IMPACT OF SPASTICITY ON BALANCE CONTROL DURING QUIET STANDING IN PERSONS POST-STROKE

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ABSTRACT

Statement of the problem: Balance impairments and falls are common among people with stroke. Muscle spasticity is also common and may influence balance control. However, the effect of spasticity on balance control among people with stroke is not well understood. **Methods:** Twenty-seven post-stroke individuals with low or high spasticity at the ankle completed quiet standing trials with eyes open and closed. Balance control was measured by estimating centre of pressure (COP) movement and trunk sway. **Results:** Individuals with high spasticity had greater COP velocity, trunk sway velocity, and trunk sway velocity frequency, particularly in the eyes closed condition. These effects were predominantly in the mediolateral direction (vision by group interaction effects $p= 0.033$, $p= 0.037$, and $p= 0.015$ respectively). Main effect of group revealed that individuals with high spasticity had higher mediolateral mean power frequency measures ($p= 0.045$). **Conclusion:** Individuals with high spasticity post-stroke demonstrated greater balance control challenges especially in absence of vision. Furthermore, these challenges were specifically noted in the frontal plane.

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CHAPTER 1

GENERAL INTRODUCTION

1.1 Rationale and Scope of Thesis

Stroke is an interruption of sufficient blood flow in the brain and is known to be the third leading cause of mortality in North America. In Canada, over 300,000 stroke survivors live with many challenges from effects of stroke. The physical challenges of stroke can include impaired motor control of movement on one side of the body, loss of sensation and dexterity, and muscle weakness. Similarly important to such physical impairments in individuals with stroke is the clinical sign of spasticity with increase muscle tone and hyperactive stretch reflexes that result in changes to postural orientation of the upper and lower limbs. As a result of these changes, managing activities of daily living (ADLs) such as standing, sitting, walking, dressing, bathing, and many more challenges including balance problems and high incidences of falls are well evident in individuals with stroke. Considering these challenges, it becomes important to understand how these factors may contribute and explain some of the difficulties that individuals with stroke might have in their daily lives. It is not surprising that individuals with stroke may develop spasticity in the upper and lower limbs, and that individuals with stroke may also have high number of falls and balance problems. However, current understanding of such problems in balance control in post-stroke persons from effects and challenges of spasticity is not well established from lack of evidence in the literature. This is in fact an important consideration since there is currently no cure for spasticity, although there are treatment options available to manage individuals' spasticity levels. Hence, if it is suggested that challenges such as balance problems, high incidence of falls, and fear of fall are related and can be explained by

the effect of spasticity in individuals with stroke, then further care and treatment of individuals with spasticity can be targeted to reduce balance problem challenges in this population.

1.2 Statement of Hypotheses

In order to investigate how spasticity can affect balance control in individuals post-stroke it is necessary to have a better understanding of the literature in the area of stroke, spasticity, and balance control research. In this project, a review of literature on the effects of stroke, spasticity, and balance control of the body is first introduced in the next chapter. Moreover, in the following chapters the affect of spasticity on balance control of individuals with stroke is studied by testing the following hypotheses:

1) Post-stroke individuals with high spasticity are expected to have greater balance control challenges as compared to post-stroke individuals with low spasticity.

2) Post-stroke individuals with high spasticity are expected to have greater fear of fall measures as compared to post-stroke individuals with low spasticity.

CHAPTER 2

LITERATURE REVIEW

2.1 Introduction to Stroke

Stroke has been defined as an interruption of adequate blood flow to an area of the brain (Kandel, Schwartz, and Jessel, 2000, p. 1302) [1]. The interruption of blood flow to the brain results in lack of oxygen, glucose and other nutrients' delivery to neurons within the brain, and further limits removal of carbon dioxide and metabolic products that can result in irreversible neuronal damages (Kandel et al., 2000, p. 1302) [1]. Strokes are either classified as occlusive from blockage of an artery of the brain or as hemorrhagic by bleeding into the brain as a result of rupture of the brain arteries. Atherosclerosis or build up of plaque in the artery of the brain can block blood flow within the brain and is responsible for occlusive strokes, whereas hypertension is responsible for most hemorrhagic type of strokes. Stroke has been reported to be the third cause of mortality in the United States (Kandel et al., 2000, p. 1306) [1] and in Canada [2]. Approximately 50,000 Canadians have a stroke per year [3], and about 315,000 Canadians have reported that they were living with challenging consequences after they had a stroke [4].

Challenges and consequences of strokes can vary significantly since different brain arteries deliver blood supply to different regions of the brain and each region of brain has unique functions. Therefore, a review of main areas of the brain and arteries supplying different brain regions can help better understand post-stroke effects and its consequences.

The brain is mainly divided to four areas: frontal lobe, parietal lobe, temporal lobe, and occipital lobe (Kandel et al., 2000, p. 8) [1]. The brain can also be mapped according to controlling

function of the brain including the primary motor and sensory cortices, primary visual cortex, primary auditory cortex, and association cortices that map the surface of the brain (Kandel et al., 2000, p. 350) [1]. Furthermore, the regions responsible for sensory and motor control of specific body parts can be mapped according to the sensory and motor homunculi of the brain (Penfield and Rasmussen, 1950, p. 344) [5]. Additionally, the cerebellum, midbrain, pons, medulla, and the spinal cord are structures near the brain that also communicate closely with different brain areas (Kandel et al., 2000, p. 8) [1]. Generally, the cerebellum is responsible for learning process of motor skills and regulating the force and range of movement. The midbrain is involved in coordination of visual and auditory reflexes as well as control of different motor and sensory functions. The pons carries information regarding movement from the cerebral hemisphere to the cerebellum. The medulla is responsible for regulation of autonomic responses including digestion, breathing and control of the heart rate. The spinal cord is responsible for receiving sensory inputs and control of movements of the limbs and the trunk. The spinal cord also receives inputs from the brain and sends information to the brain via the brainstem structures including the medulla, pons, and midbrain (Kandel et al., 2000, p. 8) [1].

The vasculature anatomy of blood flow to different brain regions can determine the consequences of stroke. The blood supply of the brain starts from the common carotid artery and the subclavian artery, which are main branches of the aortic arch of the heart (Kandel et al., 2000, p. 1303) [1]. The common carotid artery is further divided into the internal and external carotid arteries with internal branch heading up towards the head region to supply each cerebral hemisphere. In each cerebral hemisphere, the internal carotid artery is further divided to two branches namely the anterior cerebral artery, and the middle cerebral artery. The left and right subclavian arteries make up the left and right vertebral arteries that join at the level of pons and medulla to form the basilar artery. The basilar artery is then divided to left and right posterior arteries that further supply the back region of

the brain. The main branches of the anterior cerebral artery supply inferior and medial parts of the frontal lobe, medial parts of the parietal lobe, and anterior part of the corpus callosum. Main branches of the medial cerebral artery supply large areas of the cortex including frontal, parietal, temporal, and occipital lobe. The posterior cerebral artery supplies the inferior parts of the temporal lobe, medial parts of the occipital lobe, and the posterior corpus callosum. The cerebellum and the brainstem structures receive their blood supply via branches of the vertebral and basilar arteries that supply the posterior region of the brain (Kandel et al., 2000, p. 1306) [1].

As introduced earlier, the consequences of stroke depend largely on the brain area that is deprived from oxygen delivery of the affected artery causing the stroke. For example, an individual with stroke might have aphasia or difficulties in producing speech if speech areas of the brain are affected, visual impairment might be present if visual fields of the brain are affected, hemiplegia or weakness in one side of the body may be present if the motor cortex, brainstem, or the descending corticospinal tracts are affected. As a result, possible consequences of stroke can vary and depend largely on the severity of the damage within the brain (Kandel et al., 2000, pp. 1306-1314) [1]. Spasticity and frequent falls are two of many challenges commonly reported in individuals with stroke (Kandel et al., 2000, p. 1307) [1] and (Barnes and Johnson, 2008, p. 4) [6]. The post-stroke effects of these challenges will be discussed and is the main focus of the current project.

2.2 Overview on Spasticity

The neuronal damages from stroke in the motor output areas can include the upper motor neurons (UMN) which are neurons that originate from higher levels of the central nervous systems (CNS) of the cortex and brainstem and synapse on lower motor neurons at the spinal cord level (Kandel et al., 2000, p. 696) [1]. When the UMNs are damaged, a condition referred to as UMN

syndrome results and a number of negative and positive clinical signs can be present (Barnes and Johnson, 2008, p. 2) [6]. The negative signs are impaired motor control, dexterity loss and the positive signs of UMN syndrome are spasticity, clonus, spasm, positive Babinski sign, and hyperexcitable spinal reflexes [6]. Spasticity is generally accepted by Lance's definition to be "a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes ('muscle tone') with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of upper motor neurons" [7]. In this definition, the tonic stretch reflex represents a type of proprioceptive spinal reflex responsible for control of body parts based upon sensory information about body position in space and movement (Barnes and Johnson, 2008, p. 16) [6]. This reflex is active when muscle stretch is picked up by the muscle spindle receptors that transfer the signal via afferent sensory fibres to the spinal cord and activate the motor neurons for muscle contraction of the stretched muscle (P.A. Young, P.H. Young, and Tolbert, 2008, p. 60) [8] and (Barnes and Johnson, 2008, p. 16) [6].

Damage to the UMNs that control the spinal reflexes can include the pyramidal or corticospinal tracts and parapyramidal tracts which have shown to produce spasticity when damaged (Young et al., 2008, p. 65) [8] and (Barnes and Johnson, 2008, p. 11) [6]. The corticospinal tracts responsible for motor function mainly start at the level of primary motor cortex (M1) and some at the premotor regions with upper neurons located dorsally in M1 region responsible for upper limb movement and neurons located medially in the M1 region influencing the lower limbs. Both motor and premotor areas must be involved in order to produce spasticity, whereas distal lesions produced in the medullary pyramids do not produce spasticity (Barnes and Johnson, 2008, p. 11) [6]. The axons of the corticospinal tracts descend down from the cortex to pass through the internal capsule, forming bundles that descend further down to the midbrain, the pons, and the medulla. At the level of the

medulla, 90% of the fibres cross to the contralateral side of the body and descend further down towards the spinal cord. The remaining uncrossed tracts descend down the spinal tract towards the same side they originated, but as for upper limb and lower limb movement control, these fibres are believed to be completely crossed (Young et al., 2008, p. 69) [8]. The parapyramidal tracts are also important in producing spasticity since these UMNs include other brainstem areas that control spinal stretch reflex as well. The parapyramidal tracts are mainly divided to two systems of inhibition and excitation control of the spinal reflex [6]. The inhibitory system includes the cortico-reticular fibres that rise from the premotor cortex to the reticular formation of the medulla forming the dorsal reticulospinal tract (DRT) (Barnes and Johnson, 2008, p. 12) [6] which descend ipsilaterally (Kandel et al., 2000, p. 668) [1]. Lesions in this pathway can deregulate the inhibitory effect leading to hyperexcitability of the stretch reflex. The excitatory system controlled by the brainstem come from the pons and medulla namely the bulbopontine tegmentum and lateral vestibular nucleus (Barnes and Johnson, 2008, p. 12) [6] that descend ipsilaterally (Kandel et al., 2000, p. 668) [1] to form the medial reticulospinal (MRT) and lateral vestibulospinal tracts (VST), respectively. Even though these tracts are responsible for facilitation of spinal stretch reflexes, they are also responsible for inhibiting the flexor reflex afferent fibres (Barnes and Johnson, 2008, p. 13) [6]. As a result, post-stroke damages involving UMNs including the pyramidal and parapyramidal tracts controlling the spinal reflexes can result in hyperexcitability of the stretch reflex and lead to spasticity.

Spasticity can lead to a combination of functional problems seen in individuals with stroke. For example, mobility can become a major challenge due to difficulties in walking (Brashear and Elovic, 2011, p. 123) [9], and falling may become persistent (Barnes and Johnson, 2008, p. 4) [6]. Hygiene can become a serious concern as spasticity in the finger flexors can make it difficult to extend the fingers (Barnes and Johnson, 2008, p. 5) [6] and further result in skin abrasions in the palm

of the hand [9]. Spasticity may limit abilities such as propelling a wheelchair, feeding, writing, bathing, getting in and out of bed, and managing personal care to a point that access to a caregiver may be necessary for management of activities of daily living (ADLs) (Barnes and Johnson, 2008, p. 4) [6]. Therefore, it is important to be able to recognize such functional challenges so that treatment can be appropriately directed to manage spasticity. These treatment options may include different physical exercises, use of oral or intrathecal spasticity medication, focal injections of botulinum toxin, or even surgical procedures in extreme cases (Barnes and Johnson, 2008, pp. 5-7) [6].

Effective treatment and management of spasticity also depends on periodic assessment of spasticity in monitoring its progression/regression at different time points. Many different approaches from objective and subjective measures considering physiological measures, active and passive activity measures, and functional measures have been used in the past (Brashear and Elovic, 2011, p. 55) [9]. However, factors such as sensitivity, reliability, ease of use, validity, and accessibility (Brashear and Elovic, 2011, pp. 51-52) [9] make it challenging to develop a single best method to assess spasticity in different settings. In the scientific literature and clinical settings, few assessment tools including the Ashworth Scale (AS), the Modified Ashworth Scale (MAS), and the Tardieu Scale are commonly used to assess spasticity. In the literature, most widely used and recognized measure of spasticity are the AS and the MAS (Brashear and Elovic, 2011, p. 56) [9]. In these tests, the examiner passively stretches the spastic muscle to score the resistance felt at the available range of motion (ROM) (Brashear and Elovic, 2011, p. 56) [9]. A description of the MAS scoring system and its method of assessment of spasticity in the current project is provided in Table 2.1 [10].

