POSTURAL CONTROL AND FALLS IN INDIVIDUALS WITH CHRONIC STROKE: NEURAL MECHANISMS AND EFFECTS OF EXERCISE

by

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ABSTRACT

Although falls and fall-related injuries are an enormous burden for individuals with stroke, we do not know the neural mechanisms underlying these events. While impairments in postural control presumably contribute to falls, the type of exercises most effective for improving postural control in individuals with chronic stroke are unclear. The purpose of this thesis was (1) to understand how individuals with chronic stroke modulate postural control and determine the underlying neural mechanisms contributing to falls and (2) to determine the effects of two different exercise interventions on postural control and physical function. In each of the three experiments conducted, postural reflexes were evoked by unexpected translations of a platform upon which participants were standing. Experiment I examined the effects of different weight-bearing load on the modulation of lower limb postural reflexes. We revealed that individuals with stroke could modulate ankle extensor, but not ankle dorsiflexor reflex magnitude. In Experiment II we discovered that reduced tibialis anterior reflex magnitude and delayed non-paretic rectus femoris onset latency contribute to falls in individuals with stroke. In addition, reduced maximum volitional muscle strength, particularly the paretic lower limb, contributed to falls induced by platform translations. Based on the results from the first two experiments, we hypothesized that deficits in supraspinal centres are responsible for the impaired postural control observed. Experiment III was a 10-week randomized clinical trial in individuals with chronic stroke. The results demonstrated that regardless of intervention (Agility or Stretching/weight-shifting program), exercise resulted in faster paretic lower limb postural reflexes, improved functional balance and mobility, faster step reaction time, and improved balance confidence and quality of life. However, there was a greater change in paretic rectus femoris postural reflex onset latency and step reaction time for the Agility exercise group. Although there was no difference in the number of fallers between groups when the entire sample was included, a sub-analysis of those with a history falls demonstrated a reduction in the number of fallers in the Agility group. These results suggest that an Agility-based exercise intervention may be more beneficial for individuals with stroke. Further, the results suggest exercise-induced neural plasticity.
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Contribution of the Author

This thesis contains three experiments that have been conducted by the candidate Daniel S. Marigold, under the supervision of Janice J. Eng (Associate Professor, School of Rehabilitation Sciences). The collection, analysis, and documentation of all experiments were primarily the work of the candidate.

The above statement was written by Daniel S. Marigold and agreed upon by the undersigned.

Janice J. Eng, Ph.D.

References


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CHAPTER 1 - Introduction and Purpose

1.1 Overview of Thesis

The thesis begins with the overall purpose followed by introductory information regarding stroke and postural control. Subsequently, three sections are presented which are devoted to background research under the following areas, (1) falls in individuals with stroke, (2) exercise in individuals with chronic stroke, and (3) postural control following stroke. Further information for each of these sections can be obtained in the literature tables in Appendices I - III. Background research is followed by a description of the research questions and their hypotheses. Methodology is addressed in each chapter with regard to the particular experiment. Chapters two through four are experimental chapters. Chapter five integrates the findings from each experiment and provides some suggestions and implications for rehabilitation research.

1.2 Purpose

The objective of this thesis was (1) to understand how individuals with chronic stroke modulate postural control and determine the underlying neural mechanisms contributing to falls and (2) to determine the effects of two different exercise interventions on postural control and physical function.

1.3 Introduction

1.3.1 Cerebrovascular Accident

Cerebrovascular accident (or stroke) results from restricted blood supply to the brain leading to impaired neurologic function (O'Sullivan 1988). There are between 40,000 to 50,000 strokes each year in Canada and there are currently 300,000 individuals living with stroke (Heart and Stroke Foundation of Canada 2003). Stroke-related impairments include muscle weakness, spasticity, pain, sensorimotor dysfunction, visual deficits, and balance impairments (O'Sullivan
Individuals with stroke are at a high risk for falls, presumably due to stroke-related impairments. Recovery occurs fastest during the first 90 days but continues up to six months post-stroke (Duncan and Lai 1997; Jorgensen et al. 1999; Wade et al. 1985). Ninety percent of stroke survivors have some functional disability (Gresham et al. 1975).

1.3.2 Postural Control

Postural control has been defined as a complex neural process involved in the organization of the stability and orientation of the body in space (Allum et al., 1998). Postural control occurs continuously with movement; whether during quiet standing (quiet standing postural control), during preparation to move positions (anticipatory postural control), or in response to an external disturbance or perturbation applied to the body (reactive postural control) and is the primary means of maintaining stability. Postural stability (synonymous with balance) is the ability to maintain the body’s centre of mass within the base of support (i.e. the feet) or boundaries in which the base of support doesn’t require repositioning (Shumway-Cook and Woollacott 2001). During human stance postural stability is imperative, as large centre of mass excursions outside the base of support may result in a fall.

The central nervous system (CNS) utilizes somatosensory, vestibular, and visual input for postural control over a wide range of movements and in response to various destabilizing events (Allum et al., 1998; Dietz et al., 1992). This is accomplished by generating torques at the joints of the supporting legs and trunk through muscle activity and possible modifications of the base of support by stepping and/or reaching/grasping movements of the upper extremities (Maki and McIlroy 1996, 1997). The responses generated by the CNS in the form of muscle activity and/or limb movements are referred to as postural responses (or postural reflexes). The functional coupling of muscles that comprise these movements are known as muscle synergies and can change depending on the task and/or environmental constraints (Shumway-Cook and Woollacott 2001).

The effects of neurological conditions, such as stroke, on postural control are largely unknown. Reactive postural control is the first line of defense against unexpected balance-threatening disturbances that have the potential to lead to falls. Falls and fall-related injuries are major concerns in individuals who have suffered a stroke. Whether postural reflexes utilized to maintain postural stability in response to unexpected destabilizing events could be modified or improved through exercise remains uncertain. Although two studies (Eng et al. 2003;
Tangeman et al. 1990) have shown that exercise improves functional balance in individuals with chronic stroke, the advantages of different types of exercise programs are unknown.

1.4 Literature Review

1.4.1 Falls in individuals with stroke

Stroke is the number one cause of neurological disability in Canada (Mayo et al. 1999). Falls and fall-related injuries are a frequent occurrence in individuals with stroke. The incidence of falling among individuals with stroke in a hospital/rehabilitation setting ranges from 39% to 44% (Nyberg and Gustafson 1995; Ugur et al. 2000). Individuals with stroke who fall during this time are twice as likely to fall after discharge (Forster and Young 1995). The incidence of falling for community-dwelling stroke survivors has been reported to be between 23% and 73% (Forster and Young 1995; Hyndman et al. 2002; Jorgensen et al. 2002; Yates et al. 2002).

Once an individual falls, a fear of falling may develop (of course, fear of falling may also lead to falls) contributing to activity restriction (Friedman et al. 2002; Murphy et al. 2002). Unfortunately, if an individual is at high risk for falling more than one fall is a likely scenario. Forster and Young (1995) have reported an average of 3.4 falls per person with stroke during a six-month period following hospital discharge. In fact, the incidence of multiple falls among individuals with stroke is approximately 50% (Hyndman et al. 2002; Yates et al. 2002).

Nyberg and Gustafson (1995) have reported the leading cause of falls in individuals with stroke while in a rehabilitation setting is transferring or changing positions. In community-dwelling stroke survivors, 77% of falls occur during walking (Jorgensen et al. 2002).

The majority of falls in individuals with stroke appear to be directed sideways and towards their affected side or on their hands and knees (Hyndman et al. 2002). Not surprisingly, Kanis et al. (2001) found a greater than 7-fold increase in fracture risk within the first year after hospitalization with stroke. In older adults, approximately 2 – 6% suffer fractures as a result of falling (Lord et al. 2001).

Impairments such as muscle weakness, pain, spasticity, visual disturbance, and sensorimotor dysfunction may contribute to poor postural control and a high rate of falls in individuals with stroke. Yates et al. (2002) found that individuals with stroke who demonstrated motor and
sensory impairments were three times as likely to fall compared to those individuals who exhibited no impairments.

Understanding the nature of the postural control problems that result in falls and determining strategies for falls-reduction are vital in reducing health care costs and ensuring quality of life and mobility for individuals with stroke is improved.

1.4.2 Exercise in individuals with chronic stroke

There is abundant research in healthy older adults demonstrating the beneficial effects of exercise on postural control through a reduction in falls (Campbell et al. 1997; Campbell et al. 1999; Hu and Woollacott 1994a; Robertson et al. 2001a; Robertson et al. 2001b; Steinberg et al. 2000), increased muscle strength (Lord et al. 1993; Schlicht et al. 2001), and improved functional balance (Hopkins et al. 1990; Lord et al. 1993, 1995, 1996; Rogers et al. 2001). Surprisingly, there is a lack of research and implementation of community-based group exercise programs for individuals with chronic stroke even though the incidence of falls in this population is very high.

Several different approaches to exercise training have been implemented in individuals with chronic stroke including treadmill or over-ground gait training (Ada et al. 2003; Hesse et al. 1995; Macko et al. 1997, 2001; Miller 2001; Miller et al. 2002; Mudge et al. 2003; Silver et al. 2000; Smith et al. 1998b, 1999, 2000; Sullivan et al. 2002; Trueblood 2001), muscle strength training (Badics et al. 2002; Engardt et al. 1995; Kim et al. 2001; Sharp and Brouwer 1997; Weiss et al. 2000), functional exercise training (Bassile et al. 2003; Dean and Shepherd 1997; Monger et al. 2002; Potempa et al. 1995; Rodriquez et al. 1996; Tangeman et al. 1990), and combined functional exercise and muscle strength training (Bastien et al. 1998; Carr and Jones 2003; Dean et al. 2000; Eng et al. 2003; Rimmer et al. 2000; Teixeira-Salmela et al. 1999, 2001). These studies have encompassed both home-based (Dean and Shepherd 1997; Monger et al. 2002; Rodriquez et al. 1996; Tangeman et al. 1990) and community-based group programs (Ada et al. 2003; Bassile et al. 2003; Bastien et al. 1998; Dean et al. 2000; Eng et al. 2003; Rimmer et al. 2000; Teixeira-Salmela et al. 1999, 2001) as well as individual lab-based programs (Carr and Jones 2003; Engardt et al. 1995; Hesse et al. 1995; Kim et al. 2001; Macko et al. 1997, 2001; Miller 2001; Miller et al. 2002; Mudge et al. 2003; Potempa et al. 1995; Sharp and Brouwer 1997; Silver et al. 2000; Smith et al. 1998b, 1999, 2000; Sullivan et al. 2002; Trueblood 2001). These programs have ranged in duration from 2 - 24 weeks of training with
the exception of the study by Rodriquez et al. (1996) in which training at home lasted for between 10 and 65 months. Simple physical/occupational therapy assessment and treatment in the home has also been used and promising results including improved functional mobility have been reported (Green et al. 2002; Wade et al. 1992).

Exercise in individuals with chronic stroke has shown significant benefits including improvements in gait (Rodriquez et al. 1996; Teixeira-Salmela et al. 2001; Trueblood 2001), increased gait speed (Ada et al. 2003; Bassile et al. 2003; Dean et al. 2000; Eng et al. 2003; Hesse et al. 1995; Miller et al. 2002; Miller 2001; Monger et al. 2002; Sullivan et al. 2002; Teixeira-Salmela et al. 1999, 2001), increased muscle strength (Badics et al. 2002; Carr and Jones 2003; Engardt et al. 1995; Kim et al. 2001; Rimmer et al. 2000; Sharp and Brouwer 1997; Smith et al. 1998b, 1999; Teixeira-Salmela et al. 1999; Weiss et al. 2000), faster chair stand time (Weiss et al. 2000), faster stair climbing speed (Eng et al. 2003), increased peak VO$_2$ (Macko et al. 2001; Potempa et al. 1995; Rimmer et al. 2000), reduced steady state VO$_2$ (Macko et al. 1997), decreased energy expenditure (Macko et al. 2001), increased weight bearing ability or force production from the paretic limb (Dean and Shepherd 1997; Dean et al. 2000; Tangeman et al. 1990), increased balance ability (Tangeman et al. 1990), and increased muscle activation in the affected limb (Dean and Shepherd 1997). Moreover, improvements in clinical assessments including the Get Up and Go test (Silver et al. 2000), Berg Balance (Bastien et al. 1998; Eng et al. 2003; Miller 2001; Miller et al. 2002; Mudge et al. 2003; Weiss et al. 2000), Activities of Daily Living Index (Tangeman et al. 1990), and the Nottingham Health Profile (Teixeira-Salmela et al. 1999) have also been observed with exercise. While it is clear that exercise has substantial benefits for individuals with chronic stroke, whether different types of exercise programs have different effects remains uncertain.

Unfortunately, the majority of the existing research on exercise training in individuals with chronic stroke has not focused specifically on postural control and none have investigated falls-reduction. In fact, only a few studies have investigated the effects of exercise on functional balance or postural control in individuals with chronic stroke (Bastien et al. 1998; Dean and Shepherd 1997; Eng et al. 2003; Smith et al. 2000; Tangeman et al. 1990). The majority of the exercise-training studies are not controlled; rather, they have one group and are a pre-, post-test design. Consequently, it is difficult to assess whether changes are due to the exercise intervention or from the attention from the therapists or from the act of attending the exercise sessions. Nonetheless, in a study by Tangeman et al. (1990), functional balance ability (measured using a scale designed for the study) was significantly improved following a four-
week (2hr/session, 4x/week) individual home-based training program focusing on weight shifting, balance, and functional activities. In a feasibility study by Bastien et al. (1998), individuals with chronic stroke performed functional balance exercises (dance and Tai Chi) in combination with muscle strengthening tasks and group discussions. This community-based group exercise program demonstrated an increase in Berg Balance over the 6 – 8 weeks of training. However, in a later study, Smith et al. (2000) examined the influence of a 12-week (3x/wk) progressively graded treadmill aerobic exercise program on postural control. Participants were subjected to horizontal platform perturbations pre- and post-intervention and reaction and recovery times were analyzed; however, no significant differences were discovered (Smith et al. 2000). These authors suggested that task-specific training might be necessary to improve balance ability. This has been demonstrated in acute stroke survivors, where participants underwent 15 sessions over three weeks of platform translation training and demonstrated an increased ability to withstand increasing magnitude translations compared to acute stroke survivors who did not receive the intervention (Hocherman et al. 1984).

In chronic stroke survivors, Dean and Shepherd (1997) examined sitting postural control in a randomized controlled study, where the experimental group practiced exercises over two weeks designed to improve sitting and loading the affected leg while reaching and the control group performed cognitive manipulative tasks while seated. The exercise training increased the loading ability of the affected leg and led to greater muscle activation in the experimental group compared to the control group (Dean and Shepherd 1997). Recently, Eng et al. (2003) evaluated the effect of an eight-week community-based group exercise intervention (involving walking, strength training, and balance exercises) on functional capacity and balance in individuals with chronic stroke; their results demonstrated improvements in both. Interestingly, this last study is the only one to investigate retention effects and the results suggest that the exercise effects are retained for at least one month following the intervention.

Although the consequences of a fall due to an unexpected perturbation to balance are detrimental, there exist no studies on the influence of exercise on standing postural reflexes in individuals with chronic stroke. Promising results from a study in healthy older adults has shown that multi-sensory training can increase stability, decrease falls, and decrease muscle onset latencies of postural reflexes (Hu and Woollacott 1994a, b).

Therefore, one of the objectives of this thesis was to examine the influence of exercise training in individuals with chronic stroke on the reactive postural control system and determine which types of exercise programs are effective in improving postural control. A large emphasis
on community-based group exercise programs for individuals with chronic stroke is necessary, as acute phase rehabilitation is shortening and individuals have many years with their injury without easy access to these types of programs.

1.4.3 Postural control following stroke

There is very little research on postural control following stroke. In contrast, extensive literature exists for healthy young and older adults in which much of our understanding of how the body controls movement stems from. Challenging postural stability through destabilizing forces induced while standing on a moveable platform (i.e. perturbations) provides an indication about an individual’s reactive postural control capabilities. Original work by Horak and Nashner (1986) defined two types of postural responses to perturbations during stance: the ankle and hip strategies. The ankle strategy re-establishes stability by moving the centre of mass back within the base of support (i.e. the feet) through movement primarily about the ankle joint (Horak and Nashner 1986). Muscle activity originates in muscles surrounding the ankle followed by a sequential activation of muscles controlling the knee, hip, and trunk (Horak and Nashner 1986). When perturbations are larger, faster, and/or the individual is standing on a smaller surface, centre of mass is moved to re-establish stability through movement about the hip joints (Horak and Nashner 1986). Muscle activation in this case, the hip strategy, originates in the muscles surrounding the hip and controlling the trunk followed by muscles controlling the knee and minimal involvement of ankle musculature (Horak and Nashner 1986). Recent work has extended the idea of these ‘feet-in-place’ strategies and focused more on responses termed ‘change-in-support’ strategies that move the base of support to maintain stability during perturbed stance (Maki and McIlroy 1997). These researchers argue that individuals preferentially use stepping strategies, termed ‘compensatory stepping reactions’, and/or reaching/grasping movements to control stability (Maki and McIlroy 1997). Hence, there are multiple strategies available in an individuals’ repertoire for postural control.

In healthy older adults, postural control is compromised due to decreased musculoskeletal capacity (e.g. muscle weakness), sensory function, visual impairments, neural processing, and cognitive abilities, which result in a large number of falls in this population (Alexander 1994; Campbell et al. 1989; Maki and McIlroy 1996, 1997, 1999; Tinetti et al. 1988). Muscle onset latencies are delayed and the strength of the postural responses is diminished with aging (Lin
and Woollacott 2002). Furthermore, older adults often require multiple steps to recover balance following sudden platform movements (Maki and McIlroy 1999).

The investigation into the postural responses of individuals with stroke following platform perturbations that challenge the reactive postural control system have only recently started to be investigated. Early studies have sought to characterize the patterns and timing of the postural responses and to compare them with older healthy adults (Badke and Duncan 1983; Badke et al. 1987; Berger et al. 1988; Dietz and Berger 1984; Di Fabio et al. 1986; Di Fabio 1987; Jiang et al. 1998). Individuals with chronic stroke suffer from similar problems as healthy older adults although usually on a much larger scale. The additional presence of hemiparetic upper and lower extremities and a tendency for asymmetrical weight bearing results in decreased stability and greater challenges for the postural control system. Further, damage to supraspinal centers may limit cognitive resources and sensory integration ability for maintaining postural stability (unpublished observations). Individuals with stroke who undergo platform perturbations show frequent co-contraction of muscles surrounding the ankle and knee joint (Badke and Duncan 1983; Berger et al. 1988; Di Fabio et al. 1986; Di Fabio 1987; Hocherman et al. 1988), no clear muscle sequencing and greater variability of muscle activity (Badke and Duncan 1983), and frequent occurrences of zero-onset responses (absent postural muscle response bursts) (Di Fabio et al. 1986; Di Fabio 1987). Moreover, individuals with stroke exhibit delayed paretic muscle onset latencies compared to the non-paretic limb and healthy older adults (Berger et al. 1988; Dietz and Berger 1984; Di Fabio and Badke 1988; Di Fabio et al. 1986; Di Fabio 1987) and delayed paretic limb ankle muscle torque response compared to healthy older adults (Al-Zamil 1998; Ikai et al. 2003) in response to platform perturbations. Interestingly, prior knowledge of the direction of perturbation reduces the frequency of zero-onset responses and shortens the muscle onset latencies in the paretic limb (Badke et al. 1987). Recent evidence also suggests that individuals with chronic stroke have a greater tendency to use multiple steps to recover balance (majority of who step first with their loaded non-paretic limb) and/or use grasping strategies following perturbed stance (Jiang et al. 1998).

In this thesis, sudden horizontal platform translations (i.e. platform perturbations) were used to generate postural reflexes in individuals with chronic stroke and to provide a measure of their reactive postural control system.
1.5 Research Questions

Several research questions were posed in order to address the purpose of this thesis. We conducted three experiments using individuals with chronic stroke to answer these questions. Experiments II and III were from the same sample of participants (i.e. data for Experiment II came from baseline data of Experiment III). The following are the research questions, which guided this thesis along with a brief statement of how they contribute to the overall thesis and/or our research knowledge base:

1.5.1 Research Question #1 (Chapter 2)

Does weight-bearing load on lower extremities affect postural reflexes in individuals with chronic stroke?

**Hypothesis:** Muscle onset latencies will be faster and the magnitude of postural reflexes will increase as weight-bearing load is increased for both ankle dorsiflexors and extensors.

Since individuals with stroke often adopt an asymmetrical weight-bearing posture (i.e. greater weight-bearing on the non-paretic lower extremity) and exercise may alter this strategy, it is important to determine whether loading on a limb affects postural reflexes. If weight-bearing load does influence the latency of postural reflexes then weight-bearing load during post-intervention testing following the exercise intervention study would need to be matched to baseline values. This experiment also determines whether individuals with stroke can still modulate postural reflexes.

In order to determine whether an exercise intervention is capable of modifying postural reflexes, we must first establish test re-test reliability to ensure that the change can be attributed to the intervention rather than a learning effect over time. Thus, this experiment was repeated a second time within a few days. Intraclass Correlation Coefficients (Shrout and Fleiss 1979) were determined along with Standard Error of Measurement. Results of the test re-test reliability are in Chapter 4 and in Appendix IV.
1.5.2 Research Question #2 (Chapter 3)

What are the primary neural mechanisms for falls during unexpected platform translations that challenge the reactive postural control system in individuals with chronic stroke?

Hypothesis: individuals with stroke who fall will have delayed muscle onset latencies in response to unexpected platform translations and demonstrate reduced ankle muscle strength during clinical testing.

It is important to understand why some individuals with stroke fall and others do not so that rehabilitation strategies can be developed to target the appropriate aspects contributing to these falls. Postural reflexes are evoked by a translating platform upon which participants are standing.

1.5.3 Research Question #3 (Chapter 4 primary study)

What are the effects of two different exercise interventions (a fast-paced, multi-sensory, agility exercise program versus a slow-paced, stretching/weight-shifting exercise program) on the timing of standing postural reflexes, functional balance and mobility, reaction time, falls, health-related quality of life, and balance confidence in individuals with chronic stroke?

Hypothesis: the two exercise programs will improve these measures as demonstrated by (a) faster standing postural reflexes, (b) increased Berg Balance (see Appendix V) scores, (c) decreased time for the Timed Up and Go Test, (d) faster step reaction time (e) decreased Nottingham Health Profile (see Appendix VI) scores, (f) increased Activity-specific Balance Confidence (see Appendix VII) scores, (g) and reduced 12-month prospective number of falls in the community. It is hypothesized that the Agility exercise intervention will show greater improvements than the Stretching/weight-shifting program for all measures.

The knowledge obtained from this study will help guide clinicians with developing effective interventions for improving postural control following stroke. Further, if changes with exercise are demonstrated, this might suggest neural plasticity in the chronic stage of injury following stroke.
CHAPTER 2 - Experiment I

Modulation of Ankle Muscle Postural Reflexes in Stroke: Implications For Falls
(In review with Journal of Physiology)

2.1 Abstract

**Background and Purpose** - Falls are common among individuals with stroke. Postural reflexes are essential reactive control mechanisms to prevent falls when an unexpected destabilizing force is applied to the body. Given the known sensorimotor deficits and asymmetrical weight-bearing posture in stroke, the aim of this study was to determine whether stroke affects the modulation of standing postural reflexes with varying weight-bearing load.

**Methods** - Ten individuals with chronic stroke and 10 healthy older adult controls were exposed to unexpected forward and backward platform translations while standing. Three different stance conditions were imposed: increased weight-bearing load, decreased weight-bearing load, and self-selected stance. Surface EMG from bilateral ankle dorsiflexors (tibialis anterior) and extensors (gastrocnemius) were recorded and the magnitude of background muscle activity (prior to the platform translation), magnitude of a postural reflex (75 msec following reflex onset), and postural reflex muscle onset latency were determined.

**Results** - Load-dependent modulation of ankle extensors was found in controls and individuals with stroke. In contrast, load did not change the onset latency of postural reflexes of the individuals with stroke. Although controls demonstrated modulation of ankle dorsiflexors to different loads, individuals with stroke did not show this modulation and falls were most frequent when participants were required to utilize the ankle dorsiflexors.

**Conclusions** - Delayed paretic muscle onset latencies in conjunction with impaired modulation of ankle dorsiflexor postural reflexes may contribute to the instability and frequent falls observed among persons with stroke.

2.2 Introduction

The incidence of falls for community-dwelling individuals with stroke has been reported to be as high as 73% (Forster and Young 1995). Thus, an understanding of postural control in stroke is of importance to facilitate the reduction of falls in this population. Reactive postural
control entails a response, mediated by postural muscle reflexes, to a sudden unexpected perturbation applied to the body and relies on the central nervous system’s (CNS) ability to interpret afferent information to ensure that the centre of mass (COM) stays within the base of support. Proprioceptive afferents from extensor muscles in the legs and exteroceptive afferents from mechanoreceptors in the foot are among the available inputs to the CNS to modulate standing or locomotor postural reflexes (Duysens et al. 2000). It has been suggested that these afferent inputs can provide information regarding load, and thus, have been referred to as load receptors and are particularly useful in signalling COM position (Dietz et al. 1992).

Load receptor input for the regulation of stance in humans has been demonstrated in studies which found an increase in the magnitude of postural reflexes with increasing body load (via lead vests) in response to a translation of a platform upon which participants were standing under water (Dietz et al. 1989; Horstmann and Dietz 1990). Furthermore, greater load applied to the body during standing platform translations on land was associated with an increase in the magnitude of extensor muscle postural reflexes in healthy individuals (Dietz et al. 1992). It is believed that Ib afferents from golgi tendon organs (GTOs) provide load information to the spinal cord (Dietz et al. 1992; Duysens et al. 2000). In addition to Ib afferents from GTOs, cutaneous mechanoreceptors in the sole of the foot are in an optimal location to sense limb load and have been recently shown to be important in standing balance (Kavounoudias et al. 2001).

Understanding the postural responses under varying weight-bearing load is of interest since individuals with stroke tend to bear greater weight on the non-paretic limb (Eng and Chu 2002). Whether load-dependent modulation remains intact following stroke is unknown. Hassid et al. (1997) and Trueblood (2001) have studied the use of body-weight supported (BWS) treadmill training in stroke, which inherently altered limb loading and have shown improvements in gait and balance. Albeit few, studies on postural reflexes in stroke have demonstrated delayed muscle onset latencies and abnormal recovery strategies in response to external perturbations while standing (Berger et al. 1988; Dietz and Berger 1984; Di Fabio et al. 1986; Di Fabio 1987).

The objective of this study was to determine whether stroke affects the modulation of postural reflexes with weight-bearing load. Assessing the contribution of postural reflexes from weight-bearing load will determine whether the asymmetrical posture in individuals with stroke (i.e., reduced loading on the paretic limb) contributes to these altered reflexes and will quantify the extent that afferent information from the lower extremities can be utilized for modulation of postural reflexes in stroke.
2.3 Method

2.3.1 Participants

Ten individuals with hemiparesis (4 right and 6 left) due to stroke and ten healthy older adult controls were recruited from the community. Participant characteristics are described in Table 2.1. The inclusion criteria for the individuals with stroke were: (1) over 50 years of age, (2) only one stroke, (3) at least one year post stroke onset, (4) able to stand independently for at least 5 minutes without an assistive device, and (5) able to follow two-step commands. Persons with musculoskeletal or neurological disorders in addition to their stroke were excluded. Healthy older adults with musculoskeletal or neurological disorders were also excluded. Limb dominance of the healthy older adults was determined by asking which leg they used to kick a soccer ball. Following university and hospital ethics approval, informed consent was received from all participants prior to their participation (see Appendix VIII).

Table 2.1: Mean (SD) of participant characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Stroke (N = 10)</th>
<th>Controls (N = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>61.3 (8.9)</td>
<td>60.6 (5.5)</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>76.2 (17.6)</td>
<td>76.7 (10.2)</td>
</tr>
<tr>
<td>Height, cm</td>
<td>166.5 (18.2)</td>
<td>174.6 (10.0)</td>
</tr>
<tr>
<td>Stroke Duration, yrs</td>
<td>4.1 (2.9)</td>
<td>N/A</td>
</tr>
<tr>
<td>Affected Side</td>
<td>4 Right / 6 Left</td>
<td>N/A</td>
</tr>
<tr>
<td>Berg Balance, max. 56</td>
<td>44.9 (8.3)</td>
<td>N/A</td>
</tr>
<tr>
<td>Type of Stroke</td>
<td>5 Ischemic, 4 Hemorrhagic, 1 unknown</td>
<td>N/A</td>
</tr>
</tbody>
</table>
2.3.2 Protocol

Participants stood on two force plates (Bertec Corp.), one limb on each force plate, embedded in a platform and forward or backward translations of the platform were applied during three stance conditions: increased weight-bearing load (Increased Load), decreased weight-bearing load (Decreased Load), and self-selected stance (Neutral). Participants wore a full-body harness that was attached to a beam in the ceiling via a dynamic climbing rope to prevent the occurrence of a fall and at least one spotter was present. Participants were instructed to maintain their normal standing posture or shift their weight onto either the right or left leg (depending on the condition) and sustain the standing position. For the Increased or Decreased Load conditions, the experimenter visually monitored the force plate vertical forces in order to ensure that participants maintained the appropriate amount of weight-bearing (target of approximately 70% weight-bearing on one limb) prior to triggering the platform translation.

