MECHANICAL PROPERTIES OF THE LOWER EXTREMITY MUSCLES IN INDIVIDUALS WITH CHRONIC STROKE

by

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ABSTRACT

Lower extremity muscle weakness in individuals with stroke plays a significant role in limiting functional performance. In order to design more effective rehabilitation interventions to counteract this weakness, a better understanding of how stroke affects mechanical muscle properties is needed. The purpose of this thesis was to 1) quantify isometric torque and temporal parameters of torque production (times to develop and reduce torque), 2) assess isometric torque-angle relationships, and 3) determine whether concentric torque was more impaired than eccentric torque in the paretic and nonparetic legs of individuals with chronic stroke and the nondominant leg of control subjects. A dynamometer was used to assess the abovementioned muscle properties of the ankle, knee, and hip flexors and extensors. The results revealed reduced isometric torque throughout the paretic leg (ankle, knee, and hip) but only ankle plantarflexion was reduced in the nonparetic leg. Times to develop and reduce isometric torque were impaired bilaterally (ankle, knee, and hip). The paretic knee extensors revealed exaggerated weakness near terminal extension (short muscle lengths), whereas the nonparetic knee extensors and flexors were selectively stronger as the knee assumed a flexed position. Lastly, a relative preservation of eccentric torque in both the paretic and nonparetic legs was found, thus concentric torque was more affected by stroke than eccentric torque. The work of this thesis has identified muscle impairments in both the paretic and nonparetic legs of individuals with chronic stroke. Based on these findings, clinicians are encouraged to assess bilateral leg strength across joint range of motion. When designing strength training programs, exercises at high contraction speeds should be incorporated. Lastly, due to the relative preservation of eccentric torque, the 1-repetition maximum should be determined for both concentric and eccentric contractions to avoid underloading the muscle during the lengthening phase of movement.
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Chapter 1: INTRODUCTION

Improving motor performance to maximize functional recovery is an important goal of neurorehabilitation. Unfortunately, nearly 70% of individuals with stroke are left with residual impairments despite completion of rehabilitation (Jorgensen et al. 1999). Even years after the initial brain injury, individuals living with chronic stroke continue to report dissatisfaction with their mobility performance both inside and outside their homes (Harris and Eng 2004). Hence it is important for rehabilitation clinicians to fully understand the impairments leading to mobility problems and to remain involved in the care of individuals living with stroke. Due to the muscle weakness (decreased torque production) that often persists following stroke, and the surmounting evidence demonstrating a positive relationship between muscle strength and physical task performance in this population (Nakamura et al. 1985; Bohannon and Walsh 1991; Olney et al. 1994; Cameron et al. 2003; Hsu et al. 2003; Kim and Eng 2003; Lomaglio and Eng in press) clinicians have begun to incorporate strength training into neurorehabilitation programs (Carr and Shepherd 2004; Jette et al. 2005). However, clinical trials examining the effects of strength training have not always demonstrated improvements in functional performance despite improvements in leg muscle strength (Glasser 1986; Engardt et al. 1995; Kim et al. 2001; Ouelette et al. 2004). Thus a more intricate understanding of how stroke impacts muscle performance is needed so that clinicians can design more effective therapeutic exercises that are specific to the impairments identified through exploratory research.

The intention of this thesis was to therefore study muscle performance in the paretic and so-called nonparetic lower extremities of individuals with chronic stroke and add to the scientific foundation of stroke rehabilitation. More specifically, this work was divided into three related studies that examined mechanical muscle properties in individuals with stroke. The first study measured isometric torque, as well as the time to develop and reduce torque, the second measured isometric torque across joint range of motion, and the third measured relative
preservation of eccentric versus concentric torque. The following section provides background information and definitions of selected physiological and mechanical muscle properties relevant to the three studies. A literature review follows and leads to the theses purpose, research questions, and hypotheses.

SELECTED PHYSIOLOGICAL AND MECHANICAL MUSCLE PROPERTIES

Muscle force, joint torque, and muscle strength

The amount of force a muscle can generate is dependent on its cross-sectional area (muscle size), length (length-tension relationship), movement velocity (force-velocity relationship), and neural activation level (motor unit recruitment). Joint torque is the product of muscle force and moment arm and clinically represents a measure of muscle strength. Thus to increase joint torque or muscle strength one can increase muscle force, increase moment arm length, or increase the angle between muscle force application and the axis of rotation toward 90°. The joint angle at which peak torque occurs is not necessarily the same angle at which peak muscle force occurs (Lieber 2002).

Sarcomeres and contractile filaments (myosin and actin myofilaments)

Muscle fibers are composed of myofibrils arranged in parallel and myofibrils are composed of sarcomeres arranged in series. Sarcomeres are the functional units of muscle contraction. During a contraction, each sarcomere shortens approximately 1 micrometer. However, due to their series arrangement, a whole muscle will shorten several centimeters. Each sarcomere is composed of two major sets of myofilaments. One set is relatively thick and heavy and is composed of the protein myosin, and the other is relatively thin and light and is composed
of the protein *actin*. It is the active intermingling of myosin and actin that produces muscle shortening (Lieber 2002).

The myosin containing filament is a polymer of myosin molecules composed of two subunits, myosin heavy chain and myosin light chain. The myosin heavy chain consists of two discrete components, the light meromyosin (LMM) and the heavy meromyosin (HMM). The LMM makes up the heavy chain’s rod-like tail, and the HMM makes up the heavy chain’s globular head and neck. It is this important portion of the myosin molecule that forms the well known “cross-bridge” with actin to power muscle contraction. The myosin light chain possibly provides structural support for the globular head. Interestingly, both the myosin heavy chain and myosin light chain proteins exist in several isoforms. The various isoforms have different functional properties and form the basis of fiber types. Myosin isoforms may exist within the same muscle and are extremely plastic, changing in response to activity levels and muscle injury (Ricoy et al. 1998; Lutz and Lieber 2002).

The actin containing filament regulates force generation. The actin molecule is spherical in shape. Collectively, the molecules form a helix with a long groove that extends along the filaments length. Tropomyosin sits in this groove and is a regulatory protein. A second regulatory protein, troponin, is interspersed along this same groove and is responsible for initiating muscle contraction. At rest, tropomyosin prevents actin and myosin from binding by blocking the active sites of actin. During muscle contraction, troponin binds with calcium and undergoes a conformational change that pulls tropomyosin off the active sites to allow cross-bridge formation to occur (Scott et al. 2001; Lieber 2002).

**Length-tension relationship**

The amount of overlap between actin and myosin filaments predicts the amount of force generated by skeletal muscle. The *active* sarcomere length-tension curve was first described by
Gordon and colleagues in the 1960's using a single isolated fiber extracted from the muscle of a frog (Gordon et al. 1966). The force generated from sequential isometric contractions was plotted against sarcomere length and revealed a domed shaped curve with a descending limb, plateau region, and an ascending limb (see figure 1-1). The descending limb begins with the muscle fiber stretched to the point were there is no overlap of actin and myosin filaments. Thus no force is generated because cross-bridge formation cannot occur. As the fiber shortens, actin and myosin filaments begin to overlap, cross-bridge formation occurs, and force generation increases. This increase in force with fiber shortening continues until the plateau region of the curve, where despite further actin and myosin overlap no change in force occurs. This is due to the bare region in the middle of the myosin filament where no cross-bridges can be formed with the overlapping actin filament. Maximum force generation occurs over the plateau region and is thus considered the muscle’s optimal length. Further fiber shortening beyond the plateau region results in a slow decrease of force as actin filaments from one side of the sarcomere overlap with actin filaments on the other side. This “double overlap” interferes with cross-bridge formation and forms the shallow portion of the ascending limb. The steep portion of the ascending limb forms as the myosin filament begins to collide with the sarcomere Z-disk causing a rapid decline in force production. When no further shortening can occur, muscle force drops to zero and the curve is completed. It is important to note that the length-tension relationship is based off of a series of isometric single-fiber contractions, thus it does not predict force production of whole muscles contracting under dynamic conditions (for review see Rassier et al. 1999).
Determination of the active length-tension relationship is invasive and requires the difficult task of isolating a single intact muscle fiber, thus it has not been examined in any stroke model (animal or human). However, it should be mentioned that using muscle biopsies (taken during reconstructive surgery) and micromechanical testing equipment, Friden and Lieber (2004) were able to measure the sarcomere length in spastic muscle cells of children with cerebral palsy. They found that spastic sarcomeres had a shorter resting length and were twice as stiff under tension when compared with controls. Since the resting sarcomere length was shorter and thus the passive length-tension relationship was altered, it is not unreasonable to assume that the active length-tension relationship would also be altered in spastic muscles; a shorter sarcomere would change the amount of actin and myosin filament overlap for any given sarcomere length, and therefore change the amount of force generated. However, caution should be taken in applying these findings to individuals with stroke, as the above findings represented children with cerebral palsy (mean age = 8 years) compared with healthy adults (mean age = 37 years). Thus, the sarcomere adaptations reported may be due to developmental changes (i.e. response to bone growth) and not spasticity.
**Force-velocity relationship**

The velocity of shortening (concentric) and lengthening (eccentric) contractions predicts the amount of force generated by skeletal muscle under isotonic conditions (constant load) (see figure 1-2). For concentric contractions, as shortening velocity increases, muscle force rapidly decreases in a curvilinear fashion. This is explained by the rate of cross-bridge cycling. The faster actin and myosin filaments slide past each other, the less time there is for cross-bridge formation and thus lower force is produced. Conversely, for eccentric contractions, as lengthening velocity increases, muscle force rapidly increases (Lieber et al. 2004).

Unfortunately, the cross-bridge theory does not explain the mechanics of eccentric contractions at high movement velocities (Harry et al. 1990). The above relationship was originally determined using animal models and electrical stimulation (Hill 1938) and does not necessarily translate to the torque-velocity relationship in whole human muscles. For example, concentric torque significantly decreases with increasing movement velocities but eccentric torque does not significantly increase with increasing movement velocities in the human knee extensors (Westing et al. 1988, 1990).

![Figure 1-2. Force-velocity relationship. The three circled numbers represent: 1. concentric contraction, 2. isometric contraction, and 3. eccentric contraction. Adapted from Lieber (2004)]
In individuals with stroke, as shortening velocity increases, torque produced in the paretic limb decreases at a faster rate than torque produced in the nonparetic limb (Lum et al. 2004; Kim et al. 2005). Thus during concentric contractions, the torque-velocity relationship has been shown to be altered in the paretic limb. However, this has only been demonstrated for a few select upper extremity joint actions and may not generalize to all joint actions in individuals with stroke. The response to increasing lengthening velocities in individuals with stroke has not been studied.