Table 2.1
Modified Ashworth Scale (MAS) scoring system [10]

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

2.3 Introduction to Balance Control

In addition to spasticity and other complications from UMN damage, falls are a common challenge after damage to the UMNs (Barnes and Johnson, 2008, p. 4) [6]. In this section, different systems responsible for balance control of the body will be considered, especially when studying falls and balance control. In a recent review, it is well established that balance control involves complex control mechanism of multiple systems in order to maintain balance [11]. In this review, Horak explains that the balance control system depends on postural orientation and postural equilibrium. Postural orientation is described as control of the alignment of body with respect to internal references, force of gravity, surface of support, and visual environment. Postural equilibrium is further described as stabilization of the body's centre of mass by integration of sensorimotor strategies. In this review, the complexity of balance control is divided into six categories of sensory strategies, motor strategies, biomechanical constraints, orientation in space, control of dynamics, and cognitive processing [11].

Sensory strategies involve the somatosensory, vestibular, and visual systems. The somatosensory system is important in balance control via the information from the stretch reflexes within the CNS (Kandel et al., 2000, p. 817) [1] including the proprioceptive and cutaneous receptors [12]. The proprioceptive reflexes involve the proprioceptors that are in place for sensation of body position and movement in space (Barnes and Johnson, 2008, p. 16) [6]. The cutaneous receptors are non-proprioceptive receptors that are responsible for activating the sensory afferents from the skin and subcutaneous tissue to carry information from touch, pressure, temperature, and pain (Barnes and Johnson, 2008, p. 28) [6]. The vestibular system contains the vestibular labyrinths that are located in the inner ears (Kandel et al., 2000, p. 801) [1]. The labyrinths make up five receptor organs namely the saccule, utricle, and three semicircular canals. The saccule and utricle are important for detecting

linear acceleration from movement of the body, and the semicircular canals are responsible for detecting angular acceleration from rotation of the head or body (Kandel et al., 2000, p. 802) [1]. The vestibular nuclei pick up the afferent stimuli for higher processing and control of the eyes through vestibule-ocular reflexes, and control of the musculoskeletal system via the vestibulospinal reflexes (Kandel et al., 2000, p. 808) [1]. The visual system is also important in balance control in the healthy and patient population [13]. The visual system is important for control of the eyes and body in space (Kandel et al., 2000, p. 782) [1], and provides critical information for the control of gaze by keeping the fovea on target when the head is stable as well as when it is in motion (Kandel et al., 2000, p. 783) [1]. The optokinetic signals processed by the visual system are important by integrating information provided by the vestibular system in stability of posture (Kandel et al., 2000, p. 828) [1]. Visual input from the surrounding environment can also provide important information about the location of body in space and its surroundings in order to control balance (Kandel et al., 2000, p. 828) [1]. Therefore, studies have been able to test balance control in presence or absence of visual feedback for example with eyes open or eyes closed in order to better understand how other sensory systems can compensate for balance control [14-16]. This is also suggested by Horak as the ability to reweight dependence on sensory inputs under different sensory contexts or environmental conditions [11]. As a result, in studies that control for effect of vision in balance control it is possible to understand how effective other sensory systems are able to compensate for lack of visual feedback information.

Movement strategies in balance control include: 1) the feet in place namely the ankle and hip strategies; and 2) the change of base of support strategy via reaching or stepping [11]. The ankle strategy as the name implies, mainly involves the control balance via torques exerted by the ankle muscles around the ankle joint to support the body. The hip strategy involves torques mainly exerted around the hips to move the COM of the body when the ankle strategy is not sufficient. Stepping or

reaching strategy may be used in more challenging tasks including perturbations where feet in place strategies are not sufficient to maintain balance.

Biomechanical constraints of balance control involve two main concepts of the base of support (BOS) and the centre of mass (COM) [11]. The BOS of the body during quiet standing is determined by the area defined by the perimeter around the feet and its control depends on the size, strength, range of motion, and pain levels. The COM of the body is defined as the net location of the weighted average of all body segments and its position with respect to the BOS is a main factor in balance control. The role of BOS and COM will further be discussed in detail when looking at balance control during quiet standing in the next chapter.

Orientation in space is the ability to align body segments with respect to gravity, surface and the visual field that depends on different neural systems [11]. For example, perception of visual verticality is different and independent of perception of postural or proprioceptive verticality. Damage in each system can lead to misalignment of the visual or proprioceptive verticality with respect to gravity, surface of support, and the external environment that can lead to postural instability.

In addition, Horak explains that dynamic control of balance is also a complex system of controlling the body's COM out of the range of BOS such as in walking or changing body postures. Cognition in balance control is also highlighted with higher cognitive processing required with more challenging postural tasks. It is also well established that psychosocial factors such as balance confidence and fear of fall can also have influences on falls and impaired balance, and that the cerebellum plays a key role for motor learning of balance control responses by directly communicating with the vestibular and visual systems that are involved in balance control of the body as previously discussed (Kandel et al., 2000, pp. 830, 841) [1].

In summary, a general introduction to stroke highlighted that damages to the upper motor neurons can lead to post-stroke spasticity amongst different challenges of stroke. In addition to spasticity, it was noted that movement difficulties and falls can also be main challenges after stroke by better understanding the sensitivity and complexity of the balance control system of the body. Therefore, it is important to understand if post-stroke spasticity may affect the balance control challenges that are reported after stroke. Hence, the focus of the current project is to test balance control in population of individuals with spasticity after stroke as described in the following chapter.

CHAPTER 3

MANUSCRIPT TO BE SUBMITTED:

IMPACT OF SPASTICITY ON BALANCE CONTROL DURING QUIET STANDING IN PERSONS POST-STROKE

3.1 Introduction

Spasticity is one of several positive signs from damage to the UMN and clinically accepted by Lance's definition as "a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes ('muscle tone') with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of upper motor neurons" [7]. Stroke is also known to result in neurological damage in the UMNs leading to spasticity (Brashear and Elovic, 2011, p. 357) [9] with studies reporting prevalence of post-stroke spasticity of up to 43% [17]. In persons with damage to UMNs and spasticity, major changes in the postural anatomy of the upper limbs may result in excessive internal rotation and adduction of the shoulder, elbow flexion, forearm pronation, wrist flexion, finger flexion, and thumb adduction and flexion (Brashear and Elovic, 2011, p. 72) [9]. In the lower limb, spasticity may result in excessive extension, internal rotation and adduction of the leg, plantar flexion and inversion of the ankle, and flexion of the toes (Barnes and Johnson, 2008, p. 3) [6]. As a result of these changes, it has been well established that individuals with spasticity have great challenges in managing their Activities of Daily Living (ADLs) (Brashear and Elovic, 2011, p. 5) [9].

In addition to challenges of spasticity introduced earlier, another common challenge in persons with stroke that affects independence in ADLs is impaired balance control [18]. In fact, measures of balance control have been related to post-stroke falls [19], and a falls incidence rate of up

to 65% has been reported among individuals with stroke [20]. The balance control of the body is complex and involves integration of multiple systems that when damaged can result in balance control challenges [11]. For example, asymmetry is reported between distributions of forces more on the non-paretic leg to compensate for inactivity of the paretic leg in order to maintain standing balance in stroke survivors [21]. Feed forward compensatory strategies in response to internal perturbations are also known to be delayed in the paretic and non paretic legs of individuals with stroke at the time of admission as compared to healthy individuals [22]. In response to external perturbations, persons with stroke have demonstrated greater challenges in maintaining standing balance as compared to healthy and older populations [23], including abnormally low ankle muscle activity on the paretic side [24]. Furthermore, problems such as difficulties to voluntary shift their weight between their feet, and relying on other strategies that do not involve the affected limbs such as the visual system and cognitive control to maintain balance have also been reported in individuals with stroke [18]. However, although such balance control challenges do exist in individuals post-stroke, the role of spasticity on balance control post-stroke is unclear [25]. It has been suggested that spasticity is a predictor of falls in persons post-stroke [26], but only a limited number of recent studies have suggested that balance control and inter-limb temporal synchronization is impaired in persons with spasticity post-stroke [27]. During quiet standing, the body sways as seen in an inverted pendulum model, with its kinematic and kinetic validity previously presented [28]. In this model, as the centre of mass (COM) of the body moves in the anterior posterior and mediolateral directions, the centre of pressure (COP) is responsible for maintaining the position of the COM within the base of support (BOS) to prevent COM from going outside the BOS and avoid falls during quiet standing (Winter, 2005, p. 107) [29]. The COM is the weighted average of location of all body segments, where the COP is the net location from weighted average of all downward forces applied at the

support surface [29]. It is important to note that as the controlled variable (COM) moves back and forth during quiet standing, the controlling variable (COP) is maintaining standing balance via muscle activity of the ankle plantar flexors and dorsiflexors [29]. However, spasticity can induce severe postural changes in the anatomy of upper and lower limbs, including the ankle plantarflexors. Since it is established that the action of ankle musculature is important for controlling COP movements in balance control of quiet standing [29], and that spasticity can result in plantarflexion and inversion (foot turning inwards) of the ankle (Barnes and Johnson, 2008, p. 3) [6] the findings suggest that post-stroke spasticity would provoke challenges in maintaining balance control during quiet standing. There is currently no evidence in the literature that suggests severity of spasticity is related to differences in balance control in persons post-stroke, and the aim of this study was to investigate whether greater severity of spasticity affects balance control in persons with spasticity post-stroke during quiet standing.

Aside from physical challenges of balance control, psychological factors such as fear of fall are important to consider among individuals with balance problems [30]. Balance confidence measures in performing ADLs are also known to be correlated with functional balance measures [31] and balance control measures during quiet standing [32]. In persons with stroke, balance self-efficacy measures have also shown to be an important predictor of balance impairments, and hence important to consider when studying falls related challenges in this population [33].

The objective of this study was to better understand how spasticity can affect balance control of individuals after stroke, and to the best of the author's knowledge this is the first study that investigates the question on how severity of spasticity can affect balance control of individuals post-stroke during quiet standing. This question was tested in this study based on hypotheses that post-

stroke individuals with high spasticity would display greater balance control challenges and fear of fall measures as compared to post-stroke individuals with low spasticity.

3.2 Methods

Individuals with spasticity post-stroke who were receiving treatment in an outpatient spasticity clinic were invited to participate using a script (Appendices A and B) if they met the study inclusion/exclusion criteria. Twenty-nine individuals agreed to participate in the study, and 27 individuals with spasticity post-stroke met the inclusion/exclusion criteria and were recruited in this study (subjects 4 and 23 were found not to meet inclusion/exclusion criteria on testing day and were excluded in all analyses). Inclusion criteria included the ability to stand quietly with eyes open and eyes closed independently for a total testing time of 10 minutes consisting of four trials of 80 seconds quiet standing, including breaks if needed. The exclusion criteria consisted of inability to stand unassisted; inability to follow simple instructions, due to cognitive impairments determined by clinicians; had received botulinum toxin injections within the past 3 months; and diagnosed with a cerebellar stroke. The inclusion/exclusion criteria were reviewed with the clinicians who have extensive knowledge of the physical functioning of the participants. The study received ethics approval from hospital and university ethical boards, and all participants were informed and agreed to participate by providing their consent to participate in this study according to hospital and university ethical guidelines (Appendix C).

Participants were divided in two groups based on the severity of ankle spasticity: 15 individuals with high ankle spasticity, and 12 individuals with low ankle spasticity. Severity of ankle spasticity was determined using the Modified Ashworth Scale (MAS) [10], and scored by a therapist with >7 years experience in spasticity assessments. The MAS has been shown to have a high inter

rater reliability [10] and reported as one of the most clinically feasible tools in a recent study [34]. In this study, high ankle spasticity was specifically defined as MAS scores of ≥2 in the ankle plantar flexors namely in the gastrocnemius and soleus muscles, and low ankle spasticity group was defined by MAS scores of <2 in gastrocnemius and soleus muscles. MAS scores were also gathered from assessment of other muscles with spasticity in the upper limbs (UL) and lower limbs (LL). Number of muscle groups with spasticity in UL and LL were used as dependent measures to better understand the relationship between spasticity in UL and LL and balance control and balance self-efficacy measures.

Balance control was tested in all participants during 80 seconds of quiet standing using centre of pressure (COP) measures and trunk sway measures near the centre of mass (COM). The COP of the body was calculated via moment and forces measured with a single force platform; trunk sway near COM was measured with the SwayStar™ (Balance International Innovations GmbH, Switzerland), which has embedded angular velocity sensors to measure angular deviations in the pitch (anterior-posterior) and roll (medial-lateral) directions. The SwayStar has been shown to provide repeatable, reliable, and sensitive measures in trunk sway of population of individuals with balance problems [35-37]. In each trial, participants stood quietly on the force plate with the SwayStar system mounted near the lumbar region of the trunk. Participants were instructed to stand quietly with their feet shoulder width apart, arms hanging by the side, and looking straight at a marker placed on the wall 3 metres ahead. Participants were instructed not to talk and to remain as still as possible during their quiet standing trials. Two clinic staff members stood near each side of participants in order to avoid any possible falls for participants' safety reasons. COP and trunk sway measures were simultaneously collected for 80 seconds. Equipment set-up is depicted in (Figure 3.1). In post-processing, the first and last 10 seconds were removed from the analysis, leaving 60 seconds of data

to analyze quiet standing as suggested in a different study [38]. The middle 60 seconds of quiet standing trials were analysed in all trials in order to further avoid the influence of any possible small unwanted adjustments by the participant in order to keep their quiet standing posture near the very beginning and end of the trials. A total of 4 trials (2 with eyes open, and 2 with eyes closed) were conducted with each participant as demonstrated in (Figure 3.2). The order of testing was kept consistent among all participants in order to keep the level of difficulty of testing conditions the same across all participants. COP measures were then calculated offline from forces and moments obtained from force plate data collected at sampling frequency of 100Hz. COP in the AP direction was calculated from moments in the ML axis divided by vertical ground reaction force, and COP in the ML direction was calculated from moments in the AP axis divided by the vertical ground reaction force from each sample (Winter, 2005, p. 98) [29]. COP data was then filtered using Butterworth low-pass filter at cutoff frequency of 10Hz for further processing of the COP signals. Filtered COP signals were then used to calculate COP root mean square (RMS), COP velocity, and mean power frequency (MPF) in the AP and ML directions. Trunk sway angular measures in the pitch and roll directions were calculated from trapezoid integration of angular velocity from on-line analysis of the SwayStar system (100Hz, 16 bit resolution) with transducer baseline drift of 0.002 deg/sec as described in a different study [39]. On-line measures of trunk sway used for analysis include pitch and roll angle range, pitch and roll angular velocity, and pitch and roll frequency amplitude recorded at a sampling rate of 100Hz. A summary of COP and trunk sway dependent measures is as follows:

1. COP:

RMS of COP displacement in the AP and ML directions was achieved via a formula by sum of square of all COP measures divided by total number of samples and taking the square root

(Robertson, Caldwell, Hamil, Kamen, and Whittlesey, 2004, p. 172) [40]. Using the first derivative of COP displacements, COP velocity in the AP and ML directions was calculated [29]. RMS of COP velocity in AP and ML was then calculated for a single measure of COP velocity in AP and ML. MPF of COP in the AP and ML directions was also calculated using a fast fourier transformation (FFT) algorithm for frequency analysis of COP in AP and ML [41].

