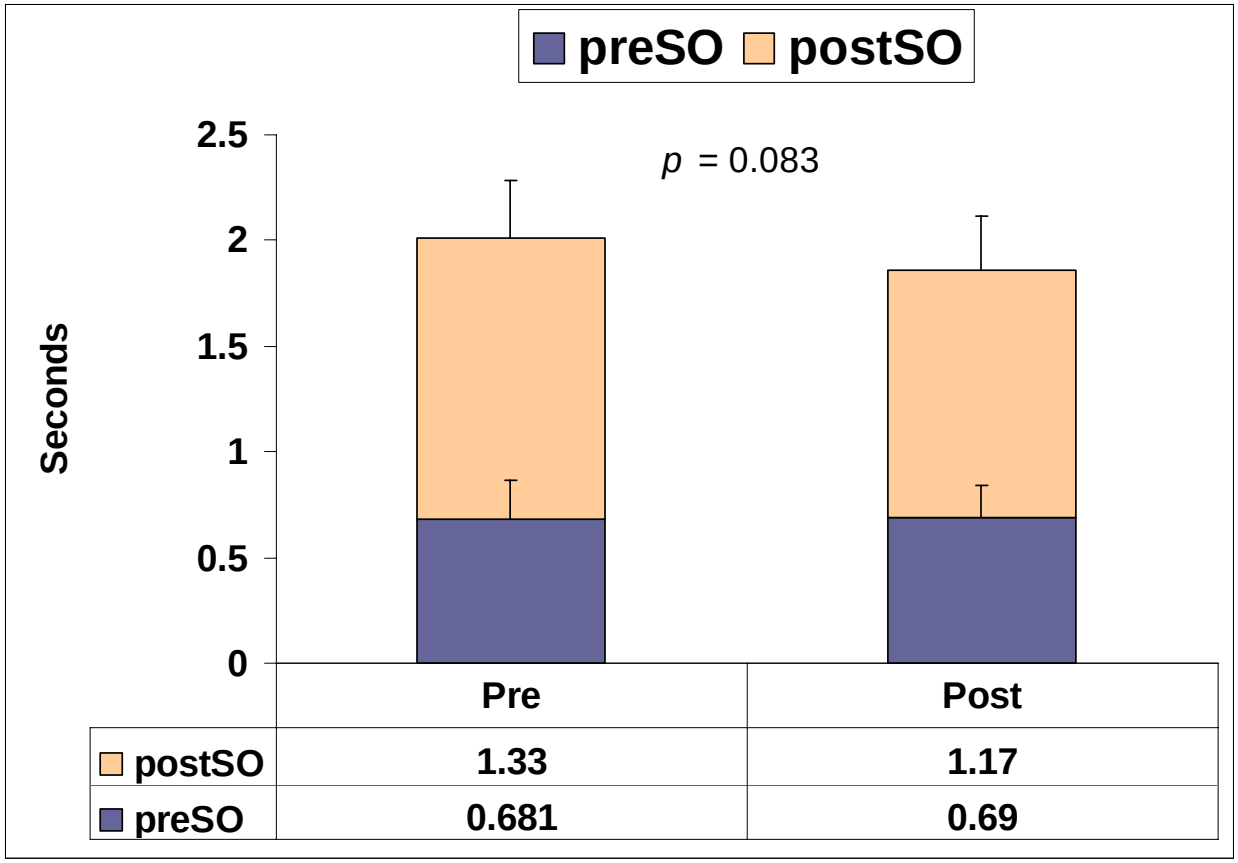

Figure 4.3. Overall rise and phase time for the STS movement Pre and Post the 16 week flexibility training program.