**Motor units and muscle fiber types**

A motor unit consists of an alpha motor neuron (originating from the spinal cord) and the muscle fibers it innervates. The number of fibers innervated by a single motor neuron is highly variable (from just a few to several hundreds) and is referred to as the innervation ratio. The muscle fibers of a single motor unit are interspersed amongst muscle fibers of other motor units, thus the forces generated by a single motor unit are spread over a large area of the muscle tissue. Muscle fibers within a motor unit all share very similar structural, contractile, and metabolic properties (Lieber 2002). Based on these properties, several different classification systems have been developed over the years (i.e. histochemical staining for myosin adenosinetriphosphatase [ATPase], biochemical identification of metabolic enzymes, and immunohistochemistry for identification of myosin heavy chain isoforms). The current gold-standard is classification by immunohistochemistry, which can identify specific cellular proteins. Using antibodies for protein identification, muscle fibers are classified based on the three different myosin heavy chain molecules (i.e. isoforms) that are expressed within different human fiber types (Lieber 2002). These myosin isoforms are referred to as MHCI, MHCIIa, and MHCIIx/d (formally identified as MHCIIb). Because the globular head region of the myosin heavy chain contains the adenosine triphosphate (ATP) binding site, which ultimately provides the energy to power
cross-bridge cycling, isoforms MHCI, MHCIIa, and MHCIIx/d determine the force and velocity of muscle fiber contraction and correspond to the well known fiber types: I, IIA, and IIB respectively. Furthermore, type I, IIA, and IIB correspond to the biochemical classification of slow-twitch, fast-twitch fatigue-resistant, and fast-twitch fatigable respectively (Scott et al. 2001).

Slow-twitch fibers (type I), as their name suggests, produce and reduce force relatively slowly. They are smaller and have fewer contractile filaments than their fast-twitch counterparts. Slow-twitch fibers are fatigue resistant, whereby low levels of force can be produced and sustained over a long period of time via aerobic or oxidative metabolism (Kandel et al. 2000).

Fast-twitch fibers (Type II A and B) produce and reduce force relatively fast. As previously alluded to, the myosin composition (isoforms) of fast-twitch fibers is different than slow-twitch fibers and is responsible for the rapid shortening velocities. Fast-twitch fatigable fibers (type IIB) are characterized by brief bursts of force and require several hours to recover due to the rapid depletion of glycogen and production of lactic acid from anaerobic or glycolytic (without oxygen) metabolism. Generally, fast-twitch fatigue-resistant fibers (type IIA) have some aerobic capacity and thus can resist fatigue for several minutes while still generating fast twitches (Kandel et al. 2000).

As previously mentioned, muscle fibers are plastic and can change from one type to another in response to changes in activity levels, exercise or rehabilitation interventions, and changes in neuronal input. In general, severe deconditioning (i.e. spinal cord injury) causes atrophy and a conversion in the slow to fast-twitch direction. Thus low levels of force are generated quickly and are only sustained for short periods of time. A conversion in the fast to slow-twitch direction occurs with both endurance and resistance training, whereby fast-twitch fibers become more efficient at utilizing oxygen while maintaining fast rates of force production and reduction (thus a conversion from type IIB to IIA can occur). Lastly, loss of alpha motor
neurons due to aging or disease can result in reinnervation of muscle fibers that leads to fiber type conversion, whereby the fiber takes on the properties of its new motor unit (for a detailed review see Scott et al. 2001).

In individuals with stroke, fast-twitch fiber atrophy occurs in both paretic and nonparetic muscles (Dattola et al. 1993; Hachisuka et al. 1997) and an increased percentage of slow-twitch fibers without atrophy occurs in paretic muscles (Dattola et al. 1993). There is also evidence that a new motor unit emerges with slow-twitch contraction times and increased fatigability (Young and Mayer 1982). However, it is important to note that this latter finding was reported for the first dorsal interosseous muscle and has not been reproduced or extended to other muscles since its initial publication.

**Motor unit recruitment and rate coding**

To increase muscle force, motor units are recruited in an orderly fashion from those with small axonal diameters to those with large axonal diameters. In general, small motor units have low firing thresholds and innervate slow-twitch motor units. Large motor units have high firing thresholds and innervate fast-twitch motor units. As muscle force decreases, motor units are derecruited in the opposite order, from large to small. This phenomenon was first described using animal data by Henneman and colleagues and it is known as the "size principle" (Henneman et al. 1965; Henneman et al. 1975). Milner-Brown and colleagues validated these findings in humans and the "size principle" provides a mechanism for the smooth increase in muscle force generated during isometric contractions (Milner-Brown et al. 1973). However, more recent evidence has shown that during eccentric contractions in both upper (Howell et al. 1995) and lower (Nardone et al. 1989) extremity muscles, large fast-twitch motor units are selectively activated while slow-twitch motor units are concurrently deactivated. Thus it is
important to note that the “size principle” of motor unit recruitment-derecruitment may not necessarily apply to dynamic muscle contractions.

Altering the firing frequency of alpha motor neurons is one way in which the central nervous system can control muscle force production. Motor unit firing frequencies are increased when high muscle forces are required and decreased when low muscle forces are required. This strategy is known as rate coding or temporal summation (Lieber 2002).

It is not known if the actual order of motor-unit recruitment is altered post-stroke. However, the motor units in the paretic muscles of individuals with stroke demonstrate reduced firing rates, reduced firing thresholds, and the range of firing thresholds are more narrow than normal. In addition, individuals with stroke do not increase motor unit discharge rates with increases in voluntary force production, thus rate coding is impaired post-stroke (Rosenfalck and Andreassen 1980; Gemperline et al. 1995).
LITERATURE REVIEW

Muscle weakness on the side of the body contralateral to the cerebral lesion (i.e. the paretic side) is a distinguishing feature of stroke. Minor reductions in strength have also been reported in the limb muscles ipsilateral to the cerebral lesion (i.e. the so-called nonparetic side) (Adams et al. 1990; Sinkjaer and Magnussen 1994; Davies et al. 1996; Andrews and Bohannon 2000; Harris et al. 2001). Adams et al., (1990) reported a combined 7% significant reduction in nonparetic leg torque of 8 isometric joint actions in individuals with stroke and suggested that deficits ipsilateral to the lesion may, in part, be due to primary damage of uncrossed corticofugal pathways. More recently, Harris et al. (2001) assessed knee extension torque of the nonparetic leg 48 hours post-stroke and again one week later and reported a 30% reduction in torque from time one to time two. This alternatively suggests that muscle weakness in the nonparetic leg is a secondary complication of stroke and may be due to inactivity. However, not all authors have reported torque deficits in nonparetic leg joints post-stroke (Newham and Hsiao 2001). Thus further study is required to determine how stroke influences torque magnitude in the nonparetic limb joints.

In addition to muscle weakness, individuals with stroke often demonstrate slowness of movement during functional tasks such as walking and rising from a chair (Olney and Richards 1995; von Schroeder et al. 1995; Cheng et al. 1998; Chou et al. 2003). To optimize performance and meet environmental demands, muscles on both sides of the body must be able to develop and reduce force in a timely manner. Unfortunately, studies examining temporal parameters of muscle function in the legs of individuals with stroke are scarce and limited to the knee joint. Bohannon and Walsh (1992) reported that the time to develop peak knee extension torque was significantly longer in the paretic leg when compared to the nonparetic leg (1.33s versus 1.18s respectively). Similarly, Tsuji and Nakamura (1987) reported that the rate of tension development was slower, and the time to reach maximum tension was longer in the paretic and
nonparetic knee extensors when compared with controls (only the paretic side reached significance). Similar findings have been reported for the paretic (Canning et al. 1999; McCrea et al. 2003) and nonparetic (McCrea et al. 2003) upper extremity, suggesting that slowness to develop torque is a widespread problem following stroke. The time to reduce torque has not been studied in the lower extremity, however the paretic arm has been shown to be 22% slower than normal when averaged across 8 joint actions (McCrea et al. 2003). The same study reported no significant impairment in the nonparetic arm.

Performance of daily tasks requires the ability to produce joint torque across range of motion, or from short to long muscle lengths. Clinically it is not uncommon for individuals with stroke to present with variable levels of muscle strength depending on the muscle length required for the task at hand. For example, individuals with stroke have been shown to have difficulty using their paretic elbow during the last 20° of extension while mid-range tasks were performed with relative ease (Bohannon 1991). Furthermore, authors examining the torque-angle relationship of the paretic elbow reported exaggerated weakness at short versus long muscle lengths in both the flexors and extensors (Ada et al. 2000; Ada et al. 2003; Koo et al. 2003). The torque-angle relationship of paretic lower extremity joints have not been reported in the literature despite the clinical observation of exaggerated weakness of the knee extensors when contracting at short muscle lengths (i.e. terminal knee extension during gait and curb climbing). In addition, the torque-angle relationship has not been examined in the nonparetic leg, even though the potential of task-specific strength changes across joint range of motion may occur due to the well documented compensatory use of this leg during functional activities (Winstein et al. 1989; Eng and Chu 2002; Chou et al. 2003; Lomaglio and Eng in press).

Lastly, it is important to point out that functional movement is accomplished through a complex interaction of isometric, concentric, and eccentric muscle contractions. Reductions in lower extremity isometric and concentric torque post-stroke have been well documented (Adams
et al. 1990; Davies et al. 1996; Andrews and Bohannon 2000; Harris et al. 2001). However, eccentric torque production has received very little attention, despite its important role during functional activities. One study tested the effects of isokinetic eccentric versus concentric strength training for paretic knee extensors. The pre-intervention relative eccentric torque (paretic torque expressed as a percentage of nonparetic torque) was 22% higher than relative concentric torque at 180°/s. Although a statistical comparison was not made, their data suggests that eccentric torque was less affected by stroke than concentric torque (Engardt et al. 1995). Interestingly, this same phenomenon has been reported in other populations with upper motor neuron lesions due to amyotrophic and primary lateral sclerosis (Griffin et al. 1994), multiple sclerosis (Ponichtera et al. 1992), and cerebral palsy (Knutsson et al. 1997; Damiano et al. 2000). Thus, understanding how stroke affects concentric and eccentric torque will have an important impact on the design of future rehabilitation interventions.
PURPOSE

The purpose of this study was to provide a better understanding of how chronic stroke affects mechanical muscle properties (times to develop and reduce torque, torque-angle relationships, and isometric, concentric, and eccentric torque) in the paretic and nonparetic lower extremities as compared to healthy control values.

RESEARCH QUESTIONS AND HYPOTHESES

Question I: Do individuals with chronic stroke demonstrate reduced isometric torque and slower than normal times to develop and reduce torque in both paretic and nonparetic lower extremity joint actions (ankle, knee, and hip flexion and extension)? (Chapter 2)

Hypothesis

Both legs will demonstrate significant isometric torque reductions and slower than normal times to develop and reduce torque across ankle, knee, and hip flexion and extension joint actions.