2. Trunk sway measures:

Trunk sway dependent measures were calculated on-line and included 90% percentile range of the trunk angle movement in pitch and roll planes. Trunk velocity was calculated using 90% percentile range of trunk angular velocity in pitch and roll directions. Spectral densities in the pitch and roll planes were measured on-line every 0.4Hz and velocity amplitude was computed by taking its square root and reporting the average amplitude of three adjacent 0.4Hz intervals. For example, trunk roll frequency amplitude at 2.4Hz consisted of the average amplitude of 2.0Hz, 2.4Hz, and 2.8Hz frequencies as described in manufacturer's manual.

Figure 3.1 A representation of simultaneous data collection of COP and trunk sway during 80 seconds of quiet standing in which the middle 60 seconds were used for further data processing

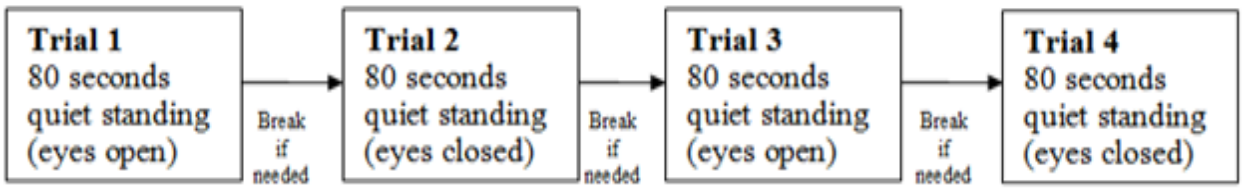

Figure 3.2 A representation of the order of testing quiet standing trials in study participants

In addition to balance control measures of quiet standing, fear of fall was also measured using the Activities-specific Balance Confidence scale (ABC) as it has been shown to be an important determinant of balance and mobility in population of individuals with spasticity [42], and highly correlated with functional balance measures [31]. The ABC scale (Appendix D) has shown to be a valid and reliable measure in individuals with stroke [43], and it is a 16 item questionnaire that asks participants to rate their confidence based on a percent score from 0% (no confidence) to a 100% (completely confident) in each item of the questionnaire. An average score from the 16 questions reflected individuals' fear of fall with lower numbers representing greater fear of fall as explained in a different study [30].

In the final clinical measure of the study, the clinic therapist assessed the motor impairment levels in the paretic side of the upper and lower limbs of all participants using the Chedoke-McMaster Stroke Assessment (CMSA). The CMSA (Appendix E) is a valid and reliable tool that is widely used to assess level of motor recovery, physical function, and rehabilitative outcomes in individuals after stroke [44]. In this study, only the arm, hand, leg, and foot motor recovery stages were assessed to better understand if outcome measures were influenced by motor impairment levels after stroke, and further explore if these scores were related to spasticity levels in the upper and lower limbs.

Statistical analyses:

All statistical analyses were conducted using JMP 12 (SAS Institute Inc. 2015. *Discovering JMP 12*®. Cary, NC: SAS Institute Inc.) A 2x2 mixed model univariate analyses of variance (ANOVA) was performed using the factors: *spasticity* (high spasticity vs. low spasticity) as the between subject factor and *vision* (eyes open vs. eyes closed) as the within subject factor. Tukey's post hoc test was used to further analyze any significant interaction effects. In the analyses of COP

and trunk sway dependent measures, statistical outliers were removed using *JMP 12*® statistical software box plot tool to achieve a more normally distributed sample across groups.

Independent Student's t-tests were used to compare differences in the ABC scores and CMSA scores between the high and low ankle spasticity groups. Additionally, Spearman's Correlation Coefficient (SCC) was used to understand the correlation between upper and lower limb spasticity with COP and trunk sway measures, and correlation between upper and lower limb spasticity with ABC scores. SCC was used since balance outcome measures and number of muscle groups with spasticity in the upper and lower limbs did not have normally distributed patterns. For all statistical testing α was set at value of 0.05.

3.3 Results

Participants characteristics:

All descriptive statistics results are reported as least square mean±SEM except when stated otherwise. Student's t-test on age between spasticity groups showed that the high spasticity group (HS) was significantly younger than the low spasticity group (LS); (HS= 61.8±3.0 years and LS= 74.3±3.4 years, p= 0.011. Eleven male and 4 female participants were in the high spasticity group, whereas 8 males and 4 females were in the low spasticity group. Participant characteristics are presented in table 3.1. Participant MAS scores in the UL and LL are summarized in table 3.2 and table 3.3, respectively.

Table 3.1

Participant characteristics table

	Low ankle spasticiy (MAS <2)	High ankle spasticity (MAS ≥2)
n	12	15
Age (years)	74.3±3.4*	61.8±3.0
Sex (F/M)	4/8	4/11
Affected side (L/R)	5/7	7/8
ABC	57±5.5	68±4.9
CMSA foot	3.83±0.25*	2.73±0.22
CMSA leg	4.08±0.17*	3.33±0.15
Stroke Type		
Ischemic	3	5
Haemorrhagic	2	2
Lacunar infarct	1	1
Not available	6	7

Note. Age (mean±SEM), ABC: Activities-specific Balance Confidence (mean±SEM), CMSA: Chedoke McMaster Stroke Assessment (mean±SEM), F: Female, M: Male, L: Left, R: Right; *represents statistically significant difference, $p < 0.05$

Table 3.2

MAS scores in UL muscle groups of participants

Subject	Sh Add	Sh Ro	Elb F	Elb E	Pro	Wrist F	Wrist E	FDS	FDP	Add P	Opp P	FPL	Lumb
1	1.5	0	1	0	1	0	0	0	0	0	0	0	0
2	1	0	1.5	2	0	1.5	0	2	1.5	2	0	0	0
3	1.5	1.5	1.5	1.5	1	1.5	0	1.5	1.5	1.5	2	1.5	0
5	1	0	1.5	0	2	1.5	0	0	0	0	0	1	1
6	1.5	1.5	1.5	1.5	1.5	1	0	0	0	0	0	0	0
7	1.5	1	2	0	2	1.5	0	1	1	0	0	1	2
8	2	2	1.5	2	1	1.5	0	2	1.5	0	2	0	0
9	0	1.5	2	0	1.5	1.5	0	1.5	1.5	0	0	0	1.5
10	1.5	0	1.5	0	1	2	0	0	0	0	1.5	0	0
11	1.5	1.5	1.5	1	1.5	1	0	1.5	1.5	0	1.5	0	0
12	1.5	1.5	2	2	1.5	1.5	0	1	1	0	0	2	0
13	1.5	1	1.5	1.5	0	2	0	2	1.5	0	0	1	0
14	1.5	2	2	0	2	3	0	2	1.5	1.5	0	0	1
15	1.5	1.5	1	0	2	2	0	0	0	2	0	0	0
16	1	0	0	0	0	0	0	2	0	0	2	0	2
17	1.5	2	1.5	0	1.5	2	0	0	0	0	0	0	0
18	2	2	1.5	1.5	2	1	0	2	1.5	1	0	1.5	2
19	1.5	1.5	0	1.5	1.5	0	1.5	1.5	1.5	1.5	2	2	1.5
20	1.5	1.5	1.5	1.5	1.5	1	0	0	0	0	0	0	0
21	0	0	0	0	0	0	0	0	0	0	0	0	0
22	0	0	1	0	1.5	1.5	0	0	0	0	0	0	0
24	2	2	2	3	3	2	0	3	2	1.5	2	2	2
25	1.5	2	2	1.5	1.5	1	0	1.5	0	0	0	1.5	2
26	2	1.5	1.5	2	2	3	0	2	2	0	1.5	2	3
27	1.5	1	1.5	0	1.5	1	0	0	0	0	0	0	0
28	1.5	1.5	1	0	1	1.5	0	1.5	1.5	2	2	0	0
29	0	1.5	1.5	1.5	2	2	0	3	0	0	0	2	3

Note. Sh Add: Shoulder Adductors, Sh Ro: Shoulder Rotators, Elb F: Elbow Flexors, Elb E: Elbow Extensors, Pro: Pronators, Wrist F: Wrist Flexors, Wrist E: Wrist Extensors, FDS: Flexor Digitorum Superficialis, FDP: Flexor Digitorum Profundus, Add P: Adductor Pollicis, Opp P: Opponens Pollicis, FPL: Flexor Pollicis Longus, Lumb: Lumbricals. MAS score of 1+ is reported as 1.5

Table 3.3

MAS scores in LL muscle groups of participants

Subject	Hip Adductors	Knee Flexors	Knee Extensors	Gastrocnemius	Soleus	Tibialis Posterior	Extensor Hallucis Longus
1	1	1.5	2	3	0	0	0
2	1	1	0	1.5	1.5	0	0
3	1.5	1.5	1.5	1.5	1.5	1.5	1.5
5	1	1.5	1.5	3	1.5	1.5	0
6	0	0	1.5	1.5	1.5	1.5	0
7	1	0	1	1.5	1.5	1	0
8	1.5	1.5	1.5	3	1.5	2	1.5
9	1.5	0	1	2	1.5	0	0
10	1.5	1.5	0	1.5	1.5	1.5	0
11	1.5	1	1.5	1.5	0	1	0
12	1	0	1.5	1	1	1	0
13	1.5	1.5	1	1.5	1.5	0	0
14	1	1	1	2	1.5	0	0
15	1	0	0	1.5	1	1	0
16	1.5	1	0	1.5	0	2	0
17	1.5	0	1.5	0	1.5	1.5	0
18	1	1	0	2	1.5	2	0
19	1.5	1	1.5	3	2	2	0
20	1	1.5	1.5	2	1.5	1.5	0
21	1.5	1.5	1.5	3	2	3	0
22	0	0	2	2	1.5	0	0
24	2	2	2	3	2	3	0
25	1.5	1.5	2	3	3	2	0
26	1	1.5	1.5	3	2	2	1.5
27	1	0	0	1.5	1.5	1	0
28	2	1.5	1	2	2	1.5	0
29	1.5	1	0	2	2	1	1

A visual representation of similar pattern of movement on COP and trunk angle in the anterior posterior direction during the same trial in 60 seconds of quiet standing with eyes open is represented (Figure 3.3).

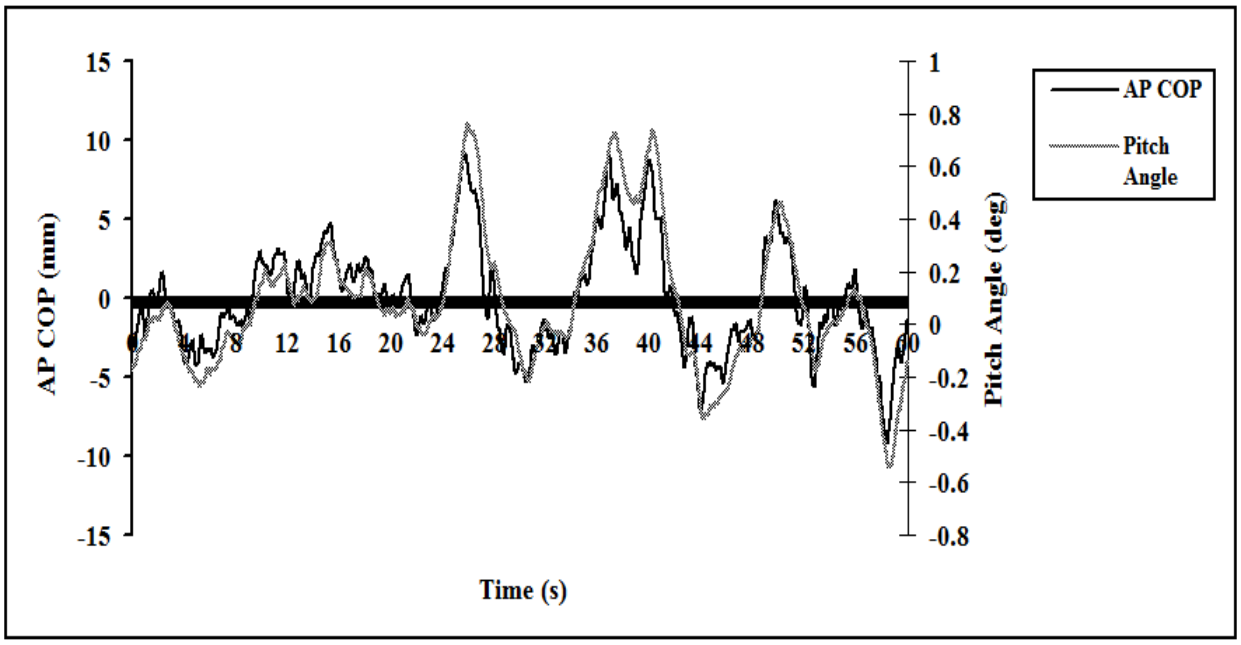

Figure 3.3 Graph represents a similar pattern of COP and trunk angle movement in the anterior (positive) and posterior (negative) direction during a trial of 60 seconds quiet standing with eyes open by an individual with low ankle spasticity post-stroke. These results further highlight the high synchronization of COP and COM movements established in the literature by the inverted pendulum model. *Note:* AP: anterior-posterior, COP: centre of pressure

COP measures:

COP RMS: Analysis revealed that the interaction effect between *vision* and *spasticity* was not statistically significant ($F(1,75.44)= 0.016$, $p= 0.90$) for COP movement in the AP direction; the main effect of *spasticity* was not significant ($F(1,24.94)= 0.0002$, $p= 0.98$), however, the main effect of *vision* indicated that COP RMS in the AP direction was greater in the eyes-closed (EC) condition than the eyes open (EO) condition (EO= 4.7±0.19mm vs. EC= 5.5±0.2mm, $F(1,75.44)= 14.63$, $p= 0.0003$). The interaction effect between *vision* and *spasticity* was not statistically significant ($F(1,70.45)= 0.097$, $p= 0.76$) for COP movement in the ML direction; the main effect of *spasticity* was not significant ($F(1,23.48)= 0.016$, $p= 0.91$), however the main effect of *vision* indicated that COP RMS in the ML direction was greater in the eyes-closed condition (EO= 3.1±0.25mm vs. EC= 3.5±0.26mm, $F(1,70.45)= 5.82$, $p= 0.019$).