A total of 15 backward and 15 forward platform translations (8 cm displacement, 30 cm/s velocity, and 300 cm/s² acceleration) were induced in three blocks (Increased Load, Decreased Load, and Neutral) of 5 trials separated by 15-30 second intervals (see Appendix IX for experimental protocol). Participants were told that the platform could move at any time prior to triggering the perturbation but the onset and direction of translation were unexpected in nature. The blocks of trials were randomly ordered for both platform directions and weight-bearing load conditions.

Surface electromyography (EMG) (Bortec) from bilateral tibialis anterior (TA) and the medial head of gastrocnemius (MG) were recorded at 600 Hz for 6 seconds (2 seconds prior to platform movement and 4 seconds after) along with force plate data (see Appendix X for EMG placement protocol). The MG muscle was used for analysis in the backward translations and the TA muscle was used in the forward translations due to their role as primary recovery muscles for these movements (Horak and Nashner 1986).

2.3.3 Data Analysis

For each condition in both directions, trials 2-5 were averaged. The first trial in response to a perturbation has been shown to be clearly different from subsequent ones (Marigold and Patla 2002). Custom MATLAB software was used to calibrate the force plate data and subsequently,
the mean vertical forces were calculated one second prior to platform onset to determine the
amount of weight-bearing for each limb for each trial.

EMG was full-wave rectified and low-pass filtered at 100 Hz. All EMG data processing
used a custom written MATLAB program. The mean EMG signal for one second prior to the
onset of platform movement was determined along with the standard deviation. Muscle onset
latency, representing a postural reflex, was defined as an increase in muscle activity that
exceeded +2 standard deviations (SD) or fell below −2 SD (depending on whether the burst
was excitatory or inhibitory) for at least 30 msec and was determined by a combination of visual
inspection and computer algorithm via an interactive program (Marigold and Patla 2002).

Background muscle activity was calculated as the area under the curve (trapezoid rule) for
one second prior to the onset of the platform movement. The magnitude of muscle activity for
75 msec following the onset of a postural reflex was also calculated as the area under the curve
(see Figure 2.1a). For each individual trial, the magnitude was obtained after removal of each
muscle's background activity from the EMG signal (on a single trial basis) so that the reflex
magnitude was not masked by any changes in background activity.

Data from the dominant and non-dominant limbs of controls were collapsed over each load
condition because there was no significant difference between them using paired t-tests. Three
separate repeated measures analyses of variance (ANOVA) determined the effect of weight-
bearing load conditions (Decreased Load, Neutral, Increased Load) on the background muscle
activity of the (1) paretic limb of the stroke group, (2) non-paretic limb of the stroke group, and
(3) controls. Post-hoc analysis consisted of Duncan's test. These statistical procedures were
also performed for the dependent variables of the postural reflex magnitude and muscle onset
latency. For graphical presentation, both background muscle activity and magnitude of postural
reflexes were normalized to the Neutral condition (Figure 2.2 and 2.3); however, statistical
analyses were performed on the non-normalized data. Paired t-tests compared the muscle onset
latencies between the paretic and non-paretic limbs of the individuals with stroke and
independent t-tests compared the stroke and control groups. All statistical analyses (alpha =
0.05) were performed with SPSS, version 11.0 for Windows.
2.4 Results

Three individuals with stroke fell (i.e. were caught by the rope and harness system or required the assistance of the spotter) during the platform translations, of which all falls occurred during the forward translations (i.e. backward induced sway that required TA activity). No control participants experienced a fall.

Table 2.2 shows the amount of weight-bearing on the paretic and non-dominant limb for the individuals with stroke and controls, respectively. In the Neutral condition the individuals with stroke had approximately 10% greater weight-bearing on their non-paretic limb while controls were nearly symmetrical.

Table 2.2: Weight-bearing load in percent body-weight (SD).

<table>
<thead>
<tr>
<th>Platform Direction</th>
<th>Condition</th>
<th>Control Non-dominant Limb</th>
<th>Control Paretic Limb</th>
<th>Stroke Non-dominant Limb</th>
<th>Stroke Paretic Limb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Backward</td>
<td>Decreased Load</td>
<td>22.9 (5.3)</td>
<td>23.8 (5)</td>
<td>22.9 (5.3)</td>
<td>23.8 (5)</td>
</tr>
<tr>
<td></td>
<td>Neutral</td>
<td>49.0 (1.9)</td>
<td>41.9 (10.4)</td>
<td>49.0 (1.9)</td>
<td>41.9 (10.4)</td>
</tr>
<tr>
<td></td>
<td>Increased Load</td>
<td>77.3 (8)</td>
<td>61.3 (17.5)</td>
<td>77.3 (8)</td>
<td>61.3 (17.5)</td>
</tr>
<tr>
<td>Forward</td>
<td>Decreased Load</td>
<td>23.0 (4.8)</td>
<td>24.0 (6.1)</td>
<td>23.0 (4.8)</td>
<td>24.0 (6.1)</td>
</tr>
<tr>
<td></td>
<td>Neutral</td>
<td>49.0 (2.4)</td>
<td>40.8 (10.3)</td>
<td>49.0 (2.4)</td>
<td>40.8 (10.3)</td>
</tr>
<tr>
<td></td>
<td>Increased Load</td>
<td>74.8 (7.3)</td>
<td>61.3 (17.8)</td>
<td>74.8 (7.3)</td>
<td>61.3 (17.8)</td>
</tr>
</tbody>
</table>
Figure 2.1: (A) Diagrammatic definitions of the onset latency and postural reflex magnitude (hatched is the postural reflex magnitude calculated from a 75 msec area from the onset latency). Typical postural reflex responses of the (B) paretic MG and (C) non-paretic MG under the three load conditions for one individual with stroke.
2.4.1 The effect of weight-bearing load on the magnitude of postural reflexes

In response to the backward platform translations, the magnitude of MG postural reflexes (i.e. after removal of background activity) increased with increasing weight-bearing load for the controls and both limbs of the individuals with stroke (Figure 2.1 and 2.2). Post-hoc analysis revealed a difference between the Increased Load and Decreased Load conditions for both groups (p < 0.05). Furthermore, there was a greater magnitude postural reflex in the Increased Load condition compared to the Neutral condition and in the Neutral condition compared to the Decreased Load condition for the controls (p < 0.05). Background muscle activity of the MG demonstrated identical results to the magnitude of MG postural reflexes for both individuals with stroke and controls (Figure 2.3).

The TA postural reflex magnitude (in response to forward platform translations) was greater in the Increased Load and Neutral conditions versus the Decreased Load condition in the control group (p < 0.05); however, TA changes were small (i.e. 20% increase in the Increased Load compared to Decreased Load versus a 90% change with the extensor muscles in controls) (Figure 2.2). In contrast, there was no difference in magnitude of TA postural reflexes among the three load conditions for both limbs in the individuals with stroke. In fact, there was a trend (p = 0.09) of increasing magnitude of postural reflexes in the non-paretic TA in the Decreased Load condition compared to the Neutral condition.

The TA background muscle activity produced similar findings to the magnitude of the postural reflex (Figure 2.3). Specifically, post-hoc analysis indicated greater (p < 0.05) background muscle activity in the Increased Load condition compared to both the Neutral and Decreased Load conditions for the controls. In contrast, non-paretic TA background muscle activity in the individuals with stroke was greater in the Decreased Load condition compared to both the Neutral and Increased Load conditions (p < 0.05).
Magnitude of MG postural reflex in response to backward translations

Controls

Magnitude of TA postural reflex in response to forward translations

Controls

Stroke

Figure 2.2: Postural reflex magnitude from MG and TA for the stroke and control groups. Values normalized to the Neutral condition are shown, although statistical analyses were performed on the non-normalized data. Note that background muscle activity was removed prior to calculating the magnitude.
Background MG activity prior to backward translations

Controls

Stroke

Figure 2.3: Background muscle activity from MG and TA for the stroke and control groups. Values normalized to the Neutral condition are shown.

2.4.2 The effect of weight-bearing load on muscle onset latency

In response to the backward platform translations, the controls demonstrated differences in MG muscle onset latency among the three weight-bearing conditions (p < 0.05). MG postural reflexes were faster as weight-bearing load increased with the Increased Load condition approximately 13 msec faster than the Decreased Load condition (Table 2.3). In contrast, no differences in MG muscle onset latency among the weight-bearing conditions were found for the individuals with stroke for either the paretic or non-paretic limb. Furthermore, there was no difference in onset latency (means less than 5 msec apart) among the three weight-bearing
conditions for the TA in the stroke and control groups during the forward platform translations (Table 2.3).

In the individuals with stroke, the paretic limb TA and MG were significantly delayed up to 15 msec and 26 msec, respectively, compared to the non-paretic TA and MG (p < 0.05). The paretic limb TA and MG latencies were also delayed compared to the control TA and MG, respectively (p < 0.05). In contrast, there was no difference for the TA or MG latencies between the non-paretic limb of the individuals with stroke and the controls.

**Table 2.3:** Mean (SD) onset latencies (msec) for backward & forward platform translations.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Controls</th>
<th>Stroke</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Paretic</td>
<td>Non-paretic</td>
<td></td>
</tr>
<tr>
<td><strong>MG onset latency in response to backward translations</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased Load</td>
<td>115.4 (20.7)*</td>
<td>141.1 (25.3)</td>
<td>115.9 (12.4)</td>
</tr>
<tr>
<td>Neutral</td>
<td>107.1 (10.1)</td>
<td>139.3 (22.7)</td>
<td>113.5 (20.1)</td>
</tr>
<tr>
<td>Increased Load</td>
<td>102.5 (17.1)</td>
<td>136.8 (28.1)</td>
<td>118.9 (21.8)</td>
</tr>
<tr>
<td><strong>TA onset latency in response to forward translations</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased Load</td>
<td>105.7 (16.5)</td>
<td>123.6 (22.8)</td>
<td>111.3 (13.2)</td>
</tr>
<tr>
<td>Neutral</td>
<td>102.4 (9.7)</td>
<td>122.8 (23.1)</td>
<td>107.8 (13)</td>
</tr>
<tr>
<td>Increased Load</td>
<td>101.3 (7.5)</td>
<td>124.5 (22)</td>
<td>109.3 (13.1)</td>
</tr>
</tbody>
</table>

* Increased Load different from Decreased Load condition for MG, p < 0.05.

2.5 Discussion

The major findings of this study suggest that: (1) load-dependent modulation of ankle extensor postural reflex magnitude remains intact following stroke, (2) load does not affect
muscle onset latencies in individuals with stroke, and (3) individuals with stroke are unable to modulate ankle dorsiflexor postural reflex magnitude under different load conditions.

### 2.5.1 Modulation of ankle muscle postural reflex magnitude following stroke

Following stroke, individuals demonstrated load-dependent modulation of the magnitude of ankle extensor muscle postural reflexes to varying levels of weight-bearing load. Dietz and colleagues (Dietz et al. 1989; Dietz et al. 1992; Horstmann and Dietz 1990) have demonstrated that the magnitude of human postural reflexes of ankle extensors is sensitive to load in healthy individuals. Thus, load modulation may not require intact supraspinal input (e.g. from corticospinal tract fibres) but rather, has substantial integration within the spinal cord. Weight-bearing load has been shown to modulate lower limb muscle activity in individuals with spinal cord injury (Harkema et al. 1997). The fact that individuals with stroke still utilize load information supports recent body-weight supported treadmill-training studies (Hassid et al. 1997; Trueblood 2001) and substantiates the rationale for their implementation for stroke rehabilitation.

For the ankle muscles of the control group and the extensor muscles of the stroke group, the concept of ‘automatic gain compensation’ was demonstrated where the muscle response (after removal of background activity) increased with increasing background activity (Matthews 1986). This phenomenon may be of benefit during perturbed stance in that a larger reflex could be used to stabilize balance through an ankle strategy that is potentially compromised in asymmetrical posture. For example, the larger reflex evoked in the loaded limb would allow it to overcome the biomechanical constraint imposed by the extra load and subsequently step forward or backward. Older adults in response to sudden platform translations often utilize compensatory steps (Maki and McIlroy 1997).

Although the TA does not directly sense load (it is non weight-bearing), the modulation of the magnitude of TA postural reflexes under different loads for the controls was likely from a combination of foot mechanoreceptors, joints receptors, and antagonistic extensor muscle feedback regarding load.

Why was modulation of the ankle dorsiflexor, but not extensor postural reflex magnitude disrupted from stroke? Clinically, individuals with stroke often present with weakness of the paretic ankle dorsiflexors. It is possible that a decreased number of motor units (Dietz et al. 1986), disturbed motor unit recruitment (Gemperline et al. 1995), and decreased motor unit
firing rates (Dietz et al. 1986; Rosenfalck and Andreassen 1980) contributed to the lack of modulation in the ankle dorsiflexor muscles. Further, Brouwer and Ashby (1992) reported that there are stronger connections of supraspinal motor centres (i.e. corticospinal tract fibres) to the distal leg flexors than the extensors as evident from transcranial magnetic stimulation studies. Given our observations of an increased incidence of falling when the condition required TA activity and the inability to modulate the magnitude of TA postural reflexes with load, we performed post-hoc analyses to examine the TA impairment. Based on ankle dorsiflexor isokinetic strength data and Chedoke-McMaster Foot scores, six of the 10 individuals with stroke had severe TA impairment (i.e. dorsiflexor torque < 0.06 Nm/kg and/or Chedoke-McMaster Foot score ≤ 3/7). Of these six individuals, five were unable to modulate TA reflex magnitude to load and three fell. The remaining four individuals had minimal TA impairment (dorsiflexors torque > 0.13 Nm/kg and/or Chedoke-McMaster Foot score of 7/7), demonstrated modulation of TA reflex magnitude, and did not fall. It is apparent that the ankle dorsiflexors are essential for the recovery from falls.

In Figure 2.2, the non-paretic TA postural reflex was not modulated and in fact, a trend toward an increased magnitude in the Decreased Load condition when it would be expected to be the reverse was found. We postulate due to the inability of the paretic limb TA to modulate magnitude in addition to its delayed muscle onset latency, a compensatory strategy results in an increase in the postural reflex magnitude of the non-paretic TA muscle. This interlimb response would serve to initiate a rapid step with the non-paretic limb if need be during a time when the paretic limb is being loaded and compromising stability.

2.5.2 Altered supraspinal control due to stroke affects timing of postural reflexes independent of load

Regardless of the group (i.e. controls or stroke), the timing of the observed postural reflexes in response to platform translations suggests long-latency reflexes that receive supraspinal input. Although others (Berger et al. 1988; Dietz and Berger 1984; Di Fabio et al. 1986; Di Fabio 1987) have reported slower postural onset latencies on the paretic side, our study is the first to control for the load taken through the limbs during the perturbation. The finding that postural reflex muscle onset latencies are independent of weight-bearing load in stroke suggests that the typical asymmetric stance adopted by individuals with stroke does not explain the delay in
muscle onset latencies observed following an external perturbation. Thus, deficits in the latency of paretic lower limb muscles can be attributed to the loss of supraspinal control and/or alterations in muscle properties resulting from the stroke.

Interestingly, MG muscle onset latency demonstrated load-dependent modulation in that postural reflexes were elicited faster when limb load was increased in the healthy, but not stroke participants. However, the functional consequences of the 13 msec faster response in controls from the Decreased to Increased Load conditions needs further exploration.

In conclusion, delayed paretic limb muscle onset latencies in conjunction with impaired modulation of dorsiflexor muscle postural reflex magnitude may contribute to the instability and frequent falls observed among individuals with stroke.

2.6 Bridging Summary

The first study found that individuals with stroke could still utilize sensory feedback regarding weight-bearing load to modulate the magnitude of postural reflexes. In addition, we identified a potential cause of falls in this population in that ankle dorsiflexor modulation is impaired following stroke. Our study has confirmed the results of others that report delayed paretic limb postural reflex muscle onset latency compared to the non-paretic lower limb and lower limbs of healthy older adults in response to standing perturbations. However, it is unknown whether delays in postural reflex onset latency and/or the magnitude of the reflex contribute to falls in individuals with stroke. Therefore, the second study sought to identify the underlying neural mechanisms contributing to falls in individuals with stroke. Evoking standing perturbations and identifying those individuals who fell accomplished this objective. This enabled us to separate out falling trials so that postural reflex muscle onset latency and magnitude could be compared.
CHAPTER 3 – Experiment II

Neural Mechanisms Contributing to Falls in Individuals with Chronic Stroke
(In review with Journal of Neurophysiology)

3.1 Abstract

Falling is a major concern in individuals with stroke. The mechanisms contributing to falls are largely unknown. The purpose of this study was twofold: (1) to determine whether the latency and magnitude of postural reflexes utilized for recovery from standing platform translations could discriminate individuals who fell in response to the translations and (2) to determine whether a clinical assessment of a stroke-related impairment, lower extremity muscle strength, could differentiate individuals who fell in response to platform translations versus those who did not. Fifty-six individuals with chronic stroke underwent unexpected forward and backward standing platform translations in which postural reflex muscle onset latency and magnitude were determined and clinical testing of isokinetic joint torque of the lower extremities. Those who fell during the standing platform translations (N = 14) and those who did not (N = 42) were compared to determine the contributors of falling in this population. Eighty-nine percent of falls occurred during the forward translations. Results demonstrated that paretic tibialis anterior postural reflex magnitude was reduced in those trials in which Fallers fell compared to the trials in which Fallers did not fall during platform translations. Furthermore, Fallers had delayed non-paretic rectus femoris postural reflex onset latency compared to Non-fallers. The altered long-latency postural reflexes suggest supraspinal involvement. In addition, the non-paretic rectus femoris response may be a strategy to compensate for the deficits in the paretic tibialis anterior. Studies using animal and human-based models are required to identify which pathways and structures may contribute to these altered reflexes and subsequent falls.
3.2 Introduction

Falls and fall-related injuries occur frequently among community-dwelling stroke survivors with many of these individuals falling multiple times within a year (Forster and Young 1995; Hyndman et al. 2002; Kanis et al. 2001). Whether injury occurs or not, fear of falling may result (Friedman et al. 2002), which could lead to activity restriction and/or further sedentary lifestyle (Murphy et al. 2002). The primary mechanisms behind a falling episode in individuals with stroke are not clear. To date, the few studies on falls in individuals with chronic stroke have focussed on predictors (Forster and Young 1995; Jorgensen et al. 2002; Lamb et al. 2003), which can identify those individuals at high risk for falls, but do not delineate the specific motor strategies which are necessary to prevent the occurrence of a fall. An understanding of strategies which prevent falls are essential in designing effective rehabilitation programs that may lead to improved quality of life and a reduced burden on the health care system.

Following an unexpected balance-threatening event (i.e. perturbation), reactive and protective strategies are initiated by postural reflexes to prevent the occurrence of a fall. The central nervous system (CNS) must utilize and integrate the available sensory and environmental information and an appropriate strategy is generated quickly with sufficient muscle strength and coordination. Studies have shown that individuals with stroke have delayed paretic limb muscle onset latencies following perturbations while standing on a moveable platform compared to their non-paretic limb and to healthy older adults (Berger et al. 1988; Dietz and Berger 1984; Di Fabio and Badke 1988; Di Fabio et al. 1986; Di Fabio 1987). This is in combination with decreased paretic ankle torque responses following platform translations (Ikai et al. 2003). Whether delays in postural reflex muscle onset latency and magnitude contribute to falls in individuals with stroke is unknown. Falls generated in a laboratory setting can be induced in such a way that every individual is exposed to the same destabilizing force. Consequently, the neurophysiological mechanisms associated with falls can be quantified. Thus, the first objective of this study was to determine whether the latency and magnitude of postural reflexes utilized for recovery from standing platform translations are different in those individuals with stroke who fall during this task versus those who do not.

Reduced lower extremity muscle strength has been found to predict falls in healthy older adults (Lord et al. 1994; Whipple et al., 1987). Lower extremity muscle strength is further reduced in individuals with stroke compared to healthy older adults, particularly in the paretic limb (Adams et al. 1990; Eng et al. 2002). In addition, both the time to initiate a muscle
contraction and time to generate torque are delayed in individuals with stroke (Chae et al. 2002; McCrea et al. 2003). Therefore, the second objective of this study was to determine whether clinical assessments of a stroke-related impairment, lower extremity muscle strength, could discriminate individuals who fell in response to standing platform translations versus those who did not.

3.3 Methods

3.3.1 Participants

Fifty-six individuals with hemiparesis due to stroke were recruited from the community. Participant characteristics are described in Table 3.1. Information on the type and location of the participants’ stroke was collected through medical records and/or physician notes. The American Heart Association Stroke Functional Classification (AHASFC) was used to provide an indication of the level of impairment of the participants (see Appendix XI). The AHASFC is based on the level of independence of an individual where level I represents complete independence in basic and instrumental daily activities of living and level V represents complete dependence (Kelly-Hayes et al. 1998). The inclusion criteria for the individuals with stroke were: (1) over 50 years of age, (2) only one stroke, (3) at least one year post stroke onset (i.e. chronic stroke), (4) able to stand independently for at least 5 minutes without an assistive device, and (5) able to follow two-step commands. Persons with musculoskeletal or neurological disorders in addition to their stroke were excluded. Following university and hospital ethics approval, informed consent was received from all participants prior to their participation in the study (see Appendix XII).
Table 3.1: Participant characteristics of the non-fallers (N = 42) and fallers (N = 14).

<table>
<thead>
<tr>
<th></th>
<th>Non-fallers</th>
<th>Fallers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD) or n</td>
<td>Mean (SD) or n</td>
</tr>
<tr>
<td>Gender, M/F</td>
<td>32/10</td>
<td>7/7</td>
</tr>
<tr>
<td>Age, yrs</td>
<td>66.9 (8.1)</td>
<td>69.9 (7.1)</td>
</tr>
<tr>
<td>Height, cm</td>
<td>171.5 (8.7)</td>
<td>165.8 (9.3)</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>81.0 (16.8)</td>
<td>77.7 (13.5)</td>
</tr>
<tr>
<td>Stroke Duration, yrs</td>
<td>4.0 (3.3)</td>
<td>3.1 (2.0)</td>
</tr>
<tr>
<td>Hemiparetic Side, R/L/NA</td>
<td>15/27/0</td>
<td>5/8/1</td>
</tr>
<tr>
<td>AHASFC, 1 - 5</td>
<td>2.2 (0.9)</td>
<td>2.9 (1.1)</td>
</tr>
<tr>
<td>Type of Stroke</td>
<td>20 ischemic</td>
<td>8 ischemic</td>
</tr>
<tr>
<td></td>
<td>15 hemorrhagic</td>
<td>2 hemorrhagic</td>
</tr>
<tr>
<td></td>
<td>2 subarachnoid hemorrhage</td>
<td>1 subarachnoid hemorrhage</td>
</tr>
<tr>
<td></td>
<td>3 lacunar</td>
<td>1 lacunar</td>
</tr>
<tr>
<td></td>
<td>2 unknown</td>
<td>2 unknown</td>
</tr>
<tr>
<td>Stroke Location</td>
<td>10 cortical</td>
<td>6 cortical</td>
</tr>
<tr>
<td></td>
<td>15 subcortical</td>
<td>2 subcortical</td>
</tr>
<tr>
<td></td>
<td>8 brainstem/cerebellum</td>
<td>3 brainstem/cerebellum</td>
</tr>
<tr>
<td></td>
<td>1 cortical/subcortical</td>
<td>3 unknown</td>
</tr>
<tr>
<td></td>
<td>8 unknown</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: M = male; F = female; R = right; L = left; NA = not applicable; AHASFC = American Heart Association Stroke Functional Classification

3.3.2 Protocol

Participants were tested over two occasions separated by one to seven days to minimize fatigue. For assessing postural reflexes, a total of 20 platform translations (8 cm displacement,
30 cm/s velocity, and 300 cm/s² acceleration), separated by 15-30 second intervals, were induced while participants stood on two force plates (Bertec Corp.), one limb on each force plate, embedded in a custom built platform (see Appendix XIII for experimental protocol). To prevent the occurrence of a fall to the ground, participants wore a full-body harness that was attached to a beam in the ceiling via a dynamic rock-climbing rope and at least one spotter stood beside them. Participants were told that the platform could move at any time but the onset and direction of the translation were unexpected in nature. The direction of the translation was counterbalanced across participants so that either 10 consecutive backward translations followed 10 consecutive forward translations or vice versa.

Surface electromyography (EMG) (Bortec) from bilateral tibialis anterior (TA), medial head of gastrocnemius (MG), rectus femoris (RF), and biceps femoris (BF) were recorded at 600 Hz for 6 seconds (2 seconds prior to platform movement and 4 seconds after) along with force plate data (see Appendix X for EMG placement protocol).

Muscle strength (i.e. isokinetic, concentric joint torque) of the ankle, knee, and hip flexors and extensors was collected using a Kin-Com Isokinetic Dynamometer (Chattanooga Group Inc). Muscle strength tested by this apparatus has been shown to be reliable in individuals with chronic stroke (Eng et al., 2002). A detailed description of participant positioning during testing can be found in Eng et al. (2002). An angular velocity of 30-degrees per second for the ankle and 60-degrees per second for the knee and hip were used. One sub-maximal and one maximal trial for each joint and direction were completed as practice. During the maximal practice trial and subsequent three test trials, participants were instructed to “push or pull as hard as possible” throughout their range of motion. Rests were given as needed.

### 3.3.3 Data Analyses

Each trial during the platform translations was classified as a fall or no-fall. A fall was defined as applying weight to the rope (i.e. caught by the rope and harness system) or requiring the assistance of the spotter to prevent a loss of balance. EMG of the postural reflexes was full-wave rectified and low-pass filtered at 100 Hz (single-pass, second-order Butterworth algorithm). All EMG data processing used a custom written MATLAB program. The mean EMG signal for one second prior to the onset of platform movement was determined along with the standard deviation (SD). Muscle onset latency, representing a postural reflex, was defined as an increase in muscle activity that exceeded +2 SD or fell below −2 SD (depending on
whether the burst was excitatory or inhibitory) for at least 30 msec and was determined by a combination of visual inspection and computer algorithm via an interactive program (Marigold et al. 2003). One second of background muscle activity prior to platform movement was calculated as the area under the curve (trapezoid rule). The magnitude of muscle activity for 75 msec following the onset of a postural reflex was also calculated as the area under the curve after removal of each muscle's background activity from the EMG signal (on a single trial basis) so that the reflex magnitude was not masked by any changes in background activity.

For each movement tested using the Kin-Com isokinetic dynamometer, a single ensemble-averaged torque-angle curve was calculated from three maximal repetitions. Subsequently, the mean torques calculated over the torque-angle curve were normalized to the participant's body mass.

### 3.3.4 Statistical Analyses

Falls occurred predominantly in response to the forward platform translations (58 falls in response to forward translations compared to only seven in response to backward translations). Thus, only the responses to forward platform translations were assessed as sufficient Fallers and Non-fallers could be identified for this condition. Consequently, only ankle dorsiflexor, knee extensor, and hip flexor strength data and the TA and RF EMG activity were analyzed since these muscles would be utilized in the primary recovery response to forward platform translations (Horak and Nashner 1986).

Those who fell at least once during the forward translations were identified as Fallers. Among the Fallers, those trials where a fall occurred (i.e. fall trial) and those trials in which a fall didn’t occur (i.e. no-fall trial) were compared using paired t-tests for the following variables: TA onset latency and magnitude and RF onset latency and magnitude for both the paretic and non-paretic limbs. To compare the Fallers (fall trials only) with the Non-fallers, independent t-tests were used for the following variables: TA onset latency and RF onset latency for the paretic and non-paretic limbs. EMG magnitude was not compared between the Non-fallers and Fallers due to methodological constraints associated with EMG collection.

Paretic limb joint torques were entered into a one-way analysis of variance (ANOVA) (ankle dorsiflexion, knee extension, and hip flexion together) to compare overall muscle strength between groups (Fallers and Non-fallers). An ANOVA for the non-paretic limb was also performed.
All statistical analyses were performed using SPSS, version 11.5, for Windows, with an alpha level set at 0.05.

3.4 Results

Figure 3.1 shows a sample of the postural reflexes evoked from the forward platform translations from a Non-faller (3.1A) and Faller (3.1B). Fourteen (25%) individuals with stroke fell during forward platform translations for a total of 58 falls.