Question II: Do the paretic and nonparetic knee joints of individuals with chronic stroke demonstrate non-uniform torque impairment across flexion and extension joint range of motion? In other words, does stroke alter the torque-angle relationships about the knee joint? (Chapter 3)

Hypothesis

The torque-angle relationships about the knee joint will be altered bilaterally in individuals with stroke. More specifically, the paretic knee extensors and flexors will demonstrate exaggerated weakness when required to contract at short muscle lengths (i.e. when the knee is straight the extensors will be relatively weaker and when the knee is bent the flexors will be relatively weaker), and the nonparetic knee extensors and flexors will demonstrate relative strength gains when the knee joint is in a flexed position.
Question III: Do individuals with chronic stroke demonstrate an abnormal imbalance between concentric and eccentric torque production in the paretic and nonparetic lower extremity joint actions? (Chapter 4)

**Hypothesis**

Both legs will demonstrate a significant relative preservation of eccentric torque. In other words, concentric torque will be relatively more impaired than eccentric torque across ankle, knee, and hip flexion and extension joint actions.
Chapter 2: Strength and temporal parameters in paretic and nonparetic leg muscles of individuals with chronic stroke

Abstract

**Background and Purpose:** Temporal parameters of muscle contraction and relaxation may be impaired bilaterally in individuals with stroke but have not been fully explored. Muscle weakness in the paretic limbs has been well documented, however there are conflicting reports as to whether or not weakness exists in the nonparetic limbs. The purpose of this study was to quantify peak isometric torque, time to develop torque, and time to reduce torque across six paretic and nonparetic lower extremity joint actions (ankle, knee, and hip, flexion and extension) in individuals with chronic stroke. **Methods:** Nineteen subjects with stroke and 19 control subjects were recruited from the community. The lower extremity muscles of the paretic and nonparetic sides of subjects with stroke were compared to the nondominant leg of control subjects using the isometric mode of a dynamometer. Group differences were assessed using a separate multivariate analysis of variance (MANOVA) for each muscle performance parameter followed by post-hoc Tukey Tests. **Results:** Peak torque was significantly reduced for all paretic joint actions except hip extension. Except for ankle plantarflexion, the nonparetic peak torques were not significantly reduced. Time to develop torque was significantly slower for all paretic joint actions and five out of six nonparetic joint actions. Time to reduce torque was significantly slower for all paretic joint actions and three out of six nonparetic joint actions. **Conclusions:** In individuals with chronic stroke temporal parameters of muscle function demonstrate widespread bilateral impairment, however significant muscle weakness appears to be present primarily in paretic leg muscles. Rehabilitation clinicians should incorporate therapeutic exercises to strengthen paretic leg muscles and methods to improve the speed of contraction and relaxation in both paretic and nonparetic leg muscles should be explored.
INTRODUCTION

Individuals living with stroke often experience chronic muscle weakness, or reduced torque production, contralateral to the cerebral lesion (i.e. the paretic side). This weakness has been shown to relate to functional performance (Nakamura et al. 1985; Bohannon and Walsh 1991; Olney et al. 1994; Cameron et al. 2003; Hsu et al. 2003; Kim and Eng 2003; Lomaglio and Eng in press) and as a result clinicians are beginning to move away from the traditional neurodevelopmental therapy approach (Davies 2000) and are including strength training into stroke rehabilitation (Engardt et al. 1995; Teixeira-Salmela et al. 1999; Ng and Shepherd 2000; Carr and Shepherd 2004; Jette et al. 2005). Muscles ipsilateral to the cerebral lesion (the nonparetic side) are often assumed by clinicians to be unaffected by stroke and the strength of these muscles are frequently assessed to determine an individual’s pre-morbid strength and recovery of the paretic side. However, some reports suggest strength in the nonparetic leg (Adams et al. 1990; Sinkjaer and Magnussen 1994; Davies et al. 1996; Andrews and Bohannon 2000; Harris et al. 2001) of individuals with stroke is reduced 7% to 35% when compared with controls. Conversely, Newham and Hsiao (2001) reported no significant differences between the control group and nonparetic knee extensors and flexors of subjects with stroke. Thus further study is required to resolve these conflicting reports.

In addition to generating sufficient joint torque, muscles on both sides of the body must be able to develop and reduce force rapidly to allow for smooth movements and balance control. Unfortunately, temporal parameters of muscle function following stroke have received only moderate attention in the literature. Slowness to develop torque has been reported for multiple joint actions of the paretic (Canning et al. 1999; McCrea et al. 2003) and nonparetic arm (McCrea et al. 2003). In the leg, the time to reach peak torque has been shown to be significantly longer for paretic knee extension (Tsuji and Nakamura 1987; Bohannon and Walsh
To our knowledge, no other leg joint actions have been studied and the time to reduce torque has not been examined in any of the leg joint actions of individuals with stroke.

In individuals with stroke, impairments in the paretic lower extremity have been shown to relate to participation restrictions (Desrosiers et al. 2003), which ultimately affect quality of life; thus a comprehensive understanding of bilateral muscle impairments are needed to improve lower extremity rehabilitation interventions for individuals living with the effects of stroke.

The purpose of this study was to quantify peak isometric torque, time to develop torque, and time to reduce torque across six lower extremity joint actions (ankle, knee, and hip flexion and extension) on the paretic and nonparetic sides of individuals with chronic stroke, and the nondominant leg of matched controls.
METHODS

Subjects. Nineteen subjects (6 women and 13 men) with residual hemiparesis following a single stroke were recruited voluntarily from the community. Subjects were at least 1 year post-stroke, 50 years of age or older, able to walk independently for 10 metres with or without an assistive device, free from severe musculoskeletal conditions, and able to follow multistep commands. In addition, 19 neurologically healthy subjects (6 women and 13 men) of similar age were recruited to serve as controls. Approval was obtained from the local university and hospital ethics committees and all subjects provided an informed consent (Appendix IV and V). For subjects with stroke, motor recovery of the paretic lower extremity was assessed with active movements using the leg and foot portion of the Chedoke-McMaster Stroke Assessment Score Form (Gowland et al. 1993); stage 1 represents flaccid paralysis and stage 7 represents normal movement patterns (Appendix VI). Disability was assessed using the Stroke Functional Classification levels from the American Heart Association Stroke Outcome Classification Score (Kelly-Hayes et al. 1998); level I represents complete independence in basic and instrumental activities of daily living, and level V represents complete dependence; requires full-time care (Appendix VII). Lower extremity muscle tone of the ankle, knee, and hip flexors and extensors was assessed using the Modified Ashworth Scale (MAS) (Bohannon and Smith 1987) which describes the resistance of muscle to manual passive movement; 0 = no increase in muscle tone, and 4 = paretic part(s) rigid in flexion or extension (Appendix VIII). The activity level of all subjects was assessed using the Physical Activity Scale for Individuals with Physical Disabilities (Washburn et al. 2002). This self-report questionnaire provides an estimate of how many days per week, and hours per day are spent being active; scores are calculated as the average hours of activity daily, multiplied by a metabolic equivalent value (MET hr/d), and summed over items 2 through 13 (Appendix IX). Group characteristics are summarized in Table 2-1. Approximately
6 weeks after the initial assessment, 9 subjects with stroke returned for repeat testing to establish test-retest intrarater reliability.

**Joint torque assessment.** The isometric mode of a Kin-Com dynamometer (Chattanooga Group, TN) was used to assess maximum voluntary joint torques of the ankle, knee, and hip flexors and extensors of the paretic and nonparetic sides of subjects with stroke, and the nondominant leg of control subjects (determined by preference for kicking a ball). Unlike the upper extremity, it has been shown that there is no dominance for maximal muscle strength (Holder-Powell and Rutherford 2000; Skelton et al. 2002) or power (Demura et al. 2001) in the lower extremity. Thus the decision to test the nondominant leg should not bias the results in either direction.

Instrument calibration was tested prior to the study with known weights and was accurate to within +/- 1 N. The ankle and knee torques were tested at a 90° sitting angle and the hip torques were tested in a 40° semi-reclined position. Three straps stabilized the trunk and pelvis (two criss-crossing over the chest and one just distal to the anterior-superior iliac spines) and the subject’s hands rested comfortably in their laps. In addition, a rigid clamp placed over the distal thigh musculature of the test leg was used for extra stabilization during measurement of knee torques. The dynamometer axis was aligned with the joint line of the ankle, knee, and hip (lateral malleolus, lateral femoral condyle, and greater trochanter respectively). A rigid metallic ankle attachment provided by the manufacturer was used for assessment of ankle torques. For knee and hip torques, the cuff of the dynamometer was positioned three finger breaths above the medial malleolus and three finger breaths proximal to the popliteal fossa respectively (for positioning details see Appendix X). All joint torque measurements were taken from a midrange contraction. Blood pressure was monitored before testing and once after each joint tested. Testing proceeded only when blood pressure was at or below 140/90.
Before each trial subjects were relaxed and instructed "At the sound of the click (an auditory cue) push (or pull) as hard and as fast as you can and hold this effort, at the second click, immediately relax." Subjects understood that they were to develop maximum torque as fast as possible, hold it, and reduce torque as fast as possible. Each trial lasted three seconds and verbal encouragement was provided to facilitate a maximum effort. One submaximal trial followed by one maximal trial were performed as practice before each new joint action tested. Three additional maximal trials were performed for analysis. During testing, analogue signals of force from the dynamometer were fed through an A-D converter (National Instruments) and collected at 100Hz for analysis. For each trial the torque profile was displayed on a computer screen and visually inspected by the same examiner; bad trials, due to pushing or pulling in the wrong direction or irregular relaxation, were repeated by the subject and subsequently eliminated before analysis (7.5% of the total number of trials across all subjects needed to be repeated). To avoid fatigue, each trial was separated by 30 seconds rest and each new joint action was separated by 5 minutes rest. To reduce a possible order effect, testing began with extension for approximately half of the subjects and flexion for the remainder.

**Joint torque analyses.** Data were processed with MATLAB using custom software. All torque measurements were normalized to body mass and corrected for the effect of gravity on the lower extremity segment and cuff of the dynamometer. Once again, individual trials were visually inspected by the same examiner and segmented trials were eliminated from analysis (2.5% were eliminated). **Peak torque** was measured as the maximum torque maintained over a period of 250ms. The **time to develop** torque was measured between 10% and 70% of peak torque and reflects the muscles' activation time. Similarly, the **time to reduce** torque was measured between 70% and 10% of peak torque and reflects the muscles' deactivation time (Figure 2-1). These definitions have been used previously in the upper extremity with good reliability (McCrea et al. 2003).
Statistical analyses. Descriptive statistics were calculated for subject characteristics (Table 2-1) and lower extremity torque parameters (peak torque, time to develop torque, and time to reduce torque).

Intraclass correlations, ICC (3,3) (Shrout and Fleiss 1979), and standard error of measurement (SEM) were used to determine intrarater reliability of peak torque, time to develop torque, and time to reduce torque for each joint action of the paretic and nonparetic legs. ICCs below 0.70 were examined for outliers using visual inspection of the appropriate scatterplots; for each joint action of the paretic and nonparetic times to develop and reduce torque, a single outlier was removed before statistical analyses were performed.