COP MPF: Analysis revealed that the *vision* by *spasticity* interaction effect was not significant ($F(1,73)= 0.069$, $p= 0.79$) for MPF measures in AP direction. There was no main effect of *spasticity* for MPF measures in AP direction ($F(1,23)= 0.79$, $p= 0.38$). A main effect of *vision* was observed in MPF measures in the AP direction (EO= 0.37±0.041Hz vs. EC= 0.43±0.041Hz, $F(1,73)= 8.68$, $p= 0.0043$). MPF measures in the ML direction did not reveal a significant *vision* by *spasticity* interaction effect ($F(1,70.14)= 0.19$, $p= 0.67$). A significant main effect of *spasticity* was observed in ML MPF results with higher measures in the HS group as compared to the LS group (HS= 0.35±0.025Hz and LS= 0.27±0.032Hz, $F(1,22.61)= 4.52$, $p= 0.045$). There was no main effect of *vision* ($F(1,70.14)= 0.0055$, $p= 0.94$) in ML MPF measures. The main findings on ML MPF analysis is further presented in (Figure 3.4).

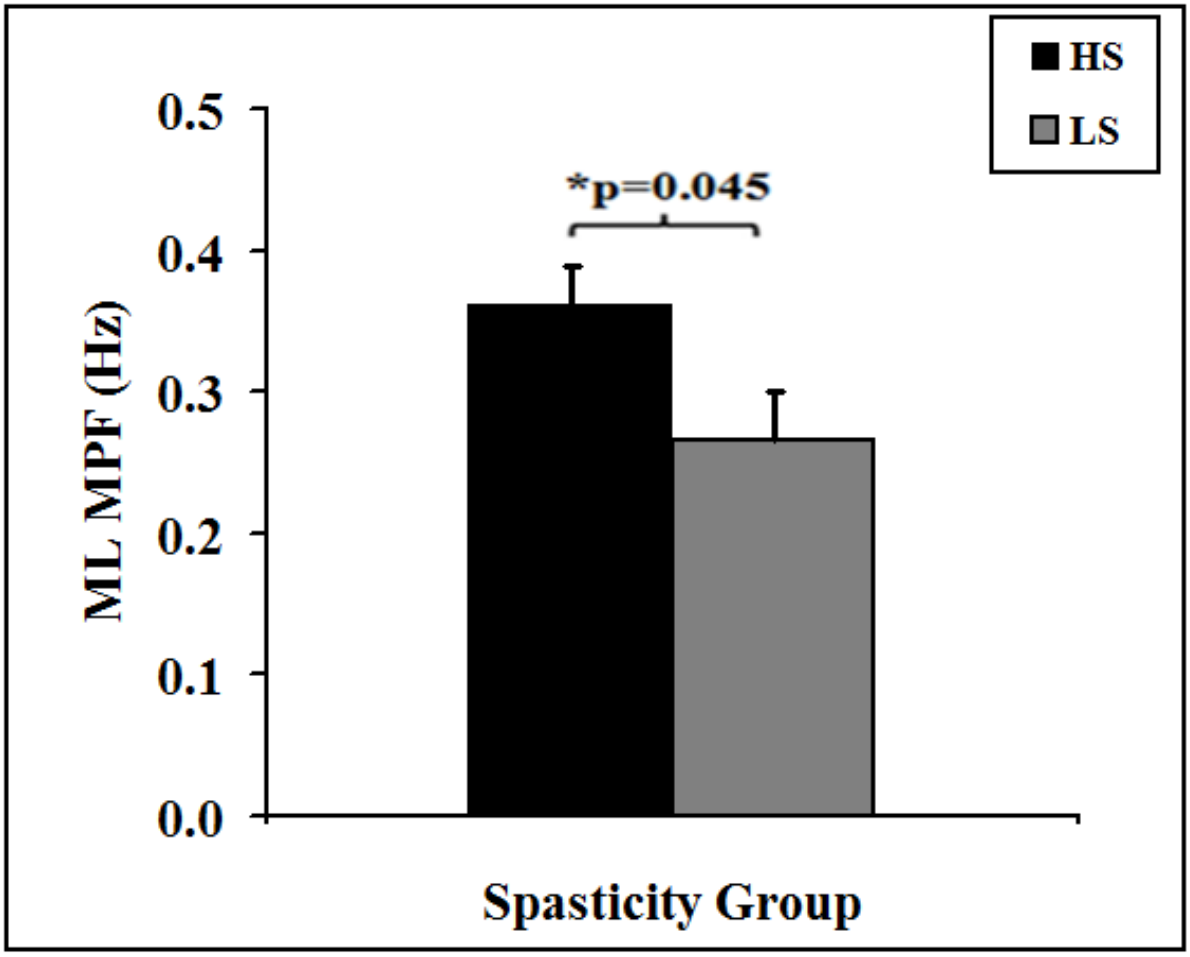

Figure 3.4 Representation of COP MPF measures between high spasticity and low spasticity groups in mediolateral direction. Significant difference was noted in ML MPF between groups (*represents statistically significant difference, $p < 0.05$). *Note.* HS: high spasticity, LS: low spasticity, ML: medial-lateral, MPF: mean power frequency

COP velocity: Results revealed that there was no *vision* by *spasticity* interaction effect in COP velocity measures in AP direction ($F(1,73)= 0.72$, $p= 0.39$). No main effect of *spasticity* was observed in AP-COP velocity measures ($F(1,23)= 1.23$, $p= 0.28$). However, there was a main effect of *vision* in COP velocity in the AP direction (EO= 15.8±1.9mm/sec and EC= 22.4±1.9mm/sec, $F(1,73)= 38.88$, $p< 0.0001$). COP velocity measures in the ML direction showed significant *vision* by *spasticity* interaction effect ($F(1,71.07)= 4.73$, $p= 0.033$). Tukey's post hoc test revealed that ML COP velocity was higher in high spasticity group in eyes closed condition versus high spasticity group in eyes open condition (HS/EC= 13.7±1.6mm/sec vs. HS/EO= 9.5±1.6mm/sec, $p< 0.0001$). There were no significant differences between the low spasticity group in eyes closed condition versus the low spasticity group in eyes open condition ($p= 0.31$). There was a trend towards significance between high spasticity group in eyes closed condition and low spasticity group in eyes closed condition (HS/EC= 13.7±1.6mm/sec vs. LS/EC= 7.7±2.0mm/sec, $p= 0.09$). There were no significant differences between high spasticity group in eyes open condition and low spasticity group in the eyes open condition ($p= 0.54$). These results are further presented in (Figure 3.5). The main effect of *spasticity* revealed that COP velocity measures in ML direction showed a trend towards significance with higher measures observed in the high spasticity group (HS= 11.6±1.6mm/sec and LS= 6.9±1.9mm/sec, ($F(1,22.83)= 3.63$, $p= 0.069$) as presented in (Figure 3.6). Also, a main effect of vision was observed in COP velocity measures in ML direction (EO= 7.8±1.2mm/sec and EC= 10.7±1.2mm/sec, $F(1,71.07)= 23.25$, $p< 0.0001$).

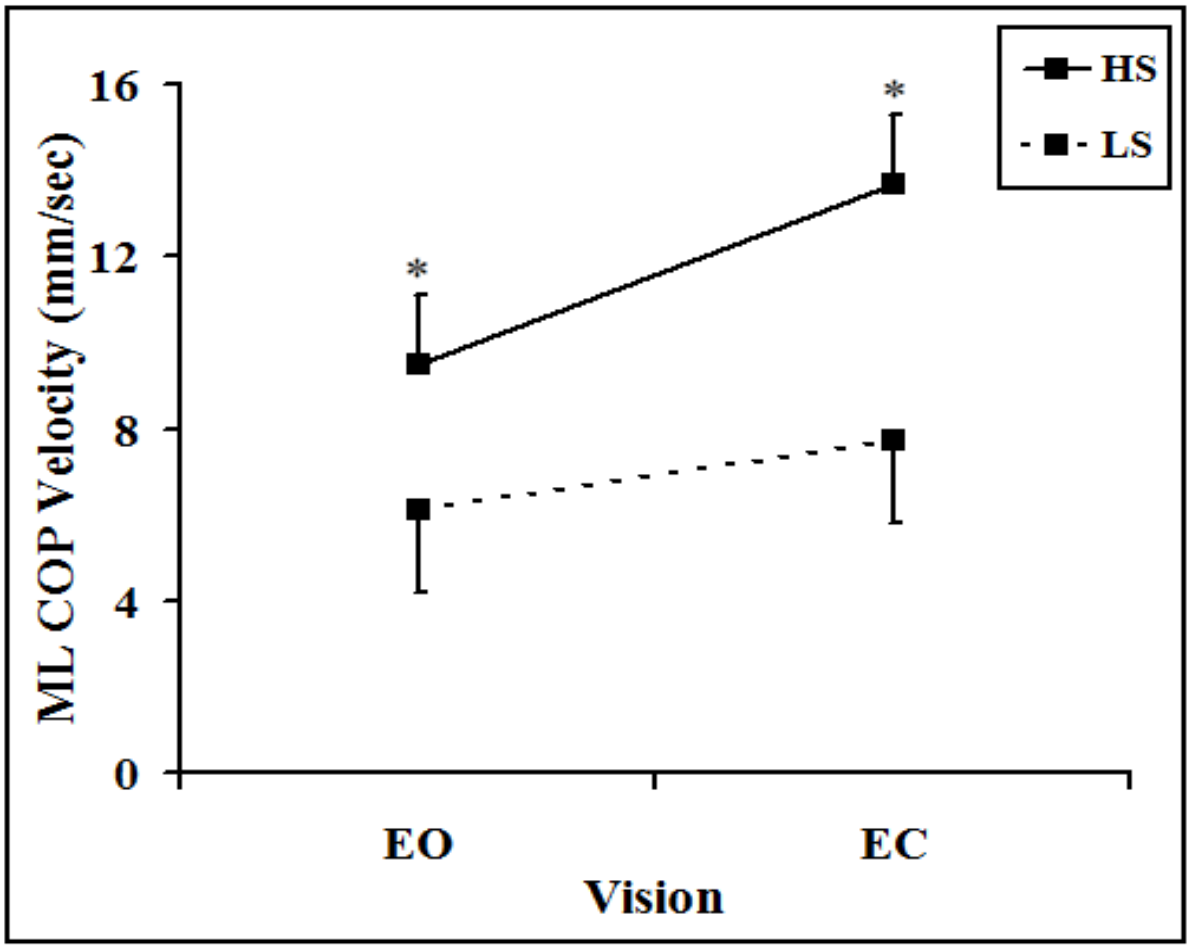

Figure 3.5 A significant vision by spasticity interaction effect representing individuals with high spasticity had significantly higher ML COP velocity measures in absence of vision, whereas the low spasticity group had relatively similar measures in the absence of vision (*represents statistically significant difference between groups marked with this symbol, $p < 0.0001$). *Note:* HS: high spasticity, LS: low spasticity, EO: eyes open, EC: eyes closed, ML: medial-lateral, COP: centre of pressure