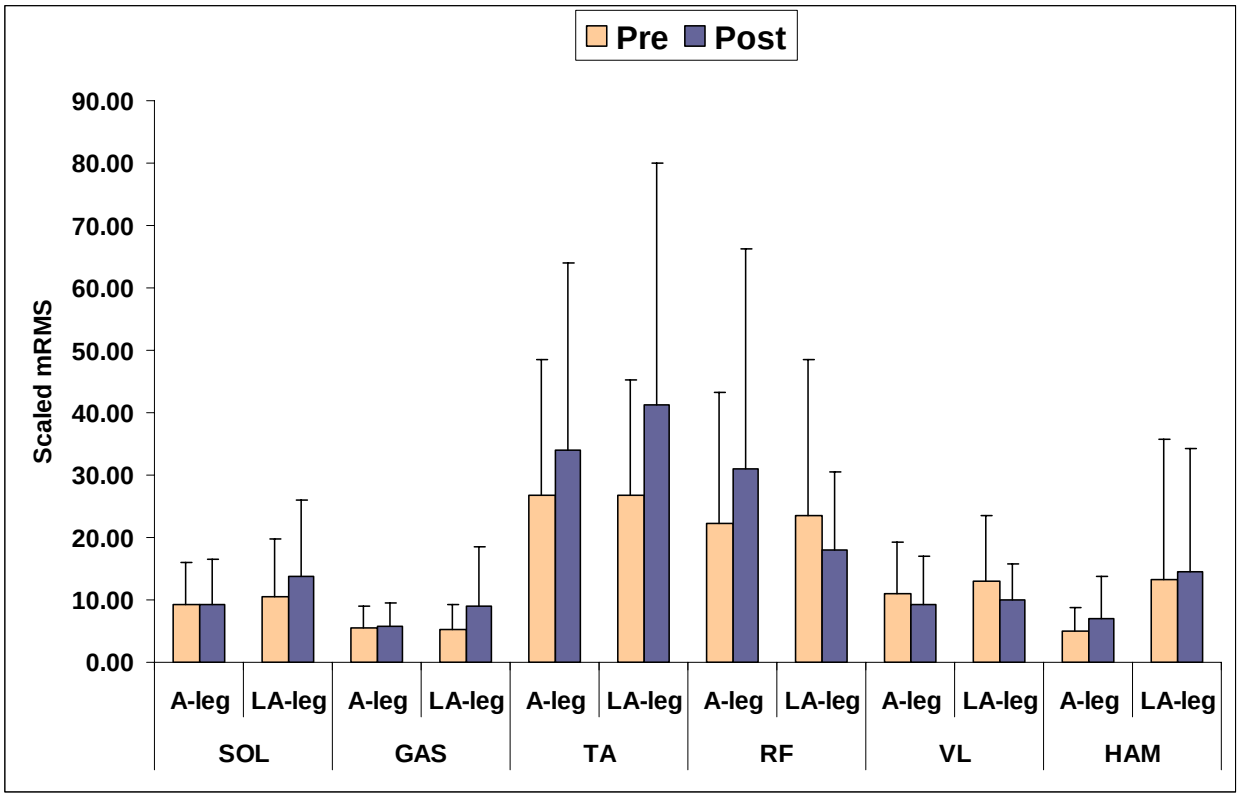

Figure 4.4. Pre and Post scaled mRMS muscle activation of the soleus (SOL), gastrocnemius (GAS), tibialis anterior (TA), rectus femoris (RF), vastus lateralis (VL) and the hamstrings (HAM) in the affected leg (A-leg) and less affected leg (LA-leg) over the entire STS movement.

CHAPTER 5

SUMMARY AND CONCLUSION

The purpose of this study was to first determine the biomechanical differences between a group of people with multiple sclerosis (MS) and non-MS control group for the STS movement and second, to determine if 16 weeks of flexibility training would improve the functional performance and biomechanics exhibited by individuals with MS during the STS. As standing up is a pre-requisite for many functional activities (Kralj et al., 1990), and mobility limitations are a primary concern for individuals with MS(M. Finlayson, 2004; Schwid et al., 1997), understanding how people with MS perform the STS, what strategies they use, and what interventions can improve the possible deficiencies exhibited when standing from a seated position is of paramount importance.

For this study 21 participants with MS and 12 controls performed five STS trials during which motion analysis, ground reaction forces, and electromyography were captured. Of the 21 MS participants 9 participated in a 16 week flexibility training program after which they once again performed five more trials of STS. A series of t-tests and repeated measures ANOVAs were used for comparisons ($\alpha = 0.05$).

People with relapsing-remitting MS who had mild to moderate levels of disability (EDSS scores: < 6.5) displayed less leg extensor strength than healthy individuals. MS individuals use a trunk flexion strategy commonly used by other clinical populations with lower extremity weakness when performing the STS movement and subsequently have an increased movement time compared to controls. Muscle activation of the rectus femoris of the affected leg was the

only muscle that displayed less mRMS than that of the CON group. No differences were found for maximum joint moment magnitudes. Therefore, it is unclear whether abnormal signal transduction or decreased leg extensor strength affected the magnitudes of the joint moments. However, the earlier times to peak moments in the MS group may suggest the increased trunk velocity aided in the initial movement just enough to get the knee to begin extension, but not enough to see changes in moment magnitudes.