Three separate multivariate analyses of variance (MANOVA) were performed to determine whether group differences (independent variables: paretic side, nonparetic side, and control group) existed across lower extremity joint actions (dependent variables: ankle, knee, and hip flexion and extension) for 1) peak torque, 2) time to develop torque, and 3) time to reduce torque. For each MANOVA, assumptions of normality and homogeneity of variance were tested; although with equal group sizes the MANOVA has been shown to be robust to violations...
of these assumptions (Pearson 1931; Horsnell 1953; Glass et al. 1972). Significant group differences for each MANOVA were followed by a Tukey honestly significant difference post-hoc analysis. All statistical analyses were performed using SPSS 11.5 software with a significance level of $P \leq 0.05$. 
# RESULTS

Table 2-1. Characteristics of subjects with stroke ($N=19$) and control subjects ($N=19$)

<table>
<thead>
<tr>
<th></th>
<th>Stroke ($n=19$)</th>
<th>Control ($n=19$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (y)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>64.9 7.6</td>
<td>53–77</td>
</tr>
<tr>
<td><strong>Gender (F/M)</strong></td>
<td>6/13</td>
<td>6/13</td>
</tr>
<tr>
<td><strong>Mass (kg)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>75.9 13.1</td>
<td>53.0–101.0</td>
</tr>
<tr>
<td><strong>Height (m)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>1.71 0.10</td>
<td>1.54–1.90</td>
</tr>
<tr>
<td><strong>BMI (kg/m²)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>26.2 4.3</td>
<td>20.2–35.0</td>
</tr>
<tr>
<td><em><em>Physical Activity</em> (MET hr/d)</em>*</td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>13.5 9.5</td>
<td>2.2–40.3</td>
</tr>
<tr>
<td><strong>Time since stroke (yrs)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>7.5 7.3</td>
<td>1–29</td>
</tr>
<tr>
<td><strong>Stroke Type</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td>(ischemic/hemorrhagic)</td>
<td>11/8</td>
<td>I-IV</td>
</tr>
<tr>
<td><strong>Paretic side (R/L)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>6/13</td>
<td>I-IV</td>
</tr>
<tr>
<td><strong>Functional Classification† (class I-V)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>I-IV</td>
</tr>
<tr>
<td><strong>Impairment‡ (stage 1–7)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td>(flexors/extensors)</td>
<td>Leg 6</td>
<td>4–7</td>
</tr>
<tr>
<td></td>
<td>Foot 4</td>
<td>2–7</td>
</tr>
<tr>
<td><strong>MAS§ (0-4; flexors/extensors)</strong></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td>Ankle</td>
<td>0/1</td>
<td>0-1/0-3</td>
</tr>
<tr>
<td>Knee</td>
<td>1/0</td>
<td>0-3/0-2</td>
</tr>
<tr>
<td>Hip</td>
<td>0/0</td>
<td>0-0/0-1+</td>
</tr>
</tbody>
</table>

* Physical Activity Scale for Individuals with Physical Disabilities  
† Stroke Functional Classification level (American Heart Association)  
‡ Chedoke-McMaster Stroke Assessment Score  
§ median is reported for the ordinal MAS, Functional Classification, and Impairment scores
Reliability. For the paretic lower extremity joint actions the mean ICCs (SEM) were 0.93 (6.3N), 0.82 (0.085s), and 0.81 (0.118s) for peak torque, time to develop torque, and time to reduce torque respectively. For the nonparetic lower extremity joint actions the mean ICCs (SEM) were 0.85 (6.3N), 0.80 (0.066s), and 0.77 (0.114s) for peak torque, time to develop torque, and time to reduce torque respectively (for details see Appendix XIII).

Peak torque. The MANOVA for lower extremity peak torque (ankle, knee, and hip flexion and extension) revealed a significant main effect of group (Wilk’s $\lambda = 0.444, P < 0.001$). The peak torques of the paretic leg muscles (mean = 0.542 Nm/kg) were all lower than the control group (mean = 0.907 Nm/kg) and post-hoc tests revealed statistical significance for all joint actions ($P = 0.001$-$0.029$) except hip extension, which approached significance ($P = 0.065$). Conversely, peak torques of the nonparetic leg muscles (mean = 0.793 Nm/kg) were not significantly lower than the control group (mean = 0.907 Nm/kg), nor did they approach significance ($P = 0.507$-$0.999$), however, nonparetic ankle plantarflexion torque was one notable exception, which was only 68% of the control value ($P = 0.009$) (Figure 2-2).

Time to develop torque. The MANOVA for lower extremity time to develop torque (ankle, knee, and hip flexion and extension) revealed a significant main effect of group (Wilk’s $\lambda = 0.384, P < 0.001$). The times to develop torque of both the paretic (mean = 0.423s) and nonparetic muscles (mean = 0.342s) were all slower than the control group (mean = 0.196s) and post-hoc tests revealed statistical significance for all paretic joint actions ($P = 0.001$-$0.010$), and five out of six nonparetic joint actions ($P = 0.003$-$0.016$); ankle dorsiflexion ($P = 0.441$) was not significant (Figure 2-3).

Time to reduce torque. The MANOVA for lower extremity time to reduce torque (ankle, knee, and hip flexion and extension) revealed a significant main effect of group (Wilk’s $\lambda = 0.525, P < 0.001$). The times to reduce torque of both the paretic (mean = 0.483s) and nonparetic (mean = 0.379s) muscles were all slower than the control group (mean = 0.200s) and
post-hoc tests revealed statistical significance for all paretic joint actions ($P = 0.001-0.003$) and three out of six nonparetic joint actions ($P = 0.004-0.016$); ankle dorsiflexion ($P = 0.170$), knee flexion ($P = 0.249$), and knee extension ($P = 0.075$) did not reach significance (Figure 2-4).
Figure 2-2. Normalized peak torques and standard deviations for each lower extremity joint action of the paretic side, nonparetic side, and control group. * = paretic joint action significantly different than control ($P \leq 0.05$), ** = nonparetic joint action significantly different than control ($P \leq 0.05$).
Figure 2-3. Times to develop torque and standard deviations for each lower extremity joint action of the paretic side, nonparetic side, and control group. * = paretic joint action significantly different than control \( (P \leq 0.05) \), ** = non paretic joint action significantly different than control \( (P \leq 0.05) \).
Figure 2-4. Times to reduce torque and standard deviations for each lower extremity joint action of the paretic side, nonparetic side, and control group. * = paretic joint action significantly different than control ($P \leq 0.05$), ** = nonparetic joint action significantly different than control ($P \leq 0.05$).
DISCUSSION

Peak torque. Not surprisingly, when compared with controls, individuals with stroke demonstrated a 40% reduction in paretic leg strength when averaged across all 6 joint actions. Paretic ankle plantarflexion demonstrated the most weakness at 63% and paretic hip flexion the least at 28% of control values. Adams et al. (1990) also reported that isometric paretic ankle plantarflexion was the most impaired and paretic hip flexion the least in individuals with stroke, although these findings were relative to the nonparetic side.

With the exception of ankle plantarflexion, lower extremity joint actions of the nonparetic side were not significantly weaker than the control group, and nonparetic knee extension strength was preserved. Although it should be mentioned that when averaged across all 6 joint actions, the combined nonparetic leg strength was reduced approximately 12%; thus with a larger and more homogeneous sample, a significant, but slight deficit might be detected for some nonparetic muscle groups. Adams et al. (1990) reported similarly a combined 7% significant reduction in nonparetic leg strength across 8 isometric joint actions in individuals with hemiparesis when compared with controls, however, unlike the present study, there was no tendency for any one joint action to be more or less affected than others.

Ankle plantarflexion strength was the only nonparetic muscle which was significantly reduced compared to controls, and it was reduced by a substantial amount (32%). This is in agreement with Sinkjaer and Magnussen (1994) who reported a reduction of 35% in the nonparetic plantarflexors of individuals with stroke. Some authors (Colebatch and Gandevia 1989; Adams et al. 1990; Sinkjaer and Magnussen 1994; McCrea et al. 2003) have suggested that strength deficits ipsilateral to the lesion may, in part, be due to damage of uncrossed corticofugal pathways. However, the ventral corticospinal tract, which has some fibers that remain uncrossed, primarily projects to axial and proximal limb muscles (Davidoff 1990) and therefore one would expect a relative sparing of the distal nonparetic ankle plantarflexors.
Reduced physical activity leading to disuse muscle atrophy may also help explain weakness on the ipsilateral side, and in fact the average physical activity scores of subjects with stroke were only 60% of control subjects; however, one would expect that physical inactivity would result in a relatively uniform impairment across joint actions, and particularly across anti-gravity muscles (Lieber 2002). Thus, this does not explain the predominant nonparetic ankle plantarflexor weakness with concurrent preservation of nonparetic knee extensor strength, which are both anti-gravity muscle groups. A more likely explanation may be due to secondary changes in nonparetic leg strength resulting from altered biomechanical strategies used during bilateral leg activities such as walking. It is well known that individuals with stroke walk slower than normal (Olney and Richards 1995; von Schroeder et al. 1995) and Olney et al. (1991) has shown that as gait velocity decreases, the power generated by the nonparetic hip and ankle also significantly decreases ($r = 0.66$, $r = 0.75$, respectively), however, nonparetic knee power does not. In addition, Olney and Richards (1995) compared a report describing hemiparetic gait due to stroke (Olney et al. 1991) with a report describing normal gait (Winter 1991), and identified reduced power generation of nonparetic ankle plantarflexors during push-off and increased power generation of nonparetic knee extensors during push-off and early swing relative to healthy subjects walking at similar speeds (Olney and Richards 1995). Thus, given that walking is a task that is performed frequently throughout the day, the above mentioned adaptations may explain why our subjects with chronic stroke displayed weakness of nonparetic ankle plantarflexion, while nonparetic knee extension strength remained preserved. Furthermore, the aforementioned study by Adams et al. (1990) who reported a small amount of uniform weakness across nonparetic lower extremity joint actions may be due to the fact that most of the subjects with stroke were in the acute phase of recovery (< 10 weeks), and thus muscle adaptations due to secondary compensations had not yet occurred.
**Time to develop torque.** When compared with controls and averaged across all 6 joint actions, the times to develop torque were 54% and 43% slower for the paretic and nonparetic sides respectively (though due to high between-subject variability, not all joint actions of the nonparetic side reached significance). In a similar study, Tsuji and Nakamura reported that the time to develop maximum tension in the paretic knee extensors of subjects with stroke was 32% slower than controls and the nonparetic knee extensors, although not significant, was 8% slower (Tsuji and Nakamura 1987). In comparison, our subjects with stroke were substantially slower across all paretic (40% - 65% slower than controls) and nonparetic (26% - 46% slower) joint actions. In addition to methodological differences, these discrepancies in severity of impairment may be due to the duration post-stroke and differences in activity levels. The subjects in the abovementioned report were on average 18 weeks post-stroke and receiving therapeutic interventions (Tsuji and Nakamura 1987). The subjects in our study were on average 7.5 years post-stroke, and were not receiving therapeutic interventions. Thus with increased chronicity and decreased activity levels, slower contraction times may occur.