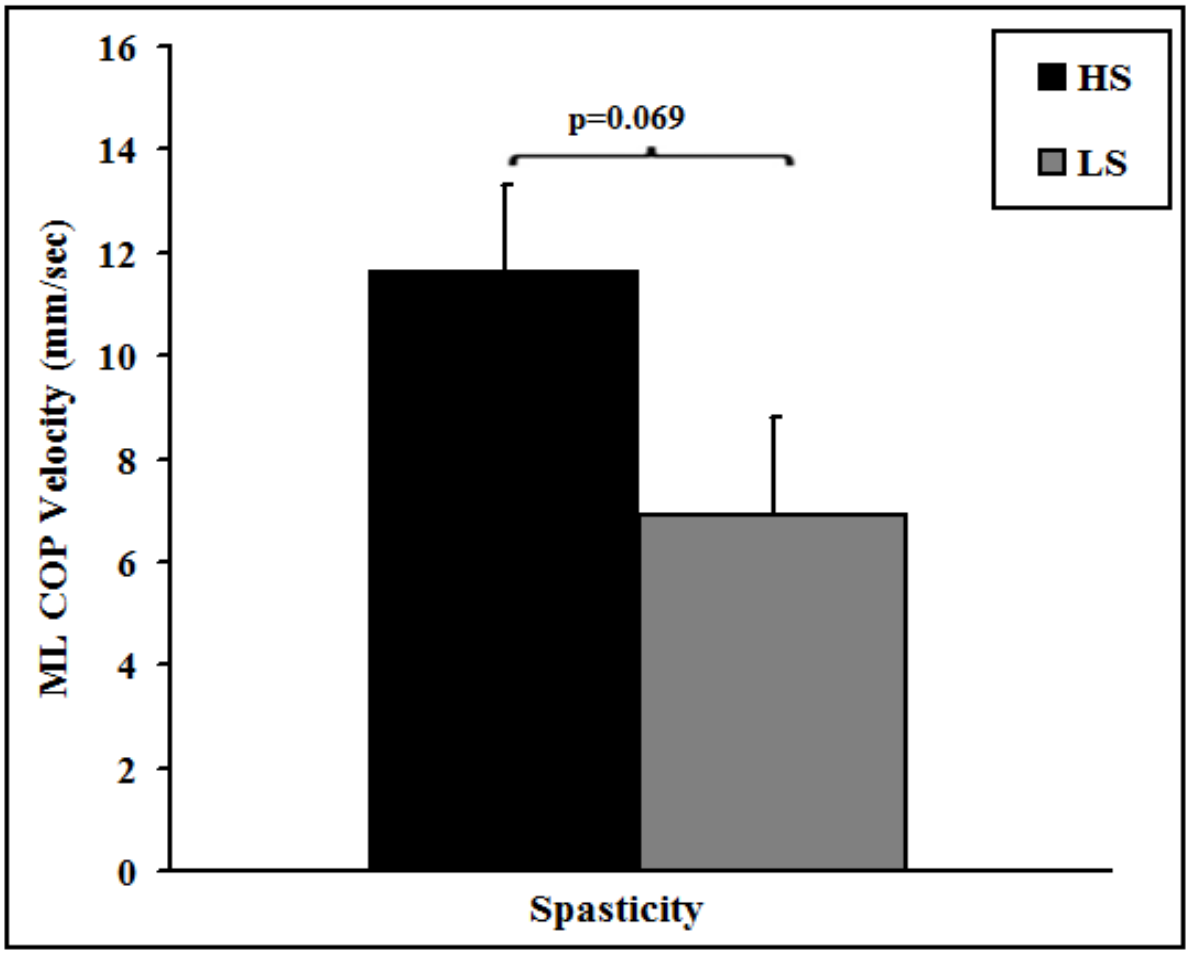

Figure 3.6 Trend towards significance showing that the high spasticity group had higher COP velocity measures compared to low spasticity group with this difference noted in the mediolateral direction. *Note:* HS: high spasticity, LS: low spasticity, ML: medial-lateral, COP: centre of pressure

Trunk sway measures:

Trunk angle: Analysis of trunk angle measures in pitch direction revealed no *vision* by *spasticity* interaction effect ($F(1,79)= 0.45$, $p= 0.50$). There was no main effect of *spasticity* ($F(1,25)= 0.22$, $p= 0.64$), and no main effect of *vision* ($F(1,79)= 1.75$, $p= 0.19$) in trunk pitch angle measures. Trunk angle measures in the roll direction revealed no *vision* by *spasticity* interaction effect ($F(1,72.23)= 0.67$, $p= 0.41$). There was no main effect of *spasticity* on trunk roll angle ($F(1,23.6)= 0.002$, $p= 0.96$), but there was a main effect of *vision* in trunk roll angle (EO= 0.55±0.069deg and EC= 0.78±0.067deg, $F(1,72.23)= 17.83$, $p< 0.0001$).

Trunk velocity: *Vision* by *spasticity* interaction effect was not significant in trunk pitch velocity measures ($F(1,74.61)= 1.76$, $p= 0.19$). The main effect of *spasticity* was not significant ($F(1,24.07)= 2.06$, $p= 0.16$), but there was a significant main effect of *vision* (EO= 1.76±0.11deg/sec, EC= 2.23±0.12deg/sec, $F(1,74.61)= 50.4$, $p< 0.0001$) in trunk pitch velocity measures. Trunk roll velocity measures revealed a significant *vision* by *spasticity* interaction effect ($F(1,71.18)= 4.52$, $p= 0.037$). Tukey's post hoc test revealed that trunk roll velocity measures were different between high spasticity eyes closed condition and high spasticity eyes open condition (HS/EC= 0.94±0.086deg/sec vs. HS/EO= 0.71±0.086deg/sec, $p= 0.0002$). There were no significant differences between low spasticity eyes closed condition and low spasticity eyes open condition ($p= 0.88$), high spasticity eyes closed condition and low spasticity eyes closed condition ($p= 0.25$), and high spasticity eyes open condition and low spasticity eyes open condition ($p= 0.94$). There was no main effect of *spasticity* ($F(1,23.08)= 1.67$, $p= 0.21$), but a significant main effect of *vision* (EO= 0.67±0.068deg/sec, EC= 0.81±0.069deg/sec, $F(1,71.18)= 10.84$, $p= 0.0015$) in trunk roll velocity measures. Trunk roll velocity measures are further presented in (Figure 3.7).

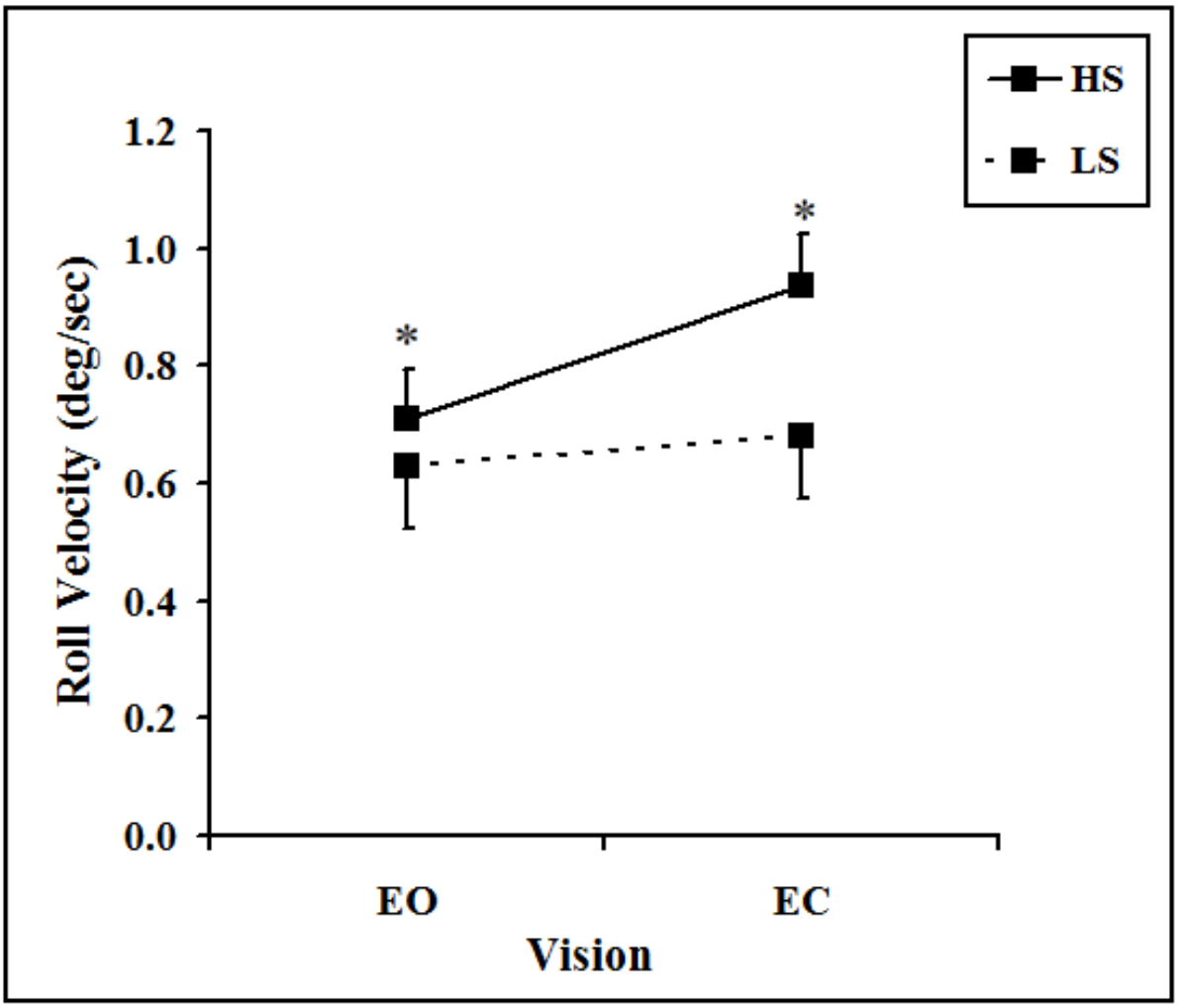

Figure 3.7 Interaction effect of vision by spasticity on trunk velocity in the roll plane. These results suggest that individuals with high spasticity had significantly higher trunk roll velocity measures in absence of vision, whereas trunk roll velocity was not significantly affected in absence of vision in individuals with low spasticity (*represents statistically significant difference between groups marked with this symbol, p= 0.0002). *Note:* HS: high spasticity, LS: low spasticity, EO: eyes open, EC: eyes closed

Frequency analysis of trunk sway amplitude revealed a significant *vision* by *spasticity* interaction effect ($F(1,62.03)= 6.24$, $p= 0.015$) in trunk roll velocity amplitude at 3.7Hz. Tukey's post hoc test revealed that trunk frequency measures were significantly higher in the high spasticity eyes closed condition versus high spasticity eyes open condition (HS/EC= 0.35±0.032deg/sec vs. HS/EO= 0.27±0.031deg/sec, $p< 0.0001$). There were no significant differences between low spasticity eyes closed condition and low spasticity eyes open condition ($p= 0.89$). There was a significant difference between high spasticity eyes closed condition and low spasticity eyes closed condition (HS/EC= 0.35±0.032deg/sec vs. LS/EC= 0.20±0.04deg/sec, $p= 0.016$). There were no significant differences between high spasticity eyes open condition and low spasticity eyes open condition ($p= 0.29$). Results showed a significant main effect of *spasticity* (HS= 0.31±0.030deg/sec and LS= 0.18±0.038deg/sec, $F(1,20.37)= 6.39$, $p= 0.02$), and a significant main effect of *vision* (EO= 0.23±0.025deg/sec, EC= 0.26±0.025deg/sec, $F(1,62.03)= 13.04$, $p= 0.0006$) in roll velocity amplitude in the 3.7Hz frequency domain. Main findings on trunk sway frequency measures are further presented in (Figure 3.8).

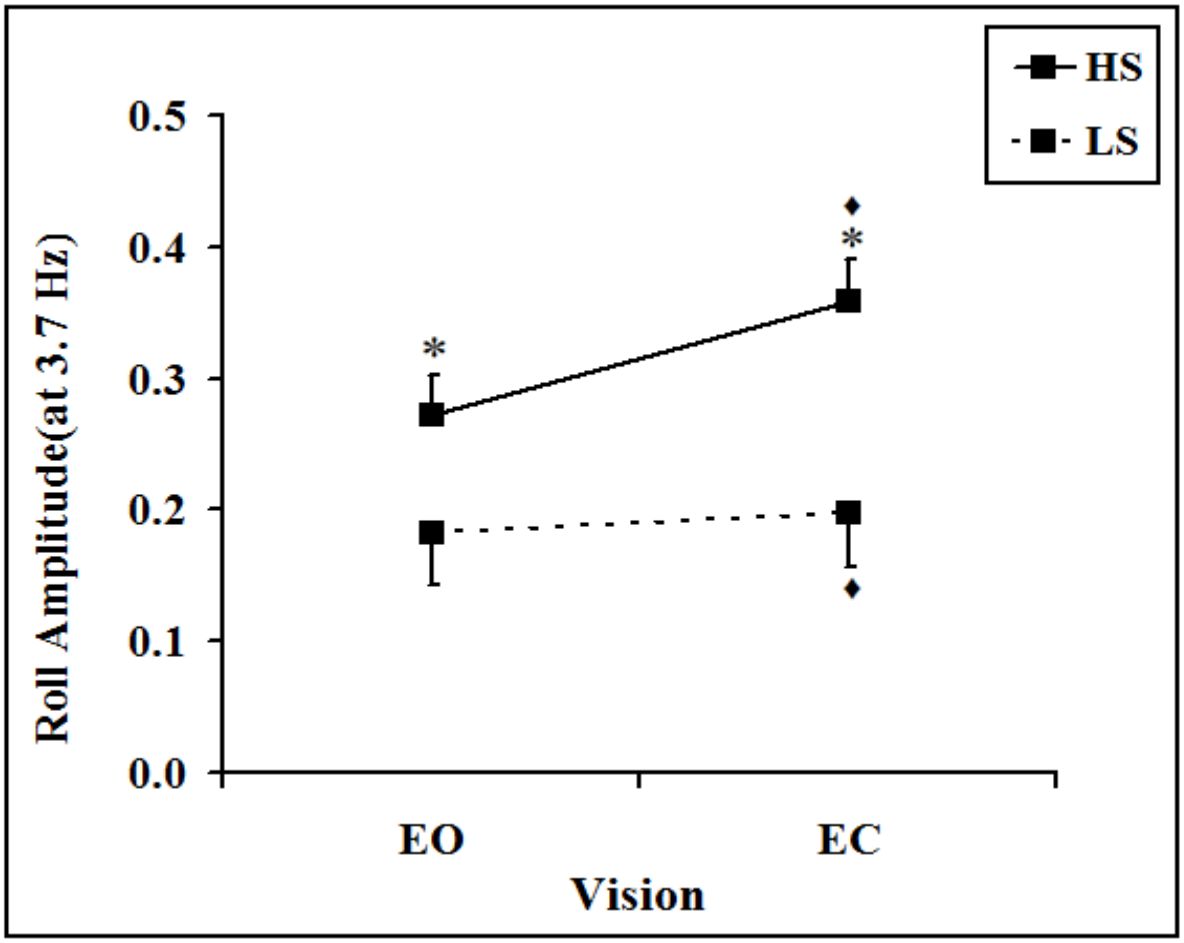

Figure 3.8 Vision by spasticity interaction effect on trunk roll velocity amplitude(deg/sec) at 3.7Hz. These results suggest that in trunk roll velocity amplitude at 3.7Hz, the high spasticity group had significantly higher values in the absence of vision, whereas this effect was not seen in the low spasticity group (*represents statistically significant difference between groups marked with this symbol, $p < 0.0001$). Furthermore, results suggest that there was a difference between high and low spasticity groups in performing the more difficult task in the eyes closed condition only (♦represents statistically significant difference between groups marked with this symbol, $p = 0.016$). *Note:* HS: high spasticity, LS: low spasticity, EO: eyes open, EC: eyes closed

ABC scores:

There was no significant difference in ABC scores between high vs. low ankle spasticity groups (HS= 68±4.9 and LS= 57±5.5, p= 0.14).