3.4.1 Muscle postural reflexes

Individuals in both groups initiated their recovery response from a forward platform translation with the TA prior to RF (i.e. an ankle strategy) and with the non-paretic TA prior to the paretic TA. The mean muscle onset latencies of the postural reflexes for the Fallers (N = 14) and Non-fallers (N = 42) are shown in Table 3.2. The paretic limb onset latencies were delayed compared to the non-paretic limb for both groups.

When comparing the fall trials (n = 58) with the no-fall trials (n = 68) within the group of individuals with stroke who fell (Table 3.2), the results demonstrated a reduced paretic TA postural reflex magnitude in the fall trials (p = 0.03). Further, there was a trend (p = 0.08) for a reduced non-paretic TA postural reflex magnitude in the fall trials compared to the no-fall trials within the individuals who fell at least once. The latency of the postural reflexes of each muscle was not different between the fall and no-fall trials for the Faller group (p > 0.05).

When comparing the postural reflex latencies of the Fallers (fall trials) versus the Non-fallers, all muscle group means were slower for the Fallers; however, only the non-paretic RF onset latency was significantly slower in the Fallers (fall trials) compared to the Non-fallers (p = 0.007; Table 3.2).
3.4.2 Muscle strength

Paretic isokinetic joint torque was reduced in individuals who fell at least once versus those who did not (p = 0.02; Figure 3.2A), with the greatest difference between groups for the ankle dorsiflexors (50% reduction) and knee extensors (34% reduction). Non-paretic isokinetic joint torque was greater than the paretic side for all muscle groups for both individuals who fell and those who did not fall. In addition, non-paretic limb joint torque was reduced in the individuals who fell compared to those who did not (p = 0.05; Figure 3.2B), with the greatest difference between groups for the ankle dorsiflexors (33% reduction).
Table 3.2: Postural reflex differences between Fallers and Non-fallers

<table>
<thead>
<tr>
<th>Measure</th>
<th>Fallers (N = 14)</th>
<th>Non-fallers (N = 42)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fall Trials (n = 58)</td>
<td>No Fall Trials (n = 68)</td>
</tr>
<tr>
<td>Muscle Onset Latency (msec)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paretic TA</td>
<td>126.8 (17.9)</td>
<td>121.6 (22.5)</td>
</tr>
<tr>
<td>Paretic RF</td>
<td>170.6 (48.6)</td>
<td>171.7 (41.0)</td>
</tr>
<tr>
<td>Non-paretic TA</td>
<td>110.8 (15.2)</td>
<td>105.8 (10.3)</td>
</tr>
<tr>
<td>Non-paretic RF</td>
<td>163.5 (36.3)</td>
<td>155.9 (33.5)</td>
</tr>
<tr>
<td>Postural Reflex Magnitude (μV·s)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paretic TA</td>
<td>8.86 (4.29)</td>
<td>10.95 (4.81)*</td>
</tr>
<tr>
<td>Paretic RF</td>
<td>3.22 (2.67)</td>
<td>3.88 (3.74)</td>
</tr>
<tr>
<td>Non-paretic TA</td>
<td>13.92 (6.43)</td>
<td>14.83 (7.01)</td>
</tr>
<tr>
<td>Non-paretic RF</td>
<td>4.66 (3.06)</td>
<td>4.44 (3.21)</td>
</tr>
</tbody>
</table>

Note: Mean (SD)
* Fallers (Fall trials) different than Fallers (No-Fall trials), p < 0.05
** Non-fallers different than Fallers (Fall trials), p < 0.05
Figure 3.2: Isokinetic joint torques normalized to body mass for the ankle dorsiflexors, knee extensors, and hip flexors. (A) Paretic limb joint torque for the Non-fallers and Fallers. (B) Non-paretic limb joint torque for the Non-fallers and Fallers.
3.5 Discussion

The purpose of this study was to determine the neural mechanisms which contribute to falls among individuals with chronic stroke. Two limitations of the study were (1) the smaller number of individuals who fell compared to individuals who did not fall and (2) the falls were generated in a laboratory. We felt it was preferable to use the entire sample rather than match the number of Non-fallers to Fallers. The proportion of fallers (i.e. 25%) within the entire sample agrees with proportions of fallers from other studies, which examine prospective community-based falls in individuals with chronic stroke (Jorgensen et al. 2002). Additionally, laboratory-generated falls provide more constant and reproducible perturbations, which allow for controlled comparisons across groups (i.e. Fallers versus Non-fallers). Future research needs to determine the relationship of laboratory-generated falls compared to those occurring in the community.

While rotational perturbations in healthy older adults have not been able to differentiate individuals who fall from those who do not using retrospective fall data (Smith et al. 1996), translational perturbations have demonstrated delayed TA postural reflex onset latency in older adults who have fallen at least once in the past year (Studenski et al. 1991). Injury to the CNS from cerebrovascular accident often has devastating consequences to sensorimotor function. Thus, characteristics of postural reflexes may be particularly important in the recovery responses for preventing a fall following an upper motoneuron lesion, as these individuals are inherently more unstable and closer to a threshold at which a fall is inevitable.

Our study suggests that the magnitude and onset latency of some lower extremity muscles in response to a forward platform translation while standing are neural mechanisms which discriminate as to whether a fall occurs or not during this perturbation. In addition, these neural mechanisms could even discriminate successful recovery and falls within consecutive trials within the same subject. Furthermore, the clinical impairment of decreased paretic and non-paretic lower extremity muscle strength increased the risk of falling in response to a platform translation.

3.5.1 Postural reflexes evoked from forward platform translations: contribution to falls

The present study has identified aspects of postural reflex magnitude and latency, which lead to falls in individuals with chronic stroke. Although others have reported delays in paretic
limb onset latency in response to platform perturbations (Berger et al. 1988; Dietz and Berger 1984; Di Fabio and Badke 1988; Di Fabio et al. 1986; Di Fabio 1987), none have attempted to relate this to falls.

One of the major differences between successful recovery and falls was the paretic TA postural reflex magnitude. Individuals with stroke often present with weak paretic ankle dorsiflexors resulting in 'foot-drop' during gait. Stroke typically affects the corticospinal tract fibres, which innervate and control predominantly distal lower limb muscles (Brouwer and Ashby 1992). Accordingly, in this study muscle strength assessment revealed severely reduced paretic limb ankle dorsiflexion strength in those who fell. We believe that stroke-specific impairments such as the poor TA postural response of the paretic limb contributes to the necessity of compensatory mechanisms such as that observed in the non-paretic hip. Di Fabio et al. (1986) have suggested that the non-paretic hip most likely compensates for paretic ankle muscle impairments in response to standing perturbations. Proximal muscle activation of the non-paretic hip (i.e. RF) likely compensated for the poor paretic TA activation. The importance of this compensation was evident by the Fallers who demonstrated a significant delay in non-paretic RF onset latency compared to Non-fallers.

It is then possible that once the centre of mass goes beyond the base of support (imminent fall), the individual attempts to recover with a late non-paretic hip response to enhance an ankle strategy, albeit too late. This compensatory strategy would be an a priori compensation due to past daily experiences or an interlimb coordination (Dietz and Berger 1984; Eng et al. 1994; Marigold et al. 2003) mediated by propriospinal pathways when an impaired ankle muscle response occurs.

There was no difference in onset latency for the non-paretic RF between fall trials and no-fall trials among the individuals who fell; however, this may be due to slight differences in the kinematic strategies employed on a trial-to-trial basis or may be due to Type II error associated with the large variability in the measure in this population.

The propensity to fall could potentially be related to the location of the stroke. For example, it has been suggested that vestibulospinal pathways regulate anterior leg (i.e. RF and TA) muscle postural reflexes (Allum et al. 1995) while corticospinal tract fibres have a greater influence over distal leg muscles such as TA (Brouwer and Ashby 1992). Additionally, extrapyramidal tract pathways such as the reticulospinal tract have differential control over leg muscles, which may be altered following stroke due to injury to the CNS and/or CNS plasticity during the acute recovery phase. Hemispheric side and location of stroke (cortical versus
subcortical) did not appear to be different between the Faller and Non-Faller group of this study (Table 3.1). However, larger samples of both groups are likely required, in addition to information regarding the integrity of individual tracts and infarct volume.

Therefore, there is a need to identify the specific neural pathways and supraspinal centres responsible for postural control following stroke. Specifically, do different neural pathways contribute to the compensatory strategies, such as the earlier non-paretic RF onset latency, or do individuals activate similar connections as prior to their stroke? Further, is the integrity of the tract used for these strategies differentially affected in those more prone to falls? Where are the residual deficits within the neural pathways (e.g. cortical, brainstem, spinal cord, peripheral nerve, neuromuscular junction, or muscle) that contribute to falls? Techniques such as transcranial magnetic stimulation to investigate motor output from the cortex or H-reflex testing to examine the integrity of the spinal reflex arc and spinal motoneuron excitability over the course of recovery and/or after an exercise intervention may provide clues to the answers to these questions. In addition, it may be useful during these investigations to separate individuals based on fall history and/or whether they utilize compensatory strategies for postural control.

Subsequently, rehabilitation clinicians would then be able to target these areas with specific exercises that require the neuronal connections associated with these pathways and centres. For example, agility-training programs may be beneficial to help elicit quick reflexive responses (Eng et al. 2003). Thus, collaborative efforts between basic and clinical scientists must be fostered to further advance our understanding of the underlying mechanisms associated with falling after an upper motoneuron lesion.

3.5.2 Muscle weakness contributes to falls

Both paretic and non-paretic muscle strength, particularly the ankle dorsiflexors and paretic knee extensors, contributed to falls with severe muscle weakness in the Faller group. Whipple et al. (1987) have shown that ankle dorsiflexor strength is particularly reduced in older adult fallers compared to non-fallers. While ankle strength is decreased in older age, the greater impairment in individuals with stroke compared to healthy older adults (Adams et al. 1990) may explain the increased risk of falling in this population.

The reduction in muscle strength in individuals with stroke may be attributed to the changes in muscle properties that follow brain injury. Studies in stroke have reported decreased type II muscle fibres (Dietz et al. 1986; McComas et al. 1973), decreased motor unit firing rates (Dietz
et al. 1986; Rosenfalck and Andreassen 1980), and decreased motor unit recruitment (Gemperline et al. 1995). Thus, individuals at risk for falls may have greater residual muscle impairments following their stroke than those with lower risk. However, a recent study by Landau and Sahrmann (2002) suggests that muscle weakness may not be due to the muscle tissue but rather a central mechanism, as maximal force of the tibialis anterior muscle elicited by electrical stimulation was similar between individuals with stroke and healthy controls.

### 3.5.3 Future directions and implications

In contrast to the older adult literature, balance, motor function, cognition, muscle strength, and activities of daily living impairment have not been able to predict falls in individuals with chronic stroke (Jorgensen et al. 2002; Forster and Young 1995; Lamb et al. 2003). Thus, identifying the neural mechanisms contributing to lab-induced falls is an alternative and promising approach to understanding falls in people with chronic stroke. The next step will be to examine the relationship of lab-induced falls to actual falls in the community. The results of this study suggest that rehabilitation programs designed to reduce falls in individuals with stroke may benefit from including functional exercises to improve muscle strength (particularly ankle dorsiflexors) and exercises that evoke postural reflexes (particularly the paretic and non-paretic ankle and non-paretic hip muscles). This in turn may improve the latency and magnitude of postural responses involved in recovery strategies. Ultimately, the identification of pathways utilized following brain injury may provide the necessary link for improving function and reducing falls in individuals with chronic stroke.

### 3.6 Bridging Summary

The results from the first and second studies demonstrate the importance of ankle dorsiflexor postural reflex magnitude for preventing falls in individuals with chronic stroke. Experiment II also demonstrated the importance of the postural reflex muscle onset latency and of volitional muscle strength, particularly with the paretic lower limb, for preventing falls. The question then becomes whether some type of intervention can alter postural reflexes. Additionally, can interventions in individuals with chronic stroke reduce falls and/or improve postural control?
Exercise interventions in individuals with chronic stroke have demonstrated improvements in physical function including balance (Bastien et al. 1998; Eng et al. 2003; Mudge et al. 2003; Tangeman et al. 1990; Weiss et al. 2000). None have investigated falls-reduction, nor are the types of exercises that are most effective known. Based on the early findings of our experiments, the known deficits in postural control and frequency of falls following stroke, and literature in healthy older adults, we designed two types of exercise interventions for individuals with chronic stroke. The following experiment was a randomized clinical trial to determine which exercise intervention was more effective in improving postural control and reducing falls.
CHAPTER 4 – Experiment III

Exercise Leads to Faster Standing Postural Reflexes and Better Functional Balance and Mobility in Persons with Chronic Stroke: A Randomized Clinical Trial
(In preparation for submission)

4.1 Summary

**Background:** Although falls and fall-related injuries in persons with chronic stroke are an enormous burden on both the individual and the health care system, the types of exercise programs that are most effective are unknown. Further, it is unknown whether interventions like exercise can alter neural circuitry, such as postural reflexes, which may be important for preventing falls. We aimed to determine the effect of two different community-based group exercise programs on functional balance, mobility, and standing postural reflexes in persons with chronic stroke.

**Methods:** We screened 109 persons with chronic stroke, of which 61 were randomized into one of two exercise groups (Agility or Stretching/weight-shifting). Persons were assessed prior to, immediately after, and one-month following a 10-week exercise intervention for clinical outcome measures including Berg Balance (primary outcome measure), Timed Up and Go, step reaction time, Activities-specific Balance Confidence, and Nottingham Health Profile. In addition, neurophysiological testing of standing postural reflexes evoked by a translating platform was also performed. Analyses were by intention-to-treat.

**Findings:** For both groups, exercise led to improvements in all clinical outcome measures. In addition, this was the first time it has been shown that exercise leads to faster paretic lower extremity postural reflexes. The Agility group demonstrated greater improvement in step reaction time and paretic rectus femoris postural reflex onset latency compared to the Stretching/weight-shifting group. Although there was no difference in the number of fallers between groups when the entire sample was included, a sub-analysis of those with a history falls demonstrated a reduction in the number of fallers in the Agility exercise group.
Interpretation: Community-based group exercise programs are effective in improving functional balance and mobility and lead to faster standing postural reflexes in persons with chronic stroke.

4.2 Introduction

One of the most devastating consequences of having a stroke is the increased risk of falling and consequent fall-related injuries. Twenty-three to 73% of community-dwelling persons with chronic stroke have been reported to fall over a 4-12 month period with approximately half falling repeatedly (Forster and Young 1995; Hyndman et al. 2002; Jorgensen et al. 2002) and there is a greater than seven-fold increase in hip fracture risk in this population (Kanis et al. 2001). Stroke-related impairments such as muscle weakness, sensorimotor dysfunction, and balance problems presumably contribute to the large number of falls. One potential way of improving balance and reducing falls is through exercise interventions.

A number of recent studies have demonstrated that exercise can improve mobility (Ada et al. 2003; Dean et al. 2000; Rodriguez et al. 1996; Silver et al. 2000; Sullivan et al. 2002; Teixeira-Salmela et al. 1999) and functional balance (Eng et al. 2003; Tangeman et al. 1990) in persons with chronic stroke. However, it is unclear what are the advantages to different types of exercise programs (i.e. specific exercises) and the mechanisms, which underlie their improvements.

Postural reflexes, in the form of coordinated muscle activity, are the first line of defence against falling subsequent to an unexpected destabilizing force (i.e. perturbation) applied to the body (e.g. collision, slip, and trip) or from self-induced movements (e.g. reaching, transferring). Persons with stroke have delayed paretic limb postural reflex muscle onset latencies compared to healthy older adults in response to unexpected perturbations during standing (Berger et al. 1988; Di Fabio et al. 1986). It is unknown whether exercise can alter the latency of postural reflexes, which could lead to improved postural control and a reduction in falls. Such a concept would support an emerging idea of the brain’s ability to adapt in terms of structural/neural changes (i.e. brain plasticity) in persons with chronic stroke (Nudo et al. 2001).

Therefore, the purpose of this study was to determine the effect of two different community-based group exercise programs (a fast-paced, multi-sensory, agility versus a slow-paced,
stretching/weight-shifting program) on functional balance, mobility, and standing postural reflexes in persons with chronic stroke.

4.3 Methods

4.3.1 Participants

Participants living in the community were recruited over a two-month period from the GF Strong Rehab Centre database, community stroke groups, and via advertisements in local community centres, newspapers and television (see Appendix XIV). Inclusion criteria to participate in the study included (1) age ≥ 50 years, (2) single stroke at least one year previously, (3) able to follow two-step commands, (4) able to walk, with or without an assistive device, for a minimum of 10 meters and have an activity tolerance of 60 minutes with rest intervals, and (5) not participating in any formal therapy programs. Exclusion criteria were (1) not medically stable (e.g. congestive heart failure, uncontrolled hypertension), (2) significant musculoskeletal or other neurological conditions not related to stroke, (3) a score of < 22 (unless language was a problem in which lower scores were re-evaluated) on the Mini-Mental State exam (see Appendix XV) (Folstein et al. 1975), and (4) a Berg Balance score over 52/56, as this indicates minimal balance deficits. Following university and hospital ethics approval, written informed consent was received from all participants prior to their participation (see Appendix XII). The participant’s physician confirmed the presence of stroke and the inclusion/exclusion criteria (see Appendix XVI). In addition, type, location, and onset of stroke were collected through medical records and/or physician information where available.

4.3.2 Study design

This study was a randomized-stratified, clinical intervention trial. Participants completed an initial screening assessment prior to participation in the intervention, which assessed six-month fall history, balance (Berg Balance), and cognition/dementia (Mini-Mental State exam). Participants were then randomly assigned alphanumeric codes through a random number generator program that allowed an unbiased randomization/stratification process. Participants
were stratified (Tate et al. 1999) into groups for factors of (1) functional balance (Berg Balance score < 40 or ≥ 40) and (2) number of falls (< 2 falls or ≥ 2 falls recalled over the past six months). Subsequently, a person independent (and blinded) of the study randomly assigned participants (using their alphanumeric codes) such that there were equal numbers of participants for each level of stratum in the two exercise groups. Participants knew they were in one of two exercise groups but were unaware of the differences between them. Exercise instructors were not aware of the outcome measures of the study. All testers of the clinical measures were blinded to the group assignment, study design, and purpose. Some of the spotters during the standing postural reflex assessment were aware of the group assignment, but not the purpose or outcome measures, and in addition, the data collection was driven by a computer system.

4.3.3 Intervention

The two exercise programs consisted of 1 hour sessions, 3x/week for 10-weeks held at a local community centre. Three instructors (physical therapist, kinesiologist, and recreation therapist) supervised the exercise programs. There were six classes (three for each of the exercise programs) with an approximate 1:3 instructor to participant ratio.

The Agility exercise program consisted of dynamic functional movement tasks (see Appendix XVII) with emphasis on multi-sensory training including standing on foam (with eyes open and closed and with one foot or both feet), sit-to-stand movements, rapid stepping, tandem walking, walking on foam, obstacle courses, standing perturbations (i.e. instructor pushing participant in a controlled manner or vice versa), and other exercises to challenge functional balance. Exercises in this program were designed so that tasks were progressively increased in difficulty on an individual basis.

The Stretching/weight-shifting exercise program consisted of slow movement tasks (see Appendix XVII) including Tai Chi-like movements, standing weight-shifting tasks, and stretching both while standing or sitting and while on mats on the floor.

4.3.4 Outcome measures

Participants were evaluated three times: before the intervention (baseline), at the end of the intervention (post-intervention), and one-month following (retention). For each of these time
periods, participants were assessed on two occasions separated by approximately two days. In one session, clinical measures including functional balance and mobility were assessed along with balance-confidence and health-related quality of life. In the other session, standing postural reflexes and step reaction time were assessed.

The Berg Balance Scale (Berg et al. 1989, 1992) was used to assess functional balance and has established validity and reliability in several different populations including stroke. It consists of 14 balance-related tasks (such as stepping, reaching, and turning) each scored on a 4-point scale (max. 56 points) (see Appendix V). The Timed Up and Go test (Podsiadlo and Richardson 1991) was used to assess functional mobility and measures the time to stand up from an arm chair, walk a distance of 3 m, turn, and walk back to the chair and sit down again.

Balance confidence and health-related quality of life were measured using the Activities-specific Balance Confidence (ABC) Scale (Powell and Myers 1995) and Nottingham Health Profile (NHP), respectively. The ABC (see Appendix VII) is a 16-item self-report questionnaire that asks individuals to rate their balance confidence in performing specific functional activities on a scale (where 100 represents complete confidence). The NHP (see Appendix VI) is a 38-item questionnaire (low scores represent higher quality of life) that has demonstrated reliability in persons with stroke (Visser et al. 1995).

To assess standing postural reflexes, a total of 20 platform translations (8 cm displacement, 30 cm/s velocity, and 300 cm/s² acceleration), separated by 15-30 second intervals, were induced while participants stood on two force plates (Bertec Corp.) embedded in a custom built platform (see Appendix XIII for experimental protocol). Participants wore a full-body harness attached to a ceiling beam via a dynamic rock-climbing rope to prevent the occurrence of a fall to the ground with at least one spotter present. Participants were told that the platform could move at any time but the onset and direction of the translation were unexpected in nature. The direction of the translation was counterbalanced across participants so that either 10 consecutive backward translations followed 10 consecutive forward translations or vice versa. The first trial from each direction was discarded from the analysis, as the first trial to an unexpected perturbation is different than subsequent ones (Marigold and Patla 2002).

Surface electromyography (EMG) (Bortec) from bilateral tibialis anterior (TA), medial head of gastrocnemius (MG), rectus femoris (RF), and biceps femoris (BF) were recorded at 600 Hz for 6 seconds (2 seconds prior to platform movement and 4 seconds after) along with force plate data (see Appendix X for EMG placement protocol). The TA and RF muscles were analyzed for the forward platform translations while MG and BF were analyzed for the backward
translations due to their roles in the primary recovery response to those translation directions (Horak and Nashner 1986). All EMG data processing used custom written software. EMG was full-wave rectified and low-pass filtered at 100 Hz and the mean signal for one second prior to the onset of platform movement was determined along with the SD. Muscle onset latency, representing a postural reflex, was defined as an increase in muscle activity that exceeded \( +2 \) SD for at least 30 msec and was determined by a combination of computer algorithm and visual inspection via an interactive program.

In order to determine whether muscle onset latencies could be reliably measured, test re-test reliability was assessed (using Intraclass Correlation Coefficients [ICC] and standard error of measurement [SEM]) using ten persons with stroke (tested on two separate occasions within seven days). ICCs ± SEMs (msec) for the paretic TA, RF, and MG demonstrated moderate to high reliability (0.92 ± 9.2, 0.87 ± 11.2, 0.79 ± 9.9, respectively), as did the non-paretic TA, RF, and MG (0.79 ± 4.3, 0.79 ± 14.1, and 0.67 ± 4.4, respectively). In addition, no learning effect was observed as evident from non-significant F-tests over the two days (see Appendix IV).

Reaction time was assessed using a simple step reaction time task. Participants stood on the platform looking forward and were instructed to step forward with the specified lower limb as fast as possible following an auditory cue. A total of five trials were performed for this task, where the first two trials and the last two trials were with the non-paretic limb while the middle trial was with the paretic limb in order to prevent any standing postural bias adopted throughout the repeated trials. Only data from the non-paretic limb was recorded because initial pilot work uncovered a tendency for individuals with stroke to step with this particular limb in recovery responses to platform translations. Reaction time, averaged over the four non-paretic limb trials, was defined as the time between the auditory cue and the time when the vertical force from the force plate reached zero due to the foot lifted off the ground.

A self-report falls diary was kept by participants and returned via mail to our research lab on a monthly basis over one year from the start of the intervention. If participants did not return the monthly diary, an experimenter phoned to remind them. Falls were recorded on a weekly basis (excluding any in the exercise classes) during the 10-week intervention period. A fall was defined as unintentionally coming to rest on the floor or another lower level but not due to seizure, stroke/myocardial infarction, or an overwhelming displacing force (e.g. earthquake).
4.3.5 Statistical analysis

Based on an average Berg Balance score (primary outcome measure) of 45.3 with a SD of 5.65 (Eng et al. 2003) and a desired 5-point change, a sample size of 21 persons per exercise group would have 80% power with a p < 0.05. Thus, thirty persons per group were sought to account for dropouts. Data were analyzed on an intention-to-treat basis. Baseline descriptive variables between the exercise groups were compared using chi-square (gender, affected limb), Mann-Whitney U (age, stroke duration), Median (AHASFC), or independent t (height, mass) tests. Outcome measures were tested for normality and, when applicable, were either log (Berg Balance, Timed Up and Go, step reaction time) or rank (NHP) transformed for subsequent analysis. Baseline outcome measures between the exercise groups were compared using independent t-tests.

Three separate repeated measures multivariate analyses of variance (MANOVA) were performed to compare the outcome measures of the two exercise groups (Group: Agility versus Stretching/weight-shifting) and at the three assessment times (Time: baseline, post-intervention, retention) to control for Type I error associated with multiple statistical tests. The first MANOVA included the clinical outcome measures: (1) Berg Balance, (2) Timed Up and Go, (3) step reaction time, (4) ABC, and (5) NHP. The second MANOVA included the paretic limb postural reflex muscle onset latencies for the: (1) TA, (2) RF, (3) MG, and (4) BF, and the third MANOVA included the non-paretic limb postural reflex muscle onset latencies for the: (1) TA, (2) RF, (3) MG, and (4) BF. Following a significant MANOVA, a two-way (Group and Time) repeated measures analysis of variance (RM ANOVAs) and, if applicable, Duncan’s post-hoc tests for a Time effect were also performed (SAS 8.2, SAS Institute Inc.). A covariate (baseline scores) was included in the RM ANOVAs when baseline differences were significant for a particular variable.

The number of falls for each participant was normalized to the number of months over which information was collected. Subsequently, the number of falls/month over the course of one year from the start of the intervention (excluding any falls during the exercise classes) for each group was compared using a Mann-Whitney U test. Additionally, the number of fallers and number of repeat fallers (≥ 2 falls) over the same time period were compared between the two groups using Chi-square tests.

A significance level of p < 0.05 was selected for all statistical analyses.
4.4 Results

4.4.1 Participant characteristics

We identified 109 potential participants between July and September 2002. We excluded 48 participants: 35 did not meet the inclusion/exclusion criteria, 2 refused to participate, and 11 could not obtain physician approval, make the exercise class times, or were planning an extended vacation during the assessment times and/or intervention. Thus, 61 persons with stroke were recruited and underwent stratification/randomization to be placed into one of the two exercise programs: 31 into the Stretching/weight-shifting and 30 into the Agility program. Two individuals discontinued the study due to time commitment issues from the Agility program prior to baseline assessment. A total of 11 individuals discontinued the intervention or were unable to attend post-intervention assessment due to time commitment reasons (n = 2), hip fracture (n = 1, during a non-challenging task in the Agility program), illness (n = 5), or personal reasons (n = 3). Six participants were lost at retention testing due to illness (n = 2), vacation (n = 3), or personal reasons (n = 1). Figure 4.1 summarizes the trial profile. The mean (SD) percent of exercise classes attended for the Stretching/weight-shifting and Agility groups were 94.4 % (5.5) and 92.6 % (10.4), respectively.

Table 4.1 describes the participant characteristics for both exercise programs. There were no differences between exercise groups for baseline descriptive variables (p > 0.2).