Fast-twitch oxidative and glycolytic fiber atrophies in the *paretic* vastus lateralis muscle with concurrent fast-twitch glycolytic fiber atrophy in the *nonparetic* muscle of individuals with stroke has been reported (Hachisuka et al. 1997). Thus, as we have shown, slower contraction times would be expected *bilaterally* in individuals with stroke. Furthermore, the paretic gastrocnemius muscle of individuals with stroke has been shown to have an increased percentage of slow twitch fibers *without* atrophy, and a decreased percentage of fast-twitch fibers *with* atrophy in the majority of subjects studied when compared with the nonparetic muscle (Dattola et al. 1993). This suggests that joint actions of the paretic leg are slower to develop torque than the nonparetic leg, which is also in line with our findings. It is currently not known whether these changes in muscle tissue result from primary damage of corticofugal tracts or are the result of secondary changes from decreased physical activity and disuse muscle atrophy.
Time to reduce torque. When averaged across all 6 joint actions, the times to reduce torque of the paretic and nonparetic leg were 59% and 47% slower than control subjects respectively (though, as with time to develop torque, not all joint actions of the nonparetic leg reached significance). In the upper extremity, the paretic arm has been reported to be 22% slower than controls across 8 different joint actions, however, in contrast to our results, the nonparetic arm showed no impairment (McCrea et al. 2003). A large percentage of individuals with stroke regain upper extremity function through compensation or substitution of the nonparetic arm (Nakayama et al. 1994), thus leaving their paretic arm mostly unused. This independent use of the nonparetic arm may help prevent temporal impairments of muscle function secondary to disuse from occurring. In the lower extremity, both limbs are required during most functional activities and although asymmetrical use of the legs has been well documented (Winstein et al. 1989; Olney et al. 1991; Eng and Chu 2002; Lomaglio and Eng in press), we know that individuals with stroke tend to walk, rise out of a chair, and climb stairs more slowly than those without stroke (Hesse et al. 1994; Olney and Richards 1995; von Schroeder et al. 1995; Chou et al. 2003). Thus despite the fact that the nonparetic leg is performing most of the work, the muscles of this leg are not required to contract or relax as quickly. In other words, the impairment in the paretic leg forces the nonparetic leg to move at a slower rate, and a task-specific training effect (i.e. slower contraction and relaxation times) may occur in the nonparetic muscles.

Limitations. The results of this study are limited to those with mild to moderate stroke deficits in the chronic phase of recovery. All of our subjects with stroke were household ambulators and most were community ambulators. Individuals who rely primarily on wheelchairs for mobility would have more disuse atrophy and would likely demonstrate more severe muscle performance impairments, particularly for nonparetic peak torques. However,
80% of individuals with chronic stroke are ambulatory (Gresham et al. 1975); thus justifying our sample.

In addition, this work cannot be generalized to eccentric or concentric muscle contractions. In fact, Davies et al. (1996) reported no significant differences in isometric knee extension torque for the nonparetic leg of individuals with stroke when compared with controls, however, isokinetic concentric torque at 30°/s was significantly reduced. Thus, further research is warranted to ascertain whether or not different types of muscle contractions demonstrate different impairments in individuals with stroke.

Clinical implications and future directions. Whilst it appears as though isometric strength in the nonparetic leg of individuals with stroke may be mildly impaired for some joint actions, it is unlikely that a relative strength loss of less than 12% would have clinical significance. A curvilinear relationship between leg strength and functional performance has been demonstrated in older adults (Buchner et al. 1996), thus once a certain threshold of strength has been met, any further gains in strength may not improve performance. This is supported by a recent randomized trial involving individuals with chronic stroke that showed significant bilateral lower extremity strength gains (up to 38% for nonparetic knee extension) following an aggressive resistance program for the paretic and nonparetic knees and ankles, but no significant differences from the control group (range of motion and stretching program) for gait speed, six-minute walk test, chair-rise test, or stair climb time (Ouelette et al. 2004). Thus, before recommendations can be made regarding nonparetic leg strengthening, further research is needed to ascertain whether or not leg strength of the paretic side is the limiting factor on functional performance.

The most novel finding of this report was that temporal parameters of muscle function (times to develop and reduce torque) were greatly impaired in both legs of individuals with chronic stroke; thus suggesting some independence between these two parameters of muscle
performance. The ability to turn muscles on and off quickly may translate into smoother movements and improved postural control. In the future it will be important to establish the relationships between temporal muscle parameters with measures of coordination, balance, and function. In addition, exercises designed to increase the speed of contraction and relaxation for both paretic and nonparetic leg muscles should be explored.
Chapter 3: Individuals with chronic stroke demonstrate non-uniform weakness in the paretic knee and compensatory strength gains in the nonparetic knee

Abstract

Background and Purpose: Recent reports suggest that the paretic elbow flexors and extensors of individuals with stroke demonstrate exaggerated weakness at short muscle lengths. The purpose of this study was to determine whether this phenomenon also exists in the paretic and nonparetic knee muscles. Methods: Nineteen subjects with stroke and 19 control subjects were recruited from the community. Maximal isometric knee extension and flexion joint torques were measured at six different angles (15, 35, 55, 75, 95, and 105° of flexion). Mean normalized torque-angle curves of the paretic and nonparetic sides were compared to controls using a two-factor (group, knee joint angle) mixed design analysis of variance for knee extension and flexion. Significant group by knee joint angle interactions were explored with post-hoc pairwise comparisons. Results: Relative to the control group, normalized paretic knee extension torque was significantly lower at 15° flexion (i.e. short muscle lengths or near full joint extension) and the nonparetic knee was significantly higher at 95 and 105° of flexion (i.e. long muscle lengths or a flexed joint position). Normalized paretic knee flexion torques were not significantly different than the controls at any joint angle, and the nonparetic knee was significantly higher at 55, 75 and 95° (i.e. moving towards shorter muscle lengths or a flexed joint position).

Conclusions: Paretic knee extensors demonstrate exaggerated weakness at short muscle lengths whereas paretic knee flexors do not. Nonparetic knee extensors and flexors are both relatively stronger as the knee joint assumes a more flexed position, possibly due to compensation. Clinicians should therefore incorporate paretic knee extensor strengthening exercises near terminal extension. In addition, symmetrical use of both legs during functional activities should be encouraged to prevent further disuse atrophy of paretic knee muscles.
INTRODUCTION

Clinical observations suggest that individuals with stroke often demonstrate variable levels of muscle strength depending on the muscle length required for the task at hand. This ability to use the paretic limb muscles successfully during some tasks but not others was recently supported by experimental evidence that demonstrated non-uniform muscle weakness across paretic elbow joint range of motion; more specifically, normalized isometric torque-angle curves of the paretic elbow flexors and extensors of individuals with stroke have been shown to be different from those without stroke, and both the flexors and extensors demonstrated exaggerated weakness when contracting at short muscle lengths (Ada et al. 2000; Ada et al. 2003; Koo et al. 2003).

The relative strength of paretic leg muscles across joint range of motion has not been examined in individuals with stroke despite clinical reports of excessive weakness and insufficient control of the paretic knee extensors when contracting at shorter muscle lengths (i.e. terminal knee extension during gait and curb climbing) (Carr and Shepherd 2004). Thus, the common practice of examining paretic leg strength with a single isometric midrange contraction may not always reveal the full clinical picture. There has also been recent interest in the nonparetic leg of individuals with stroke and some reports suggest that bilateral motor impairments may exist (Adams et al. 1990; Davies et al. 1996; Newham and Hsiao 2001). On the other hand, we know that individuals with stroke often put more weight through their nonparetic leg during functional activities (Winstein et al. 1989; Eng and Chu 2002; Lomaglio and Eng in press). This may lead to task-specific strength changes at different joint angles in the nonparetic leg muscles, particularly in the chronic stage; however, this has not been previously studied. Thus, knowledge of alterations in the torque-angle curves of both the paretic and nonparetic legs of individuals with stroke may help guide clinicians in selecting appropriate rehabilitation exercises and treatment strategies.
The purpose of this study was to assess isometric torque production across multiple knee joint angles to determine whether or not alterations of the flexion and extension torque-angle curves exist on the paretic and nonparetic sides of individuals with chronic stroke when compared to a healthy control group.
METHODS

Subjects. Nineteen subjects (6 women and 13 men) with residual hemiparesis following a single stroke were recruited voluntarily from the community. Subjects were at least 1 year post-stroke, 50 years of age or older, able to walk independently for 10 metres (with or without an assistive device), able to actively achieve full paretic knee extension and 105° of knee flexion in sitting, and able to follow multistep commands. In addition, 19 neurologically healthy subjects (6 women and 13 men) of similar age were recruited to serve as controls. Approval was obtained from the local university and hospital ethics committees and all subjects provided an informed consent (Appendix IV and V). For subjects with stroke, motor recovery of the paretic lower extremity was assessed with active movements using the leg and foot portion of the Chedoke-McMaster Stroke Assessment Score Form (Gowland et al. 1993); stage 1 represents flaccid paralysis and stage 7 represents normal movement patterns (Appendix VI). Disability was assessed using the Stroke Functional Classification levels from the American Heart Association Stroke Outcome Classification Score (Kelly-Hayes et al. 1998); level I represents complete independence in basic and instrumental activities of daily living, and level V represents complete dependence; requires full-time care (Appendix VII). Lower extremity muscle tone of the ankle, knee, and hip flexors and extensors was assessed using the Modified Ashworth Scale (MAS) (Bohannon and Smith 1987) which describes the resistance of muscle to manual passive movement; 0 = no increase in muscle tone, and 4 = paretic part(s) rigid in flexion or extension (Appendix VIII). The activity level of all subjects was assessed using the Physical Activity Scale for Individuals with Physical Disabilities (Washburn et al. 2002). This self-report questionnaire provides an estimate of how many days per week, and hours per day are spent being active; scores are calculated as the average hours of activity daily, multiplied by a metabolic equivalent value (MET hr/d), and summed over items 2 through 13 (Appendix IX). Group characteristics are summarized in Table 3-1.
Joint torque assessment. The isometric mode of a Kin-Com dynamometer (Chattanooga Group, TN) was used to assess extension and flexion maximum voluntary joint torques across six different angles (15, 35, 55, 75, 95, and 105° of flexion; note: 115° could not be tested due to mechanical constraints of the dynamometer) of the paretic and nonparetic knee joints of subjects with stroke, and the nondominant knee joint of control subjects (determined by preference for kicking a ball). Unlike the upper extremity, it has been shown that there is no dominance for maximal muscle strength (Holder-Powell and Rutherford 2000; Skelton et al. 2002) or power (Demura et al. 2001) in the lower extremity. Thus the decision to test the nondominant leg should not bias the results in either direction.