CMSA scores:

Analysis of motor impairment levels between groups showed no significant difference between groups in arm CMSA (LS= 2.83±0.24 and HS= 3.13±0.23, p= 0.39) and hand CMSA (LS= 2.83±0.37 and HS= 3.07±0.33, p= 0.64), but a significant difference in leg CMSA (LS= 4.08±0.17 and HS= 3.33±0.15, p= 0.0024) and foot CMSA (LS= 3.83±0.25 and HS= 2.73±0.22, p= 0.003) between groups.

Effects of Age, sex, and motor impairment on balance measures

The effect of age on COP velocity ML measure was not significant in the low spasticity group (r^2= 0.027, p= 0.31) or high spasticity group (r^2= 0.001, p= 0.81). The effect of foot impairment from CMSA foot on COP velocity ML was also not significant in the low spasticity (r^2= 0.003, p= 0.75) or high spasticity group (r^2= 0.036, p= 0.15). The effect of age on roll velocity was not significant in the low spasticity (r^2= 0, p= 0.99) and high spasticity group (r^2= 0.0007, p= 0.84). The effect of foot CMSA on roll velocity was also not significant in the low spasticity group (r^2= 0.003, p= 0.74) and high spasticity group (r^2= 0, p= 0.99). The effect of age was significant on MPF ML of low spasticity group (r^2= 0.22, p= 0.0032) but not significant for the high spasticity group (r^2= 0.012, p= 0.41). The effect of CMSA foot was not significant on MPF ML of low spasticity group (r^2= 0.015, p= 0.46) and high spasticity group (r^2= 0.037, p= 0.15). The effect of age on roll velocity amplitude at 3.7Hz was significant in the low spasticity group (r^2= 0.27, p= 0.0013) but not significant in the high spasticity

group (r^2= 0.004, p= 0.63). The effect of foot CMSA on roll velocity amplitude at 3.7Hz was also not significant in the low spasticity group (r^2= 0.0071, p= 0.63) and high spasticity group (r^2= 0.049, p= 0.11). Since the effect of age and foot impairment was not significant on the outcome measures in both groups, a further analysis to include age or foot impairment as a covariate was not necessary.

The difference in balance control measures based on sex was not controlled, but the number of females in the low and high spasticity groups was equal with females (n= 4) in HS and LS groups.

Correlation analyses:

The correlation between COP and trunk sway measures with number of muscles with spasticity in the UL and LL revealed weak but significant relationships. Number of muscles with spasticity in UL was weakly correlated with AP-COP velocity (rho= 0.21, p= 0.039), AP-MPF (rho= -0.26, p= 0.0082), trunk pitch velocity (rho= -0.29, p= 0.0037), trunk roll frequency 2.5Hz (rho= -0.39, p< 0.0001), and trunk pitch frequency 1.4Hz (rho= -0.34, p= 0.0005), and 2.5Hz (rho= -0.28, p= 0.0047). Number of muscles with spasticity in LL was weakly correlated with ML-MPF (rho= 0.21, p= 0.035), COP RMS in ML (rho= -0.25, p= 0.013), trunk roll frequency 2.5Hz (rho= -0.21, p= 0.034) and 3.7Hz (rho= 0.27, p= 0.012), and trunk pitch velocity (rho= -0.22, p= 0.026), trunk pitch frequency 2.5Hz (rho= -0.22, p= 0.029) and 3.7Hz (rho= 0.22, p= 0.036). These results are presented in tables 3.4, 3.5, and 3.6. Correlation between ABC scores and number of muscle groups in UL and LL showed no significant correlation between ABC scores and number of muscles with spasticity in UL (rho= 0.061, p= 0.76) or LL (rho= -0.15, p= 0.47).

Table 3.4

Correlation analysis between number of muscle groups with spasticity in upper and lower limbs with COP measures

	AP COP		ML COP		AP COP Velocity		ML COP Velocity		AP MPF		ML MPF	
	rho	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**
UL (MAS >0)	-0.021	0.83	-0.13	0.2	**0.21**	**0.039***	-0.046	0.65	**-0.26**	**0.0082***	-0.054	0.6
LL (MAS >0)	-0.0084	0.93	**-0.25**	**0.013***	0.015	0.89	0.065	0.53	0.006	0.95	**0.21**	**0.035***

Note. MAS: Modified Ashworth Scale; AP: anterior-posterior; ML: medial-lateral; COP: centre of pressure; MPF: mean power frequency; UL: upper limb; LL: lower limb; *represents statistical significant correlation, $p < 0.05$

Table 3.5

Correlation analysis between number of upper and lower limb muscle groups with spasticity and trunk sway measures in pitch direction

	Pi Angle		Pi Velocity		Pi Amp 1.4Hz		Pi Amp 2.5Hz		Pi Amp 3.7Hz		Pi Amp 4.9Hz	
	rho	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**
UL (MAS >0)	0.03	0.76	**-0.29**	**0.004***	**-0.34**	**0.0005***	**-0.28**	**0.0047***	-0.05	0.64	-0.09	0.36
LL (MAS >0)	-0.074	0.44	**-0.22**	**0.026***	-0.17	0.096	**-0.22**	**0.029***	**0.22**	**0.036***	0.13	0.22

Note. MAS: Modified Ashworth Scale; UL: upper limb; LL: lower limb; Pi: pitch; Amp: amplitude; *represents statistical significant correlation, $p < 0.05$

Table 3.6

Correlation analysis between number of upper and lower limb muscle groups with spasticity and trunk sway measures in roll direction

	Ro Angle		Ro Velocity		Ro Amp 1.4Hz		Ro Amp 2.5Hz		Ro Amp 3.7Hz		Ro Amp 4.9Hz	
	rho	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**	**rho**	**p**
UL (MAS >0)	-0.038	0.7	-0.12	0.25	-0.15	0.13	**-0.39**	**<0.0001***	-0.08	0.46	-0.07	0.47
LL (MAS >0)	-0.12	0.24	-0.08	0.46	0.06	0.55	**-0.21**	**0.034***	**0.27**	**0.012***	0.18	0.08

Note. MAS: Modified Ashworth Scale; UL: upper limb; LL: lower limb; Ro: roll; Amp: amplitude; *represents statistical significant correlation, $p < 0.05$

3.4 Discussion

The main findings of this study suggest that balance control of persons with stroke is further impaired with higher spasticity severity. Moreover, the results indicate that such balance control challenges are mainly noted in the frontal plane affecting mediolateral control of balance in this population. The changes in balance control measures noted from COP and trunk sway measures are further discussed with respect to preexisting findings in the literature.

COP differences from severity of spasticity:

The results of this study indicated that net COP RMS in AP and ML directions was similar among individuals with respect to severity of spasticity. This finding closely resembles the finding in Singer et al., study where they also report no differences in AP/ML net COP RMS of stroke individuals with and without spasticity [27]. However, COP velocity results in ML direction showed a *vision* by *spasticity* interaction effect suggesting that individuals with high spasticity have greater challenges under the eyes closed condition than individuals with low spasticity. This suggests that individuals with high ankle spasticity may be more dependent on their vision for balance control, than individuals with low ankle spasticity. It is important to note that COP measures including COP velocity in the ML direction is an important measure to consider in balance control as it is reported to be strongly associated with and predictive of future falls among the elderly in a review [15, 45]. COP velocity has been shown to be highly reliable amongst other COP measures in healthy and stroke individuals [46, 47]. COP velocity has been reported to be correlated with functional balance measures using the Berg Balance Scale (BBS) [48, 49] and the Functional Standing Balance scale (FSB) [50]. The higher COP velocity measures observed in the high spasticity group within the eyes closed condition resembles greater balance control challenges as previously noted in other studies of

individuals with stroke. The AP and ML COP velocity measures of stroke persons with spasticity reported in the current study are similar to COP velocity measures previously observed to be greater than controls [51]. Furthermore, the higher COP velocity numbers observed in the absence of vision in this study are similar to findings of Pyoria et al., in their study in individuals with acute and chronic stroke [50] representing greater balance control challenges in the absence of vision.

The MPF of the COP was another measure that was found to be significantly different in the group of individuals with high vs. low spasticity. In this study, individuals with higher spasticity levels demonstrated higher MPF measures in the ML as compared to the low spasticity group. The MPF findings are important to consider since greater MPF measures are known to represent greater balance control challenges in population of individuals with balance problems. For example, in a study by Demura et al., [52], the older adults were shown to have higher COP MPF measures than younger adults. In their study, they explained that COP sway characteristic of the younger adults were presented in the lower 0.02-0.2Hz domain whereas COP sway of older adults were in the higher frequency range of 0.2-2.0Hz during standing trials. It is interesting to note that the MPF measures in our study are closer to the higher frequency ranges between the 0.2-2.0Hz observed within the elderly population. In another study [53], MPF measures during standing trials were shown to be higher in individuals with stroke as compared to matched healthy controls. The increase in MPF has been related to a stiffening strategy; Carpenter et al. reported that MPF measures were higher with greater postural threat while standing on higher versus lower heights in order to control the stability of the body [54]. Since higher balance control measures were noted in the high spasticity group, especially in the eyes closed trials, which generally represent a more challenging task to control balance as compared to trials with eyes open, a stiffening strategy may explain the higher MPF measures observed in the high spasticity group in this study. Therefore, an indirect interpretation from study

results could be that stroke persons with greater severity of spasticity may have used a stiffening strategy to maintain their balance during quiet standing trials when compared to individuals with low spasticity levels. Since higher frequency measures in the high spasticity group were also observed near the trunk, it may be well possible that a behavioral stiffening strategy was used as increase in muscle tone around trunk muscle is less likely and not measured in the current study. Alternatively, it has been demonstrated that during quiet standing, the frequency of oscillations is proportional to velocity of oscillations which is also proportional to a stiffness measure of a spring model that represents the tone of muscles in control of balance [55]. Therefore, another interpretation from observing higher MPF, COP velocity and trunk velocity measures in the high spasticity group may be that an increase in ankle muscle tone from high level of spasticity around the ankle may be responsible for the higher COP frequency measures in the high spasticity group. Future studies may further investigate the possibility of these underlying mechanisms for increased frequency measures observed in individuals with high spasticity post-stroke.

Severity of spasticity on trunk sway

The findings of this study also suggested that trunk control stability in persons with high spasticity was different than low spasticity group trunk control based on vision by spasticity interaction effect on angular velocity in the roll plane. These results suggest that among individuals with high ankle spasticity, trunk angular velocity is significantly higher in absence of vision compared to when vision is available. However, the low spasticity group does not demonstrate this challenge as they present similar trunk roll velocity in presence or absence of vision. It is important to note here that higher trunk velocity measures represent greater difficulties in balance control as higher numbers were reported in elderly as compared to young controls [56] as well as in individuals with

balance problems versus healthy matched controls [16]. Also, absence of vision is reported to result in higher trunk velocity measures when compared to measures in presence of vision [56], highlighting greater balance control challenges with higher trunk angular velocity measures in the absence of vision in the high spasticity group only. It is worth noting that trunk velocity findings between spasticity groups and conditions were similar to COP velocity findings in that these measures were higher in ML especially during the eyes closed conditions. These findings may further highlight the high correlation of COP and COM movements during quiet standing that is already established in the literature [28]. In addition to trunk roll velocity, a significant vision by spasticity interaction effect on trunk roll velocity amplitude near the 3.7Hz frequency was also observed. It was noted that individuals with high spasticity have greater trunk roll frequency measures in the absence of vision than when vision was present, whereas individuals with low spasticity had similar frequency measures regardless of vision condition. As introduced earlier, this may suggest that individuals with high spasticity may be more dependent to use their vision in order to control their balance whereas this effect is not shown in individuals with low spasticity. In this study, spasticity was not specifically measured near the trunk and frequency measures were higher in the eyes closed versus eyes open trials in the high spasticity group. This can suggest that individuals with high spasticity might have been using a stiffening strategy to control their balance in the eyes closed trials, which potentially translated to higher frequency measures near the trunk. However, higher trunk roll frequency measures in the absence of vision were also noted between the high spasticity group versus the low spasticity group whereas similar frequency measures were observed between the high and low spasticity groups in presence of vision. This suggests that in a more challenging task of eyes closed condition, greater balance control challenges were evident in individuals who had higher level of spasticity. Hence, these findings generally present that balance control during quiet standing was

more challenging among individuals with high spasticity especially during the more difficult task when measures were compared to those individuals with low spasticity and measures taken during the easier task. Perhaps these findings reiterate Horak's explanation of sensory strategies, which includes contributions from intact somatosensory and visual systems in balance control of the body. In conditions that vision is absent, it is conceivable that the body would rely more on the somatosensory control system. However, since the high spasticity group have higher muscle tone and stretch reflexes and possibly less somatosensory control contribution to balance control as compared to the low spasticity group, the effect of visual system in balance control of the body seem to be crucial in individuals with high spasticity as they have demonstrated greater challenges in absence of vision. Higher trunk velocity amplitude at frequencies in the 2.4Hz and 4.8Hz ranges have also been shown in individuals with neurological conditions such as Parkinson's disease who are known to have challenges in their balance control and high risk of falling [16]. The higher frequency measures observed in that study have been explained by Adkin et al., who suggest that higher trunk velocity amplitude frequency measures in those individuals with greater balance control challenges can be from increased stiffness due to rigidity or increase muscle tone. Greater stiffness in the population of this study is quite possible as increased MPF measures in the same plane were also observed in COP movements of individuals with high spasticity. As suggested earlier, future studies may further investigate the role of increase muscle tone or a behavioural stiffening strategy observed from high trunk sway and COP frequencies in the high spasticity group. The trunk sway findings within this study further suggests that the kinematic role of trunk control near COM in standing balance of individuals with stroke is important and cannot be ignored as there are currently limited number of studies in the literature that focus on kinematics of trunk control in both planes during standing balance control in this population.