4.4.2 Clinical outcome measures

With the exception of step reaction time (p = 0.01), there were no baseline differences in the clinical outcome measures (p > 0.2, except ABC p = 0.09). Thus, baseline values for step reaction time were entered as a covariate. The MANOVA demonstrated an overall Group by Time interaction (Wilk's $\lambda$ = 0.76, p = 0.04), Time main effect (Wilk's $\lambda$ = 0.33, p < 0.0001), but no Group main effect (Wilk's $\lambda$ = 0.83, p = 0.18). Step reaction time was decreased in the Agility exercise group to a greater extent than the Stretching/weight-shifting group following the intervention (Table 4.2). There was also a trend for a Group by Time effect for the Timed Up and Go test (Table 4.2). All clinical measures showed improvements after the intervention, which with the exception of step reaction, were retained at follow-up (Table 4.2).
109 persons screened for eligibility

48 persons were excluded

61 persons were randomly assigned to one of the two exercise groups

30 persons assigned to Agility exercise program
31 persons assigned to Stretching/weight-shifting exercise program

2 persons declined to participate

28 persons assessed at baseline
22 persons assessed post-intervention
19 persons assessed for one-month retention

31 persons assessed at baseline
26 persons assessed post-intervention
23 persons assessed for one-month retention

2 persons declined to participate

28 persons assessed at baseline
22 persons assessed post-intervention
19 persons assessed for one-month retention

31 persons assessed at baseline
26 persons assessed post-intervention
23 persons assessed for one-month retention

Figure 4.1: Trial profile.
Table 4.1: Participant characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Stretching/weight-shifting (n = 26)</th>
<th>Agility (n = 22)</th>
<th>Dropouts (n = 11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender, M/F</td>
<td>18 (69) / 8 (31)</td>
<td>17 (77) / 5 (23)</td>
<td>6 (55) / 5 (45)</td>
</tr>
<tr>
<td>Age, yrs</td>
<td>67.5 (7.2)</td>
<td>68.1 (9.0)</td>
<td>69.6 (10.8)</td>
</tr>
<tr>
<td>Height, cm</td>
<td>168.9 (8.9)</td>
<td>171.0 (9.4)</td>
<td>169.7 (12.9)</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>78.4 (15.9)</td>
<td>83.5 (17.7)</td>
<td>76.3 (16.3)</td>
</tr>
<tr>
<td>Stroke Duration, yrs</td>
<td>3.8 (2.4)</td>
<td>3.6 (1.8)</td>
<td>4.1 (5.7)</td>
</tr>
<tr>
<td>Affected Side, R/L/NA</td>
<td>8 (31) / 18 (69) / 0 (0)</td>
<td>10 (45) / 11 (50) / 1 (5)</td>
<td>7 (64) / 1 (9)</td>
</tr>
<tr>
<td>AHASFC, 1 - 5</td>
<td>2.5 (2 - 3)</td>
<td>2.0 (1 - 3)</td>
<td>3.0 (2.5 - 3)</td>
</tr>
<tr>
<td># of Fallers (retrosp.)</td>
<td>15 (58)</td>
<td>15 (68)</td>
<td>6 (55)</td>
</tr>
</tbody>
</table>

**Stroke Location**

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortical</td>
<td>10 (39)</td>
<td>4 (18)</td>
<td>2 (18)</td>
</tr>
<tr>
<td>Subcortical</td>
<td>8 (31)</td>
<td>7 (32)</td>
<td>2 (18)</td>
</tr>
<tr>
<td>Brainstem/cerebellum</td>
<td>4 (15)</td>
<td>6 (27)</td>
<td>3 (27)</td>
</tr>
<tr>
<td>Cortical-subcortical</td>
<td>0</td>
<td>0</td>
<td>1 (9)</td>
</tr>
<tr>
<td>Unknown</td>
<td>4 (15)</td>
<td>5 (23)</td>
<td>3 (27)</td>
</tr>
</tbody>
</table>

Values are (1) mean (SD) for age, height, and mass, (2) are number (%) for gender, affected side, and # of Fallers, and (3) are median (IQR) for AHASFC.

**Abbreviations:** R = right; L = left; NA = not applicable; AHASFC = American Heart Association Stroke Functional Classification; retrosp. = 6-month retrospective data
4.4.3 Muscle onset latencies

The paretic RF and non-paretic MG demonstrated baseline differences between exercise groups (p = 0.03 and 0.04, respectively) while the remaining muscles' baseline measures were not different (p > 0.2, except non-paretic BF p = 0.08). Thus baseline values for the paretic RF and non-paretic MG were entered as a covariate for their respective analyses. The MANOVA for the paretic limb muscle onset latencies demonstrated an overall Group by Time interaction (Wilk’s \( \lambda = 0.68 \), p = 0.05), Time main effect (Wilk’s \( \lambda = 0.50 \), p = 0.0004), but no Group main effect (Wilk’s \( \lambda = 0.87 \), p = 0.31). The paretic RF onset latency was significantly faster by 27.5 msec following the Agility exercise program compared to 11 msec following the Stretching/weight-shifting exercise program. Onset latencies were faster in all paretic muscles ranging between 4.7 and 27.5 msec (Figure 4.2). Changes in latency were not due to different recovery strategies employed, as muscle sequencing was similar in all test sessions. Table 4.3 summarizes the results of the interventions on the standing postural reflex muscle onset latencies.

The MANOVA for the non-paretic limb did not show a Group by Time interaction (Wilk’s \( \lambda = 0.84 \), p = 0.21) or Group main effect (Wilk’s \( \lambda = 0.86 \), p = 0.20), but did show a Time main effect (Wilk’s \( \lambda = 0.64 \), p = 0.0008). Of the non-paretic musculature, only the RF showed faster onset latency over time.

4.4.4 Falls

Table 4.4 illustrates the number of falls and fallers in the two exercise groups over one year following the start of the intervention. No significant differences were observed (p > 0.05). A sub-analysis on those who fell prior to the intervention (15 in each exercise group) using a Chi-square test revealed that only eight continued to fall in the Agility group compared to 13 in the Stretching/weight-shifting group (p = 0.046).
Figure 4.2: Changes in paretic limb postural reflex muscle onset latencies following the exercise intervention. A typical filtered EMG profile (sample from one participant within the Agility group) demonstrating the faster postural reflexes with exercise training. The solid thick line represents the postural reflex during baseline testing and the dashed line represents the postural reflex during post-intervention testing.
Table 4.2: Changes over time with clinical measures for both exercise groups.

<table>
<thead>
<tr>
<th>Outcome Measure</th>
<th>Stretching/weight-shifting Group (N = 26)</th>
<th>Agility Group (N = 22)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Post-intervention</td>
<td>Retention</td>
</tr>
<tr>
<td>Berg Balance, max. 56</td>
<td>44.8 (7.1)</td>
<td>48.1 (5.7)</td>
<td>47.5 (6.0)</td>
</tr>
<tr>
<td>Timed Up &amp; Go, sec</td>
<td>18.4 (13.1)</td>
<td>17.0 (10.7)</td>
<td>17.5 (11.0)</td>
</tr>
<tr>
<td>Step Reaction Time, msec*</td>
<td>590 (171)</td>
<td>540 (144)</td>
<td>659 (175)</td>
</tr>
<tr>
<td>ABC, %</td>
<td>58.0 (21.2)</td>
<td>68.3 (19.4)</td>
<td>64.8 (20.0)</td>
</tr>
<tr>
<td>NHP, max. 38</td>
<td>10 (6.4)</td>
<td>7.9 (8.0)</td>
<td>8.7 (7.8)</td>
</tr>
</tbody>
</table>

Data are mean (SD).

* Baseline differences between exercise groups, p < 0.05.
† Post-intervention and retention different than baseline assessment
‡ Post-intervention different than baseline and retention assessment
Table 4.3: Effects of the exercise interventions on postural reflex muscle onset latencies.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Stretching/weight-shifting Group (N = 26)</th>
<th>Agility Group (N = 22)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Post-intervention</td>
<td>Retention</td>
</tr>
<tr>
<td>Paretic TA</td>
<td>115.7 (18.8)</td>
<td>109.5 (15.9)</td>
<td>115.9 (18.4)</td>
</tr>
<tr>
<td>Paretic RF*</td>
<td>140.3 (32.2)</td>
<td>129.3 (26.6)</td>
<td>138.0 (28.4)</td>
</tr>
<tr>
<td>Paretic MG</td>
<td>130.0 (33.7)</td>
<td>117.7 (18.0)</td>
<td>120.2 (18.3)</td>
</tr>
<tr>
<td>Paretic BF</td>
<td>170.9 (37.8)</td>
<td>164.6 (21.5)</td>
<td>156.7 (33.6)</td>
</tr>
<tr>
<td>Non-paretic TA</td>
<td>107.1 (13.0)</td>
<td>105.1 (11.3)</td>
<td>106.5 (14.5)</td>
</tr>
<tr>
<td>Non-paretic RF</td>
<td>139.6 (33.0)</td>
<td>134.5 (26.5)</td>
<td>129.0 (23.4)</td>
</tr>
<tr>
<td>Non-paretic MG*</td>
<td>109.1 (15.3)</td>
<td>109.2 (20.5)</td>
<td>107.6 (14.0)</td>
</tr>
<tr>
<td>Non-paretic BF</td>
<td>149.9 (30.7)</td>
<td>145.7 (22.7)</td>
<td>152.7 (31.5)</td>
</tr>
</tbody>
</table>

Data are mean (SD) in msec.

* Baseline differences between exercise groups, p < 0.05.
† Post-intervention different than baseline and retention assessments
‡ Three time periods different than each other
§ Post-intervention different than baseline assessment
¶ Retention different than baseline assessment
|| Retention different than baseline and post-intervention assessments
Table 4.4: Fall data over one year from the start of the exercise interventions for the Stretching/weight-shifting and Agility groups.

<table>
<thead>
<tr>
<th>Fall Measure</th>
<th>Stretching/weight-shifting (N = 26)</th>
<th>Agility (N = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td># of Total Falls</td>
<td>75</td>
<td>25</td>
</tr>
<tr>
<td># of Falls/Month/Person</td>
<td>0.26</td>
<td>0.10</td>
</tr>
<tr>
<td># of Fallers</td>
<td>16</td>
<td>11</td>
</tr>
<tr>
<td># of Repeat Fallers</td>
<td>11</td>
<td>7</td>
</tr>
</tbody>
</table>

4.5 Discussion

Regardless of the type, exercise training improved functional balance and mobility, led to faster standing paretic limb postural reflex muscle onset latencies, and resulted in greater balance confidence and health-related quality of life in persons with chronic stroke. In addition, this study showed that a fast-paced, multi-sensory, agility exercise program results in greater improvements in step reaction time and paretic RF postural reflex onset latency in persons with chronic stroke compared to a slow-paced, stretching/weight-shifting exercise program.

This was only the second study to examine the retention effects of a community-based group exercise program: our results and those of Eng et al. (2003) suggest that the exercise effects are maintained for at least one month. It is imperative to develop effective community-based exercise programs to offset earlier acute care discharge. The group aspect of the programs enhances social contact, which is important considering 20% of this population suffers from depression (Jorgensen et al. 2002).

The task-specific nature of the Agility exercise program may have contributed to the greater improvements in postural control. For example, the vestibular stimulation in this multi-sensory program may have contributed to the faster paretic RF onset latency as it strengthened those descending pathways responsible for proximal limb control (Allum et al. 1995). However, the weight-shifting components of the Stretching/weight-shifting program may have aided in the improvements of many of the clinical outcome measures. Further, for many individuals getting'
to the floor for mat exercises was a major challenge, as it was the first time they had performed this task since their injury.

This is the first time postural reflex muscle onset latency has been shown to change with exercise. For example, the postural reflex onset latency of the paretic RF was over 27 msec faster post-intervention in the Agility exercise group and this magnitude is well outside the SEM on a repeated test. Further, this is approximately the latency of the monosynaptic stretch reflex for this muscle (Bergui et al. 1992) and is highly suggestive of functional significance when coupled with the concomitant increase in functional balance.

The behavioural and neurophysiological changes in this study are most likely the result of neuronal circuitry remodelling. Exercise has been suggested to promote this brain plasticity through mechanisms such as increased expression of brain-derived neurotrophic factor (Cotman and Berchtold 2002; Gomez-Pinilla et al. 2001). Additionally, exposure to enriched rehabilitative training has demonstrated that increased dendritic arborisation accompanies increased motor performance in rats (Biernaskie and Corbett 2001). In humans, forced-use of the paretic limb in persons with chronic stroke has shown cortical reorganization and improved motor performance (Liepert et al. 2000). It has been suggested that disinhibition of gamma-aminobutyric acid neuron activity with a concomitant enhancement of N-methyl-D-aspartate receptor activation may allow plastic changes within the human brain (Bütefisch et al. 2000; Ziemann et al. 2001).

Given our results, we suggest that exercise programs in persons with chronic stroke should include balance training, with emphasis on multi-sensory tasks. The reduced number of falls following the Agility exercise program compared to the Stretching/weight-shifting program is encouraging, although larger studies are required to investigate this finding, as this trial was powered for balance rather than falls. In addition, the relationship of faster standing postural reflexes to falls-reduction warrants further study.

4.6 Platform-induced falls

Falls that occurred during the platform translations for baseline, post-intervention, and retention testing were recorded. The results are shown in Appendix XVIII; however, no statistical analyses were performed.
CHAPTER 5 - Conclusions and General Discussion

5.1 General Findings

The purpose of this thesis was (1) to understand how individuals with chronic stroke modulate postural control and determine the underlying neural mechanisms contributing to falls and (2) to determine the effects of two different exercise interventions on postural control and physical function.

The results of the three studies collectively contributed to four main conclusions. First, the control of the paretic ankle dorsiflexors (i.e. tibialis anterior) is severely impaired in individuals with chronic stroke, which along with delayed non-paretic rectus femoris postural reflex onset latency contributes to falls in this population. We propose that deficits in supraspinal control are responsible for these findings. Second, an Agility exercise program is more beneficial than a Stretching/weight-shifting program, although both demonstrate improvements. Third, community-based group exercise programs for individuals with chronic stroke are effective and methods should be explored for their implementation within the community. And fourth, exercise in individuals with chronic stroke induces neural plasticity. The discussion to follow expands on these conclusions.

5.2 Evidence of altered supraspinal control contributing to impaired postural control in stroke

Reactive postural control entails a rapid coordinated muscular response (i.e. postural reflexes), which is evoked following an unexpected destabilizing event (or perturbation) such as a slip, trip, or collision (Marigold and Patla 2002; Patla 2003). If the central nervous system (CNS) does not react with sufficient speed or strength a fall may occur. In our experiments we utilized a servomotor driven translating platform to elicit standing postural reflexes. Postural control is clearly impaired following brain injury, such as stroke, and we have shown that this is a contributing factor for falls in this population. For example, we demonstrate in all three experiments that the latency of paretic lower limb postural reflexes is delayed in individuals with chronic stroke following unexpected perturbations while standing.
We propose central mechanisms are responsible for the impaired postural control observed following stroke. Specifically, deficits in supraspinal control contribute, in large part, to postural control dysfunction in stroke survivors. Postural control mechanisms require (1) the detection of external stimuli and (2) transmission of this afferent input to spinal cord, brainstem, subcortical, and cortical regions through neuronal circuits. Supraspinal centres can then contribute to the response by, for example, modulating reflex gain or selecting an appropriate muscle synergy. Stroke may affect the detection or transmission of afferent information or alternatively, disrupt cognitive processing centres. Several converging lines of research lend support that the latter is more detrimental and responsible for impaired postural control.

5.2.1 Peripheral mechanisms contribute minimally to the impaired postural control following stroke

Our first experiment demonstrated load-dependent modulation of ankle extensor muscles (i.e. gastrocnemius) in response to backward platform translations during different standing weight-bearing load conditions. Dietz and colleagues (Dietz et al. 1992) argue what has already been suggested in cats (Duysens and Pearson 1980; Pearson and Collins 1993) that golgi tendon organs (GTO) within extensor muscles contribute to load-dependent modulation. Thus, GTO function appears to be intact following stroke. In addition, Wilson et al. (1999) have recently shown muscle spindle activity in individuals with stroke is similar to healthy controls suggesting that muscle spindle function remains intact after stroke as well. Muscle spindle function is important for recovering after platform translations, as these perturbations are thought to be somatosensory-triggered (Inglis et al. 1994) and the stretch of ankle musculature in response to the translations would evoke spindle activity.

Several muscle property changes have been observed following stroke including a decreased number of motor units (Dietz et al. 1986), disturbed motor unit recruitment (Gemperline et al. 1995), decreased motor unit firing rates (Dietz et al. 1986; Rosenfalck and Andreassen 1980), and delayed time to generate peak torque (McCrea et al. 2003). Furthermore, motor conduction velocities to paretic leg muscles are slower, albeit in the range of approximately 3-4 msec, than non-paretic motor conduction velocities (Cruz-Martinez 1983). However, a recent investigation in individuals with stroke (Landau and Sahrmann 2002) demonstrated that maximal voluntary muscle contraction elicited by electrical stimulation was no different than healthy age-matched controls, which led these authors to suggest that muscle weakness may stem from central
impairments (such as corticospinal tract involvement) rather than a problem with the muscle tissue itself. Additionally, Chae et al. (2002) have recently shown in individuals with chronic stroke that the time to initiate a muscle contraction in the paretic limb following an auditory cue (a task which would require cortical processing) is delayed compared to the non-paretic limb.

5.2.2 Supraspinal deficits are responsible for the impaired postural control following stroke

In order to initiate a postural response, the CNS integrates incoming sensory and environmental information. This process requires sufficient cognitive processing resources and thus, is attention demanding (Redfern et al. 2001). Attention relies on a complex interaction of many cortical, subcortical, and brainstem areas (Banich 1997). Our hypothesis that deficits in supraspinal control contribute to the impairment in postural control following stroke is supported by several recent findings in studies dealing with cognitive processing. First, individuals with chronic stroke do suffer from permanent cognitive impairments (Hochstenbach et al. 2003). Second, Hyndman and Ashburn (2003) have recently reported that attention deficits correlate with falls in individuals with chronic stroke. Third, using a dual-task paradigm in individuals with chronic stroke, Brown et al. (2002) demonstrated increased reaction time (verbal response to a visual stimulus) during various postural tasks compared to healthy older adults. Fourth, individuals in the acute phase of stroke who demonstrate slower reaction time of finger tapping following a visual stimulus are at higher risk for falls than those individuals with acute stroke who have faster reaction time (Mayo et al. 1990). Fifth, step reaction time is delayed in individuals who fall in response to platform perturbations versus those who do not (unpublished observations). Sixth, sensory integration is impaired following stroke compared to healthy older adults, as demonstrated by increased postural sway and falls during manipulation of ankle proprioception and visual cues while standing (unpublished observations). Lastly, premotor time but not mean motor time for an ankle dorsiflexion task with the paretic leg was delayed compared to that in the non-paretic limb, supporting the notion that stroke affects central, premotor time processing centres rather than peripheral deficits (Smith et al. 1998a).

The lack of cognitive resources for postural control duties may be the result of increased attention to the hemiparetic side. In other words, in order to function, individuals with stroke devote a large amount of conscious effort in ensuring stability in the face of sensorimotor
dysfunction on one side of their body. In particular, the compensatory strategies adopted over the course of recovery from brain injury may require extra resources.

Therefore, deficits in the supraspinal control over postural reflexes following stroke likely contributed to the results observed in Experiment II. Specifically, these deficits likely contributed to the delay in non-paretic rectus femoris latency, attenuated tibialis anterior postural reflex magnitude, and reduced volitional muscle strength (assessed through an isokinetic dynamometer) in individuals with stroke who fell in response to platform translations.

5.2.3 Implications for rehabilitation

What then does this mean for rehabilitation? Clinicians treating individuals with stroke and those involved in developing exercise protocols must ensure that supraspinal centres are stimulated. This may be accomplished through multi-sensory training (as performed in Experiment III with great success) whereby the brain would be required to integrate multiple sources of sensory information. This concept is discussed in more depth in later sections.

Additionally, the use of dual-task paradigms may be beneficial. Here, two tasks are performed simultaneously, competing for attention and processing resources. This may include walking or standing in various challenging postures while counting or performing arithmetic, object recognition during challenging balance tasks, or even having individuals perform a circuit in which obstacle avoidance is required. The results of obstacle course training in exercise programs for individuals with chronic stroke are promising (Ada et al. 2003; Bassile et al. 2003).

5.3 Exercise interventions for individuals with chronic stroke

There is an urgent need to develop safe and effective community-based group exercise programs for individuals with chronic stroke to offset earlier discharge from a rehabilitation setting and to maintain and/or improve functional balance and mobility. Further, these programs can help reduce sedentary lifestyle and secondary complications as well as improve quality of life. Barnett et al. (2003) have recently shown that a community-based group exercise program in healthy older adults results in improved balance and a reduction in falls over one year. The benefits of a community-based group exercise program in individuals with chronic
stroke have also been reported (Ada et al. 2003; Bassile et al. 2003; Batien et al. 1996; Dean et al. 2000; Eng et al. 2003; Rimmer et al. 2000; Teixeira-Salmela et al. 1999, 2001).

5.3.1 Program adherence, safety, and exercise instructors

Although the attrition rate for the exercise intervention (Experiment III) was 18.6%, the average attendance for the Agility exercise group was 92.6% (range 51.7 - 100%) and for the Stretching/weight-shifting exercise group was 94.4% (range 82.8 - 100%). Thus, program adherence was excellent for those who remained in the interventions. The most common reason for discontinuing the intervention was illness. These results are encouraging as the Stretching/weight-shifting program was minimally challenging and although it was difficult to keep the high functioning individuals challenged our instructors did an excellent job to keep them entertained. In addition, the Agility program was highly challenging and low functioning individuals still attended and gave their best. This latter aspect may have been facilitated by the fact that exercise difficulty was graded on an individual basis.

Adherence in both exercise programs may also have been due to the collegiality that developed among participants. The programs were an excellent way for the participants to interact with one another and connect with individuals with similar impairments. The finding that perceived health-related quality of life, assessed by the Nottingham Health Profile, was improved for both exercise groups supports this. Therefore, both exercise programs appear to be feasible for maintaining participation and improving quality of life, presumably from a combination of social interaction and the perception of improved physical function.

The class length (one hour sessions) and frequency (3x/wk) seemed to have worked out well. Bastien et al. (1998) investigated class length and frequency of exercise programs and found that moderate improvements were noticeable even with a 90-minute class once per week or two 60-minute classes per week. However, to show meaningful improvement in balance we recommended that the length and frequency be maintained as in Experiment III.

Safety is a major concern when running an exercise program, particularly with individuals at high risk for falls and fall-related injuries. The 3:1 instructor-to-client ratio was chosen based on a previous study by Eng et al. (2003). This ratio proved to be sufficient for both exercise groups. We do not recommend this ratio being adjusted especially if agility exercises are being performed. Since exercises were implemented in a graded fashion on an individual basis for the Agility program, a large number of clients did not perform all types of exercises. For example,
very few utilized the tilt-board or the most difficult piece of foam in the Agility program. Instructors spotted those individuals at higher risk for falls and during exercises that were particularly challenging regardless of the client's functional level. There was one injury related to the Agility exercise intervention group (i.e. a hip fracture). However, it must be emphasized that this adverse event was unavoidable. The client involved had a very high Berg Balance score and did not typically require spotting. Furthermore, the event occurred during a non-challenging task (i.e. standing balloon toss). Only one hip fracture out of the 59 individuals who started the exercise intervention represents less than 2% of the sample, which is well within the percent of individuals with stroke that fracture their hip in a given year (Dennis et al. 2002; Kanis et al. 2001). In the future, it may be beneficial to have clients utilize hip protectors as a precaution and/or have high absorbent flooring in the exercise classroom to minimize the chance of serious injury.

The exercise instructor team consisted of a physiotherapist (head instructor), kinesiologist, and recreation therapist. The combination worked exceptionally well, with each instructor providing a different level and kind of expertise. Of particular benefit was the presence of a recreation therapist. A recreation therapist provides expertise on using leisure and recreation for achieving optimal health and quality of life. They not only focus on the exercises to improve function but also on adapting the program based on the goals and interests of each individual (e.g. social interaction, making exercises fun and meaningful, instilling confidence). In an ideal situation, future community-based group exercise programs would utilize all three of these professionals.

5.3.2 Types of exercises used in the intervention and their effectiveness

The selection of appropriate exercises for the Agility exercise group were based, in large part, on the work of Fitzgerald et al. (2002), Gardner et al. (2001), and Hu and Woollacott (1994a, b) in healthy older adults and modifying them to suit a lower functioning population. The major foci for this exercise program were the multi-sensory and agility components (see Appendix XVII). A variety of densities of foam were used to alter somatosensation, thereby enhancing afferent input from the visual and vestibular systems. Removing visual input by closing the eyes was also used to further emphasize the need for vestibular regulation of postural control.
Agility training has been shown to be effective in older adults (Fitzgerald et al. 2002). Rapid forward and backward stepping was a major task in our program, as compensatory stepping is a common strategy employed in response to standing perturbations in older adults (Maki and McIlroy 1997). Standing perturbation training was also utilized. In this task, the instructor would attempt to knock the client off balance to force them to step (in a highly controlled and safe manner). Additionally, clients were given the chance to attempt to knock the instructor off balance (a self-induced perturbation). This, not surprisingly, was one of the more enjoyable tasks for the clients. Both the rapid stepping and standing perturbation tasks may have contributed to the faster standing postural reflexes and step reaction time outcome measures. The braiding and tandem walking were also very effective, as demonstrated by the improved functional mobility seen after the intervention. It is recommended that clients be carefully spotted during the braiding task because of the risk of tripping on their feet.

The major foci of the Stretching/weight-shifting exercise program were, as the name implies, stretching and weight-shifting (see Appendix XVII). Stretching of major muscle groups was performed during standing, sitting, and while on mats on the floor. This latter location was particular important for this exercise program as getting down to and up from mats on the floor proved to be a challenge for many of the clients. In fact, several clients told us that they had not been down on the floor since their injury. Although none of the instructors involved in the exercise intervention were trained in the art of Tai Chi, similar weight-shifting movements were incorporated and provided a constant driving force for this program. Increasing the ability to take load through the paretic limb is important for many activities of daily living, such as standing up from a chair and reaching forward, and for postural stability (Cheng et al. 1998; Eng and Chu 2002; Sackley 1990).

Although the Agility exercise group demonstrated additional benefits over the Stretching/weight-shifting exercise group, a combination of the two programs is likely important. We recommend that mat exercises be incorporated into the Agility exercise program. Further, group discussions (and even educational sessions) would also be of benefit to this program. The results of this thesis should facilitate the design of rehabilitation programs for individuals with chronic stroke. It is clear from Experiments I and II that clinicians need to focus on the ankle dorsiflexors, particularly on the paretic limb, to improve muscle strength. This in turn may then help reduce the incidence of falling in this population. Furthermore, it is important to incorporate exercises that evoke postural reflexes, such as standing perturbation training, and exercises that require fast movements, such as rapid stepping. Multi-sensory
training would also be effective as this type of training might improve cognitive processing and may have contributed to some of our clinical trial (Experiment III) results (see neural plasticity section below).

Balance training has not been extensively studied in individuals with chronic stroke. Exercise tasks that have been used include reaching tasks while seated or standing with feet together or in tandem (Dean et al. 2000), stepping onto blocks or steppers (Dean et al. 2000; Eng et al. 2003), walking over obstacles (Ada et al. 2003; Bassile et al. 2003; Dean et al. 2000), walking on different surface terrain (Ada et al. 2003; Dean et al. 2000; Eng et al. 2003), and varying step length or speed during walking (Ada et al. 2003; Eng et al. 2003). Unfortunately, Tangeman et al. (1990) did not report the type of functional balance exercises performed during their intervention, although they did mention weight-shifting tasks. Foam (walking task) was used in only one study (Eng et al. 2003); however, not to the extent of this clinical trial. Furthermore, no other study in individuals with chronic stroke has utilized standing perturbation tasks. We have demonstrated that these tasks are not only feasible, but may also have contributed to the better performance of the Agility exercise group.

Overall, both exercise programs were highly effective in improving functional balance and mobility among other measures. The cost of equipment used was minimal and readily available in most communities. We argue that it is time to implement community-based group exercise programs for individuals with chronic stroke.

5.4 Exercise-induced Neural Plasticity

Neural plasticity refers to a persistent nervous system modification (whether it be structural, molecular, or cellular) that results from past experience and affects future behaviour (Wolpaw and Tennissen 2002). How the brain adapts to recovery from injury is one form of plasticity. In addition, motor learning, which is a set of internal processes associated with practice or experience leading to relatively permanent changes in the capability for motor skill (Schmidt and Lee 1999), is another form of plasticity. Until recently, it was believed that functional recovery of individuals with stroke plateaus after about 6-months following injury (Duncan and Lai 1997; Jorgensen et al. 1999; Wade et al. 1985). However, recent studies of exercise training and constraint-induced therapy in individuals with chronic stroke (i.e. greater than 6-months post-stroke) suggest that improvements in function are still possible (Liepert et al. 1998, 2000a).
We showed that functional balance and mobility were improved following our exercise interventions (Experiment III). Further, we demonstrate for the first time that paretic limb postural reflexes become faster following exercise, particularly following an Agility intervention (e.g. approx. 28 msec change in latency of the paretic rectus femoris). We argue that the faster standing postural reflexes are functionally significant as there was a concomitant improvement in functional balance and mobility. Moreover, we argue that the investigation of postural reflexes provide a means to speculate on whether plastic changes occur within the nervous system and the faster postural reflexes represent neural plasticity. The faster step reaction time for the Agility exercise group also supports the notion of neural plasticity. This task requires individuals to process the auditory cue for initiating the step forward and integrate this afferent information within multiple cortical association areas so that a motor command can be generated and subsequently sent through descending pathways to the muscle itself. The onset latency of the postural reflexes (> 100 msec) and especially the time to initiate a step (> 500 msec) for the step reaction task are highly suggestive of cortical involvement (Di Fabio et al. 1992). Although the small changes in the paretic tibialis anterior, gastrocnemius, and biceps femoris may have occurred through spinal cord plasticity and/or alterations in peripheral nerve conduction velocity and muscle fibre type, the larger change with the paretic rectus femoris is too great not to have been from plasticity within cortical neuronal networks. This is especially true for the change in step reaction time (> 100 msec faster for the Agility exercise group post-intervention).