In addition to increasing joint ROM, it was surmised that 16 weeks of flexibility training would have secondary benefits including increased muscle strength and decreased muscle spasticity. Consequently, greater muscle strength and less spasticity would improve functional performance and change the biomechanics demonstrated for the STS movement. Although MS participants were able to increase ROM at the ankle and knee joints and exhibit some biomechanical changes with low-intensity flexibility training, no increases in strength or decreases in spasticity were detected. However, to improve flexibility of other joints and overall function of the STS movement, that is, movement time, and reduce atypical movement kinematics and kinetics, it is possible that a more intense flexibility training program may be needed**.**

Although significant differences were not detected for leg strength or the biomechanics displayed for the STS movements, a majority of participants reported anecdotally that they felt stronger. They commented that they were able to perform other activities, such as washing the dishes or taking a bath with less trepidation. Hence, engaging in this flexibility training may have improved the performers' confidence. Future investigations may also find that some individuals do, in fact, improve strength with this program, while others do not.

In conclusion MS participants display different STS biomechanics during the STS movement using the trunk flexion strategy commonly found in clinical populations with lower extremity muscle weakness. Furthermore, although the flexibility training program in this study did not improve strength or decrease spasticity, flexibility training can induce increased ROM for the joints of the lower extremity of people with MS. Further research is warranted to determine how different levels of MS perform the STS movement and if strength training can improve functional performance of the STS movement.

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APPENDIXES

Appendix A

Kurtzke Expanded Disability Status Scale

0.0	Normal neurological examination
1.0	No disability, minimal signs in one FS
1.5	No disability, minimal signs in more than one FS
2.0	Minimal disability in one FS
2.5	Mild disability in one FS or minimal disability in two FS
3.0	Moderate disability in one FS, or mild disability in three or four FS. Fully ambulatory
3.5	Fully ambulatory but with moderate disability in one FS and more than minimal disability in several others
4.0	Fully ambulatory without aid, self-sufficient, up and about some 12 hours a day despite relatively severe disability; able to walk without aid or rest some 500 meters
4.5	Fully ambulatory without aid, up and about much of the day, able to work a full day, may otherwise have some limitation of full activity or require minimal assistance; characterized by relatively severe disability; able to walk without aid or rest some 300 meters.
5.0	Ambulatory without aid or rest for about 200 meters; disability severe enough to impair full daily activities (work a full day without special provisions)
5.5	Ambulatory without aid or rest for about 100 meters; disability severe enough to preclude full daily activities
6.0	Intermittent or unilateral constant assistance (cane, crutch, brace) required to walk about 100 meters with or without resting
6.5	Constant bilateral assistance (canes, crutches, braces) required to walk about 20 meters without resting
7.0	Unable to walk beyond approximately five meters even with aid, essentially restricted to wheelchair; wheels self in standard wheelchair and transfers alone; up and about in wheelchair some 12 hours a day
7.5	Unable to take more than a few steps; restricted to wheelchair; may need aid in transfer; wheels self but cannot carry on in standard wheelchair a full day; May require motorized wheelchair
8.0	Essentially restricted to bed or chair or perambulated in wheelchair, but may be out of bed itself much of the day; retains many self-care functions; generally has effective use of arms
8.5	Essentially restricted to bed much of day; has some effective use of arms retains some self care functions

9.0	Confined to bed; can still communicate and eat.
9.5	Totally helpless bed patient; unable to communicate effectively or eat/swallow
10.0	Death due to MS

Appendix B

Modified Ashworth Spasticity Scale

TABLE 1 *Modified Ashworth Scale for Physical Therapy—Spasticity Evaluation*

Date/Time														
	R	L	R	L	R	L	R	L	R	L	R	L	R	L
Shoulder Flexors														
Shoulder Extensors														
Elbow Flexors														
Elbow Extensors														
Wrist Flexors														
Wrist Extensors														
Hip Flexors														
Hip Extensors														
Hip Abductors														
Hip Abductors														
Knee Flexors														
Knee Extensors														
Ankle Dorsiflexors														
Ankle Plantarflexors														
Average for UE														
Average for LE														