Frontal plane specific challenges

In the previous sections, it was noted that COP and trunk sway measures were different between the groups (low and high spasticity) in the current study, but only in the mediolateral direction, or the frontal plane. In fact, the frontal plane specific balance control challenges in stroke individuals is not new and has been reported in many studies [14, 18]. For example, in a review of standing balance among individuals with stroke, multiple studies have reported greater postural sway and balance control challenges in the mediolateral direction with greater weight bearing asymmetry towards the non-affected limb during quiet standing [18]. In a study by de Haart et al., they looked at recovery of standing balance among individuals with stroke and used COP distance and COP velocity as measure of AP and ML instability using platforms [57]. Specifically, they noted greater instability challenges in the frontal plane, and interestingly reported greater stability improvements in frontal plane COP velocity following rehabilitation. Furthermore, the authors argued that asymmetry quotient from both legs in stroke participants was specifically high in the frontal plane in a similar fashion to frontal plane asymmetry quotient of amputee patients with greater weight bearing towards the unaffected limb. In another study, Marigold et al., tested the affect of vision (eyes open versus eyes closed) on quiet standing among individuals with stroke compared with healthy elderly individuals [14]. In their results they reported that only COP velocity in ML was significantly different between groups with stroke individuals being greatly dependant on vision. Furthermore, they found a significant correlation between weight bearing asymmetry index and ML COP velocity in stroke individuals where ML COP velocity was negatively correlated with weight bearing asymmetry index representing those who put more weight on the non affected leg have higher ML COP velocities. In another study, Mansfield et al., studied between-limb synchronization among individuals with stroke versus healthy controls and explained that reduced between-limb synchronization in persons with

stroke was related to greater mediolateral sway [58]. In their study, the authors suggested that reduced synchronization observed in individuals with stroke may be due to the action of more proximal muscles responsible for mediolateral control of balance control during quiet standing. This concept was in fact studied in detail by Winter et al., where authors explained that the AP control of standing balance is under the control of the ankle muscles. Moreover, their findings suggested that the loading/unloading of weight distribution under the feet are profound in the mediolateral direction but not in the anterior posterior control of balance and are under a motor response better explained by the action of hip abductor/adductors and not the ankle plantarflexor/dorsiflexors [59]. Since greater weight bearing asymmetry is commonly noted in individuals with stroke and the loading/unloading mechanism has been shown to contribute to movements in the ML plane, it is not surprising that greater balance control challenges were observed in this direction in the high spasticity group. Interestingly, another study on balance control of individuals with multiple sclerosis and spasticity had grouped individuals with high and low ankle spasticity based on cluster analysis of H-reflex measures for spasticity group determination [60]. In their findings, authors reported that high spasticity group had significantly greater sway measures compared to low spasticity and control groups. Furthermore, they reported higher sway length and MPF measures in the high spasticity versus low spasticity groups noted in the ML direction. The authors concluded two main findings in that high spasticity group had higher sway compared to low spasticity group and controls, and that these challenges were recognized in the ML direction. In their discussion, they too suggested that ML differences may be from action of the hips and knees rather than the ankles and suggested that their participants may also have had spasticity in other muscle groups although they did not have this data available. In the current study design, hip adductor and knee flexor/extensor spasticity scores were also included in the assessment of lower limb spasticity, and it was observed that all of the

participants had some level of spasticity in lower limb muscle groups other than the ankle muscle groups. Interestingly, 5 out of 15 participants in the high spasticity group had severe spasticity (MAS of 2 or greater) in the knee/hip muscles whereas none of the participants in the low spasticity group had severe spasticity in the knee/hip muscle groups. The observation that the low spasticity group had no severe spasticity (MAS<2) in any of the lower limb muscle groups may also explain why they had lower postural sway measures during standing balance control compared to the high spasticity group. Finally, since the COP is the controlling variable and the COM of the body is the controlled variable during quiet standing with high correlations between them in both AP and ML planes [28, 29], a greater mediolateral sway near the COM may be related to greater mediolateral COP movements among individuals in the high spasticity group.

Sagittal plane findings

The findings in this study suggested that there were no differences in COP and trunk sway in the sagittal plane between the high and low ankle spasticity groups. This finding was surprising since greater balance control challenges among individuals with high spasticity in the sagittal plane was anticipated in COP and trunk sway measures mainly because the role of ankle plantarfexor/dorsiflexors are to control the balance of the body in the sagittal plane according to the inverted pendulum model [29]. Additionally, individuals with spasticity post-stroke had shown greater challenges when compared to individuals without spasticity in anterior posterior control of balance between lower limbs [27]. A possible explanation may be that net AP measures were not different based on severity of ankle spasticity because all of the participants in the current study were hemiparetic, with spasticity on one side, but also had plantar/dorsiflexors without spasticity on the unaffected side. Perhaps the unaffected side contributed enough control in the AP direction to

compensate for the different levels of spasticity in both groups. A better understanding of AP balance control in this case would have been possible had there been a force plate under each foot, to differentiate between the non affected and affected leg with spasticity. Additionally, the role of other muscles with spasticity may be contributing in different balance control strategies as noted earlier, and cannot be ignored in overall understanding of balance control in this population. Therefore, the study findings also suggest considering hip and trunk control in the overall balance control of population of individuals with spasticity post-stroke in future investigations.

Discussion on other findings

Balance self-efficacy was not found to be different between groups, and results suggested that fear of fall was not different based on severity of ankle spasticity among individuals with stroke. The influence of age and foot motor impairment levels was not found to be significant in balance control measures in both groups. Hence, no further analysis was required to control for age and foot impairment levels. The influence of sex differences on balance control measures is believed to be minimal as there was equal number of female participants in both groups. Lastly, the relationship between UL and LL muscle groups with spasticity and balance control measures suggested no correlations to weak correlations. Furthermore, the weak correlations observed did not suggest a uniform pattern of relationships between UL and LL spasticity and balance control measures since a combination of positive and negative relationships were observed between these measures.

3.5 Limitations

In this study it was demonstrated that individuals with high spasticity have greater mediolateral balance control challenges than individuals with low ankle spasticity in COP movements

using a single force platform. However, since stroke affects one side of the body, current results with a single force plate cannot further explain the kinetics under each foot as oppose to having a single force plate under each foot. Had this study included use of two force plates, it could have been possible to further investigate the spatiotemporal COP characteristics of the limb with spasticity versus the limb without spasticity in addition to net COP characteristics. Another limitation of the study is the lack of a control group to compare the results with individuals without spasticity. This is important as Singer et al., had shown that post-stroke individuals with spasticity use different temporal balance control strategies than post-stroke individuals with spasticity. Having a control group composed of individuals with stroke without spasticity could have helped better understand balance control differences between post-stroke individuals without spasticity, with low spasticity, and with high spasticity. However, since the hospital spasticity clinic is for treatment of individuals with spasticity it was not possible to recruit post-stroke individuals without spasticity.

CHAPTER 4

GENERAL DISCUSSION

4.1 Recalling Study Objective

Previous knowledge of balance control challenges in persons with stroke had suggested enough evidence in the literature that post-stroke individuals have high risk of falls and greater balance control challenges compared to healthy adults. It was also supported in the literature that individuals with stroke have high fear of fall measures. However, although some individuals with stroke do have spasticity, the sample of individuals with stroke in the previous studies may or may not have had spasticity and those studies had not controlled for severity of spasticity in their design. Hence, it was known that post-stroke individuals have balance control challenges and high fear of fall measures, but the objective of this study was to understand the impact of spasticity on balance control measures and strategies in individuals post-stroke. Therefore, to understand the impact of spasticity on balance control measures in individuals post-stroke, study hypotheses were tested in this project.

4.2 Revisiting Study Hypotheses

Hypothesis #1: Post-stroke individuals with high spasticity are expected to have greater balance control challenges as compared to post-stroke individuals with low spasticity.

ACCEPTED

The study hypothesis that post-stroke individuals with high spasticity were expected to have greater balance control challenges as compared to post-stroke individuals with low spasticity was accepted. This hypothesis was accepted since the high spasticity group demonstrated significantly

greater balance control challenges as compared to the low spasticity group in the mediolateral direction.

The findings that high spasticity group had greater challenges in balance control in the mediolateral direction only is interesting of itself as it may suggest specific strategies to control standing balance. Since the mediolateral control of the body has been suggested to be under the control of the hips, it may be that those with higher lower limb spasticity have greater challenges with the control of balance around the hips. Another explanation with observed differences in this study may be that control of the trunk may be more challenging in individuals with higher spasticity in frontal plane. Since the activity of COM and COP are closely related in both planes, greater ML COP movement may be related to greater ML COM sway in this population. Also a stiffening strategy may be suggested from the sway frequency measures in COP and trunk sway in this population as a stiffening strategy in order to control balance have been suggested from higher sway frequency measures in other population of individuals with high risk of falls. Furthermore, it can be suggested that the higher frequency measures observed in the high spasticity group may be representing a measure of higher muscle tone and stiffness that is expected in individuals with high level of spasticity. Lastly, it should be noted that some of the observed differences in balance control between spasticity groups were only evident in performing the more challenging task in the eyes closed trials as balance control typically gets more difficult in the absence of visual feedback during quiet standing

Hypothesis #2: Post-stroke individuals with high spasticity are expected to have greater fear of fall measures as compared to post-stroke individuals with low spasticity.

REJECTED

The study hypothesis that post-stroke individuals with high spasticity were expected to

have greater fear of fall measures as compared to post-stroke individuals with low spasticity was rejected. This hypothesis was rejected since there were no significant differences in balance confidence scores between the high and low spasticity groups. This finding suggests that balance control challenges observed between the high and low spasticity groups cannot be explained from fear of fall measures between groups.

4.3 Conclusion

This project was the first pilot study to investigate standing balance control challenges in population of individuals with stroke specifically based on severity of spasticity. In this study, severity of ankle spasticity on balance control of individuals post-stroke demonstrated that individuals with high spasticity levels have greater balance control challenges when compared to individuals with lower spasticity levels during quiet standing especially during the more challenging task in absence of vision. Moreover, the greater challenges observed in individuals with high spasticity are specifically noted in the medial lateral direction suggesting a possible strategy to control their balance around the hips and the trunk. It can be suggested that a stiffening strategy to control balance may have been used in individuals with high spasticity, as higher power and amplitude frequency measures were evident in individuals with high spasticity. Future studies may also consider if the observed findings in frequency measures could be related to an increase in muscle tone within this population. Lastly, the findings of this study can further be implemented in clinical research and rehabilitation of individuals with spasticity post-stroke for a better understanding and treatment of balance control challenges in this population.

4.4 Future Areas of Study

It is highly recommended for future studies to further look at COP characteristics under each foot separately to better understand the role of the affected limb with spasticity and the unaffected limb without spasticity in balance control of post-stroke individuals with different levels of spasticity. Addition of a control group of post-stroke individuals without spasticity may also suggest different balance control strategies in individuals who have no spasticity versus those with different levels of spasticity. Future studies can also consider studying balance recovery of individuals with spasticity in response to dynamic balance control by looking at stretch reflex response of individuals with spasticity in response to balance perturbations. Although current results demonstrated that individuals with spasticity have challenges in static balance control, strategies in response to dynamic balance control are studied differently and are of great interest in balance control literature. Since some individuals with spasticity need to receive treatment to reduce the symptoms of spasticity, future studies may be considering a treatment effect on spasticity and balance control. For example, it may be interesting to look at the before and after treatment effects of botulinum toxin as one line of treatment in spasticity and its effect on balance control of individuals who get this type of treatment. The findings of this study may also be used in rehabilitation settings with more focus on mediolateral balance control training of post-stroke individuals with spasticity for preventative measures in loss of balance and risk of falling in this population.

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LIST OF APPENDICES

Appendix A- Invitation Letter

Dear Sir/Madam,

My name is Reza Rahimzadeh, and I am a research assistant at the spasticity clinic in West Park Healthcare Centre. We are doing a research study in the spasticity clinic to better understand the kind of balance problems that persons with stroke might face. I have got your contact information from our spasticity clinic coordinator and would like to further explain our study to you.

We are looking to study balance performance in persons with stroke by measuring how their body's centre moves, which tells us about potential balance problems. To measure body centre, we will use a belt like device that is worn on the waist (figure 1) and weight scale like platform that requires simply standing (figure 2). If you are interested to participate, you are required to stand on the platform for a total of 10 minutes. Following these measurements, we will fill out a short questionnaire to assess your balance confidence and fear of falling during day to day activities. Lastly, one of our clinic therapists will assess how well you are able to move your arm and leg. Total testing time can be up to 20 minutes. If you are interested to participate, we would like to ask you to arrive 20 minutes prior to your scheduled appointment in 2 weeks. I will call you next week to ask you about your interest in participating in this study and to answer any questions you might have.

If you have further questions and are interested to participate, please feel free to contact us via email: (removed in thesis), or call us at west park: (removed in thesis).

Figure 1: Waist belt **Figure 2**: Standing on a platform

(Figures 1 and 2 were solely used for illustration of testing procedure for research study participants and have been removed by the author for copyright purposes)

Sincerely,
Reza Rahimzadeh, Research Assistant- West Park Healthcare Centre
Study Principal Investigator: Dr. Chetan P. Phadke
Spasticity Research Program,
West Park Healthcare Centre
82 Buttonwood Ave., Toronto, M6M 2J5
Phone: (removed in thesis), and Fax – (removed in thesis)

Appendix B- Phone Script Invitation

Hello Mr/Ms/Mrs. _____
My name is Reza and I am a research assistant in the spasticity clinic at West Park healthcare centre. I got your number from our clinic coordinator. We are doing a study in the spasticity clinic to understand the kind of balance problems that stroke patients with spasticity might face.