Further support comes from the fact that the changes are not from spontaneous recovery, as this would have occurred well before entry into the intervention. In terms of postural reflex onset latency, the change is not due to an alteration in muscle sequencing or recovery strategy as individuals continued to demonstrate the same responses before and after the intervention. In addition, the faster postural reflexes are not the result of learning as we demonstrated that postural reflex muscle onset latencies have moderate to high test re-test reliability (without improvement) (see Appendix IV). Thus, it appears as though exercise induces neural plasticity in individuals with chronic stroke.

5.4.1 Reasons for exercise-induced neural plasticity

Why might these changes have occurred? The faster step reaction time and paretic rectus femoris postural reflex onset latency occurred in the Agility exercise group, which might be
explained by the task-specific training involved in this program. Task-specific training is thought to drive neuronal reorganization (Shepherd 2001). The use of the rectus femoris muscle to step forward during the rapid stepping tasks and the standing perturbations performed in the Agility exercise program may have facilitated the faster paretic limb rectus femoris onset latency and faster non-paretic limb step reaction time.

Strengthening of redundant or alternative pathways could be responsible for the faster onset latency of the paretic rectus femoris and step reaction time in the Agility exercise group. Several studies using transcranial magnetic stimulation (TMS) or functional magnetic resonance imaging (fMRI) in individuals with stroke during recovery have demonstrated that activation of the unaffected hemisphere (which would stimulate ipsilateral pathways) occurs during thumb or hand movements; although whether this correlates with functional recovery is still under debate (Caramia et al. 2000; Cramer et al. 1997; Cramer and Bastings 2000; Feydy et al. 2002; Netz et al. 1997). Lee and van Donkelaar (1995) have reported that after gradual improvement over several years from a left hemispheric stroke resulting in right-sided hemiparesis, a patient experienced a second stroke in the opposite hemisphere in almost the identical location. The result was a mild sensorimotor deficit in the left arm but a marked worsening of the original right hemiparesis. The authors argue that the most plausible explanation is that the original unaffected hemisphere was responsible for the improved right hemiparesis following the first stroke. Animal studies have also demonstrated neural plasticity in both the affected and unaffected hemispheres (Frost et al. 2003; Jones et al. 1996; Kozlowski and Schallert 1998; Stroemer et al. 1995).

The exercise tasks encompassing the Agility program, for example, the use of multi-sensory tasks, enhanced vestibular stimulation and hence, the vestibulospinal tract. It is believed that the vestibulospinal tract, particularly the lateral vestibulospinal tract, has a large influence on proximal leg muscles (e.g. rectus femoris) for postural control (Allum et al. 1995). The lateral vestibulospinal tract remains uncrossed and thus, is stimulated by the unaffected hemisphere (Fredericks 1996). Uncrossed pontine reticulospinal tract fibres, which receive ipsilateral cortical projections, also have a large influence on proximal leg muscles (Fredericks 1996). It is possible that a combination of neural plasticity in these pathways contributed to the faster paretic rectus femoris postural reflex. Since all paretic limb muscles exhibited faster postural reflexes, albeit to different amounts, these pathways may have influenced these other muscles. Alternatively, ipsilateral corticospinal tract fibres may have been the driving force; particularly
with the paretic tibialis anterior and gastrocnemius as corticospinal tract fibres have strong influences over distal leg muscles (Brouwer and Ashby 1992).

The Agility exercise program presumably stimulated proprioceptive pathways as well. Thus, improvements in functional balance and mobility as well as the faster postural reflexes and step reaction time may have been facilitated by neural pathways and supraspinal centres associated with this sensory system.

5.4.2 Potential mechanisms for exercise-induced neural plasticity

The question then arises as to what are the underlying mechanisms involved in the neural plasticity induced through exercise training? Exercise can activate the molecular and cellular cascades involved in neural plasticity (Cotman and Berchtold 2002). Wheel running or treadmill exercise in rats for multiple days leads to an increase in brain-derived neurotrophic factor (BDNF) expression within the soleus muscle, spinal cord, hippocampus, and cerebral cortex (Cotman and Berchtold 2002; Gomez-Pinilla et al. 2001; Neeper et al. 1996). BDNF has many characteristics that make it suitable for neural plasticity: its expression is activity-dependent (Black 1999; Cotman and Berchtold 2002; Kohara et al. 2001), it can be transported to a post-synaptic neuron from a pre-synaptic neuron (Kohara et al. 2001), it enhances synaptic transmission (Black 1999; Cotman and Berchtold 2002), and stimulates synaptophysin for synaptogenesis (Cotman and Berchtold 2002). Recently, Kwon et al. (2002) discovered that rubrospinal neurons do not die in spinal cord injured rats but rather they are in a state of severe atrophy, which can be reversed with BDNF treatment one-year post-injury. It is possible that following stroke, rubrospinal tract neurons as well as additional pathways such as corticospinal tract neurons are also severely atrophied and exercise-induced BDNF expression reverses this process and contributes to the observed neural plasticity.

Exercise also increases nerve growth factor (NGF) expression in wheel running rats (Cotman and Berchtold 2002; Neeper et al. 1996) and increases neurotrophin-3 (NT-3) expression in treadmill exercising rats (Gomez-Pinilla et al. 2001). Kolb et al. (1996) has shown that NGF treatment prevents dendritic atrophy and promotes dendritic arborisation and increases spine density following unilateral lesions in rats. In addition, van Praag et al. (1999a, 1999b) have reported enhanced neurogenesis in conjunction with better performance on the Morris water maze task following wheel running in mice.
Plastic changes to neurons must be accompanied by local increases in oxygen and glucose (Kleim et al. 2002). This may be accomplished by increased growth of capillaries (angiogenesis) within regions undergoing plasticity. Kleim et al. (2002) and Swain et al. (2003) have recently shown exercise (wheel running) induces angiogenesis in the motor cortex of rats. Thus, BDNF, NGF, and NT-3 expression along with angiogenesis may facilitate exercise-induced neural plasticity.

Few studies have investigated neural plasticity in individuals with chronic stroke. The cortical reorganization seen in animal models has also been demonstrated in individuals with chronic stroke following forced-use of the paretic arm through constraint-induced (CI) therapy (Liepert et al. 1998, 2000a). Individuals with chronic stroke experienced two-weeks of CI therapy and motor function was improved with a concomitant increase in motor output area size of the affected hemisphere, which was retained up to 6-months (Liepert et al. 1998, 2000a).

Cortical reorganization and changes in cortical excitability may stem from several processes including long-term potentiation (LTP), changes in N-methyl-D-aspartate (NMDA) receptor activity, changes in gamma-aminobutyric acid (GABA)_A receptor activity, through intracortical connections, or unmasking silent synapses. LTP refers to a brief high-frequency train (or tetanus) stimulus causing a prolonged increase in amplitude and excitatory potential in a connecting neuron (Bliss and Lomo 1973). The activity-dependent induction of LTP, its longevity, and property of associativity (which is the ability of a strong LTP inducing stimulation to cause a non-LTP inducing stimulation with which it is paired to exhibit LTP) remain compelling reasons why LTP may be one mechanism of neural plasticity (Martinez et al. 1998). LTP is also involved in motor learning (Martinez et al. 1998) and exercise in rats has shown enhanced LTP (van Praag et al. 1999a).

Following stroke, changes in excitatory receptor activity (i.e. up-regulation of NMDA receptors) and inhibitory receptor activity (i.e. down-regulation of GABA_A receptors) occur (Qu et al. 1998). The intracortical connections, which are supposed to modulate cortical representation (i.e. regions within the cortex responsible for specific movements) area sizes, are mainly GABAergic (Jones 1993). Early studies in rat brains by Donoghue and colleagues support the notion of unmasking latent intracortical (horizontal) connections for reorganizing cortical representations (Hess and Donoghue 1994; Hess et al. 1996; Jacobs and Donoghue 1991). After application of bicuculline methobromide, a GABA antagonist, to a remote region in the primary motor cortex from the electrical stimulations site, forelimb movements could be evoked (Jacobs and Donoghue 1991). In rat primary motor cortex slice preparations, LTP could
be produced with theta burst stimulation only after application of the GABA_\text{A} receptor antagonist, bicuculline methiodide, in layer II/III horizontal projections (Hess and Donoghue 1994; Hess et al. 1996). Furthermore, application of 2-amino-5-phosphonovaleric acid, a NMDA receptor antagonist, blocked LTP in these connections (Hess et al. 1996).

Recent experiments in human subjects have confirmed the plausibility of GABA_\text{A} and NMDA receptor mediated neural plasticity. In a study by Bütefisch et al. (2000), TMS was used to evoke thumb movements in one direction. Subsequently, participants practiced for 30-minutes thumb movements in the opposite direction, which TMS post-training then induced thumb movements in the practiced direction. This process was repeated over several days in which oral doses of lorazepam, a GABA receptor agonist, or dextromethorphan, a NMDA receptor blocker, were given to participants prior to testing. The results demonstrated both these drugs decreased thumb movements in the practiced direction evoked by TMS post-training.

More recently, Ziemann et al. (2001) found that motor practice of ballistic contractions of the biceps brachii during ischemic nerve block (to decrease GABA inhibition) increased motor evoked potentials assessed by TMS whereas motor practice after an oral dose of lorazepam decreased motor evoked potentials. Thus, neural plasticity appears to involve an increase in NMDA receptor density and/or efficiency with a concomitant decrease in inhibitory GABA_\text{A} receptor density and/or efficiency.

The knowledge gained from studies on the effects of rehabilitative training following ischemic infarct to the sensorimotor cortex may also serve to be fruitful in providing potential mechanisms for plasticity in the chronic stage of injury. Environmental enrichment and task-specific rehabilitation in rats following unilateral middle cerebral artery occlusion improved motor function and increased dendritic arborisation of layer V pyramidal cells within the undamaged motor cortex (Biernaskie and Corbett 2001). Jones and colleagues (Chu and Jones 2000; Jones et al. 1999) have shown that an acrobat task (i.e. obstacle course) following unilateral forelimb sensorimotor lesion in rats increases dendritic growth and synaptogenesis compared to rats undergoing repetitive task training and those who received the lesion but no intervention. Our Agility exercise program may be the human equivalent to enhanced rehabilitative training in animals.

In monkeys, a lesion to the motor cortex hand area with subsequent behavioural rehabilitation training (food pellet retrieval task) increased performance and cortical reorganization (Nudo et al. 1996). This form of ‘rehab’ prevented the loss of spared hand cortical representations in adjacent, intact cortex. In humans, a single physiotherapy session
during the subacute phase of rehabilitation in individuals with stroke resulted in an increased cortical representation (i.e. motor output map) of the paretic hand muscle in the affected hemisphere one-hour after therapy as assessed by TMS (Liepert et al. 2000b). Thus, increased dendritic arborisation, dendritic spine density, synaptogenesis, and on a larger scale, cortical reorganization, may contribute to the exercise-induced neural plasticity.

It would be interesting to utilize TMS or fMRI to investigate neural plasticity in individuals with chronic stroke during the course of an exercise intervention similar to ours.

5.5 Limitations

This was the first study to investigate falls-reduction following a community-based group exercise program in individuals with chronic stroke. There were three times as many falls in the Stretching/weight-shifting group than the Agility group over the one-year follow-up; however, this did not reach statistical significance. Further, the number of fallers was not different between exercise groups. A limitation of the clinical trial was that it was not powered for falls. However, following a sub-analysis on just the individuals who fell prior to the intervention, the number of fallers was significantly lower in the Agility group versus the Stretching/weight-shifting group. There is an obvious need to examine strategies for falls-reduction in individuals with chronic stroke. The results from our clinical trial are encouraging; however, multi-centre clinical trials with a larger sample size are required to firmly establish whether an exercise intervention can reduce falls in this population.

Several additional limitations to this thesis exist. First, data for Experiment II was obtained from the baseline assessment of the clinical trial (Experiment I). Hence, the knowledge gained on the neural mechanisms of falling from Experiment II was not used in the development of the exercise interventions. Although this information may have been useful, the exercises still targeted the aspects associated with falls that we identified. Exercises were based on previous studies in individuals with chronic stroke, from knowledge of the clinical presentation of individuals with chronic stroke, and studies in healthy older adults. We would not have altered the exercise interventions in any way had we run Experiment II before designing the clinical trial.

Second, we were unable to obtain information regarding the location of the stroke for approximately 20% of our sample. Further, we did not have information on the volume of the
stroke. Thus, we could not determine which specific areas within the brain were damaged and responsible for our observations. It would be fruitful to have future studies obtain this information.

Third, changes in the latency of postural reflexes but not the magnitude following exercise were investigated in Experiment III. Unfortunately, this is due to methodological constraints associated with EMG collection (e.g. skin impedance) that may influence magnitude from one day to the other. Changes in kinematics during perturbations following exercise interventions would also be interesting and warrants further study.

Fourth, we restricted our perturbations to the anterior-posterior direction. Individuals with stroke often load their non-paretic lower limb to a greater extent. Further, falls do occur to a large extent in the medial-lateral direction, thereby increasing the chance for hip fracture. Unfortunately, no study has used medial-lateral surface translations in individuals with chronic stroke and thus, little is known about how these individuals react in this direction.

Fifth, screening and recruitment were limited. Not every person screened for our clinical trial was eligible. Cognitive deficits and the inability to ambulate were among the reasons for exclusion. Since we required participants to ambulate, this might have excluded those with severe motor impairments, sensory impairments, or spasticity. Fortunately, the majority of individuals with stroke do become ambulatory after their injury (Jorgensen et al. 1999; Wade et al. 1985) and thus, a large portion would be able to participate in community-based exercise programs. Due to the amount of questionnaires and testing which required several instructions, we excluded individuals with cognitive deficits. However, these individuals may still be able to participate (possibly with an extra assistant present during the exercise classes) in exercise programs in the community where laboratory testing is not required. In addition, we recruited participants on a voluntary basis. Thus, the fact that these people wanted to get better may have influenced our results.

And sixth, the difficulty in transportation to the community centre for the exercise classes made it impossible for those living outside the Vancouver area (such as Powell River) to participate. Since a portion of the population does live in these areas, methods for including these individuals must be explored. Fortunately, transportation in Vancouver for individuals with impairments, such as stroke, is available and effective.

A final concern must be addressed regarding the lack of a true control group in Experiment III. Although we cannot exclude the possibility that the simple act of attending the exercise classes (i.e. getting to and from the classes) may have contributed to the changes, there are
several reasons why the exercise interventions were the primary cause of the observations. First, test re-test reliability was established (and learning was not evident) for the standing postural reflexes and the changes (particular the rectus femoris muscle of the paretic limb) were outside the SEM range. Second, Eng et al. (2003) report that Berg Balance scores are stable over time and also found improvements with exercise. And third, the Agility exercise group demonstrated additional improvements in some outcome measures over the Stretching/weight-shifting group. In addition, the changes were not due to recovery over time as a plateau of recovery is reached within 6 months post-stroke (Duncan and Lai 1997; Jorgensen et al. 1999; Wade et al. 1985). Thus, the changes observed in Experiment III were in fact due to the exercise interventions.

5.6 Future directions

We propose a three-tier community-based group exercise program system be implemented in the community for individuals with chronic stroke. This exercise program should follow the tasks outlined in the Agility exercise program with the addition of (1) the task of getting down to and up from the floor, (2) group discussions, and (3) educational sessions such as fall prevention and cardiovascular health. Additional social events and/or outings may also be included. Each of the three tiers would represent a separate exercise class/program held for one-hour, three times per week throughout the year. In terms of the exercise instructor team, a 3:1 instructor-to-client ratio should be maintained. In addition, we recommend the team consist of a physiotherapist, kinesiologist, and recreation therapist; although a physiotherapist could act as a consultant and be replaced as an instructor by another kinesiologist or rehab assistant trained to deal with individuals with stroke.

The difficulty of tasks in each tier would be progressively more challenging. Tier one would be tailored to clients predominantly wheelchair bound and/or very low functioning. Tier two would be tailored to clients of medium functioning. And tier three would be tailored to high functioning clients. This last and final tier would also be considered an exercise class for maintaining any improvements in functional balance and mobility. Thus, multiple classes may be required to accommodate the larger amount of individuals that will eventually be at a tier three level.
Participation in this recreation/exercise program would be voluntary. However, prior to entry all individuals would be required to undergo an assessment to determine which tier is most suitable for them and to determine any goals of the individual. A composite score based on clinical testing of Berg Balance, Timed Up and Go, and step reaction time is recommended due to the speed and ease in which these tests can be administered as well as their relevance to the purpose of the program. At certain points throughout the year (for example, every two months) clients would be re-evaluated to determine whether they could graduate to the next tier level. This assessment period would also serve as a point in which new clients could enter the program.

5.7 Final thoughts

In conclusion, we have identified aspects of impaired postural control and the neural mechanisms contributing to falls in individuals with chronic stroke. Further, we have shown the benefits of exercise in this population. It is time to implement community-based group exercise programs for individuals with chronic stroke.
CHAPTER 6 - References


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### Appendix I: Literature table – Falls in individuals with stroke

#### Table I.1: Falls in individuals with stroke

<table>
<thead>
<tr>
<th>ARTICLE</th>
<th>PURPOSE</th>
<th>SUBJECTS</th>
<th>PROTOCOL</th>
<th>OUTCOME MEASURES</th>
<th>RESULTS</th>
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<tbody>
<tr>
<td>Denis et al. 2002</td>
<td>To establish the rate of fracture after stroke and compare that rate with that of the general population</td>
<td>N=2696 stroke; x=68 yrs old; 1426M, 1270F</td>
<td>- Followed stroke patients admitted between 1990-1998 and referred to a clinic between 1994-1998 - Followed up at 6 months and 1 and 2 years - Calculated risk of admission with hip fracture to hospital after stroke</td>
<td>- Falls - Fractures</td>
<td>- 4% experienced fracture (30% of hip and 75% due to a fall) for hospital referred patients - Hospital discharge data to 2% had fracture by 1 year and 10.6% by 10 years - 1.2 times the rate of hip fracture compared to general population and 2.3 times compared to patients with MI</td>
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<td>DSM 510</td>
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<td>Forster and Young 1995</td>
<td>To undertake a systematic inquiry into the incidence and consequences of falls in a cohort of elderly patients with stroke after discharge from hospital</td>
<td>N=108 stroke; 57M, 51F; 46 right, 57 left hemi; x=70 yrs old;</td>
<td>- Community-dwelling stroke survivors recruited</td>
<td>- Falls - Motor club assessment - Barthel Index - Frenchay activities index - NHP</td>
<td>- 73% fell within six months after discharge from hospital for a total of 270 falls reported - Fallers were less socially active at six months and more had depressed mood - Patients who fell in hospital were more like to fall after discharge</td>
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<tr>
<td>DSM 172</td>
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<tr>
<td>Hyndman and Ashburn 2003</td>
<td>To describe levels of attention deficits and explore relationships between attention, ADL ability, balance, and falls in chronic stroke</td>
<td>N = 48 chronic stroke; 30M, 18F; 21 right, 26 left hemi; x=68.4 yrs old; x=46 months post-stroke;</td>
<td>- Fall information collected from retrospective (12 months) data - Performed measures of attention, ADL, and balance</td>
<td>- Berg Balance - ADL - Attention (sustained attention, auditory selective, visual selective, visual inattention) - Falls</td>
<td>- Attention deficits in stroke common - Balance was correlated with ADL (r=0.83), elevator counting (r=0.4), telephone searching while counting (r=0.51), and fall status (r=0.42) - Fall status also correlated with ADL (r=0.38), elevator counting (r=0.37), and telephone search</td>
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<tr>
<td>DSM 1079</td>
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<tr>
<td>Study</td>
<td>Objective</td>
<td>Participants</td>
<td>Fall Definition</td>
<td>Additional Details</td>
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<tr>
<td>Hyndman et al. 2002</td>
<td>To determine whether circumstances of falls and fall risk differ among a community sample of stroke patients whose time since stroke varies and to compare characteristics of fallers and non-fallers</td>
<td>N=41 strokes; 26M, 15F; x=69.7 yrs old; x=50.4 months post; 16 right, 23 left hemi and 2 brainstem</td>
<td>Fall → defined as “an event that results in a person coming to rest unintentionally on the ground or other lower level, not as a result of a major intrinsic event or overwhelming hazard” (quoted from another article)</td>
<td>Falls - ADL (Nottingham extended ADL) - Rivermead Mobility Index (RMI) - Rivermead Motor assessment upper limb scale (RMA) - Hospital anxiety and depression (HAD) scale</td>
<td></td>
</tr>
<tr>
<td>Jorgensen et al. 2002</td>
<td>To compare the incidence of falls among non-institutionalized long-term stroke survivors with healthy older adults</td>
<td>N=111 stroke; 57%M; x=68 yrs old; x=10 yrs post-stroke; N=143 controls; 57%M; x=67 yrs old;</td>
<td>Recruited from community from 1994-1995. Descriptive collected - 4-month prospective fall history</td>
<td>Risk of first fall - Risk of recurrent falls - Type and characteristics of falls</td>
<td></td>
</tr>
<tr>
<td>Kanis et al. 2001</td>
<td>To examine whether stroke</td>
<td>N=273,288 strokes;</td>
<td>Followed subjects from time</td>
<td>Depression predicted falls in stroke - 23% of stroke fell during a 4-month period while living in the community (50% of fallers fell multiple times) - 77% of falls were during walking</td>
<td></td>
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</tbody>
</table>

while counting (r=-0.41)
- Those with normal scores on sustained and divided attention tests had significantly better balance and ADL ability
- Repeat fallers had lower balance scores and ADL than non-fallers with no near-falls and non-fallers with near-falls

Repeat fallers had lower balance scores and ADL than non-fallers with no near-falls and non-fallers with near-falls
<table>
<thead>
<tr>
<th>study</th>
<th>purpose</th>
<th>sample</th>
<th>methods</th>
<th>findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lamb et al. 2003</td>
<td>To investigate the relevance of known predisposing risk factors for falling and stroke-specific factors in a population of women with a history of stroke</td>
<td>N=94 stroke; all women; x=76 yrs old; x=48 months post-stroke;</td>
<td>Recruited women from community of which people with stroke included (N=124 started study) - 12-month prospective fall history</td>
<td>Falls - ADL - BMI - Depression - Cognition - Balance - Visual acuity - Knee extensor strength - Pinch grip strength - Walking speed - Chair rising - 48% fell during 1 year follow-up - 29% of sample fell more than once - Frequent balance problems while dressing was the strongest risk factor for falls (OR = 7) - Residual balance, dizziness, or spinning stroke symptoms were also a strong risk factor for falling (OR = 5.2)</td>
</tr>
<tr>
<td>Nyberg and Gustafson 1995</td>
<td>To investigate the incidence, characteristics, and consequences of patient falls in a stroke rehabilitation setting</td>
<td>N=161 stroke patients; x=23 days post-stroke; 84M, 77F; x=75.2 yrs old;</td>
<td>Falls were recorded while in rehab setting (x=48 days in study) - Descriptive collected</td>
<td>Frequencies and incidence rates of falling - 39% of patients fell and 24% of entire sample fell more than once - Most frequent location for falls was patients room - Transferring (37%) was the most frequent activity being performed when fall occurred. - 28% of falls resulted in injury</td>
</tr>
<tr>
<td>Ugur et al. 2000</td>
<td>To investigate the incidence of falling, to identify the risk factors for falling after stroke, and to evaluate the effect of stroke on the incidence of falling</td>
<td>N=131 (44% of all patients) stroke fallers; x=62.5 yrs old; 70M, 61F, 48 left</td>
<td>Patients were followed in stroke unit between 1992-1996</td>
<td>Barthel Index - Depression scale - Risk factors - Stroke location - Affected side - Falling occurred most often in the oldest age group - Right CVA (i.e. left CVA) were more likely</td>
</tr>
<tr>
<td>Relationship between lesion localization and falling</td>
<td>hemi, 38 right hemi; N=162 non-faller stroke patients; x=59.88 yrs old; 78F, 84M; 39 left hemi, 57 right hemi</td>
<td>Falls</td>
<td>Fallers were more depressed and lower Barthel Index scores</td>
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<tr>
<td>Yates et al. 2002</td>
<td>To examine the relationship between accumulated neurological impairments following stroke and the increased risk of falling in community-dwelling stroke survivors</td>
<td>Follow-ups were completed 1, 3, &amp; 6 months post-stroke</td>
<td>- Stroke severity - Motor impairment (Fugl-Meyer) - Sensory and visual impairments - Falls</td>
<td></td>
</tr>
<tr>
<td>DSM 898</td>
<td>N=280 stroke patients; x=68.3 yrs old; 50%M;</td>
<td></td>
<td>- 51% patients fell while in the community - 35% of those fell multiple times - Risk of falling in stroke was increased 2x when motor impairment present and 3x when motor and sensor impairments were present compared to stroke fallers with no impairments</td>
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</tbody>
</table>

- Falls
## Appendix II: Literature table – Exercise in individuals with stroke

### Table II.1: Persons with Chronic Stroke

<table>
<thead>
<tr>
<th>ARTICLE</th>
<th>PURPOSE</th>
<th>SUBJECTS</th>
<th>PROTOCOL</th>
<th>OUTCOME MEASURES</th>
<th>RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ada et al.</td>
<td>To determine whether 4 weeks of treadmill and overground walking increases speed and capacity of walking, improve quality of walking, and decrease handicap and are these changes maintained 3 months after</td>
<td>N = 13 stroke in experimental group; x=66 yrs old; x=28 months post-stroke; 9M, 4F; 8 right, 5 left hemi; N = 14 stroke in control group; x=66 yrs old; x=26 months post-stroke; 10M, 4F; 6 right, 8 left hemi;</td>
<td>- Experimental group intervention → 3x/wk, 45-minute sessions over 4-weeks</td>
<td>- Walking speed 6-min walk test - Handicap measure (SA-SIP30) - Step length, cadence, and step width</td>
<td>- Walking speed and walking capacity (6-min walk test) were increased in experimental group at post-test and retention - Step length for both legs increased by post-test in experimental group and remained at retention testing</td>
</tr>
<tr>
<td>DSM 1026</td>
<td></td>
<td></td>
<td>- Intervention was treadmill training (decreasing by 10% each week from 80%) and overground walking; walking speed was varied and dual cognitive task was introduced; overground walking was done forward, backward, or sideways, and up and down stairs and had target stepping involved; in addition, overground walking involved an outdoor circuit of curbs, slopes, stairs and rough terrain</td>
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</tr>
<tr>
<td>Study</td>
<td>Objective</td>
<td>Sample</td>
<td>Intervention</td>
<td>Outcome Measures</td>
<td>Comments</td>
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<tr>
<td>Badics et al. 2002</td>
<td>To examine the effects of sequential exercising on muscle tone and strength in stroke</td>
<td>N = 56 stroke; 3 wks – 10 yrs post-stroke; 34M, 22F; x=61 yrs old</td>
<td>Exercise program was a residential rehab program of 4 weeks of upper and lower limb strengthening (exercise units → 30-50% of max., 3-5 series of 20 reps)</td>
<td>- Exercise strength</td>
<td>- No increase in muscle tone strength - No increase in muscle tone strength increased in all patients - Intensity and number of exercising units positively correlated with strength gain</td>
</tr>
<tr>
<td>Bassile et al. 2003</td>
<td>To develop an obstacle training program for people with stroke and assess its effects</td>
<td>N=5 chronic stroke; 41-88 yrs old; 0.5-6 yrs post-stroke; 4 right, 1 left hemi</td>
<td>Pre-, post-, 1-month retention testing - 4-week training (group) program of walking over obstacles</td>
<td>- Pre-, post-, 1-month retention testing - 4-week training (group) program of walking over obstacles</td>
<td>- 6-min walk test and walking velocity improved - Only walking velocity showed retention - MAS time improved and was retained - SF-36 improved but not significant</td>
</tr>
<tr>
<td>Bastien et al. 1998</td>
<td>To determine (1) acceptability of various exercises, activities, games, (2) optimal class time, (3) instructor/client ratio, (4) outcome measures, (5) compliance, and (6) cost or running an exercise program for individuals with stroke</td>
<td>N =24 chronic stroke; 17M (x=64.2 yrs old), 7F (x=67.4 yrs old); greater than 9-months post-stroke; Only 15 had post-test assessment</td>
<td>Multi-location trial - Community-based group exercise program - Intervention → N = 14, 2x/wk, 60-min over 6 wks, 2 instructors; N = 4, 1x/wk, 90-min over 8 wks, 1 instructor and 1 assistant; N = 6, same as previous but with 2 instructors</td>
<td>- Berg Balance trial - Get Up and Go test - Grip strength - RNL - Fall events during classes - Falls self-efficacy - Social and leisure involvement</td>
<td>- Berg Balance improved by 4-points - Get Up and Go improved by about 4 sec.</td>
</tr>
<tr>
<td>Reference</td>
<td>Study Details</td>
<td>Results/Findings</td>
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<tr>
<td>Carr and Jones 2003</td>
<td>To investigate the long-term effects of moderate exercise training on physiological and metabolic outcomes for stroke survivors. N = 40 stroke; 22M, 18F; greater than 6-months post-stroke</td>
<td>- Baseline testing of VO₂ and blood testing. - Randomly assigned into one of two groups after baseline testing: aerobic training (ATO) and aerobic and strength training (A&amp;ST). - 16-week program. - Pre- and post-testing. - Individual lab-based training 3x/wk. - ATO group did training on upper and lower limb ergometer; Phase 1 (5 wks) – 40-50% of original test wattage (from VO₂ testing) for 20-min; Phase 2 (5 wks) – 50-60% for 30-min; Phase 3 (6 wks) – 60-70% for 40-min; also did flexibility. - A&amp;ST group did same protocol but also strength training (2 sets of 10 reps for upper and lower limbs using free weights and machines); weight progressively increased.</td>
<td>- VO₂ max - Peak torque on knee flexion/extension and shoulder extension/flexion. - Cholesterol - High-density lipoprotein (HDL) levels. - Glucose levels (fasting).</td>
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<tr>
<td>DSM 1074</td>
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<td></td>
<td>- A&amp;ST group increased VO₂ max. - Knee flexion and shoulder extension increased in both groups. - Shoulder extension increased in both groups and flexion in A&amp;ST group. - Cholesterol decreased for A&amp;ST group.</td>
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<tr>
<td>Dean and Shepherd 1997</td>
<td>To examine the effect of a training program designed to improve the ability to balance in sitting after stroke. N=20 stroke; Exp. (n=10) ( \rightarrow x=68.2 \text{ yrs old}; 7M, 3F; 5 \text{ right, 5 left hemi}; x=6.7 \text{ yrs post}; \text{Control (n=10) } \rightarrow x=66.9 \text{ yrs old}; 7M, 3F; x=5.9 \text{ yrs post}; 6 \text{ left, 4 right hemi}</td>
<td>- Randomly assigned into experimental or control group. - 10 sessions over two weeks in-home. - Experimental ( \rightarrow \text{designed to improve sitting balance, loading affected leg while reaching, etc.} ). - Control ( \rightarrow \text{cognitive-} ).</td>
<td>- Max. reaching difference. - Ground reaction force. - EMG of anterior deltoid (non-paretic) and bilateral lateral vastus, TA, and soleus.</td>
<td>- Increased max. reaching distance and shorter time after training and vs. controls. - Increased vertical GRF through affected limb after training and vs. controls. - Experimental group could activate affected leg muscles more after training than</td>
<td></td>
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</tbody>
</table>
### Dean et al. 2000