Date/Comments

Modified Ashworth Scale for Grading Spasticity

Grade	*Description*
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension.
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM.
2	More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved.
3	Considerable increase in muscle tone, passive movement difficult
4	Affected part(s) rigid in flexion or extension

Appendix C

Participant Information and Health Questionnaire

Medical History
Participant Information

Name: _____ Date of Birth: _____

Address: _____ Phone number: (w) _____

_____ (h) _____

Email: _____ (cell) _____

Blood Pressure:__/_____ Hear rate _____

Height: _____ Measured weight: _____

Gender: Male Female (circle)

Ethnicity : Caucasian African American Hispanic Asian Other

Emergency contact name and number: _____

Family Physician name and number: _____

Please answer the following questions:

I. GENERAL HEALTH

1. Have you been diagnosed with diabetes? **Yes** **No**
   If "yes", please explain _____
2. Have you ever had an oral glucose tolerance test? **Yes** **No**
   If "yes", please explain _____
3. Have you ever been told by a physician that you have Osteoporosis/Osteopenia? **Yes** **No**
4. Have you ever been told by a physician that you have a heart condition? **Yes** **No**
3. Have you or anyone in your immediate family had a heart attack, stroke, or cardiovascular disease before age 50 yrs? If "yes," please explain. **Yes** **No**
   _____
5. Have you ever been told by a physician that you have high blood pressure? **Yes** **No**
6. Have you ever been told by a physician that you have high cholesterol? **Yes** **No**
7. Have you ever been told by a physician that you have thyroid problems? **Yes** **No**
8. Have you ever been told by a physician that you have kidney disease? **Yes** **No**

9. Do you feel angina-like symptoms (pain or pressure in your chest, neck, shoulders, or arms) **Yes** **No**

10. Do you ever lose your balance because of dizziness? **Yes** **No**

11. Do you ever lose consciousness? **Yes** **No**

12. Do you consider most of your days very stressful? **Yes** **No**

13. Do you consider your eating habits healthy overall? **Yes** **No**
(Lower in fats and fried foods, higher in fruits, veggies and grains)

14. Have you had any major surgeries? **Yes** **No**
If "yes", please explain: _____

15. Do you consider yourself to be generally healthy? **Yes** **No**

16. Do you currently smoke cigarettes or cigars or chew tobacco? **Yes** **No**
If "yes", how often and how much: _____

17. Are you a former smoker? **Yes** **No**
If so, how long has it been since you quit smoking? _____

18. Has your weight changed more than 5 pounds in the last 6 months? **Yes** **No**

EARS:

____ hearing difficulty
____ ringing
____ pain
____ discharge
____ other

NOSE:

____ bleeding
____ difficulty smelling
____ nasal congestion
____ sinus problems
____ other

Please explain _____

PULMONARY

____ shortness of breath
____ wheezing
____ asthma
____ chronic cough
____ allergies
____ other

Please explain_____

19. Are there any other health related issues we should know about? _____
Please explain _____

_____

II. MEDICATION/SUPPLEMENTS

1. Please list all of the **prescription medication** you are currently taking.

Medicine name	Amount taken per day	Months/years on the Medication	Reason
a. _____	_____	_____	_____

b. _____ _____ _____ _____

c. _____ _____ _____ _____

d. _____ _____ _____ _____

e. _____ _____ _____ _____

f. _____ _____ _____ _____

2. Any known drug allergies? Explain _____

3. Have you been on steroid medication in the past? **Yes** **No**
If so, please explain in detail _____

_____

_____

4. Please list all of the **over-the-counter medicines or supplements** (including vitamins that you take regularly)

Item name	Amount taken per day	Months/years on Medication	Reason
a. _____	_____	_____	_____
b. _____	_____	_____	_____
c. _____	_____	_____	_____
d. _____	_____	_____	_____
e. _____	_____	_____	_____
f. _____	_____	_____	_____

III. REPRODUCTIVE STATUS

1. Have you reached menopause? (if NO skip to Section III) **Yes** **No**

2. How long has it been since you reached menopause? _____ **Yes** **No**

3. Do you still have your ovaries? _____ **Yes** **No**
   a. If not, how old were you when they were removed? _____.

4. Have you ever been on hormone replacement therapy? **Yes** **No**

   a. If so, are you still taking hormone replacement therapy? **Yes** **No**

   b. If you have previously taken hormone replacement therapy, but have since stopped, when did you stop taking hormone replacement therapy? _____