Instrument calibration was tested prior to the study with known weights and was accurate to within +/- 1 N. Subjects were positioned at a 90° sitting angle. Three straps stabilized the trunk and pelvis (two criss-crossing over the chest and one just distal to the anterior-superior iliac spines) and the subject’s hands rested comfortably in their laps. A rigid clamp placed over the distal thigh musculature of the test leg was used for additional stabilization. The dynamometer axis was aligned with the lateral femoral condyle and the cuff was positioned three finger breaths above the medial malleolus (for positioning details see Appendix X - knee). Blood pressure was monitored before, during, and after testing; testing proceeded only when blood pressure was at or below 140/90.

Before each trial, subjects were relaxed and instructed “At the sound of the click (an auditory cue) push (or pull) as hard and as fast as you can and hold this effort. At the second click, immediately relax”. Each trial lasted 3 seconds and verbal encouragement was provided to facilitate a maximum effort. Before extension and flexion joint actions, one submaximal trial followed by one maximal trial were performed as practice. Analogue signals of force from the dynamometer were fed through an A-D converter (National Instruments) and collected at 100Hz. During testing, the torque profile of each trial was displayed on a computer screen and visually...
inspected by the same examiner. Bad trials due to pushing or pulling in the wrong direction or irregular torque profiles were repeated by the subject and subsequently eliminated before analysis (5% of the total number of trials across all subjects needed to be repeated). To avoid a fatigue effect, each trial was separated by 30 seconds rest and each joint angle was tested one at a time from short to long muscle lengths before being repeated (i.e. knee extension torque testing was as follows: 15, 35, 55, 75, 95, 105, 15, 35, 55, 75, and 105° of flexion). In addition, 5 minutes rest was provided between extension and flexion joint actions. To reduce a possible order effect, testing began with extension for approximately half of the subjects and flexion for the remainder.

**Data analyses.** Data were processed with MATLAB using custom software. Once again, individual trials were visually inspected by the same examiner and segmented trials were eliminated from further analysis (2% were eliminated). All torque measurements were corrected for the effect of gravity on the lower extremity segment and cuff of the dynamometer. Peak torque was measured as the maximum torque maintained over a period of 250ms. The best of two contractions for each angle was used for analysis. Knee extension and flexion torque-angle curves of the mean *absolute* torques (Nm/kg) were plotted to descriptively illustrate the strength loss or gain of the paretic and nonparetic knees when compared with controls. Due to the variability of muscle strength across subjects, *relative* torque production (%) across the 6 joint angles was calculated by expressing each subject’s torque values as a percentage of their *own* peak torque (each subject’s highest torque value across joint range of motion was designated as 100%). Knee extension and flexion torque-angle curves were again plotted to allow for a comparison of *relative* strength across joint range of motion between subject groups (paretic, nonparetic, control). Lines of fit were added to all to torque-angle curves to aid visual inspection.
Statistical analyses. Descriptive statistics were calculated to depict subject’s characteristics, clinical scores, and measured torque values.

Mean relative torque-angle curves of the paretic and nonparetic sides of individuals with stroke were compared with the control group using a 2-factor (Factor 1: group; paretic, nonparetic, control. Factor 2: joint angle; 15, 35, 55, 75, 95, 105°) mixed design analysis of variance (3 x 6 ANOVA). Separate analyses were performed for knee extension and knee flexion torques. If significant subject group by joint angle interactions were revealed, post-hoc pairwise comparisons for independent samples were performed for each joint angle tested to determine relative strength differences between paretic and nonparetic knee joints when compared with controls. All statistical analyses were performed using SPSS 11.5 software with a significance level of $P \leq 0.05$. 
RESULTS

Table 3-1. Characteristics of subjects with stroke (N = 19) and control subjects (N = 19)

<table>
<thead>
<tr>
<th></th>
<th>Stroke</th>
<th></th>
<th>Control</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean or SD</td>
<td>Range</td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td>Age (y)</td>
<td>64.9</td>
<td>7.6</td>
<td>53-77</td>
<td>63.4</td>
</tr>
<tr>
<td>Gender (F/M)</td>
<td>6/13</td>
<td></td>
<td></td>
<td>6/13</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>75.9</td>
<td>13.1</td>
<td>53.0-101.0</td>
<td>82.0</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.71</td>
<td>0.10</td>
<td>1.54-1.90</td>
<td>1.77</td>
</tr>
<tr>
<td>BMI (kg/m^2)</td>
<td>26.2</td>
<td>4.3</td>
<td>20.2-35.0</td>
<td>26.0</td>
</tr>
<tr>
<td>Physical Activity*</td>
<td>13.5</td>
<td>9.5</td>
<td>2.2-40.3</td>
<td>23.1</td>
</tr>
<tr>
<td>(MET hr/d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time since stroke (yrs)</td>
<td>7.5</td>
<td>7.3</td>
<td>1-29</td>
<td></td>
</tr>
<tr>
<td>Stroke Type</td>
<td>11/8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(ischemic/hemorrhagic)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paretic side (R/L)</td>
<td>6/13</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Functional Classification† ‡ (class I-V)</td>
<td>II</td>
<td>I-IV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impairment‡ ‡ (stage 1-7)</td>
<td>Leg</td>
<td>6</td>
<td>4-7</td>
<td>Foot</td>
</tr>
<tr>
<td>MAS* (0-4; flexors/extensors)</td>
<td>Ankle</td>
<td>0/1</td>
<td>0-1/0-3</td>
<td>Knee</td>
</tr>
<tr>
<td></td>
<td>Hip</td>
<td>0/0</td>
<td>0-0/0-1+</td>
<td></td>
</tr>
</tbody>
</table>

* Physical Activity Scale for Individuals with Physical Disabilities
† Stroke Functional Classification level (American Heart Association)
‡ Chedoke-McMaster Stroke Assessment Score
* median is reported for the ordinal MAS, Functional Classification, and Impairment scores
Knee extension torque-angle curves. For knee extension the absolute torque-angle curves (Nm/kg) of the paretic and nonparetic sides of individuals with stroke and the control subjects are presented in figures 3-1 and 3-2. The mean absolute torque produced across all 6 joint angles was 0.67Nm/kg (SD = 0.27) for the paretic side, 1.05Nm/kg (SD = 0.33) for the nonparetic side, and 1.04Nm/kg (SD = 0.21) for the controls. Thus, the figures demonstrate absolute torque reduction across joint range of motion for the paretic extensors and preservation of torque for the nonparetic extensors when compared to controls.

For statistical analysis the mean relative extension torques (normalized to each subject's own peak torque; %) at each joint angle were used and are presented in figures 3-3 and 3-4. The ANOVA revealed a significant interaction between group (paretic, nonparetic, and control) and joint angle (6 angles) (P = 0.001). Post-hoc T-tests for independent samples revealed paretic extensors to be significantly lower than controls at 15° of flexion (P = 0.002) (i.e. relatively weaker at short muscle lengths or near full joint extension); no other joint angles were significant (P = 0.224-0.943) (Figure 3-3). Nonparetic extensors were significantly higher than controls at 95 and 105° of flexion (P = 0.015, 0.004 respectively) (i.e. relatively stronger at long muscle lengths or near full joint flexion); no other joint angles were significant (P = 0.143-0.633) (Figure 3-4). On average, peak knee extension torque occurred at 55° for the paretic side and 75° for both the nonparetic side and control group.
Figure 3-1. Comparison of *absolute* knee extension torques (Nm/kg) between the paretic side and control group at 6 different angles. The error bars represent 1 standard deviation.

Figure 3-2. Comparison of *absolute* knee extension torques (Nm/kg) between the nonparetic side and control group at 6 different angles. The error bars represent 1 standard deviation.
Figure 3-3. Comparison of relative knee extension torques (%) between the paretic side and control group at 6 different angles. The error bars represent 1 standard deviation. * indicates paretic relative torque significantly different than control ($P < 0.05$).

Figure 3-4. Comparison of relative knee extension torques (%) between the nonparetic side and control group at 6 different angles. The error bars represent 1 standard deviation. * indicates nonparetic relative torque significantly different than control ($P \leq 0.05$).
**Knee flexion torque-angle curves.** For knee flexion the absolute torque-angle curves (Nm/kg) of the paretic and nonparetic sides of individuals with stroke and the control subjects are presented in figures 3-5 and 3-6. The mean absolute torque produced across all 6 joint angles was 0.35Nm/kg (SD = 0.17) for the paretic side, 0.55Nm/kg (SD = 0.18) for the nonparetic side, and 0.60Nm/kg (SD = 0.21) for the controls. Thus, the figures demonstrate absolute torque reduction across joint range of motion for the paretic flexors. The nonparetic flexors produced less torque than controls when the knee joint was extended, but torque remained preserved as the knee joint moved towards flexion; thus the nonparetic flexors demonstrated a flattened torque-angle curve.

For statistical analysis the mean relative flexion torques (normalized to each subjects own peak torque; %) at each joint angle were used and are presented in Figures 3-7 and 3-8. The ANOVA revealed a significant interaction between group (paretic, nonparetic, and control) and joint angle (6 angles) ($P = 0.041$). Post-hoc T-tests for independent samples revealed no significant differences between paretic flexors and controls for any joint angles tested ($P = 0.389-0.641$) (Figure 3-7). Nonparetic flexors were significantly higher than controls at 55, 75, and 95° ($P = 0.050, 0.014, 0.016$ respectively), and 105° approached significance ($P = 0.057$) (i.e. relatively stronger as the muscle moved towards shorter lengths or more joint flexion). Due to the flattening of the absolute nonparetic torque-angle curve and the weakness demonstrated near full joint extension, the relative torque-angle curves crossed between 15 and 35° with the nonparetic flexors being significantly lower than controls at 15° of flexion ($P = 0.044$) (Figure 3-8). On average, peak knee flexion torque occurred at 15° for all 3 groups.
Figure 3-5. Comparison of *absolute* knee flexion torques (Nm/kg) between the paretic side and control group at 6 different angles. The error bars represent 1 standard deviation.

Figure 3-6. Comparison of *absolute* knee flexion torques (Nm/kg) between the nonparetic side and control group at 6 different angles. The error bars represent 1 standard deviation.
Figure 3-7. Comparison of relative knee flexion torques (%) between the paretic side and control group at 6 different angles. The error bars represent 1 standard deviation.