**DSM 6**  
To investigate the feasibility and efficacy of an exercise class aimed at improving performance of locomotor-related tasks in individuals with stroke  
N=12 chronic stroke  
- 4-week training program  
  - Experimental group focused on strengthening the affected lower limb & practicing functional tasks involving the lower limbs (sit-to-stand, walking, reaching sitting & standing, stair ascent & descent)  
  - Control group upper limb tasks  
  - 2 month follow-up  
- Walking speed and endurance  
- 6-min walk test  
- Step test (repetitions of stepping onto 7.5 cm block in 15 seconds)  
- Timed Up and Go  
- Peak vertical GRF through affected foot during sit-to-stand and the step test  
- Significant & retained improvement for experimental group vs. control in walking speed and 6-min walk test, force production through affected limb during sit-to-stand, and # of repetitions of step test

### Eng et al. 2003

**DSM 918**  
To evaluate a community-based exercise intervention on both balance and functional capacity in stroke. Also, to evaluate the effect of the intervention on measures of health-related quality of life  
N=25 chronic stroke; x=63.13 yrs old; x=4.24 yrs post-stroke; 13 right, 12 left hemi  
- 2 baseline test sessions  
- 8-week exercise program (1hr, 3x/wk) of balance training, walking, and strength  
  - 1 post-test and 1 one-month retention test  
- Berg balance (BBS)  
- 12-min walk test  
- Self-paced and fast-paced gait speed  
- Self-paced and fast-paced stair climbing speed  
- RNL  
- COPM  
- BBS, gait speed, 12-min walk test, stair climbing, and COPM improved with exercise  
- RNL did not improve  
- Improvements demonstrated retention

### Engardt et al. 1995

**DSM 1051**  
To investigate the effect of isokinetic maximal voluntary knee extensions in persons with chronic stroke  
N = 10 stroke in eccentric group; x=62.2 yrs old; 7M, 3F; 7 left, 3 right hemi; x=26.5 months post-stroke;  
N = 10 stroke in concentric group; x=64.6 yrs old; 8M, 2F; 5 left, 5 right hemi;  
- Two strength groups: eccentric training and concentric training  
  - Trained paretic leg for 6-weeks (2x/wk) with Kin-Com isokinetic dynamometer  
- Integrated EMG  
- Concentric and eccentric muscle strength (joint torque) of knee extensors and flexors  
- Body-weight distribution on the legs while rising and sitting down  
- Gait speed (self-selected and fast-paced)  
- After training, knee extensor strength increased in eccentric and concentric actions in both groups  
- Eccentric and concentric strength in the paretic limb relative to the non-paretic increased in eccentric but not concentrically trained group  
- Antagonist
<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
<th>Participants</th>
<th>Intervention</th>
<th>Measures</th>
<th>Findings</th>
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<tbody>
<tr>
<td>Hesse et al. 1995</td>
<td>To investigate the efficiency of treadmill training with partial body-weight support (PBW) compared with gait training within regular physiotherapy in nonambulatory patients with chronic hemiparesis</td>
<td>N = 7 stroke inpatients; 6M, 1F; x=60.3 yrs old; x=176.8 days post-stroke (range 91-362 days...4 of 7 seven in chronic stage)</td>
<td>Treadmill training phase, regular PT based Bobath phase, another treadmill phase each lasting 3-weeks (15 sessions, 30-minute for treadmill and 45-min for physiotherapy)</td>
<td>Functional Ambulation Category (FAC), Rivermead Motor Assessment Index, Modified Ashworth Spasticity scale, Gait speed</td>
<td>Nearly perfect symmetrical body-weight distribution on the legs in the eccentric but not concentric group. No change in gait speed between groups. Eccentric training has some advantages over concentric training in persons with stroke.</td>
</tr>
<tr>
<td>Kim et al. 2001</td>
<td>To determine the effect of a 6-week maximal isokinetic strength program on the paretic lower extremity</td>
<td>N=20 chronic stroke (10 per group) Exp./ x=60.4 yrs old; x=4.9 yrs post-stroke; 7M, 3F; 4 right, 6 left hemi; Control/ x=61.9 yrs old; x=3.2 yrs post-stroke; 7M, 3F; 7 right, 3 left hemi</td>
<td>Pre and post-test measures</td>
<td>Functional Ambulation Category (FAC), Rivermead Motor Assessment Index, Modified Ashworth Spasticity scale, Gait speed</td>
<td>Lower extremity muscle strength (composite score), Walking velocity (slow and fast-paced), Health-related quality of life (SF-36), Stair climbing speed</td>
</tr>
<tr>
<td>Macko et al. 1997</td>
<td>To investigate the safety and efficacy of graded treadmill as an aerobic</td>
<td>N=9 stroke; x=67 yrs old; 0.7-6.7 yrs post;</td>
<td>Stress test</td>
<td>VO2, HR, Energy expenditure</td>
<td>Significant decrease in energy expenditure, HR, and VO2 after training</td>
</tr>
<tr>
<td>Study</td>
<td>Objective</td>
<td>Participants</td>
<td>Measures</td>
<td>Main Findings</td>
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<tr>
<td>Macko et al. 2001</td>
<td>To determine whether treadmill training will improve peak fitness while lowering energy cost of hemiparetic gait in chronic stroke</td>
<td>N=23 stroke; x=67 yrs old; 6-81 (x=28) months post; 19M, 4F</td>
<td>- Training consisted of 3, 40 min sessions weekly of treadmill walking at 60% HRR for 6 months - Treadmill exercise testing at sub-max with open circuit spirometry used to measure economy of gait at baseline, after 3 &amp; 6 months of training</td>
<td>- Gait economy - VO2 - RER - 21 completed 3-month &amp; 19 the 6-month - Economy of gait decreased &amp; peak VO2 &amp; fractional utilization increased after training</td>
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<tr>
<td>Miller 2001</td>
<td>To determine if combined body-weight supported (BWS) treadmill and overground training was feasible and effective in improving the functional capabilities of a patient with chronic stroke</td>
<td>N = 1 female, 71 yrs old, right hemi, 19 months post-stroke</td>
<td>- Baseline testing, treatment intervention introduced and removed, second baseline test, and one-month retention test - 3 sessions per day → BWS treadmill ambulation, overground ambulation with BWS, and overground ambulation without BWS - Each session was between 5 and 10 minutes - BWS percent was decreased over time - 3x/wk for 8 weeks of training</td>
<td>- Orpington Prognostic Scale (OPS) - FIM - Berg Balance - Step lengths - Motor Assessment Scale (MAS) - Gait speed (10-m walk test and 6-min walk test) - Berg Balance, MAS, Gait speed improved and retained</td>
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<tr>
<td>Miller et al. 2002</td>
<td>To report the feasibility and patient tolerance for using body-weight supported (BWS) treadmill and overground ambulation training and to measure the</td>
<td>N = 2 chronic stroke; 87 yrs old female, 10 yrs post-stroke; 93-yrs old female, 14 yrs post-stroke</td>
<td>- BWS ambulation 2-3x/wk for 6-7 weeks - 4 bouts of ambulation per session → first 3 consisted of ambulation on the treadmill with BWS and the</td>
<td>- Berg Balance - Tinetti Gait and balance assessment - 10-m timed walk - Step length - Step length ratio - One patient improved in Berg balance and 10-m walk time while the other improved in step length and 10-m walk time</td>
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<tr>
<td>Study</td>
<td>Aim</td>
<td>Design</td>
<td>Intervention Details</td>
<td>Outcome Measures</td>
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<tr>
<td>Monger et al. 2002</td>
<td>To investigate the feasibility and efficacy of a task-specific and home-based exercise protocol for improving sit-to-stand in stroke</td>
<td>N = 6 chronic stroke; x=65 yrs old; 4M, 2F; x=3.6 yrs post-stroke; 3 left, 3 right hemi</td>
<td>3-week home-based exercise program under supervision (3x/wk, 20 min)</td>
<td>- Sit-to-stand (STS) item of Motor Assessment Scale (MAS) - Gait speed - Grip strength - Mean vertical ground reaction force (VGRF) through limbs during STS task</td>
<td></td>
</tr>
<tr>
<td>Mudge et al. 2003</td>
<td>To investigate the influence of a period of body-weight supported treadmill training in a single subject with stroke on gait, balance, trunk control and general function</td>
<td>N = 1 chronic stroke; Male, 48 yrs old, left hemi, 30 months post-stroke</td>
<td>No treatment during initial 4-weeks, then body-weight supported treadmill training for 4-weeks (3x/wk), then no training for 4-weeks</td>
<td>- Gait velocity - Trunk control (Trunk control test) - Berg Balance - Functional Independence Measure (FIM) - Lateral reach test - Berg Balance increased during and at follow-up - Lateral reach for both arms improved during intervention (right also at follow-up) - Trunk control and FIM didn’t change</td>
<td></td>
</tr>
<tr>
<td>Potempa et al. 1995</td>
<td>To describe the responses of stroke patients to intense exercise and to determine the effect on cardiovascular and functional outcome measures</td>
<td>N=42 chronic stroke (n=19 in experimental group and n=23 in control group); 43-72 yrs old; at least 6-months post-stroke;</td>
<td>Experimental group = 10-week aerobic exercise program - Control group = 10-week passive ROM exercises</td>
<td>- HR - BP - VO2, VCO2, VE, RER - Exercise time - Sensorimotor function (Fugl-Meyer) - Workload - Experimental group increased in VO2, workload, and exercise time and decreased SBP at sub-max</td>
<td></td>
</tr>
<tr>
<td>Rimmer et al. 2000</td>
<td>To determine the effects of a 12-week exercise training program in a predominantly African-American group of stroke survivors with multiple comorbidities</td>
<td>N=35 strokes; 9M, 26F; x=53.2 yrs old; 31 African-Americans</td>
<td>Screened blood, performed graded exercise test and fitness testing - Peak VO2 recorded electronically via a braked upright stationary cycle - Strength tested on LifeFitness bench press and seated leg press machine</td>
<td>- Peak VO2 - 10RM for bench press and leg press - Flexibility - Body composition - Increase in absolute and relative peak VO2 after training and decrease for controls - Increase in gains of time to exhaustion and max. workload post training - Increase in bench and leg press post training</td>
<td></td>
</tr>
<tr>
<td>Rodriguez et al. 1996</td>
<td>1) Further substantiate the benefits of post-acute gait training, (2) evaluate the efficacy of a home-based gait training model (Wisconsin Gait Scale, WGS), (3) assess the relation of improved gait to patients’ perceptions of their health status and well being, (4) characterize which factors associated with stroke &amp; rehab are predictors of outcome, &amp; (5) determine cost of home-based training</td>
<td>N=18 stroke; 31-78 (x=54) yrs old; 12M, 6F; 2.2 yrs post</td>
<td>- Pre/post test lag control group design - Exercise program → 1hr/session, 3x/wk for 12 wks; cardiovascular endurance (30min), muscle strength and endurance (20 min) and flexibility (10min); during 1st 2 wks, participants went through educational program; 70% 10RM for one set of 15-20 reps than increased 10% 10RM later</td>
<td>- WGS → see paper for tasks - Falls Efficacy Scale (FES) and HSQ test - Cost</td>
<td>- Increase in gains in hamstring and low back flexibility - Decrease in body weight, BMI, and total skin folds - Suggest education on signs of stopping are important</td>
</tr>
</tbody>
</table>

| DSM 5 |  |  |  |  |  |

<p>| Sharp and Brouwer 1997 | To determine whether persons with chronic hemiparesis due to stroke can improve function and muscle strength at an isolated | N = 15 chronic stroke; 10M, 5F; x=67 yrs old; 0.9-18 yrs post-stroke; 8 left, 7 right hemi | - Tested baseline, post-intervention, and 4 weeks after training - Intervention → 3x/wk over 6-weeks consisting of a warm-up, | - Muscle strength - Spasticity (pendulum test) - Gait speed - Timed Up and Go test - Timed stair climbing | - Paretic muscle strength improved after training while tone remained consistent but only quadriceps torque was still increased at follow-up - Gait velocity |</p>
<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Intervention</th>
<th>Measures</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Silver et al. 2000</td>
<td>N=5 stroke; 5M; x=60.4 yrs. old; x=26 months post stroke; 4 right, 1 left hemi</td>
<td>- Aerobic exercise on treadmill 3x/wk, progressed to 40 min/session at 60-70% max HRR for 3 months - Pre- and post-testing</td>
<td>- Human Activity Profile scores</td>
<td>- Increased after training and at follow-up - Stair climbing and Timed Up and Go were not different - Paretic spasticity remained constant</td>
</tr>
<tr>
<td>Smith et al. 1998b</td>
<td>N = 14 chronic stroke; 12M, 2F; x=66 yrs old; x=19 months post-stroke: 6 left, 7 right hemi, 1 bilateral</td>
<td>- Exercise consisted of progressive low to moderate intensity aerobic exercise treadmill training 40-min/day, 3x/wk, for 3-months at 60-70% HRR - Pre- and post-testing</td>
<td>- Reflexive (passive) &amp; volitional (concentric &amp; eccentric) torque from bilateral quadriceps obtained using isokinetic dynamometer with torque measured at 30°, 60°, 90°, &amp; 120°/s angular velocity - Torque was determined between 35° and 45° of knee flexion</td>
<td>- Increase in mean eccentric torque generation following the exercise intervention of 50% and 25% for the paretic and non-paretic limb, respectively - Increase in mean concentric torque following intervention of 38% and 17% for the paretic and non-paretic limb, respectively - Reflexive torque decreased in both limbs but didn’t reach significance - Eccentric torque higher at each angular velocity than concentric torque - “Task-oriented” treadmill training good for individuals with chronic stroke</td>
</tr>
</tbody>
</table>

Note: The table provides a summary of the study designs, participants, interventions, measures, and findings related to joint strength training of the affected lower extremity following a training program and whether gains are associated with alterations in muscle spasticity.
<table>
<thead>
<tr>
<th>Year</th>
<th>Study Details</th>
<th>Participants</th>
<th>Intervention</th>
<th>Outcome Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>1999</td>
<td>Smith et al.</td>
<td>N=11 stroke; 10M, 1F; x=23.7 months post</td>
<td>Exercise protocol → 3 months (i.e. 12 weeks), 3x/wk, progressively graded treadmill aerobic exercise (AEX) training program; 5 min warm-up and cool-down at 30% heart rate reserve (HRR); limited to 40% HRR for 10-20 min at onset of study to 60-70% HRR for 40 min later</td>
<td>Reaction times (RXT) → initial loss of contact after perturbation; Recovery times (RCT) → post-perturbation time at which full foot contact is reestablished bilaterally for at least 300 msec; Movement times → (RXT-RCT)</td>
</tr>
<tr>
<td>2000</td>
<td>DSM 225</td>
<td>12M, 2F; x=66 yrs old; greater than 6 months post; 7 right, 6 left, 1 bilateral</td>
<td>Intervention → treadmill walking 3x/wk, progressed to 40 min/session for 3 months (i.e. 12 weeks) at 60-70% heart rate reserve</td>
<td>Pre- and post-testing (passive) &amp; volitional (concentric &amp; eccentric) torque from bilateral hamstrings obtained using isokinetic dynamometer with torque measured at 30°, 60°, 90°, &amp; 120°/s angular velocity</td>
</tr>
<tr>
<td>DSM 97</td>
<td>To investigate the effect of a treadmill exercise training paradigm on equilibrium reactions and recovery times after standardized translational balance perturbations</td>
<td>N=11 stroke; 10M, 1F; x=23.7 months post</td>
<td>- Exercise protocol → 3 months (i.e. 12 weeks), 3x/wk, progressively graded treadmill aerobic exercise (AEX) training program; 5 min warm-up and cool-down at 30% heart rate reserve (HRR); limited to 40% HRR for 10-20 min at onset of study to 60-70% HRR for 40 min later</td>
<td>No change pre-to post in all measures (RXT, RXT, MVT)</td>
</tr>
<tr>
<td>Study</td>
<td>Objective</td>
<td>Participants</td>
<td>Methods</td>
<td>Findings</td>
</tr>
<tr>
<td>-------------------------------</td>
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<td>-----------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Sullivan et al. 2002</td>
<td>To determine whether faster treadmill training speeds would result in better transfer to overground walking in stroke patients</td>
<td>N = 24 stroke; 5F, 19M; 34-81 (x=67) yrs; 6-62 (x=25.8) months post; 16 left, 8 right hemi</td>
<td>Stratified into a category by locomotor severity based on self-selected gait speed. Groups: slow, variable, and fast velocity. Training program: 12 sessions over 4-5 wks, 20 min/session. Pre-, post-, and 3-month retention testing. No orthosis allowed.</td>
<td>- Total Fugl-Meyer motor score (TFM) - Lower extremity Fugl-Meyer (LEFM) - SF-36 - Self-selected overground walking speed over 10m (SSV)</td>
</tr>
<tr>
<td>Tangeman et al. 1990</td>
<td>To investigate the changes in functional level that would occur after intensive rehabilitation therapy for stroke patients who were at least one year post stroke</td>
<td>N=40 stroke; 13M, 27F; 27-77 (x=65.6) yrs old; 1-23 (x=3.09) yrs post;</td>
<td>4-week training program → 2 hrs/session, 4x/wk individual treatment by PT &amp; OT. 1 month pre (to serve as control period), pre-, post-, &amp; 3 months post-testing. Therapy program focused on weight shift to the affected side, balance, and functional activities.</td>
<td>- Balance using a 10-point scale - Weight shift → # of objects moved in 20 seconds - ADL</td>
</tr>
<tr>
<td>Teixeira-Salmela et al. 1999</td>
<td>To evaluate the impact of a combined program of physical conditioning &amp; muscle</td>
<td>N=13 stroke; 1-34 yrs post; 7 left, 6 right hemi; 7M, 6F</td>
<td>Randomly assigned into experimental or control group. Pre- &amp; post testing. 10-week</td>
<td>- Gait speed (22m) - Human activity profile (HAP) → adjusted using metabolic energy</td>
</tr>
</tbody>
</table>
strengthening on reducing impairments and disability in subjects with chronic stroke

training program → 3x/wk, 60-90 min/session
- Control group no intervention and then did training protocol after
  - Training program → 5-10 min warm-up, aerobic exercises consisting of graded walking plus stepping or cycling at 70% max HR, strength training, cool-down for 5-10 min
- Strength training → isometric, concentric & eccentric contractions; body weight, sandbag weights, and elastic bands
- Consumption
  - Quality of life by Nottingham Health Profile (NHP)
  - Lower-extremity muscle strength using Cybex II isokinetic dynamometer
  - Muscle tone

Teixeira-Salmela et al. 2001

To evaluate the impact of a combined program of muscle strengthening and physical conditioning on gait performance in individuals with chronic stroke

N = 13 chronic stroke; 6F, 7M; x=67.7 yrs old; x=7.7 yrs post-stroke; 7 left, 6 right hemi
- Pre- and post-testing
- Intervention → 3x/wk, 60-90 min, over 10-weeks
- Were given a list of exercises to do at home and told to do them at least 3x/wk
- Community-based group program of warm-up, aerobic exercise, strength training, and cooldown
  - Aerobic exercise → 1st 5 wks at 50-70% of aerobic capacity for 10-20 minutes and increased to 70% capacity for 2nd 5 wks; consisted of graded walking plus stepping or cycling
  - Strength training → progressive

- Gait speed
- Gait kinetics and kinematics

DSM 408

- Gait speed improved
- Gait improved
- Able to generate higher levels of powers during gait
- Demonstrated increases in positive work performed by ankle plantar flexors and hip flexor/extensor muscles
<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Study Details</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trueblood</td>
<td>2001</td>
<td>To examine the effects of partial body-weight (PBW) ambulation in people with chronic stroke</td>
<td>Series of pilot studies with chronic stroke for 30-min for hip, knee, and ankle joint</td>
<td>Compared gait characteristics during 3 modes of walking: level ground ambulation, level ground ambulation with PBW, and treadmill ambulation with PBW. Examined effects of repeated (6-8 weeks) PBW treadmill training during level ground ambulation. See article for details</td>
</tr>
<tr>
<td>Weiss et al.</td>
<td>2000</td>
<td>To evaluate the effects of a high intensity resistance strength training intervention on bilateral lower limb strength, performance, &amp; clinical outcome measures in individuals with stroke</td>
<td>N=7 stroke; x=70 yrs old; x=2.3 yrs. post; 5 right, 2 left hemi</td>
<td>Baseline testing twice separated by 5 days. Exercise training program: 12 wks, 2x/wk. Standing hip flexion, abduction, and extension with machines, sitting knee extension and press, 3 sets of 8-10 reps at 70% 1 rep max (1RM). 1RM measured every 2 wks.</td>
</tr>
<tr>
<td>ARTICLE</td>
<td>PURPOSE</td>
<td>SUBJECTS</td>
<td>PROTOCOL</td>
<td>OUTCOME MEASURES</td>
</tr>
<tr>
<td>----------------------</td>
<td>--------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
<td>--------------------------------------------</td>
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</tbody>
</table>
| Barbeau and Visintin | To identify stroke patients who are most likely to benefit from locomotor training with body-weight support (BWS), to determine the extent of carryover from treadmill training to overground locomotion, and to determine the variables that are most likely to influence the recovery of locomotion | N = 50 stroke patients (43 finished) in BWS group; x=66.5 yrs old; 19F, 31M; 20 right hemi, 30 left; x=68.1 days post-stroke; N = 50 stroke patients (36 finished) in no-BWS group; x=66.7 yrs old; 22F, 28M; 29 right, 21 left hemi; 78.4 days post-stroke | - Both groups trained 4x/wk for 6-weeks (max. 3 walking trials for no more than 20-min)  
- BWS group trained with BWS on treadmill  
- BWS progressively decreased over time  
- Tested pre-, post-, and 3-months retention | - Berg Balance  
- Motor recovery (STREAM)  
- Walking speed  
- Walking endurance (10m walk) | - BWS group scored significantly higher in all clinical measures  
- When stratified according to initial walking speed, endurance, balance, and motor recovery, more severely impaired stroke patients showed Time effect  
- Older adults in BWS group increased walking speed more than older adults in no-BWS group |
| DSM 1027             |                                                                          |                                                                          |                                                                          |                                            |                                                                          |
| Duncan et al. 1998   | 1) To develop an exercise intervention based on principles of exercise physiology & motor learning and to deliver it in the home to individuals with mild or moderate stroke, (2) evaluate the feasibility and (3) assess the effects | N=20 stroke; 30-90 days post; 11 left, 8 right hemi | - After baseline assessments, the subjects were randomly assigned to experimental or control group  
- Intervention → exercise program for 12 weeks, 3x/wk, 1.5 hrs/session that was home-based (supervised for first 8 weeks – all 3x/week); 10 min warm-up, assistive or resistive exercises using PNF patterns or theraband, balance exercises, upper extremity functional activities, progressive walking or bicycle ergometer  
- Control → 2x/wk visit to home for assessment of activity & exercise level only | - 6 min walk  
- BBS  
- Jebson test of hand function  
- Barthel and Lawton ADL scales  
- 10 meter walk  
- Fugl-Meyer motor score | - Increase in lower-extremity Fugl-Meyer motor score for experimental group  
- Gait speed increased more for experimental group  
- ADL showed no differences |
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Participants</th>
<th>Measurements</th>
<th>Findings</th>
</tr>
</thead>
</table>
| Duncan et al. 2003 | To determine the effect of a structured, reproducible, physiologically based exercise program on strength, balance, endurance, and upper limb function in persons with stroke | N=50 acute stroke (intervention); x=68.5 yrs old N=50 acute stroke (control); x=70 yrs old; Approximately 75 days post-stroke | - Pre- and 3-months following intervention testing  
- Intervention = home-based for 36 sessions of 90-min. over 12-14 weeks focusing on balance and strength | - Both groups improved but control no in endurance  
- Intervention group improved more in VO2, BBS, 6-min walk distance, and gait speed |
| DSM 956          |                                                                         |                                                                                |                                                   |
| Hocherman et al. 1984 | To train hemiparetic individuals to sustain posture by using platform movements | N=24 stroke; anterior cerebral circulation; 10-21 days post; x=72.7 yrs old  
Treatment group = N=13; 6F, 7M; 8 right and 5 left CVA  
Control group = N=11; 5F, 6M; 6 right, 5 left CVA | - Platform moved at 0.5 Hz within a range of 1-16 cm  
- Treatment group underwent 15 training sessions over 3 wks consisting of two 5 min parts (one with patients standing parallel to axis of movement and one with patients perpendicular)  
- AP and ML perturbations | - MMA change in treatment group was larger than control (i.e. better stability)  
- Patients with lowest initial ability showed better improvements  
- IEMG of TA was similar in both legs during training but affected limb TA was minimal during quiet standing  
- Improved weight distribution on the feet in both groups (more in treatment group) |
| DSM 481          |                                                                         |                                                                                |                                                   |
| Moreland et al. 2003 | To investigate whether lower-extremity strength-training exercises plus conventional physiotherapy is more effective than conventional therapy alone | N = 68 stroke in experimental group; 39M, 29F; 27 left, 35 right, 5 bilateral, 1NA hemi; x=69.1 yrs old; x=36.8 months post-stroke;  
N = 65 stroke in control group; 42M, 23F; 31 left, 27 right, 6 bilateral, 1 NA hemi; x=72 yrs old; x=38.1 months post-stroke | - Tested at 4-weeks, discharge, and 6-months following  
- Intervention ➔ 3x/wk, 30-minutes for duration of rehab stay; progressive resistance exercises with weights  
- Control group exercises same but with no resistance | - No differences between groups |
| DSM 1025         |                                                                         |                                                                                |                                                   |
| Richards et      | To determine                                                             | N = 27 acute                                                                   | - Randomly                                        | - Fugl-Meyer - PT time |
whether early and intensive physiotherapy that emphasized gait training promoted a gait outcome and an early return of functional mobility that was superior to that obtained following either early and intensive conventional physiotherapy that did not focus on ambulation or conventional physiotherapy

Suzuki et al. 1999

To investigate biomechanical determinants and predictors of walking speed in early gait training after stroke