5. Have you ever taken osteoporosis medications? **Yes** **No**

Which ones and for how long? _____
_____

IV. OSTEOPOROSIS/FRACTURE/BONE HEALTH SECTION

1. Have you ever had a bone scan? **Yes** **No**
   If so, what year _____
   What was the outcome_____

2. Please provide a list of any bone fractures you have had in the past.

Bone	Cause (fall, accident, etc)	Year
_____	_____	_____

3. Did a doctor tell you that any of these fractures were due to osteoporosis/osteopenia? **Yes** **No**

4. Is your diet low in dairy products? **Yes** **No**

5. Do you take calcium supplements? **Yes** **No**
   If so, how much per day? _____

6. In a typical week, how many alcoholic drinks do you consume? _____

7. Do you drink coffee, tea, or cola products routinely? **Yes** **No**

   About how much coffee, tea, or cola do you drink on an average day? _____-
   _____

8. Do you have a heart valve or implant devices such as knee, hip ect.? **Yes** **No**

9. Do you get claustrophobic in small spaces? **Yes** **No**

V. SUN EXPOSURE

1. How many times a week do you spend more than 10 minutes outside? _____
2. How much time do you spend outdoors (minutes) per week?_____
3. How much of your outdoor time is spent without sunscreen on (minutes)?_____
4. How much of your outdoor time is spent "fully exposed" (minutes)?_____
   ("fully exposed" is defined as uncovered face, arms, and hands)

VI. EXERCISE HABITS

1. How many times per week do you generally exercise? _____

   a. What **<u>type(s) of exercise</u>** do you generally perform? (circle all that apply)
   Walking Running bicycling swimming

Weight lifting aerobics spinning tennis

Other _____

b. In a typical week, **how may days** do you exercise? (circle)

0-1 time/week 2-3 times week 4-6 times/week daily

c. How many **minutes** do you typically exercise per session? (circle)

<15min 15-30min 30-45 >45
Other_____

d. What is the typical **level of exertion** during your exercise?

Light Moderate Moderate/heavy Heavy

e. When you are exercising do you ever feel limited by the following?

	Yes	No	Activity
Breathing	____	____	_____
Chest arm neck pain	____	____	_____
Low back pain	____	____	_____
Side ache	____	____	_____
Leg pain	____	____	_____
Foot drop	____	____	_____

Other? Please explain _____

_____

VII. MULTIPLE SCLEROSIS STATUS

1. How long have you been diagnosed with Multiple Sclerosis? (If not diagnosed with MS skip to Section VIII) _____
2. When did you have your first MS symptoms? _____
3. Has your physician ever discussed what type of MS you have? **Yes** **No**

Relapsing remitting **Primary progressive** **Secondary progressive** **Progressive relapsing**

4. Briefly describe your current MS symptoms _____

_____

5. Does MS affect your legs? **Yes** **No** Does MS affect your arms? **Yes** **No**

If yes, which leg is more involved? Right Left Both same
If yes, which arm is more involved? Right Left Both same

6. Do you fatigue easily? **Yes** **No**
If yes, what causes it to be worse? _____

7. Do you ever experience worsening of symptoms? **Yes** **No**

	Yes	**No**	**How often?**
Bath/shower	____	____	_____
Physical activity	____	____	_____
Hot outside	____	____	_____