Figure 3-8. Comparison of relative knee flexion torques (%) between the nonparetic side and control group at 6 different angles. The error bars represent 1 standard deviation. * indicates nonparetic relative torque significantly different than control ($P \leq 0.05$).
DISCUSSION

Torque-angle relationships of the paretic knee. The clinical observation of excessive weakness and insufficient control of the paretic knee near terminal extension in individuals with stroke was supported by our findings. After normalization to peak torque (relative strength), paretic knee extensors were 10% weaker than controls when tested at short muscle lengths (15° of knee flexion), thus demonstrating non-uniform weakness of the paretic knee extensors. Although further research is necessary we postulate, and agree with Ada et al. (2003), that non-uniform weakness may be due to the primary cerebral damage that occurs with stroke. In healthy individuals, it has been shown that twitch duration is reduced during voluntary isometric contractions at short muscle lengths in the biceps brachialis (Gandevia and McKenzie 1988; Christova et al. 1998) and tibialis anterior (Gandevia and McKenzie 1988; Vander Linden et al. 1991). Thus to achieve fusion of twitches when contracting at short lengths, a healthy central nervous system (CNS) will increase motor unit discharge rates. However, in individuals with stroke, there is evidence of reduced motor unit firing rates in both upper (Gemperline et al. 1995) and lower (Rosenfalck and Andreassen 1980) extremity paretic muscles. Thus, as suggested by Ada et al., exaggerated weakness at short muscle lengths in individuals with stroke may be due to an inability to increase motor unit discharge rates to achieve fusion of twitches (Ada et al. 2003). This reduction in central drive was indirectly supported by Koo et al., who reported no significant differences in EMG levels of the brachioradialis muscle of subjects with stroke when tested isometrically across short to long muscle lengths, whereas control subjects demonstrated significantly higher EMG levels at short lengths relative to long lengths (Koo et al. 2003). In other words, to maintain force production, healthy subjects increased brachioradialis muscle activation levels when contracting at short lengths where as subjects with stroke did not. However, as previously mentioned, this mechanism requires further empirical evidence, as the effect of muscle length on twitch durations have not been studied in the quadriceps muscle of...
healthy individuals, and rate coding is not the only mechanism in which the CNS relies on to increase force production.

An alternative mechanism for non-uniform weakness in paretic knee extensors may be due to the influence of the fusimotor system (muscle spindles and gamma motor neurons) on motor unit recruitment and discharge rates across short to long muscle lengths in individuals with stroke. Recent evidence suggests that the fusimotor system in individuals with stroke is not impaired during volitional efforts; Wilson et al. (1999a) demonstrated that stimulating paretic wrist flexor muscle spindles (via tendon tap) in a neutral position immediately following isometric contractions at both short and long muscle lengths produced a stretch reflex response that was not different than that produced by healthy control muscles. Furthermore, during voluntary isometric contractions of paretic wrist extensors, muscle spindle discharge rates in individuals with stroke were the same as controls (Wilson et al. 1999b). It is interesting to apply these findings to our results where a knee flexion angle of 105° would place the muscle spindles of the paretic quadriceps on stretch. This loading of the spindles would cause an increase in the discharge rate of primary and secondary sensory afferents (Ia and group II sensory fibers) and increase excitatory drive to alpha motor neurons innervating the quadriceps, thus helping to maintain force production at longer lengths (Hulliger 1984). It is possible, albeit purely speculative, that there is an imbalance between fusimotor and alphamotor neuron drives in individuals with stroke and that overactivity of the fusimotor system occurs to compensate for reduced central drive. This mechanism would help preserve muscle strength at long lengths, however, at short lengths, it would be unavailable as the muscle spindles would be relatively unloaded and fire at lower rates; therefore lower force production and exaggerated weakness would result. This explanation also warrants further investigation as muscle spindle drive during voluntary isometric contractions across joint angles has not been investigated, and the
aforementioned results reported by Wilson et al. (1999a, 1999b) have not been demonstrated in
the lower extremity muscles of individuals with stroke.

Unexpectedly, our results for the paretic knee flexors did not demonstrate any evidence of
selective weakness at short muscle lengths or any other length. This is in contrast to the paretic
elbow where selective weakness at short lengths was demonstrated for both flexor and extensor
muscle groups (Ada et al. 2003; Koo et al. 2003). This negative finding may be due to the
differences in architectural and contractile properties between the quadriceps and hamstring
muscles. The quadriceps is characterized by higher pennation angles, shorter fibers, and a larger
cross-sectional area relative to the hamstrings; thus the knee extensors are designed for high
force production, and the knee flexors are designed for large excursions and velocity
(Wickiewicz et al. 1983; Lieber and Friden 2000). As a result there may be substantial
differences in how the CNS controls motor unit firing to muscles with vastly different structural
properties.

Also, it is important to note that the upright test position of the subjects at a 90° sitting
angle may not have caused sufficient hamstring shortening to demonstrate non-uniform
weakness in the paretic knee flexors. Each of the hamstring muscles crosses both the hip and the
knee joints (with the minor exception of the short head of biceps femoris), thus, when sitting
upright, the hamstring muscles remain lengthened over the hip joint. In contrast, for the knee
extensors, only the rectus femoris of the quadriceps muscle crosses both the hip and the knee
joint (vastus medialis, lateralis and intermedius originate below the hip joint) and a 90° sitting
angle, combined with a 15° knee flexion angle, would cause considerable shortening of this
muscle group. Furthermore, normal knee flexion range of motion is 135° and due to mechanical
limitations of the dynamometer, the maximum knee flexion angle tested was limited to 105°;
thus approximately 30° of available hamstring shortening was not tested. It can be argued that a
reclined position with the hip joint in 0° flexion may have been a more appropriate position for
knee flexor testing, as considerably more muscle shortening would have occurred. However, lower extremity resisted isometric exercise is known to cause a transient increase in systolic and diastolic blood pressure (Lind et al. 1964; Bezucha et al. 1982) and the magnitude of this cardiovascular response is proportional to the mass of the contracting muscles (Seals et al. 1983). Given that most individuals with stroke have hypertension, and the knee extensors and flexors are relatively large muscle groups, cardiovascular complications are of great concern. Thus the upright test position used in this study was considered safer and more comfortable than a reclined position for this population.

**Torque-angle relationships of the nonparetic knee.** Relative weakness at short muscle lengths was not demonstrated on the nonparetic side for either the knee extensors or flexors. In fact, after normalization to peak torque (relative strength), the nonparetic knee extensors and flexors were up to 12% stronger than controls when tested over flexed joint angle positions (95° to 105° of flexion). Our subjects were tested, on average, 7.5 years post-stroke and this relative strength gain over flexed knee joint positions may be due to task-specific changes in nonparetic knee musculature. For example, individuals with stroke often compensate for paretic leg weakness by placing more of their body mass through their nonparetic leg during biomechanically demanding tasks such as stair climbing (Eng and Chu 2002) and sit-to-stand (Engardt and Olsson 1992; Lomaglio and Eng *in press*). Furthermore, the sit-to-stand task is performed several times throughout the day and older adults use up to 87% of their available knee torque to stand up from a chair (Alexander et al. 1995). Thus, it can be assumed that if the nonparetic leg is performing the majority of work during this transfer, the knee musculature is being loaded to near maximal levels several times throughout the day. Additionally, the quadriceps and hamstring muscle activity in older adults peaks during the critical transition phase of the sit-to-stand transfer; i.e. when the knee is *flexed beyond 90°* the quadriceps and hamstrings co-contract to lift the body up against gravity (Millington et al. 1992). Thus
supporting compensatory strength gains of the nonparetic extensors and flexors over flexed knee joint positions.

**Limitations.** These findings are reported for isometric torque-angle curves and may not necessarily extend to concentric or eccentric contractions, which are most often employed during functional activities. However, there is a relationship between knee flexion and extension isometric strength measures with isokinetic and isotonic measures taken at the same joint angle (mean $r = 0.75$) (Knapik et al. 1983). In a clinical setting, angle specific weakness may be more difficult to assess during concentric and eccentric contractions then during isometric contractions. Thus weakness identified from isometric strength testing across joint range of motion will help form the knowledge foundation for the development of appropriate angle specific concentric and eccentric strengthening exercises. Further research is warranted and it should be noted that the mechanisms underlying torque generation will differ between contraction types.

In addition, generalization of these findings can only be made to individuals with chronic stroke and mild to moderate residual hemiparesis. Similar studies should be performed during the acute stage which would help to discern whether changes in the torque-angle curves are due to primary changes from cerebral damage or secondary compensatory mechanisms.

**Clinical implications and future directions.** The main finding of exaggerated paretic knee extensor weakness near terminal extension should encourage clinicians to design exercises that strengthen the quadriceps over short muscle lengths. Increasing strength over the last 15-20° of extension may enhance performance during functional activities such as gait and stair climbing. Furthermore, the finding that both nonparetic extensors and flexors tend to be relatively stronger as the knee assumes a more flexed position suggests that individuals with stroke rely heavily on their nonparetic leg during everyday tasks, possibly leading to further strength loss in the paretic leg muscles due to disuse atrophy. Thus, clinicians should encourage
symmetrical use of the paretic and nonparetic legs, which may enhance paretic leg strength throughout joint range of motion and improve functional performance.

The information gained by analyzing isometric strength across joint range of motion in individuals with stroke is important for identifying impairments and designing appropriate rehabilitation programs. However, due to differences in architectural properties and functional use across different muscle groups, the findings of this study can not be generalized to the hip or ankle. Thus to assist clinicians in designing more effective stroke rehabilitation programs, further study is required to identify whether or not non-uniform weakness exists in other lower extremity muscle groups.
Chapter 4: Relative preservation of eccentric strength in the paretic and nonparetic leg muscles of individuals with chronic stroke

Abstract

Background and Purpose: It has been well documented that most individuals with stroke have reduced isometric and concentric muscle strength. However, reports examining eccentric muscle performance in this population are scarce despite its importance during daily tasks. The purpose of this study was to measure and compare concentric and eccentric muscle strength in the paretic and nonparetic ankle, knee, and hip flexors and extensors of individuals with chronic stroke.

Methods: Eighteen subjects with stroke were each age and sex matched to one of 18 healthy control subjects. Peak maximum voluntary concentric and eccentric joint torques were measured using an isokinetic dynamometer at 30°/s. Relative concentric and eccentric torque (% of control subject torque) across all 6 joint actions was compared using separate multivariate analyses of variance (MANOVA) for the paretic and nonparetic legs of individuals with stroke. Significant differences were followed by post-hoc paired T-Tests and Wilcoxon Signed Ranks tests for each measured joint action. Results: Relative eccentric torque was significantly higher than relative concentric torque for the paretic ($P = 0.032$) and nonparetic legs ($P = 0.012$). Across all joint actions, paretic leg concentric and eccentric torque was 58% and 74% of control torque respectively; five out of six joint actions were significant ($P = 0.001 - 0.014$). Across all joint actions, nonparetic leg concentric and eccentric torque was 87% and 101% of control torque respectively; four out of six joint actions were significant ($P = 0.001 - 0.010$).

Conclusions: Individuals with stroke demonstrate a relative preservation of eccentric strength in both the paretic and nonparetic leg muscles compared to concentric strength. Knowledge of both concentric and eccentric strength ability will assist clinicians in choosing the appropriate amount of resistance for both the shortening and lengthening phases of rehabilitation exercises. Further study on the response to eccentric training in individuals with stroke is required.
INTRODUCTION

Muscle weakness, or impaired torque production, is a common and persistent consequence of stroke and is due to both primary cerebral damage and secondary muscle adaptations from disuse and inactivity (for reviews see Bourbonnais and Vandon Noven 1989; Ng and Shepherd 2000; Patten et al. 2004). Fortunately strength training is becoming an integral part of stroke rehabilitation due to consistent reports demonstrating positive relationships between leg muscle strength (joint torque production) and functional performance (Nakamura et al. 1985; Bohannon and Walsh 1991; Olney et al. 1994; Eng and Chu 2002; Cameron et al. 2003; Lomaglio and Eng in press). However, clinical trials involving high intensity lower extremity resistance training in individuals with stroke have not always resulted in improvements in functional tasks such as gait speed, sit-to-stand time, or stair climbing ability despite significant improvements in muscle strength (Glasser 1986; Engardt et al. 1995; Kim et al. 2001; Ouelette et al. 2004). This suggests that additional research is necessary to further elucidate how stroke impacts muscle strength, and in turn more effective resistance training programs can be developed.