N=34 stroke; 34M; 19 right, 15 left hemi; x=8.6 wks post

- Received computer-assisted gait training (CAGT) for 8 wks, 4-5x/wk
- Pre- & post-testing
- Testing after 4 wks as well

Balance, arm, and leg scores
- Barthel Ambulation
- Berg Balance
- Gait velocity

Balance, arm, and leg scores
- Gait velocity

highest in experimental group
- Gait velocity similar between two control groups after 6-weeks but faster in experimental group (thus timing not important)
- Effects not retained at retention testing of 3 and 6 months
## Appendix III: Literature table – Postural control following stroke

### Table III.1: Postural control following stroke

<table>
<thead>
<tr>
<th>ARTICLE</th>
<th>PURPOSE</th>
<th>SUBJECTS</th>
<th>PROTOCOL</th>
<th>OUTCOME MEASURES</th>
<th>RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Al-Zamil 1998</td>
<td>To identify posturographic methods that can be used to quantify symptoms associated with postural deficit</td>
<td>N=24 stroke; 13M, 11F; x=63 yrs old; x=2.9 weeks post; N=25 age-matched controls; x=62.4 yrs old</td>
<td>Motor control test using dynamic posturography platform (Equitest, Neurocom Int.) 9 forward &amp; 9 backward movements of 3 magnitudes (1.25, 3.15, and 5.7 cm) in 250, 300, and 400 msec</td>
<td>Onset latency of response, strength (force relative to rate of increase in ankle torque over 150 msec), and symmetry (force exerted by each leg against force plates) of limb response</td>
<td>Latencies were abnormal in 75% of patients; Non-affected limb had normal latencies but affected limb latencies were significantly delayed and of decreased amplitude if present at all</td>
</tr>
<tr>
<td>DSM 364</td>
<td>To identify posturographic methods that can be used to quantify symptoms associated with postural deficit</td>
<td>N=10 stroke; 6M, 4F; right hemi; x=54 yrs old; 8 of 10 -&gt; 6 months (&lt;1 yr) post; 2 of 10 -&gt; &gt; 1 yr post</td>
<td>Platform on wheels that displaced 15 cm in AP at constant acceleration EMG -&gt; MG, TA, Hams, Quads Sway recorded Stood on platform barefoot Perturbation order for all subjects: 1) 2 trials forward displacement, 2) 1 trial backward, 3) 1 trial forward, 4) 2 trials backward</td>
<td>Fugl-Meyer used to group patients EMG onset, amplitude and sequencing Sway and weight distribution</td>
<td>Hemiparetics showed frequent co-contraction of all 4 muscles in both perturbation directions; Inconsistent ratios of intermuscular activation in hemiparetics; Distal to proximal sequence in normals; No clearly defined sequence in hemiparetics; Large variability with hemiparetics; Lower the lower-extremity Fugl-Meyer, the more abnormal findings of postural adjustments</td>
</tr>
<tr>
<td>Badke &amp; Duncan 1983</td>
<td>To describe the patterns of postural adjustments during induced body sway in healthy and hemiparetic subjects</td>
<td>N=10 normals; 3M, 7F; x=35 yrs old</td>
<td>Platform on wheels that displaced 15 cm in AP at constant acceleration EMG -&gt; MG, TA, Hams, Quads Sway recorded Stood on platform barefoot Perturbation order for all subjects: 1) 2 trials forward displacement, 2) 1 trial backward, 3) 1 trial forward, 4) 2 trials backward</td>
<td>Fugl-Meyer used to group patients EMG onset, amplitude and sequencing Sway and weight distribution</td>
<td>Hemiparetics showed frequent co-contraction of all 4 muscles in both perturbation directions; Inconsistent ratios of intermuscular activation in hemiparetics; Distal to proximal sequence in normals; No clearly defined sequence in hemiparetics; Large variability with hemiparetics; Lower the lower-extremity Fugl-Meyer, the more abnormal findings of postural adjustments</td>
</tr>
<tr>
<td>DSM 214</td>
<td>To determine whether prior knowledge of the direction of the balance perturbation improved neuromuscular sequencing during postural adjustments</td>
<td>N=10 stroke; 6M, 4F; x=56 yrs old; right hemi; 3-29 months post; N=5 normals; 2M, 3F; x=47 yrs old</td>
<td>3 phases: (1) voluntary AP sways, (2) balancing during unexpected support surface displacements, &amp; (3) support surface displacements with prior knowledge of direction</td>
<td>Bilateral EMG of TA, Quads, Gastrocs, and Hamstrings for latency, sequencing, and amplitude</td>
<td>Hemiparetics had longer and more variable onsets in the paretic limb during voluntary AP sway; Prior knowledge had no effect on responses in normals; Prior knowledge showed</td>
</tr>
</tbody>
</table>
Berger et al. 1984

<table>
<thead>
<tr>
<th>Trials</th>
<th>Shorter onsets in paretic limb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase 1: 6 randomized trials where verbally told to rapidly shift posture anteriorly or posteriorly</td>
<td>Default responses (no clear onsets) in both groups less with prior knowledge</td>
</tr>
<tr>
<td>Phase 2: 12 trials of 5 cm/s AP perturbations</td>
<td>Proximal-distal for paretic limb even with knowledge whereas reverse for normals</td>
</tr>
<tr>
<td>Phase 3: 12 trials with warning tone and prior knowledge of which 8 were told correct info</td>
<td></td>
</tr>
</tbody>
</table>

To analyze the activity and functional significance of mono- and poly-synaptic reflexes during normal and disturbed gait, and to evaluate their contributions to the development of tension in the triceps surae to compensate for body load after impact.

N=15 spastic hemiparesis (11 from stroke); 39-74 (x=54) yrs old

- Treadmill walking (1.5-2.5 km/hr)
- Gauge fixed laterally near Achilles tendon
- 3 patients randomly displaced at distinct step cycle phases (increase to 7.5 km/hr in 100ms)
- Comparison using acceleration during standing on treadmill

- TA and MG activity
- Ankle joint angle
- Tension in Achilles tendon

- Mean amplitude of spastic side ankle joint movement was half of that of unaffected side
- Reduction of EMG strength was correlated with the severity of paresis
- When perturbation induced to unaffected limb, strong MG response (65-75 ms onset with respect to ankle joint movement) but when induced to spastic limb, small biphasic potentials appeared with 40 ms onset
- Reciprocal modulation of mono- and poly-synaptic EMG responses during gait → normals have reduced mono but not spastic hemiparetics
- No connection between exaggerated monosynaptic reflexes and hypertonia → may be due to change in

DSM 497

- TA and MG activity
- Ankle joint angle
- Tension in Achilles tendon

- Mean amplitude of spastic side ankle joint movement was half of that of unaffected side
- Reduction of EMG strength was correlated with the severity of paresis
- When perturbation induced to unaffected limb, strong MG response (65-75 ms onset with respect to ankle joint movement) but when induced to spastic limb, small biphasic potentials appeared with 40 ms onset
- Reciprocal modulation of mono- and poly-synaptic EMG responses during gait → normals have reduced mono but not spastic hemiparetics
- No connection between exaggerated monosynaptic reflexes and hypertonia → may be due to change in
<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Experiment Description</th>
<th>Participants</th>
<th>Results</th>
<th>Notes</th>
</tr>
</thead>
</table>
| Berger et al. 1988 | To evaluate the extent to which reflexes and the programmed pattern are impaired in spastic paresis following treadmill perturbations | N = 11 patients; x = 52.1 yrs old; 8 had vascular lesion | Forward and backward treadmill accelerations during standing (random order)  
- 4 ramp accelerations →  
  5.5 m/s², 4 cm; 11.1 m/s², 7 cm; 16.6 m/s², 10.5 cm; 22 m/s², 15 cm | TA and MG muscle activity  
- Delayed MG response of spastic side  
- Steepness of increase in activity depended on acceleration for TA and MG for respective directions  
- Co-activation with higher accelerations  
- Two parts to compensatory response to stance perturbations: functionally directed early EMG response mediated by stretch reflexes which are impaired in spastic paresis, followed by a triggered complex component which is unchanged in patients (motor programs are intact) |
| Chaudhuri & Aruin 2000 | To determine whether a lift applied to the nonparetic limb would result in improved symmetry of weight bearing during dynamic postural perturbations in hemiparetic patients | N = 10 stroke; 7M, 3F; 68.7 yrs old; 8.2 wks post; 7 right, 3 left hemi; | Used EquiTest system with medium (0.026 m/deg) and large (0.048 m/deg) perturbations  
- Stood on force plates  
- 1st series → no lifts  
- 2-4 series → stood with lifts under non-affected limb  
- 4 translations for no lift and each of the 3 lifts (0.6, 0.9, & 1.2 cm) and was repeated 3x with order of lifts random | Weight symmetry scores = (RF + RR / LF + LR + RF + RR) *200, where right and left frontal and rear load cells used  
- Latency of response → onset of active response  
- Strength of response → active force generated | Lifts improved symmetry of weight bearing  
- No lifts → faster onset latency and stronger response for non-paretic limb to perturbations  
- Tendency for small lift to decreased latency in paretic limb  
- Tendency for lifts to increase strength of response of paretic limb |
| Di Fabio & Badke 1988 | To examine the effects of a rapid length change on the moveable platform that moved unexpectedly in a | N = 4 stroke; right hemi; 3M, 1F; 43-71 yrs old | Stood on moveable platform  
- TA and MG EMG → onset, zero onset frequency,  
- Normals agonist-antagonist burst onset separated by −10 to |
<table>
<thead>
<tr>
<th>Elongated and N=4 normals; horizontal amplitude -15 msec</th>
<th>Shortened ankle 1M, 3F; 39-45 yrs old</th>
<th>12-15 msec separated by 200 msec</th>
<th>Right and left limbs</th>
<th>4M, 4F; 4-31 yrs old</th>
<th>N=8 stroke; 31-73 yrs old; see Di Fabio et al. 1986</th>
</tr>
</thead>
<tbody>
<tr>
<td>3M, 4F; 4-31 yrs old; right hemiplegia</td>
<td>4M, 4F; 4-31 yrs old; right hemiplegia</td>
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<td>Supraspinal and segmental mechanisms related to phasing of opposing muscles in a stroke population.</td>
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<td>Initial long-latency response (LLR) in muscle segmental stretch by gastrocnemius corresponded with initial LLR.</td>
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</tr>
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<td>Muscle onset latency and latency response relationship were explored in normals and stroke patients.</td>
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</tr>
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<td>Amplitude of short-latency response (AR) was significantly delayed with respect to non-paretic limb and antagonist limb in paretic limb.</td>
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<td>Greater disassociation between MG and TA was observed in normal control subjects compared to stroke patients.</td>
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<tr>
<td>To examine the interaction of the hemiplegia on muscle onset latency and latency response relationship.</td>
<td>To examine the interaction of the hemiplegia on muscle onset latency and latency response relationship.</td>
<td>To examine the interaction of the hemiplegia on muscle onset latency and latency response relationship.</td>
<td>To examine the interaction of the hemiplegia on muscle onset latency and latency response relationship.</td>
<td>To examine the interaction of the hemiplegia on muscle onset latency and latency response relationship.</td>
<td>To examine the interaction of the hemiplegia on muscle onset latency and latency response relationship.</td>
</tr>
</tbody>
</table>
Di Fabio and Badke 1989

DSM 716

<p>| To evaluate the consistency of postural muscle recruitment in several different directions of volitional body sway in a non-choice reaction time task | N = 4 stroke; x=54 yrs old; right hemiparesis N = ?? healthy controls; x=52 yrs old | - Simple reaction time task of visual tracking target via body sway - Measured EMG from bilateral TA and MG | - Default response (i.e. no EMG activity) - Postural reaction time (time between visual target and change in force for body sway) - EMG onset | - Stroke had higher number of default or zero onset responses - Normals got faster with practice but stroke got slower - Stroke tended to recruit muscles in an all-or-none fashion - Tonic rather than phasic burst patterns dominated in stroke - Suggest stroke have to rely more on conscious cortical modulation apparent to control amplitudes and latencies and LLR - AR coupling rather than segmental or vestibular pathology - Difficulty recruiting proximal synergists |</p>
<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
<th>Participants</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Di Fabio et al. 1986</td>
<td>To study symmetry and adaptability of a postural response following stroke</td>
<td>N=4 stroke; x=60 yrs old; 3M, 1F; 5-30 months post; lower extremity Fugl-Meyer score = 78-98; right hemi; N=5 normals; x=46.4 yrs old; 2M, 3F;</td>
<td>- Participants stood on platform and had 6-12 practice trials - 3 horizontal displacements (5cm/s) given at random intervals within 2 min with 3 rotational perturbations (8°/s) randomly interspersed - EMG of bilateral TA, Quads, Gastrocs, and hamstrings</td>
<td>- Integrated EMG amplitudes over 100 ms - EMG onsets and activation patterns - Tendency for strokes to initiate from non-paretic limb (proximal muscles) - Latency modulation appeared to be organized in a diagonal fashion - Significantly delayed non-paretic distal limb activation - Non-paretic proximal limb latencies were shorter - Latencies ranged from 124-187 ms - Normal attenuation of synergistic muscle activity to rotations in strokes - Distal to proximal activation (i.e. inverted pendulum) in both groups - Greater onset difference between distal and proximal muscles for non-paretic limb indicating co-contraction for paretic - Normal paretic proximal onset and non-paretic distal onset - Delayed paretic distal onset with compensation via early non-paretic proximal onset</td>
</tr>
<tr>
<td>DSM 95</td>
<td></td>
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</tr>
<tr>
<td>DSM 715</td>
<td>To determine how the onset of postural reflexes compared to the conscious identification of stroke</td>
<td>N = 5 stroke; x=58 yrs old; 3F, 2M; 1-24 month post-stroke</td>
<td>- Backward platform displacements → 1-9 cm displacement, 5-45 cm/s velocity, 80-</td>
<td>- Thenar muscle and MG activity - Zero (default) responses - Reaction time - Although not significant, there was a delay in RT for stroke - No difference between groups for</td>
</tr>
<tr>
<td>Dickstein et al. 1989a</td>
<td>Assess the effect of weight shift over the affected leg of hemiparetic patients on two attributes of postural responses during continuous AP sinusoidal movements of the base of support: (1) adaptation of the activation pattern of the MG and TA muscles to the imposed movements and (2) relative amount of integrated EMG activity of these muscles</td>
<td>N=10 strokes; 5M, 5F; x=61 yrs old; 7 right, 3 left hemi N=9 healthy adults: 4M, 5F; x=55 yrs old</td>
<td>- AP perturbations at 0.5 Hz level and with one leg elevated on a step monitoring MG and TA</td>
<td>- Modulatory Index (MI) → ratio between IEMG (area under curve) of each muscle during the time corresponding to forward half cycle of movement and the total IEMG during the whole cycle</td>
</tr>
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<td>---</td>
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</tr>
</tbody>
</table>
| Dickstein et al. 1989b | To examine the effect of motor set on the postural adjustments required to maintain balance during continuous displacements of the base of support and to compared these adjustments to hemiplegic patients | N=17 stroke; x=71.8 yrs old; x=2.4 months post; 11 left hemi, 6 right hemi; 11M, 6F; N=42 normals; 20-84 yrs old; 21M, 21F; | - Subjects stood on moveable platform → AP translations (sinusoidal) fixed at 0.5 Hz, 1-11 cm amplitude, 3.22-35.42 cm/s velocity, 10.37-114.07 cm/s² acceleration | - Bilateral TA and MG EMG responses | - Bilateral TA and MG EMG responses | - Descriptive responses mainly | - Anticipatory responses in normals | - MMA was 3cm for cortical stroke and 1.25 cm for vertebral artery stroke | - Affected limb cyclic reciprocal pattern was lost in strokes → complete (or nearly) silence of TA and variable amounts of cyclicity of MG; low amplitude co-contraction of
<p>| Dietz &amp; Berger 1984 | To analyze the impairment of interlimb coordination in patients with a supraspinal lesion of the motor system to obtain information as to the influence of higher motor centers on the spinal coordinating mechanisms | N=15 normals N=12 spastic hemiparetics due to stroke (7) or other causes (5) N=12 spastic paraparetics due to cervical spondylotic myelopathy (6) or myelitis (3) or unknown Age for patients 30-65 (x=46 yrs old) | - Stood eyes open for 90 seconds on seesaw and then single supramaximal electrical stimuli (2msec) were randomly applied to tibial nerve in either spastic or healthy leg - 5 patients also had see-saw apparatus they were standing on move suddenly that mimicked the displacement caused by electrical stimulation | - EMG of TA and MG bilaterally - Force changes on force plate - Twitch contraction of triceps surae | antagonists - 4-5 Hz balancing oscillations on unaffected side and under 1 Hz for spastic side - Less activity in affected side - Healthy showed simultaneous bilateral responses and same amplitude to unilateral displacement for both stimulation and see-saw triggered movements - Reduced amplitude and delayed responses in spastic side (20-30 msec) regardless of side perturbed - Not simultaneous activation when stimulated or triggered for hemiparetics - Latency of H-reflex not different between sides (i.e. nerve conduction velocities same) - Reduced amplitude and delayed onset for paraparetics - Twitch amplitude same but delayed for spastic side - Impaired responses in spasticity due to a dysfunction of a spinal interneuronal system from a loss of supraspinal control |
| DSM 428 | Hocherman et al. 1984 | To train hemiparetic | N=24 stroke; anterior | - Platform moved at 0.5 Hz | - Average integrated EMG | - MMA change in treatment group |</p>
<table>
<thead>
<tr>
<th>DSM 481</th>
<th>individuals to sustain posture by using platform movements</th>
<th>cerebral circulation; 10-21 days post; x=72.7 yrs old</th>
<th>within a range of 1-16 cm of TA</th>
<th>was larger than control (i.e. better stability)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment group = N=13; 6F, 7M; 8 right and 5 left CVA</td>
<td>- Treatment group underwent 15 training sessions over 3 wks consisting of two 5 min parts (one with patients standing parallel to axis of movement and one with patients perpendicular)</td>
<td>- Maximal movement amplitude (MMA)</td>
<td>- Patients with lowest initial ability showed better improvements</td>
</tr>
<tr>
<td></td>
<td>Control group = N=11; 5F, 6M; 6 right, 5 left CVA</td>
<td>- Maximal movement amplitude (MMA)</td>
<td>- Body weight distribution (i.e. asymmetry)</td>
<td>- IEMG of TA was similar in both legs during training but affected limb TA was minimal during quiet standing</td>
</tr>
<tr>
<td>Hocherman et al. 1988</td>
<td>To see whether anticipatory adjustments (and strategies used) occur with repetitive reciprocal perturbations during stance and is the inability of hemiplegic patients to withstand these perturbations due to a reduction in the effectiveness of postural responses or a change in the strategy</td>
<td>N=21 elderly; x=68 yrs old; N=15 stroke; 9 right, 6 left hemi; 5-12 weeks post;</td>
<td>- Stood on platform for 5 min that moved at 0.5 Hz</td>
<td>- Anticipatory reactions from TA and MG were seen in the elderly</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Collected EMG for 60 sec after 2 min into time (thus allowed practice time)</td>
<td>- TA and MG activity onsets, average EMG, area under curve, modulation index (ratio between backwards and forwards displacements)</td>
<td>- Subjects adapted a leaning strategy</td>
</tr>
<tr>
<td>Holt et al. 2000</td>
<td>To compare balance responses of stroke vs. controls to external perturbations to the lateral side of the pelvis</td>
<td>N=21 (13 used) strokes; right hemi; range 22-77 yrs old; x=86.4 wks post; N=15 controls; range 17-80 yrs.</td>
<td>- Lateral perturbations applied via a waist belt by a machine</td>
<td>- Stroke differed from elderly in that tonic contractions were present in MG or TA, co-contractions were seen</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Alternate blocks of 5 pushes (left, right, etc.) until 20 pushes each direction</td>
<td>- Pelvic displacement (sway)</td>
<td>- Stroke had abnormal stance strategies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- 3% body weight force applied</td>
<td>- Ground reaction force onset</td>
<td>- GRF onset increased for stroke compared to controls</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- 27-30 cm apart at 5th toes and 10 cm at heel</td>
<td>- 10 m timed walk</td>
<td>- No correlations between measures and functional assessments</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Functional reach with unaffected hand</td>
<td>- Message: latency of GRF</td>
</tr>
</tbody>
</table>
| **Ikai et al. 2003** | **To evaluate dynamic postural control in patients with hemiparesis and in normal subjects matched for age** | **N = 59 stroke; 39M, 20F; x=61.5 yrs old; 32 right, 27 left hemi; x=11.3 months post-stroke** | **- EquiTest system used and motor control and adaptation tests used**  
- Motor control test consisted of 3 trials of small (5 cm/s, 250 msec), medium (10 cm/s, 300 msec), and large (15 cm/s, 400 msec) forward and backward perturbations  
- Adaptation test consisted of 5 toes-up and toes-down rotation perturbations at 8°/s for 400 msec | **- Weight symmetry during perturbations during motor control test**  
- Latency and amplitude (strength) of response based on force plate measures during motor control test  
- Sway response scores during adaptation test | **onset after a push at the hips is related to sway**  
- Symmetry was deviated toward non-paretic limb in left hemi patients but same in right hemi and controls  
- Strength of response more in non-paretic limb for both right and left hemi patients  
- Latency of response delayed in paretic limb for both right and left hemi patients  
- Strength of response reduced in paretic limb of right and left hemi patients  
- Longer adaptation in stroke with toes-down rotation  
- Sway response greater in stroke than controls for toes-down rotation |
| **Jiang et al. 1998** | **To examine the influence of chronic hemiparesis on the control of compensatory stepping and grasping reactions in stroke patients** | **N=8 stroke; 20-72 months post**  
N=8 healthy older adult controls | **- Healthy subjects instructed to weight bear at 70% on dominant leg to mimic hemiparetics**  
- Stepping and grasping reactions were evoked by unpredictable platform translations  
- Handrails were at the perimeter  
- Random order perturbations in AP direction  
- Light-cued voluntary grasping reactions also tested  
- First set of 10 trials, compensatory stepping elicited | **- Step timing**  
- COM  
- Arm EMG | **- Both groups relied on compensatory stepping but strokes stepped with loaded (non-paretic) leg (68%)**  
- Stroke had greater tendency to use multiple steps, and/or grasp handrail  
- APAs in ML direction also observed in stroke but led to delayed step and increased AP COM displacement |
<p>| Kirker et al. 2000a | To describe the different patterns of activation in stroke subjects and changes during recovery | N=13 stroke; range 33-75 yrs old; 7M, 8F; 8 right, 5 left hemi N=18 controls; x=46 yrs old | - Subjects stood on force plate and received up to 30% body weight sideways pushes via a computer-controlled linear motor in blocks of 5 alternating left and right - Tracked subjects over course of recovery - Received physiotherapy in hospital over recovery 5 days/wk and 1-2 times after discharge | - Rivermead mobility Index (RMI) - 10m walk time - Motricity Index - EMG of GM and adductor bilaterally observing onset and presence of response and magnitude and patterns - 4 patterns found: (1) no response in any hip muscle after push to either direction, (2) little or no response in hemiparetic GM after push to weak side but minimal or increased activity in unaffected adductor. Push to strong side showed normal activation of unaffected GM but no response in hemiparetic adductor, (3) push to weak side - normal hemiparetic GM, increase or normal unaffected adductor; push to strong side - normal unaffected GM, no response in hemiparetic adductor, (4) push to weak side - normal hemiparetic GM, increase or normal unaffected adductor; push to strong side - normal unaffected GM, normal hemiparetic adductor - 8 strokes showed change towards normal recruitment with recovery |
| DSM 392 | To compare the pattern of pelvic girdle muscle activation in normal subjects and hemiparetic patients while stepping and maintaining | N=17 stroke; x=54 yrs old; 12M, 5F; 13 right, 4 left hemi; median=74 wks post; N=16 controls | - Stood on force plate and received sideways push via waist belt in blocks of 5 alternating sides up to 20 each - Gait initiation - 40 total, blocks of 5 alternating | - EMG amplitude (area under curve) - EMG onsets - Push to hemiparetic side showed smaller and later increase in activity vs. controls in GM - Hemiparetic GM and adductor recruitment smaller |</p>
<table>
<thead>
<tr>
<th>Study</th>
<th>Details</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
</table>
| Leonard et al. 1998 | To determine whether or not soleus H-reflexes were reciprocally inhibited during TA muscle contractions elicited by a balance platform-induced postural perturbation | N = 4 CP
N = 6 chronic stroke
N = 12 healthy controls | - While H-reflex of soleus and EMG of TA was being recorded, a forward platform translation of 15 cm at 88.9 cm/s was evoked (350 to 889 cm/s² random accelerations)
- 15 trials collected
- Voluntary dorsiflexion trials also collected | - H-reflex
- EMG onset |
| DSM 13 | - Healthy subjects demonstrated inhibition of soleus (assessed through H-reflex) during both voluntary dorsiflexion and in response to perturbation requiring TA activation |
| Wing et al. 1993 | To describe the effects of applying left-right direction horizontal forces to the hips in stroke patients | N=11 stroke; x=65 yrs old; greater than 12 months post; 4M, 7F; 5 right, 6 left hemi
N=12 elderly controls; x=67 yrs old | - Perturbation applied via an actuator attached to waist belt providing up to +/-100 N over 400 ms and max speed of 350 mm/s
- Feet slightly apart with hips centered
- Force determined based on 1/2 force subject could exert on machine; control subjects all got 31.7N force compared to 26.4N average in strokes
- 13 sec trials; 6 trials in a block alternating force
- Keep feet in | - Functional impairment of balance and correlations between other measures
- Peak displacement
- Stabilization times
- Offsets (on push and release) |
| DSM 261 | - No differences in offsets
- Larger peak displacements on push and release for stroke vs. controls
- Release of push towards involved side the peak displacement (i.e. sway over the noninvolved side), was less than on release from a push towards the noninvolved side
- Release and push (only on 1st session) stabilization times increase in stroke
- Correlations \(\Rightarrow\) longer |
| place and not move them | stabilization time from push to noninvolved side decreases activity rating for reach (noninvolved) and reach involved |
## Appendix IV: Test re-test reliability for standing postural reflexes

Table IV.1: Test re-test reliability

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Condition</th>
<th>Mean 1 (msec)</th>
<th>Mean 2 (msec)</th>
<th>SD1</th>
<th>SD2</th>
<th>ICC (3,1)</th>
<th>F-test P-value</th>
<th>SEM (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P_MG</td>
<td>Backward</td>
<td>139.63</td>
<td>124.90</td>
<td>24.08</td>
<td>19.11</td>
<td>0.79</td>
<td>0.05</td>
<td>9.90</td>
</tr>
<tr>
<td>NP_MG</td>
<td>Backward</td>
<td>107.59</td>
<td>106.15</td>
<td>7.65</td>
<td>7.66</td>
<td>0.67</td>
<td>0.46</td>
<td>4.40</td>
</tr>
<tr>
<td>P_TA</td>
<td>Forward</td>
<td>125.83</td>
<td>105.83</td>
<td>22.33</td>
<td>43.04</td>
<td>0.92</td>
<td>0.25</td>
<td>9.24</td>
</tr>
<tr>
<td>P_RF</td>
<td>Forward</td>
<td>151.09</td>
<td>155.83</td>
<td>28.04</td>
<td>33.92</td>
<td>0.87</td>
<td>0.68</td>
<td>11.17</td>
</tr>
<tr>
<td>NP_TA</td>
<td>Forward</td>
<td>104.55</td>
<td>109.17</td>
<td>8.17</td>
<td>10.67</td>
<td>0.79</td>
<td>0.12</td>
<td>4.32</td>
</tr>
<tr>
<td>NP_RF</td>
<td>Forward</td>
<td>140.55</td>
<td>134.96</td>
<td>32.24</td>
<td>29.17</td>
<td>0.79</td>
<td>0.53</td>
<td>14.07</td>
</tr>
</tbody>
</table>

**Abbreviations:**

P_MG = paretic gastrocnemius
NP_MG = non-paretic gastrocnemius
P_TA = paretic tibialis anterior
NP_TA = non-paretic tibialis anterior
P_RF = paretic rectus femoris
NP_RF = non-paretic rectus femoris

SD = standard deviation
ICC = Intraclass Correlation Coefficient (ICC$_{3,1}$)

SEM = Standard Error of Measurement

Formula: $SEM = \left( \frac{SD1 + SD2}{2} \right) \times \sqrt{1 - ICC}$
Appendix V: Berg Balance Scale

1. Sitting to Standing
   Instruction: Please stand up. Try not to use your hands for support.
   Grading:
   4: Able to stand no hands and stabilize independently
   3: Able to stand independently using hands
   2: Able to stand using hands after several tries
   1: Needs minimal assist to stand or to stabilize
   0: Needs moderate or maximal assist to stand

2. Standing Unsupported
   Instruction: Stand for two minutes without holding.
   Grading:
   4: Able to stand safely 2 minutes
   3: Able to stand 2 minutes with supervision
   2: Able to stand 30 seconds unsupported
   1: Needs several tries to stand 30 seconds unsupported
   0: Unable to stand 30 seconds unassisted

   If subject able to stand 2 minutes safely, score full marks for sitting unsupported. Proceed to position change standing to sitting.