8. Do you drive yourself independently? **Yes** **No**

9. Do you walk (circle) w/o aid with cane walker wheelchair

9. Has your physician ever recommended that you get a bone scan? _____

10. Has your physician ever recommended that you exercise? _____

Neurologist _____ Phone _____

Family Practice Physician _____ Phone _____

Other _____ Phone _____

VIII. EMPLOYMENT STATUS

1. Full-time employed □
2. Part-time employed □
3. Retired □
4. Not working □

Please describe employment status_____
_____

IX. EDUCATION

1. None □
2. High School □
3. College □
4. Masters □
5. Ph.D. □
6. Other □

X. MARITAL STATUS **Single** **Married**

I certify that these answers are accurate and complete

_____ _____
YOUR SIGNATURE DATE
Witness _____ date: _____

Appendix D
ANOVA Results of all dependent variables for Study #1

Variables	Source	F(1,31)	p-value	η^2	power
1 RM Leg press	Group	8.392	0.007	0.211	0.796
Max trunk inclination angle	Group	7.340	0.011	0.192	0.747
Max trunk flexion velocity	Group	5.540	0.025	0.152	0.626
Max trunk extension velocity	Group	0.680	0.417	0.021	0.126
Max COM displacement	Group	0.032	0.86	0.001	0.053
Movement Time	Phase	67.90	<0.001	0.687	1.000
	Group	5.887	0.020	0.160	0.830
	Phase × Group	9.050	0.005	0.226	0.652
Knee extension velocity	Leg	0.085	0.773	0.003	0.059
	Group	4.754	0.037	0.133	0.561
	Leg × Group	0.311	0.581	0.010	0.084
Max dorsiflexion angle	Leg	1.806	0.189	0.055	0.256
	Group	<0.001	0.987	<0.001	0.050
	Leg × Group	8.828	0.006	0.222	0.821
Max dorsiflexion velocity	Leg	0.292	0.593	0.009	0.082
	Group	0.293	0.592	0.009	0.082
	Leg × Group	0.350	0.559	0.011	0.088
Max plantarflexion velocity	Leg	1.338	0.256	0.041	0.202
	Group	3.241	0.082	0.095	0.415
	Leg × Group	4.206	0.049	0.119	0.511
Max vertical GRF	Leg	0.839	0.367	0.026	0.144

	Group	0.394	0.535	0.013	0.093
	Leg × Group	0.166	0.687	0.005	0.068
Max vertical GRF RT	Leg	0.002	0.962	<0.001	0.050
	Group	0.411	0.526	0.013	0.095
	Leg × Group	1.242	0.274	0.039	0.191
Max posterior GRF	Leg	0.241	0.627	0.008	0.076
	Group	0.914	0.347	0.029	0.153
	Leg × Group	0.079	0.780	0.003	0.059
Max posterior GRF RT	Leg	4.671	0.039	0.131	0.553
	Group	0.766	0.388	0.024	0.136
	Leg × Group	0.504	0.483	0.016	0.106
Max dorsiflexor moment	Leg	0.508	0.481	0.016	0.106
	Group	0.028	0.867	0.001	0.053
	Leg × Group	0.287	0.596	0.009	0.081
Max plantarflexormoment	Leg	0.338	0.565	0.011	0.087
	Group	1.020	0.320	0.032	0.165
	Leg × Group	1.231	0.276	0.038	0.189
Max knee extensor moment	Leg	0.444	0.510	0.014	0.099
	Group	0.679	0.416	0.021	0.126
	Leg × Group	0.221	0.641	0.007	0.074
Max knee extensor moment RT	Leg	0.175	0.678	0.006	0.069
	Group	6.939	0.013	0.183	0.723
	Leg × Group	0.083	0.775	0.003	0.059
Max hip extensor moment	Leg	0.462	0.502	0.015	0.101
	Group	0.010	0.921	<0.001	0.051
	Leg × Group	1.465	0.235	0.045	0.217
Max hip extensor moment RT	Leg	0.477	0.495	0.015	0.103
	Group	10.238	0.003	0.248	0.873
	Leg × Group	1.594	0.216	0.049	0.231
Max positive ankle joint power	Leg	0.715	0.404	0.023	0.130

	Group	0.643	0.429	0.020	0.122
	Leg × Group	3.012	0.093	0.089	0.391
Max negative ankle joint power	Leg	0.020	0.888	0.001	0.052
	Group	4.552	0.041	0.128	0.543
	Leg × Group	0.431	0.516	0.014	0.098
Max positive knee joint power	Leg	3.553	0.069	0.103	0.447
	Group	2.297	0.140	0.069	0.312
	Leg × Group	0.888	0.353	0.028	0.150
Max positive knee joint power RT	Leg	0.007	0.932	<0.001	0.051
	Group	5.505	0.026	0.151	0.623
	Leg × Group	0.193	0.664	0.006	0.071
Max positive hip joint power	Leg	1.064	0.310	0.033	0.170
	Group	2.253	0.143	0.068	0.307
	Leg × Group	0.687	0.413	0.022	0.127
Max positive hip joint power RT	Leg	0.232	0.634	0.007	0.075
	Group	6.148	0.019	0.166	0.671
	Leg × Group	0.126	0.725	0.004	0.064
Gastrocnemius mRMS	Leg	0.019	0.890	0.001	0.052
	Group	1.098	0.303	0.034	0.174
	Leg × Group	1.926	0.175	0.059	0.270
Soleus mRMS	Leg	0.578	0.453	0.018	0.114
	Group	0.036	0.850	0.001	0.054
	Leg × Group	0.485	0.491	0.015	0.104
Tibialis anterior mRMS	Leg	1.038	0.316	0.032	0.167
	Group	0.373	0.546	0.012	0.091
	Leg × Group	2.209	0.147	0.067	0.302
Rectus Femoris mRMS	Leg	0.127	0.724	0.004	0.064
	Group	1.630	0.211	0.050	0.236
	Leg × Group	10.242	0.003	0.248	0.873
Vastus Lateralis mRMS	Leg	0.087	0.770	0.003	0.059