We are doing a study to understand if there is any relationship between spasticity, and balance problems in stroke patients. If you participate in this study you will be asked to simply step up on a plate (2 inch height and 2.6 square feet) that measures the weight you put under your foot. You will be asked to stand on this plate comfortably for up to 10 minutes while maintaining your balance with your eyes open as well as with your eyes closed. While you are standing on the plate, we also require you to wear a belt like device on your waist that allows us to measure small waist movements. The test is simple and involves keeping your balance while standing as you would regularly do on a daily basis. Following this, we will fill out a 16 item questionnaire that asks about your balance confidence and fear of falling. At the end of our session, one of our clinic therapists will assess how well you are able to move your arm and leg.

The total testing time can be up to a maximum of 20 minutes, and if you are willing to participate then we want to request you to come 20 minutes before your scheduled appointment next week. If you have further questions, then I can answer them for you.

Would you be interested in participating in this study?

WEST PARK HEALTHCARE CENTRE
REHABILITATION, COMPLEX CONTINUING AND LONG-TERM CARE

Appendix C- PATIENT INFORMATION AND INFORMED CONSENT FORM

Title: Impact of Spasticity on Balance Impairment and Postural Sway During Quiet Standing in Persons with Stroke

INVESTIGATORS
Chris Boulias MD, Farooq Ismail MD, Reza Rahimzadeh Khiabani MSc student, William Gage PhD, Chetan P. Phadke PhD

CONTACT INFORMATION
West Park Healthcare Centre
82 Buttonwood Avenue, Toronto, Ontario M6M 2J5
Tel: (removed in thesis)

SPONSOR
None

BACKGROUND
Spasticity is excessive tightness of muscles, making smooth movements difficult.
To maintain proper balance during quiet standing, our body needs to be centred. By measuring how much our bodies sway, we can tell how imbalanced someone is. Balance problems and spasticity levels in stroke patients have been previously shown to be correlated. However, the underlying mechanisms explaining this relationship are still missing.

PURPOSE OF THE STUDY
In this study, we want to look at the postural sway in persons with stroke with spasticity during quiet standing to compare the sway measurements of patients with - 1) severe ankle spasticity and 2) mild degree of ankle spasticity to better understand if spasticity complications are putting persons post-stroke at a greater risk of fall.

PROCEDURES
For our study we are looking to recruit 30 stroke participants with spasticity. You are being invited to participate in this research study because you are being treated for spasticity by your physician in the Spasticity Clinic at West Park Healthcare Centre. If you choose to participate in this research study, you will be asked to step on a plate and stand on it with both feet while maintaining your balance. This is a common widely used test of balance used in various clinics and you may have done it in the past. While standing, you are required to wear a belt like device on your waist for measurement of your waist movements. We will observe you closely to measure how well you are able to maintain your balance during this task with and without your eyes closed. Following this, we will go through a 16 item questionnaire to assess your balance confidence and fear of falling as you wait to see your doctor. Our clinic therapists will then assess how well you are able to move your arm and leg. The

total testing time is up to 20 minutes. Clinicians will assess your arm and leg spasticity. We will gather basic information such as your age, neurological condition from your medical chart.

RISKS & BENEFITS
There is a minimal risk of falling during the balance testing (especially with eyes closed) and the researcher will be close at hand to assist you if you feel like you are losing balance. There are no direct benefits to you by participating. At the end of the study we would be able to better understand whether spasticity may affect balance impairments. At the end of the study the results will be available to you upon request.

CONFIDENTIALITY
It is important to note that any personal information collected in this study and all information you supply in this research will be kept confidential, and your name will not appear in any report or publication of the research unless you have specifically indicated your consent. To help protect storage of electronic data, name and contact information with a unique alphanumeric code assigned to each patient will be placed on paper and stored in a locked cabinet in West Park Healthcare Centre. This paper will be destroyed 2 years after the end of the study. Data stored on the computer will not have patient names, but just the code; each patient's results will be named using their unique alphanumeric code. Thus, the data on the computers in the West Park Healthcare Centre facilities will not have any personal health identifiers and confidentiality will be provided to the fullest extent by law. Only the study investigators and the JREB will have access to the data. JREB helps protect the participants such as you and ensures that research studies are carried out with utmost respect, consideration, and care for the participants. The findings will be analysed and reported in a medical journal, and will not be linked to your name.

COMPENSATION/REIMBURSEMENT:
No compensation or reimbursement for time or any other expenses incurred to participate in this study will be provided.

CONSENT
I, the participant, have read and received a copy of the Informed Consent form. The research study including harms and benefits has been explained to me and my questions have been answered to my satisfaction. I understand that I may not experience personal benefit through my participation in the study. I have been assured that all personal information collected in the study will be kept confidential. I understand that my participation is completely voluntary and that I am free to decide not to participate in this study or to withdraw from the study at any time. I understand that my treatment and relationship with West Park Health Care Centre, York University, and researchers associated with this projected will not be affected by whether or not I choose to participate in the study, refuse to answer any questions, or withdraw from the study. I understand that if I choose to withdraw from the study, all data collected will be destroyed immediately wherever possible. I agree to participate in this research study. I know that I may ask any questions I have about the study at any time, either now or in the future. At the end of the study the study results will be available to me upon request.

QUESTIONS

I know that if I have any concerns in the future about my participation in the study, I can contact: Dr. Chetan Phadke, (Principal Investigator) 82 Buttonwood Ave., Toronto, ON, M6M 2J5, Phone # (removed in thesis). If I have any concerns about my ethical rights as a research participant I can contact Dr. Ron Heslegrave (Joint Research Ethics Board Chair) Phone # (removed in thesis).

I understand that this research has also been reviewed and approved by the Human Participants Review Sub-Committee, York University's Ethics Review Board and conforms to the standards of the Canadian Tri-Council Research Ethics guidelines. I know that if I have any concerns and questions about this process, or about my rights as a participant in the study, I can contact the Sr. Manager & Policy Advisor for the Office of Research Ethics, 5th Floor, Kaneff Tower, York University (telephone (removed in thesis), or e-mail (removed in thesis)).

_____	_____	_____
Name of participant	Signature	Date
_____	_____	_____
Person obtaining consent	Signature	Date

Appendix D- Activities-specific Balance Confidence Scale

The Activities-specific Balance Confidence (ABC) Scale

Administration:
The ABC can be self-administered or administered via personal or telephone interview. Larger typeset should be used for self-administration, while an enlarged version of the rating scale on an index card will facilitate in-person interviews. Regardless of method of administration, each respondent should be queried concerning their understanding of instructions, and probed regarding difficulty answering specific items.

Instructions to Participants:
For each of the following, please indicate your level of confidence in doing the activity without losing your balance or becoming unsteady from choosing one of the percentage points on the scale form 0% to 100%. If you do not currently do the activity in question, try and imagine how confident you would be if you had to do the activity. If you normally use a walking aid to do the activity or hold onto someone, rate your confidence as it you were using these supports. If you have any questions about answering any of these items, please ask the administrator.

Instructions for Scoring:
The ABC is an 11-point scale and ratings should consist of whole numbers (0-100) for each item. Total the ratings (possible range =0 - 1600) and divide by 16 to get each subject's ABC score. If a subject qualifies his/her response to items #2, #9, #11, #14 or # 15 (different ratings for "up" vs. "down" or "onto" vs. "off'), solicit separate ratings and use the lowest confidence of the two (as this will limit the entire activity, for instance the likelihood of using the stairs.)

- 80% = high level of physical functioning
- 50-80% = moderate level of physical functioning
- < 50%= low level of physical functioning
  Myers AM (1998)
- < 67% = older adults at risk for falling; predictive of future fall
  Lajoie Y (2004)

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The Activities-specific Balance Confidence (ABC) Scale

For each of the following activities, please indicate your level of self-confidence by choosing a corresponding number from the following rating scale:

0% 10 20 30 40 50 60 70 80 90 100%

no confidence **completely confident**

"How confident are you that you will not lose your balance or become unsteady when you...

1. …walk around the house?___%

2. …walk up or down stairs?_%

3. …bend over and pick up a slipper from the front of a closet floor ___%

4. …reach for a small can off a shelf at eye level?____%

5. …stand on your tiptoes and reach for something above your head?____%

6. …stand on a chair and reach for something? _____%

7. …sweep the floor? ____%

8. …walk outside the house to a car parked in the driveway? _____%

9. …get into or out of a car? ____%

10. …walk across a parking lot to the mall? ____%

11. …walk up or down a ramp?_____%

12. …walk in a crowded mall where people rapidly walk past you? _____%

13. …are bumped into by people as you walk through the mall? ____%

14. …step onto or off an escalator while you are holding onto a railing?____ %

15. …step onto or off an escalator while holding onto parcels such that you cannot hold onto the railing?____%

16. …walk outside on icy sidewalks? ____%

Appendix E- Chedoke-McMaster Stroke Assessment (arm, hand, leg, foot impairment inventory)

Chedoke-McMaster Stroke Assessment
SCORE FORM Page 2 of 4
IMPAIRMENT INVENTORY: STAGE OF RECOVERY OF ARM AND HAND

ARM and HAND: Start at Stage 3. Starting position: sitting with forearms in lap or supported on a pillow in a neutral position, wrist at 0° and fingers slightly flexed. Changes from this position are indicated by underlining. Place an X in the box of each task accomplished. Score the highest Stage in which the client achieves at least two Xs.

	ARM	HAND
1	☐ not yet Stage 2	☐ not yet Stage 2
2	☐ resistance to passive shoulder abduction or elbow extension	☐ positive Hoffman
	☐ facilitated elbow extension	☐ resistance to passive wrist or finger extension
	☐ facilitated elbow flexion	☐ facilitated finger flexion
3	☐ touch opposite knee	☐ wrist extension > ½ range
	☐ touch chin	☐ finger or wrist flexion > ½ range
	☐ shoulder shrugging > ½ range	☐ <u>supination, thumb in extension:</u> thumb to index finger
4	☐ extension synergy, then flexion synergy	☐ finger extension then flexion
	☐ shoulder flexion to 90°	☐ thumb extension > ½ range, then lateral prehension
	☐ <u>elbow at side, 90° flexion:</u> supination, then pronation	☐ finger flexion with lateral prehension
5	☐ flexion synergy, then extension synergy	☐ finger flexion, then extension
	☐ shoulder abduction to 90° with pronation	☐ <u>pronation:</u> finger abduction
	☐ <u>shoulder flexion to 90°:</u> pronation then supination	☐ <u>hand unsupported:</u> opposition of thumb to little finger
6	☐ hand from knee to forehead 5X in 5 sec	☐ <u>pronation:</u> tap index finger 10X in 5 sec
	☐ <u>shoulder flexion to 90°</u>: trace a vertical figure 8	☐ <u>pistol grip</u>: pull trigger, then return
	☐ <u>arm resting at side of body</u>: raise arm overhead with full supination	☐ <u>pronation</u>: wrist and finger extension with finger abduction

7 ☐ clap hands overhead, then behind back 3X in 5 sec

☐ <u>shoulder flexion to 90°:</u> scissor in front 3X in 5 sec

☐ <u>elbow at side, 90° flexion:</u> resisted shoulder external rotation

☐ **STAGE OF ARM**

7 ☐ thumb to finger tips, then reverse 3X in 12 sec

☐ bounce a ball 4 times in succession, then catch

☐ pour 250 ml. from 1 litre pitcher, then reverse

☐ **STAGE OF HAND**

Chedoke-McMaster Stroke Assessment
SCORE FORM Page 3 of 4
IMPAIRMENT INVENTORY: STAGE OF RECOVERY OF LEG AND FOOT

LEG: Start at Stage 4 with the client in lying on back with knees bent and feet flat. FOOT: Start at Stage 3 with the client in supine. Test position is beside the item or underlined. If not indicated, the position has not changed. Place an X in the box of each task accomplished. Score the highest stage in which the client achieves at least two Xs. For "standing" test items, light support may be provided but weight bearing through the hand is not allowed. Shoes and socks off.

	LEG		FOOT	
1		☐ not yet Stage 2		☐ not yet Stage 2
2	Crook Lying	☐ resistance to passive hip or knee flexion	Crook Lying	☐ resistance to passive dorsiflexion
		☐ facilitated hip flexion		☐ facilitated dorsiflexion or toe extension
		☐ facilitated extension		☐ facilitated plantarflexion
3		☐ <u>abduction:</u> adduction to neutral	Supine	☐ plantarflexion > ½ range
		☐ hip flexion to 90°	Sit	☐ some dorsiflexion
		☐ full extension		☐ extension of toes
4		☐ hip flexion to 90° then extension synergy		☐ some eversion
		☐ bridging hips with equal weightbearing		☐ full inversion
	Sit	☐ knee flexion beyond 100°		☐ <u>legs crossed:</u> dorsiflexion, then plantarflexion
5	Crook Lying	☐ extension synergy, then flexion synergy		☐ <u>legs crossed:</u> toe extension with ankle plantarflexion
	Sit	☐ raise thigh off bed		☐ <u>sitting with knee extended:</u> ankle plantarflexion, then dorsiflexion
	Stand	☐ hip extension with knee flexion	Stand	☐ <u>heel on floor:</u> eversion
6	Sit	☐ lift foot off floor 5X in 5 sec		☐ <u>heel on floor:</u> tap foot 5X in 5 sec
		☐ full range internal rotation		☐ <u>foot off floor:</u> foot circumduction
		☐ trace a pattern: forward, side, back, return		☐ <u>knee straight, heel off floor:</u> eversion

7 Stand

☐ unsupported: rapid high stepping 10X in 5 sec

☐ unsupported: trace a pattern quickly: forward, side, back; reverse pattern

☐ on weak leg with support: hop on weak leg

☐ **STAGE OF LEG**

7

☐ heel touching forward, then toe touching behind, repeat 5X in 10 sec

☐ foot off floor: circumduction quickly, reverse

☐ up on toes then back on heels 5X

☐ **STAGE OF FOOT**