3. Sitting Unsupported Feet on Floor
   Instruction: Sit with arms folded for two minutes.
   Grading:
   4: Able to sit safely and securely 2 minutes
   3: Able to sit 2 minutes under supervision
   2: Able to sit 30 seconds
   1: Able to sit 10 seconds
   0: Unable to sit without support 10 seconds

4. Standing to sitting
   Instructions: Please sit down.
   Grading:
   4: Sits safely with minimal use of hands
   3: Controls descent by using hands
   2: Uses back of legs against chair to control descent
   1: Sits independently but has uncontrolled descent
   0: Needs assistance to sit
5. Transfers
Instructions: Please move from chair to bed and back again. One way toward a seat with
armrests and one way toward a seat without armrests.
Grading:
4: Able to transfer safely with minor use of hands
3: Able to transfer safely definite use of hands
2: Able to transfer with verbal cuing and/or supervision
1: Needs one person to assist
0: Needs two people to assist or supervise to be safe

6. Standing Unsupported with Eyes Closed
Instructions: Close your eyes and stand still for 10 seconds.
Grading:
4: Able to stand 10 seconds safely
3: Able to stand 10 seconds with supervision
2: Able to stand 3 seconds
1: Unable to keep eyes closed 3 seconds but stays steady
0: Needs help to keep from falling

7. Standing Unsupported with Feet Together
Instructions: Place your feet together and stand without holding.
Grading:
4: Able to place feet together independently and stand for 1 minute safely
3: Able to place feet together independently and stand for 1 minute with supervision
2: Able to place feet together independently but unable to hold for 30 seconds
1: Needs help to attain position but able to stand 15 seconds feet together
0: Needs help to attain position and unable to hold for 15 seconds

8. Reaching Forward with Outstretched arm.
Instructions: Lift arm to 90 degrees. Stretch out your fingers and reach forward as far as you
can. (Examiner places a ruler at end of fingertips when arm is at 90 degrees. Fingers should not
touch the ruler while reaching forward. The recorded measure is the distance forward that the
fingers reach while the subject is in the most forward lean position.)
Grading:
4: Can reach forward confidently more than 10 inches
3: Can reach forward more than 5 inches safely
2: Can reach forward more than 2 inches safely
1: Reaches forward but needs supervision
0: Needs help to keep from falling
9. Pick Up Object from the Floor
   Instructions: Pick up the shoe/slipper, which is placed in front of your feet.
   Grading:
   4: Able to pick up slipper safely and easily
   3: Able to pick up slipper but needs supervision
   2: Unable to pick up but reaches 1 to 2 inches from slipper and keeps balance independently
   1: Unable to pick up and needs supervision while trying
   0: Unable to try/needs assistance to keep from falling

10. Turning to Look Behind Over Left and Right Shoulders
    Instructions: Turn to look behind you over toward left shoulder. Repeat to the right.
    Grading:
    4: Looks behind form both sides and weight shifts well
    3: Looks behind one side only; other side shows less weight shift
    2: Turns sideways only but maintains balance
    1: Needs supervision when turning
    0: Needs assist to keep from falling

11. Turn 360 Degrees
    Instructions: Turn completely around in a full circle. Pause. Then turn a full circle in the other direction.
    Grading:
    4: Able to turn 360 degrees safely in less than 4 seconds each side
    3: Able to turn 360 degrees safely one side only – less than 4 seconds
    2: Able to turn 360 degrees safely but slowly
    1: Needs close supervision or verbal cuing
    0: Needs assistance while turning

12. Count Number of Times Step Touch Measured Stool
    Instructions: Place each foot alternately on the stool. Continue until each foot has touched the stool four times.
    Grading:
    4: Able to stand independently and safely and complete 8 steps in 20 seconds
    3: Able to stand independently and complete 8 steps in more than 20 seconds
    2: Able to complete 4 steps without aid with supervision
    1: Able to complete more than 2 steps – needs minimal assist
    0: Needs assistance to keep from falling – unable to try
13. Standing Unsupported One Foot in Front
Instructions: (DEMONSTRATE to subject.) Place one foot directly in front of the other. If you feel that you cannot place your foot directly in front, try to step far enough ahead that the heel of your forward foot is ahead of the toes of the other foot.
Grading:
4: Able to place foot tandem independently and hold 30 seconds
3: Able to place foot ahead of the other independently and hold 30 seconds
2: Able to take small step independently and hold 30 seconds
1: Needs help to step but can hold 15 seconds
0: Loses balance while stepping or standing

14. Standing on One Leg
Instructions: Stand on one leg as long as you can without holding.
Grading:
4: Able to lift leg independently and hold more than 10 seconds
3: Able to lift leg independently and hold 5 to 10 seconds
2: Able to lift leg independently and hold at least 3 seconds
1: Tries to lift leg, unable to hold 3 seconds but remains standing independently
0: Unable to try or needs assist to prevent fall

Additional Instructions

All questions should be scored in the lowest category that applies. All furniture or chairs should be normal height, not very low sofas or easy chairs. All questions after #5 are performed standing unsupported. Subjects may be permitted a rest between items as required.

4. The instructions do not specify that the subject refrain from using hands. It would not have been consistent with the clinical training given most patients.

5. This item tests how well the subjects are able to transfer from and towards seats with and without arm rests. The purpose is not to test the strength of either side. Therefore, persons with a definite side preference may be positioned so as to go in the same direction twice.

11. The subject is awarded 3 points if he or she can turn 360 degrees independently and safely in both directions but one side took longer than 4 seconds.

12. This item can be performed using the bottom step of a staircase or a stool of comparable height.
Appendix VI: Nottingham Health Profile (NHP)

Please check the appropriate box ("Yes" or "No") to each question.

Table VI.1: NHP

<table>
<thead>
<tr>
<th></th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>I'm tired all the time.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I have pain at night.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Things are getting me down.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I have unbearable pain.</td>
<td></td>
<td></td>
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<tr>
<td>I take pills to help me sleep.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I've forgotten what it’s like to enjoy myself.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I'm feeling on edge.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I find it painful to change position.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I feel lonely.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I sleep badly at night.</td>
<td></td>
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</tr>
<tr>
<td>I'm finding it hard to get along with people.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I need help to walk about outside (e.g. a walking aid or someone to support me).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I'm in pain when going up or down stairs.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| I wake up feeling depressed.  
| I'm in pain when I'm sitting.  
| I can walk about only indoors  
| I find it hard to bend.  
| Everything is an effort.  
| I'm waking up in the early hours of the morning.  
| I'm unable to walk at all.  
| I'm finding it hard to make contact with people.  
| The days seem to drag.  
| I have trouble getting up and down stairs and steps.  
| I find it hard to reach for things.  
| I'm in pain when I walk.  
| I lose my temper easily these days.  
| I feel there is nobody that I am close to.  
<p>| I lie awake for most of the night.  |</p>
<table>
<thead>
<tr>
<th>I feel as if I'm losing control.</th>
</tr>
</thead>
<tbody>
<tr>
<td>I'm in pain when I'm standing.</td>
</tr>
<tr>
<td>I find it hard to get dressed by myself.</td>
</tr>
<tr>
<td>I soon run out of energy.</td>
</tr>
<tr>
<td>I find it hard to stand for long (e.g. at the kitchen sink, waiting in line.)</td>
</tr>
<tr>
<td>I'm in constant pain.</td>
</tr>
<tr>
<td>It takes me a long time to get to sleep.</td>
</tr>
<tr>
<td>I feel I am a burden to people.</td>
</tr>
<tr>
<td>Worry is keeping me awake at night.</td>
</tr>
<tr>
<td>I feel that life is not worth living.</td>
</tr>
</tbody>
</table>

'Yes' responses summated for total score out of 38. Lower scores represent better health-related quality of life.
Appendix VII: 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. Answer all items even if there are activities you would not do or are unsure about.

<table>
<thead>
<tr>
<th>0%</th>
<th>10%</th>
<th>20%</th>
<th>30%</th>
<th>40%</th>
<th>50%</th>
<th>60%</th>
<th>70%</th>
<th>80%</th>
<th>90%</th>
<th>100%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not Completely Confident</td>
<td>Completely Confident</td>
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<td></td>
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<td></td>
<td></td>
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</tr>
</tbody>
</table>

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

A) walk around the house? ______%  
B) walk up and down stairs?______%  
C) pick up a slipper from the floor?_____%  
D) reach at eye level?______%  
E) reach while standing on your tiptoes?_____%  
F) stand on a chair to reach?_____%  
G) sweep the floor?______%  
H) walk outside to nearby car?_____%  
I) get in and out of a car?_____%  
J) walk across a parking lot?_____%  
K) walk up and down a ramp?_____%  
L) walk in a crowded mall?_____%  
M) walk in a crowd or get bumped? _____%  
N) ride an escalator holding the rail?____%  
O) ride an escalator not holding the rail?____%  
P) walk on icy sidewalks?_____%
Appendix VIII: Informed consent for Experiment I

Informed Consent Form
Posture and Locomotion Database

Principal Investigator: Dr. Janice Eng
School of Rehabilitation Sciences
University of British Columbia
(604) 714-4105

Purpose:

I understand that I am being invited to participate in this study so that my balance, walking patterns, and strength can be evaluated and entered into a posture and locomotion database. This database will be used to provide information as to the effects of aging, neurological conditions and musculoskeletal conditions on posture and locomotion.

Study Procedures:

I may be asked to stand for one minute, stand and balance on a moveable platform, and/or raise my arms while standing. Electrodes will be attached to the surface of my limbs and trunk with double-sided tape to record the activity of my muscles. Small markers will also be attached to my limbs and trunk with double-sided tape so that cameras can record my movements.

The whole evaluation procedure will take approximately 2.0 hours. Rest breaks can be taken at any time.

Exclusions:

Subjects who are not able to rise from a chair and stand for 10 seconds with minimal assistance from one person will be excluded from this study.

Confidentiality:

Any information resulting from this research study will be kept strictly confidential. All documents will be identified only by a code number and kept in a locked filing cabinet. I will not be identified by name in any reports of the completed study. My medical record may, however, be inspected by the Health Protection Branch HPB Canada) in the presence of the Investigator or her designate. Copies of relevant data which identify me only by code number may be required by the HPB, but I will not be identified by name, initials or date of birth.

Contact:

I understand that if I have any questions or desire further information with respect to this study, or if I experience any adverse effects, I should contact Dr. Janice Eng or one of her associates at (604) 714-4105. If I have any concerns about my treatment or rights as a research subject I may
contact the Director of Research Services at the University of British Columbia at (604) 822-8598.

**Patient Consent:**

I understand that participation in this study is entirely voluntary and I may refuse to participate or I may withdraw from the study at any time without any consequences. I have received a copy of this consent form for my own records. I consent to participate in this study.

<table>
<thead>
<tr>
<th>Subject Signature</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Witness Signature</td>
<td>Date</td>
</tr>
<tr>
<td>Investigator Signature</td>
<td>Date</td>
</tr>
</tbody>
</table>
Appendix IX: Platform translation protocol for Experiment I

Table IX.1: Experiment I protocol

Increased WtBr = 30% on non-paretic leg, 70% on paretic leg
Decreased WtBr = 70% on non-paretic leg, 30% on paretic leg
Neutral = 50% on each leg

**Interval between perturbations (random) = 30 seconds to 1 minute

<table>
<thead>
<tr>
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<th>Displacement</th>
<th>Velocity</th>
<th>Acceleration</th>
</tr>
</thead>
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<tr>
<td>Magnitude:</td>
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<td>30 cm/s</td>
<td>300 cm/s²</td>
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</table>

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<th>Trial #</th>
<th>Condition</th>
<th>Platform Direction</th>
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<td>6</td>
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<td>7</td>
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<td>Decreased WtBr</td>
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### Appendix X: EMG electrode placement guidelines

#### Table X.1: Electrode placement

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>Placement Guidelines</th>
<th>Segment Measurements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tibialis Anterior (TA)</td>
<td>Over the greatest muscle bulk just lateral to tibial crest; most proximal half of the shank</td>
<td>Right leg: ____ cm down from inferior border of patella</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Left leg: ____ cm down from inferior border of patella</td>
</tr>
<tr>
<td>Medial Gastrocnemius (MG)</td>
<td>Over greatest muscle bulk on medial side of calf (proximal posterior shank) → line from the popliteal fossa to the heel: 1/3 down from popliteal fossa</td>
<td>Right leg: line ____ cm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Left leg: line ____ cm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1/3 = ____ cm</td>
</tr>
<tr>
<td>Biceps Femoris (BF)</td>
<td>On lateral posterior distal thigh: line from 2 cm lateral to the spinal cord at the level of iliac crests to the head of the fibula → 1/3 up from the head of the fibula</td>
<td>Right leg: line ____ cm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Left leg: line ____ cm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1/3 = ____ cm</td>
</tr>
<tr>
<td>Rectus Femoris (RF)</td>
<td>Midway between a line from the ASIS to the superior border of the patella</td>
<td>Right leg: line ____ cm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Left leg: line ____ cm</td>
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<tr>
<td></td>
<td></td>
<td>1/2 = ____ cm</td>
</tr>
</tbody>
</table>
Appendix XI: American Heart Association Stroke Functional Classification (AHASFC)

Function Level

I → **Independent** in BADL and IADL activities and tasks required of roles of patient had before stroke. Patient is able to live alone, maintain a household, and access the community for leisure and/or productive activities such as shopping, employment, or volunteer work.

II → **Independent** in BADL but partially dependent in routine IADL. Patient is able to live alone but requires assistance/supervision to access the community for shopping and leisure activities. Patient may require occasional assistance with meal preparation, household tasks, and taking medication.

III → **Partially dependent** in BADL (<3 areas) and IADL. Patient is able to live alone with substantial daily help from family or community resources for more difficult BADL tasks such as dressing lower extremities, bathing, or climbing stairs. Patient requires assistance with such IADL tasks as meal preparation, home maintenance, community access, shopping, handling finances, and/or taking medications.

IV → **Partially dependent** in BADL (≥3 areas). Patient is unable to live alone safely and requires assistance with IADL except for simple tasks such as answering the telephone.

V → **Completely dependent** in BADL (≥5 areas) and IADL. Patient is unable to live alone safely and requires full-time care.

BADL indicates *Basic Activities of Daily Living*: feeding and swallowing, grooming, dressing, bathing, continence, toileting, and mobility.

IADL indicates *Instrumental Activities of Daily Living*: using the telephone, handling money, shopping, using transportation, maintaining a household, working, participating in leisure activities, etc.
Appendix XII: Informed consent for Experiment II and III

Informed Consent
Postural Control in Individuals with Stroke

Principle Investigator:
Dr. Janice Eng, PhD, PT/OT, School of Rehabilitation Sciences, Contact: (604) 714-4105

Co-investigators:
Daniel Marigold, BSc. Kin, Graduate Program in Neuroscience
Dr. Tim Inglis, PhD, Human Kinetics
Dr. Drew Dawson, MD, FRCP, Acquired Brain Injury
Dr. Heather McKay, PhD, Human Kinetics
Jocelyn Harris, BA, OT, School of Rehabilitation Sciences

Background:
You are being invited to participate in this study because the exercise programs involved in this study may improve your physical function, which has been affected by a stroke.

Purpose:
The purpose of this study is to determine whether exercise interventions affect physical function in individuals with chronic stroke.

Study Procedures:
Should you choose to participate in this study, you will be assigned by chance (i.e. like flipping a coin with a 50% chance of being assigned to a particular group) to one of two exercise groups: 1) balance-based exercise group or 2) posture-based exercise group. Physical therapists, occupational therapists, and/or kinesiologists will supervise the exercise programs. Your involvement in the study means that you will participate in your assigned exercise program (1 hour/session, 3 sessions/week for 10 weeks) aimed at improving physical function. Your primary care physician must consent to your participation in this study before you are officially accepted into the study. Your primary care physician will also release to the principle investigator the type (e.g. ischemic versus hemorrhagic stroke) and the location in the brain of your stroke if this information is known. You will be provided with a release-of-information form to give to your physician. Both exercise programs will consist of a warm-up, stretching, exercises that challenge posture and balance, and a cool-down. Rest breaks can be taken at any time.

You will be asked to come for assessments at three different weeks (prior to the exercise program, immediately following the exercise program, and one month after the end of the exercise program). At each of these weeks, there will be 3 test sessions separated by one or two days. Each test session will last 2 hours. One session will have you perform standing tasks for 60 seconds with eyes open and eyes closed, standing and raising your arms to the side, and maintaining standing balance on a platform that will suddenly move horizontally.

During these tasks, you will be fitted with a harness for safety and have markers attached to your body segments to monitor your movement and surface electrodes on leg and trunk muscles.
to measure their muscle activity. The second session will assess your functional balance, walking speed and muscle strength and have you answer questionnaires on balance confidence and quality of life. The third session will measure your bone density and structure using a bone densitometer and computerized tomography system. You will also maintain a diary in which you will record any time you experience a fall and the circumstances surrounding the incident and return by mail to the GF Strong Rehab Centre every month for the following year.

Exclusions:
Individuals who have had their stroke for less than 1 year or who have had more than 1 stroke will be excluded from the study. In addition, individuals will be excluded if they are not medically stable (e.g. have congestive heart failure, unstable cardiovascular status, uncontrolled hypertension, atrial fibrillation, or left ventricular failure), and have significant musculo-skeletal problems (e.g. active inflammatory arthritis) due to conditions other than stroke.

Risks:
There is a chance that you may feel tired or experience some muscle soreness after the exercise and/or testing sessions. These symptoms should disappear within a few days. These symptoms can be minimized with stretching following the testing and exercise sessions. In addition, there is a slight chance that the electrodes used to monitor muscle activity and/or the tape used to secure infrared emitting diodes to monitor movement during the testing sessions may cause some minor skin irritation.

Although the bone measurements are X-ray based, the total effective dose per session will be approximately 10 millirem. This is less than you receive on an airplane flight across the country.

Benefits:
The balance-based and posture-based exercise programs may have the potential to improve your physical function.

Confidentiality:
Any information resulting from this research study will be kept strictly confidential. All documents will be identified only by a code number and kept in a locked filing cabinet. You will not be identified by name in any reports of the completed study. Your medical records may be inspected by the Health Protected Branch (HPB Canada) in the presence of the investigator or her designate. Copies of relevant data, which identify you only by code number, may be required by the HPB, but name, initials, or date of birth will not identify yourself.

Remuneration/Compensation:
You will be reimbursed for bus or parking charges to attend the sessions.

Compensation for Injury:
Signing this consent form in no way limits your legal rights against the sponsor, investigators, or anyone else.

Contact:
If you have any questions or desire further information with respect to this study or experience any adverse effects, please contact Dr. Janice Eng or one of her associates at (604) 714-4105. If
you have any concerns about your treatment or rights as a research participant you may contact the Director of Research Services at the University of British Columbia at (604) 822-8598.

Participant Consent:
I understand that participation in this study is entirely voluntary and I may refuse to participate or I may withdraw from the study at any time without any consequences to my continuing medical care.

I have received a copy of this consent form for my own records.

I consent to participate in this study.

Participant Signature _______________________________ Date __________

Witness Signature _______________________________ Date __________

Investigator Signature _______________________________ Date __________
Table XIII.1: Experiment II and III protocol

**TR Protocol**

Subject Code: ______________

*Interval between perturbations (random) = 15-30 seconds*

Perturbation Magnitude

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<thead>
<tr>
<th>Displacement</th>
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<td>30 cm/s</td>
<td>300 cm/s²</td>
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<th>Perturbation #</th>
<th>Trial #</th>
<th>Platform Direction</th>
<th>Step</th>
<th>Limb</th>
<th>Direction</th>
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</table>
Appendix XIV: Recruitment for Experiment II and III

Newspaper Advertisement

Persons with stroke are invited to take part in a study undertaken by the School of Rehabilitation Sciences, University of British Columbia in conjunction with the GF Strong Rehab Centre. This study will examine the effect of 10-week balance-based and posture-based exercise programs on physical function. Physical therapists, occupational therapists, recreational therapists and/or kinesiologists will supervise the exercise programs. For more information or to participate in this study, contact: Daniel Marigold (Kinesiologist) at the GF Strong Rehab Centre at (604) 714-4109.

Flyer

***RESEARCH STUDY***

Exercise Program for Individuals with Stroke

Persons with stroke are invited to take part in a study undertaken by the School of Rehabilitation Sciences, University of British Columbia in conjunction with the GF Strong Rehab Centre. This study will examine the effect of 10-week balance-based and posture-based exercise programs on physical function. Physical therapists, occupational therapists, and/or kinesiologists will supervise the exercise programs. Participants will be assigned by chance to either the balance-based or posture-based exercise program groups. Participants will be assessed at three difference weeks (prior to the exercise programs, immediately following the exercise programs, and again one month after the programs). During each of these weeks, there will be three test sessions separated by one or two days. During the tests sessions, you will be asked to perform various standing tasks, undergo muscle strength, balance testing and bone density testing. In addition, you will be asked to complete questionnaires about your balance ability and quality of life. Both exercise programs will last 10 weeks and consist of 3 sessions/week, maximum of 1 hr/session. Transportation can be arranged.

For more information or to participate in this study, contact: Daniel Marigold (Kinesiologist) at the GF Strong Rehab Centre at (604) 714-4109.
Appendix XV: Mini-Mental State Exam

Score 1 for every correct answer:

1. What year is it? _____
2. What season are we in? _____
3. What month are we in? _____
4. What is today’s date? _____
5. What day of the week is it? _____
6. What country are we in? _____
7. What province are we in? _____
8. What city are we in? _____
9. What hospital are we in? _____
10. What floor of the hospital are we on? _____

Name three objects ("Ball," "Car," "Man"). Take a second to pronounce each word. Then ask the patient to repeat all 3 words. Take into account only correct answers given on the first try. Repeat these steps until the subject learns all the words.

11. Ball? _____
12. Car? _____
13. Man? _____

Either “please spell the word WORLD and now spell it backwards” or “Please count from 100 subtracting 7 every time”

14. “D” or 93 _____
15. “L” or 86 _____
16. “R” or 79 _____
17. “O” or 72 _____
18. “W” or 65 _____
What were the 3 words I asked you to remember earlier?

19. Ball? 
20. Car? 
21. Man? 

Show the subject a pen and ask: “Could you name this object?”

22. Pen. 

Show the subject your watch and ask: “Could you name this object?”

23. Watch 

Listen and repeat after me:

24. “No ifs, ands, or buts.” 

Put a sheet of paper on the desk and show it while saying: “Listen carefully and do as I say.”

25. Take the sheet with your left/right (unaffected) hand. 
26. Fold it in half. 
27. Put in on the floor. 

Show the patient the visual instruction page directing him/her to “CLOSE YOUR EYES” and say:

28. Do what is written on this page. 

Give the subject a blank sheet and a pen and ask:

29. Write a sentence, whatever you want, but a complete sentence. 

Give the patient the geometric design page and ask:

30. Could you please copy this drawing? 

**Total Score: (30)** ___
Appendix XVI: Experiment II and III physician consent

Physician’s Consent

Dear Doctor:

Your patient has expressed interest in participating in our Postural Control in Individuals with Stroke study examining the effectiveness of a 10-week exercise program focused on improving balance, posture, and functional mobility in stroke survivors. This study is being undertaken at the G.F. Strong Rehab Centre in collaboration with the School of Rehabilitation Sciences at the University of British Columbia and has been approved by the UBC and hospital ethics committees.

All participants will undergo assessments at three different weeks (prior to the exercise program, immediately following the exercise program, and one month after the completion of the exercise program). At each of these weeks, there will be 3 test sessions separated by one or two days with each session lasting approximately 2 hours.

One session will have your patient perform standing tasks that challenge their balance. Another session will assess your patient’s function and muscle strength and have them answer questionnaires on balance confidence and quality of life. The other session will measure your patient’s bone density and structure using a bone densitometer and computerized tomography system.

Your patient will be assigned by chance (i.e. like flipping a coin with a 50% chance of being assigned to a particular group) to either a balance-based or posture-based exercise program group. Both exercise programs will last 10 weeks (3 sessions/week, 1hr/session) and consist of a warm-up, stretching, moderate intensity exercises that challenge posture and balance (e.g. rapid stepping, muscle strength training, and brisk walking), and a cool-down.

Individuals with stroke who have residual weakness on one side of the body and can walk independently shall be included in the study. Theses individuals must have had their stroke for greater than one year and have only suffered from one stroke. However, those with uncontrolled hypertension, congestive heart failure, unstable cardiovascular status, atrial fibrillation, left ventricular failure, or significant musculo-skeletal problems (e.g. active inflammatory arthritis) due to conditions other than stroke will be EXCLUDED.

We would be grateful if you would decide whether your patient would be suitable to participate in this exercise research study and complete the relevant medical information regarding the type and location of their stroke.

Sincerely,

Dr. Drew Dawson, MD, FRCPC
Acquired Brain Injury
G.F. Strong Rehab Centre

Dr. Janice Eng, Ph.D, PT/OT
Research Scientist, GF Strong Rehab Centre
Assistant Professor, University of BC

Daniel Marigold, BSc. Kin
MSc. Candidate
Graduate Program in Neuroscience, University of BC
Patient Release

I agree to the release of the following medical information to the Postural Control in Individuals with Stroke Study conducted by the School of Rehabilitation Sciences, UBC and G.F. Strong Rehab Centre.

Relevant Medical Information of Patient (Physician Please fill out this information)

Type of Stroke (e.g. ischemic versus hemorrhagic stroke): __________________________

Location in Brain of Stroke (e.g. middle, anterior, or posterior cerebral artery): __________

Signed: ___________________________________________________________________

Patient Signature

Please print patient's full name

Date: ______________________________________________________________________

Physician's Assessment

The above patient is suitable for the Postural Control in Individuals with Stroke Study described on page 1 of this letter and I have filled out the above information related to the type and location of the patient’s stroke.

Signed: ___________________________________________________________________

Physician’s Signature

Please print Physician’s full name

Address of Physician’s Office

Date: ______________________________________________________________________
Appendix XVII: Exercise interventions

Exercise Interventions (*major component)

Agility Exercise Group
- Exercises for many of the components were performed in stations (1 instructor to approximately 3 clients)
- Stations allowed for more challenging exercises to be performed since small group easier to handle for instructor and since groups could be assigned in such a way to have a low functioning client with a high functioning client or a group of high functioning clients could be formed

⇒ Warm-up (5 min)
- Walking (little steps, big steps, side steps, knee high steps)
- Weight-shifting while standing

⇒ Multi-sensory Component*
- Walking on foam (little steps, big steps, tandem, backwards)*
- Standing on foam (separate or combination of: eyes open, eyes closed, weight-shifting, head turning, head back, feet together, feet staggered, feet parallel, one foot)*
- Rapid stepping onto foam
- Note: several different types of foam were used with various densities

⇒ Strength Component (functional)
- Sit-to-stand (repetitions of 10)
- Ankle strengthening (heel raises and toe raises while supported by chair)
- Rapid knee raises (hip and knee flexion) with ankle weights

⇒ Agility Component*
- Rapid stepping forward and backward*
- Side stepping across room
- Braiding (alternating front and back crossover steps during side stepping) across room
- Tandem walking (heel-to-toe walking) across room
- Figure-8 eight (drawn in chalk on floor) walking (tandem, regular, and backward)
- Stepping onto and over steppers
- Target stepping (into circles of varying size and distance) across room
- Standing balloon toss games
- Standing on tilt-board
- Standing perturbations (instructor pushing client or vice versa)*
Cool-down (5 min)
- Light stretching

Stretching/weight-shifting Exercise Group

- Small group discussions were also done at the beginning of class occasionally

Warm-up (5 min)
- Walking (little steps, big steps, and side steps)

Weight-shifting*
- Side-to-side weight-shifting*
- Reaching up and down tasks
- Tai Chi-like movements*
- Seated balloon toss

Mat exercises*
- On all fours (shifting side-to-side, superman stretch – opposite arm and leg raised)
- Arm weight exercises
- Stretching various muscle groups

Cool-down (5-10 min)
- Relaxation
Appendix XVIII: Platform-induced falls for Experiment III

Table XVIII.1: Falls on platform

<table>
<thead>
<tr>
<th>Measure</th>
<th>Stretching/weight-shifting Group</th>
<th>Agility Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Post-intervention</td>
</tr>
<tr>
<td># of Falls</td>
<td>19</td>
<td>30</td>
</tr>
<tr>
<td># of Falls/person</td>
<td>0.76</td>
<td>1.20</td>
</tr>
<tr>
<td># of Fallers</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td># of Repeat Fallers</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>