	Group	0.253	0.618	0.008	0.078
	Leg × Group	3.095	0.088	0.091	0.399
Hamstring mRMS	Leg	4.165	0.050	0.118	0.507
	Group	0.454	0.505	0.014	0.100
	Leg × Group	0.018	0.894	0.001	0.052

Appendix E

ANOVA Results of all dependent variables for Study #2

Variables	Source	F(1,8)	p-value	η^2	power
Ankle dorsiflexion ROM	Pre-post	6.025	0.044	0.463	0.561
	Leg	0.299	0.601	0.041	0.076
	Pre-post × Leg	5.470	0.052	0.439	0.522
Ankle plantarflexion ROM	Pre-post	8.105	0.025	0.537	0.686
	Leg	0.036	0.855	0.005	0.053
	Pre-post × Leg	5.890	0.046	0.457	0.552
Knee flexion ROM	Pre-post	10.837	0.013	0.608	0.805
	Leg	0.686	0.435	0.089	0.111
	Pre-post × Leg	0.012	0.914	0.002	0.051
Knee extension ROM	Pre-post	0.737	0.419	0.095	0.116
	Leg	4.487	0.072	0.391	0.448
	Pre-post × Leg	0.255	0.629	0.035	0.072
Hip flexion ROM	Pre-post	2.029	0.197	0.225	0.235
	Leg	2.001	0.200	0.222	0.232
	Pre-post × Leg	1.975	0.203	0.220	0.230
Hip extension ROM	Pre-post	3.692	0.150	0.552	0.271
	Leg	2.126	0.241	0.415	0.180
	Pre-post × Leg	0.327	0.608	0.098	0.070
Lower extremity spasticity	Pre-post	0.865	0.405	0.178	0.112
	Leg	<0.001	1.000	<0.001	0.050
	Pre-post × Leg	2.667	0.178	0.400	0.243
Max Isokinetic leg extension 60°/s	Pre-post	0.366	0.562	0.044	0.083
	Leg	0.015	0.905	0.002	0.051
	Pre-post × Leg	1.287	0.289	0.139	0.171
Max Isokinetic leg extension 180°/s	Pre-post	0.384	0.553	0.046	0.085
	Leg	0.845	0.385	0.095	0.128
	Pre-post × Leg	1.036	0.339	0.115	0.147

1 RM Leg press	Group	0.397	0.546	0.047	0.086
Max trunk inclination angle	Group	0.347	0.572	0.042	0.082
Trunk flexion velocity	Group	0.566	0.474	0.066	0.102
Trunk extension velocity	Group	6.593	0.033	0.452	0.616
Max COM displacement	Group	4.162	0.076	0.342	0.435
Movement Time	Phase	3.935	0.083	0.330	0.415
	Pre-post	3.107	0.116	0.280	0.342
	Phase × Pre-post	34.531	<0.001	0.812	0.999
Max Knee extension velocity	Pre-post	1.167	0.311	0.127	0.159
	Leg	1.351	0.279	0.144	0.177
	Pre-post × Leg	0.739	0.415	0.085	0.177
Max dorsiflexion angle	Pre-post	0.117	0.741	0.014	0.061
	Leg	2.610	0.145	0.246	0.297
	Pre-post × Leg	0.156	0.703	0.019	0.064
Max Dorsiflexion velocity	Pre-post	0.216	0.654	0.026	0.070
	Leg	0.884	0.375	0.099	0.132
	Pre-post × Leg	0.002	0.967	<0.001	0.050
Max Plantarflexion velocity	Pre-post	0.992	0.348	0.110	0.142
	Leg	1.121	0.321	0.123	0.155
	Pre-post × Leg	0.512	0.495	0.060	0.097
Max vertical GRF	Pre-post	0.627	0.451	0.073	0.108
	Leg	0.509	0.496	0.060	0.097
	Pre-post × Leg	0.306	0.596	0.037	0.078
Max vertical GRF RT	Pre-post	0.113	0.745	0.014	0.060
	Leg	2.602	0.145	0.245	0.296
	Pre-post × Leg	0.321	0.587	0.039	0.079

Max posterior GRF	Pre-post	1.996	0.195	0.200	0.239
	Leg	0.032	0.862	0.004	0.053
	Pre-post × Leg	0.009	0.925	0.001	0.051
Max posterior GRF RT	Pre-post	6.197	0.038	0.436	0.589
	Leg	0.379	0.555	0.045	0.085
	Pre-post × Leg	3.870	0.085	0.326	0.410
Max dorsiflexor moment	Pre-post	1.891	0.206	0.191	0.229
	Leg	0.410	0.540	0.049	0.088
	Pre-post × Leg	1.904	0.205	0.192	0.230
Max plantarflexor moment	Pre-post	0.182	0.681	0.022	0.067
	Leg	0.080	0.784	0.010	0.057
	Pre-post × Leg	1.309	0.286	0.141	0.173
Max knee extensor moment	Pre-post	0.040	0.846	0.005	0.054
	Leg	0.075	0.792	0.009	0.057
	Pre-post × Leg	0.033	0.860	0.004	0.053
Max knee extensor moment RT	Pre-post	3.131	0.115	0.281	0.345
	Leg	0.770	0.406	0.088	0.121
	Pre-post × Leg	0.598	0.462	0.070	0.105
Max hip extensor moment	Pre-post	0.227	0.646	0.028	0.071
	Leg	0.845	0.385	0.096	0.128
	Pre-post × Leg	2.279	0.170	0.222	0.265
Max hip extensor moment RT	Pre-post	6.835	0.031	0.461	0.631
	Leg	4.933	0.057	0.381	0.497
	Pre-post × Leg	0.053	0.823	0.007	0.055
Max positive ankle joint power	Pre-post	0.751	0.411	0.086	0.120
	Leg	0.487	0.505	0.057	0.095
	Pre-post × Leg	0.317	0.589	0.038	0.079
Max negative ankle joint power	Pre-post	0.407	0.541	0.048	0.087
	Leg	<0.001	0.995	<0.001	0.050
	Pre-post × Leg	1.649	0.235	0.171	0.205

Max positive knee joint power	Pre-post	0.007	0.935	0.001	0.051
	Leg	0.715	0.422	0.082	0.116
	Pre-post × Leg	0.344	0.574	0.041	0.081
Max positive knee joint power RT	Pre-post	0.967	0.354	0.108	0.140
	Leg	0.798	0.398	0.091	0.124
	Pre-post × Leg	2.318	0.166	0.225	0.269
Max positive hip joint power	Pre-post	0.459	0.517	0.054	0.092
	Leg	0.886	0.374	0.100	0.132
	Pre-post × Leg	4.302	0.072	0.350	0.446
Max positive hip joint power RT	Pre-post	2.602	0.145	0.245	0.296
	Leg	0.003	0.958	<0.001	0.050
	Pre-post × Leg	1.061	0.333	0.117	0.149
Gastrocnemius mRMS	Pre-post	1.911	0.204	0.193	0.231
	Leg	0.850	0.383	0.096	0.129
	Pre-post × Leg	0.914	0.367	0.103	0.135
Soleus mRMS	Pre-post	0.561	0.475	0.065	0.102
	Leg	0.886	0.374	0.100	0.132
	Pre-post × Leg	1.375	0.275	0.147	0.179
Tibialis anterior mRMS	Pre-post	2.250	0.172	0.220	0.263
	Leg	1.396	0.271	0.149	0.181
	Pre-post × Leg	0.979	0.352	0.109	0.141
Rectus Femoris mRMS	Pre-post	0.329	0.582	0.040	0.080
	Leg	0.957	0.357	0.107	0.139
	Pre-post × Leg	0.303	0.597	0.036	0.078
Vastus Lateralis mRMS	Pre-post	0.064	0.807	0.008	0.056
	Leg	1.498	0.256	0.158	0.191
	Pre-post × Leg	2.520	0.151	0.240	0.288
Hamstring mRMS	Pre-post	0.345	0.573	0.041	0.082
	Leg	1.502	0.255	0.158	0.191
	Pre-post × Leg	0.091	0.770	0.011	0.058