We know that stroke significantly reduces isometric (static contraction) and concentric (shortening contraction) torque productions in the paretic leg joints of individuals with stroke, and minor reductions in the nonparetic leg joints have also been reported (Adams et al. 1990; Sinkjaer and Magnussen 1994; Davies et al. 1996; Andrews and Bohannon 2000; Harris et al. 2001). However, research examining the effect of stroke on eccentric (lengthening contraction) torque are scant despite its importance for the performance of daily tasks. One study examined the effects of isokinetic eccentric versus concentric resistance training in individuals with stroke and the pre-intervention relative torque values of the paretic knee extensors (expressed as a percentage of the nonparetic extensors) revealed that eccentric torques were 7, 18, and 22% higher than concentric torques at movement velocities of 60, 120, and 180°/s respectively.
These data suggest that eccentric torque production was relatively less affected by stroke than concentric torque production and that this relative preservation of eccentric torque was more pronounced at faster movement velocities. However, a statistical comparison was not made as this was not the focus of the study. Currently there is no data which examines eccentric torque production in the nonparetic leg joints.

Similarly in individuals with spastic paresis due to amyotrophic and primary lateral sclerosis (Griffin et al. 1994), multiple sclerosis (Ponichtera et al. 1992; Knutsson et al. 1997), and cerebral palsy (Knutsson et al. 1997; Damiano et al. 2000), concentric torque has been shown to be significantly more impaired than eccentric torque about the knee joint. These findings have been attributed to the hyperactive stretch reflexes in individuals with upper motor neuron lesions, whereby torque production during shortening concentric contractions would be reduced, in part, by the stretch reflex elicited in the lengthening antagonist muscle (Griffin et al. 1994; Damiano et al. 2000).

These studies present a growing body of interesting evidence to suggest that individuals with upper motor neuron lesions will demonstrate a relative preservation of eccentric strength in paretic limb joints. Thus the first purpose of this study was to extend these findings to individuals with chronic stroke by conducting a comprehensive evaluation of concentric and eccentric torque productions for paretic ankle, knee, and hip flexion and extension joint actions. The second purpose was to conduct the same evaluation in the nonparetic leg joints to determine whether or not similar findings would exist.
METHODS

Subjects. Eighteen subjects (6 women and 12 men) with residual hemiparesis following a single stroke were recruited voluntarily from the community. Subjects were at least 1 year post-stroke, 50 years of age or older, able to walk independently for 10 metres (with or without an assistive device), and able to follow multistep commands. In addition, 18 neurologically healthy subjects (6 women and 12 men) were recruited and matched for sex and age to each of the subjects with stroke. Approval was obtained from the local university and hospital ethics committees and all subjects provided an informed consent (Appendix IV and V). For subjects with stroke, motor recovery of the paretic lower extremity was assessed with active movements using the leg and foot portion of the Chedoke-McMaster Stroke Assessment Score Form (Gowland et al. 1993); stage 1 represents flaccid paralysis and stage 7 represents normal movement patterns (Appendix VI). Disability was assessed using the Stroke Functional Classification levels from the American Heart Association Stroke Outcome Classification Score (Kelly-Hayes et al. 1998); level I represents complete independence in basic and instrumental activities of daily living, and level V represents complete dependence; requires full-time care (Appendix VII). Lower extremity muscle tone of the ankle, knee, and hip flexors and extensors was assessed using the Modified Ashworth Scale (MAS) (Bohannon and Smith 1987) which describes the resistance of muscle to manual passive movement; 0 = no increase in muscle tone, and 4 = paretic part(s) rigid in flexion or extension (Appendix VIII). The activity level of all subjects was assessed using the Physical Activity Scale for Individuals with Physical Disabilities (Washburn et al. 2002). This self-report questionnaire provides an estimate of how many days per week, and hours per day are spent being active; scores are calculated as the average hours of activity daily, multiplied by a metabolic equivalent value (MET hr/d), and summed over items 2 through 13 (Appendix IX). Group characteristics are summarized in Table 4-1. Approximately
5 weeks after the initial assessment, 9 subjects with stroke returned for repeat testing to establish test-retest intrarater reliability.

**Isokinetic joint torque assessment.** A Kin-Com isokinetic dynamometer (Chattanooga Group, TN) was used to assess maximum voluntary concentric and eccentric joint torques of the ankle, knee, and hip flexors and extensors of the paretic and nonparetic sides of subjects with stroke and the nondominant leg of control subjects (determined by preference for kicking a ball). Unlike the upper extremity, it has been shown that there is no dominance for maximal muscle strength (Holder-Powell and Rutherford 2000; Skelton et al. 2002) or power (Demura et al. 2001) in the lower extremity. Thus the decision to test the nondominant leg should not bias the results in either direction.

Instrument calibration was tested prior to the study with known weights and was accurate to within +/- 1 N. All joint torques were assessed at an angular velocity of 30°/second as our lab has previously shown that subjects with stroke have difficulty achieving higher speeds at the knee and ankle joints for concentric joint actions on the paretic side (Eng et al. 2002). Furthermore, the risk for muscle damage and impaired force production increases as the velocity of eccentric contractions increases (McCully and Faulkner 1986).

The ankle and knee torques were tested at a 90° sitting angle and the hip torques were tested in a 40° semi-reclined position; an upright position was considered safer and more comfortable than a reclined position due to the incidence of hypertension in this population (blood pressure was monitored throughout the test protocol and testing proceeded only when blood pressure was at or below 140/90). For stabilization, three straps crossed the trunk and pelvis (two over the chest and one just distal to the anterior-superior iliac spines), and for measurement of knee torques an additional rigid clamp was placed over the distal thigh musculature of the test leg. The dynamometer axis was aligned with the joint line of the ankle, knee, and hip (lateral malleolus, lateral femoral condyle, and greater trochanter respectively) by
the same examiner throughout testing. A rigid metallic ankle attachment provided by the
manufacturer was used for assessment of ankle torques. For knee and hip torques, the cuff of the
dynamometer was positioned three finger breaths above the medial malleolus and three finger
breaths proximal to the popliteal fossa respectively (for positioning details see Appendix X).
The subject’s hands rested comfortably in their laps during testing.

Joint angle or muscle length affects torque production; therefore to allow for an
equivalent comparison, the paretic, nonparetic, and control leg joint actions were all tested
through the same limited range of motion. Ankle torques were tested through a range of 10-35°
of plantarflexion (0° = neutral; foot perpendicular to shank), knee torques through a range of 15-
95° of flexion (0° = full knee extension), and hip torques through a range of 55-105° of flexion
(0° = neutral; pelvis aligned with thigh). The selection of these joint angle values were based
from mean values obtained from the paretic leg during pilot testing.

Gravity compensation was performed in accordance with the manufacturer’s instructions
before each new joint action. As warm-up, and to ensure the subject understood the requested
action, two submaximal and one maximal practice contractions were performed before each
muscle group tested. Preload (used to decrease variability in the time taken to reach peak torque
and defined as the preset torque which must be overcome before movement of the dynamometer
can occur) was individualized for each subject, joint action, and contraction type. For concentric
actions it was set at 75% of the peak torque value obtained from the maximal practice concentric
contraction. For eccentric actions it was set at 75% of the final torque value obtained from the
maximal practice concentric contraction (which was the start position for the eccentric
contraction). This was done because muscle strength is length-dependent and during eccentric
testing, for most joint actions, the subjects could not overcome the preload value used for
concentric testing (i.e. the eccentric start position often placed the joint in a relatively weaker
position).
During testing subjects were instructed to push (or pull) as hard as possible and both visual and verbal feedback was provided during the trials. For both concentric and eccentric contractions, 3 consistent maximum effort trials were recorded (the maximum number of repetitions performed by any subject was 5 per contraction type). A 5 second rest was used between the concentric-eccentric cycles. To prevent fatigue, each concentric-eccentric cycle was separated by 30 seconds of rest, and each new joint action was separated by 5 minutes of rest. Testing began with extension for approximately half of the subjects and flexion for the remainder, thus reducing a possible order effect.

**Data and statistical analyses.** A single ensemble-averaged torque-angle curve was calculated from the three recorded curves for each joint action (ankle, knee, and hip flexion and extension) and contraction type (concentric and eccentric). Both the mean average torque and the mean peak torque were taken from these curves and assessed for intrarater reliability using intraclass correlations, ICC (3,3) (Shrout and Fleiss 1979), and standard error of measurement (SEM). Overall, peak torque values were slightly more reliable than average torque values (see results) and therefore only peak torque values were used for further analyses. Each subject’s absolute torque values were normalized by body mass. To describe paretic and nonparetic relative concentric and eccentric torque preservation, the normalized torques for the paretic and nonparetic sides of subjects with stroke were divided by the normalized torques of their matched control subjects for each joint action (thus paretic and nonparetic torque values were expressed as a percentage of control torque values).

A multivariate analysis of variance (MANOVA) was performed to determine whether concentric relative torque preservation was different than eccentric preservation (independent variables: concentric relative torque and eccentric relative torque) across all lower extremity joint actions (6 dependent variables: ankle, knee, and hip flexion and extension) for the paretic side of individuals with stroke. A second MANOVA used the same variables for the nonparetic side.
Significant group differences (i.e. concentric relative torque was significantly different than eccentric relative torque across ankle, knee, and hip joint actions) for each MANOVA were followed by post-hoc paired T-tests (parametric) and Wilcoxon Signed Ranks Tests for matched pairs (nonparametric) depending on the distribution of the variable.

All statistical analyses were performed using SPSS 11.5 software with a significance level of $P \leq 0.05$. 
# RESULTS

Table 4-1. Characteristics of subjects with stroke ($N = 18$) and control subjects ($N = 18$)

<table>
<thead>
<tr>
<th></th>
<th>Stroke</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean or SD</td>
<td>Range</td>
</tr>
<tr>
<td><strong>Age (y)</strong></td>
<td>64.9</td>
<td>7.8</td>
</tr>
<tr>
<td><strong>Gender (F/M)</strong></td>
<td>6/12</td>
<td></td>
</tr>
<tr>
<td><strong>Mass (kg)</strong></td>
<td>76.7</td>
<td>13.0</td>
</tr>
<tr>
<td><strong>Height (m)</strong></td>
<td>1.71</td>
<td>0.10</td>
</tr>
<tr>
<td><strong>BMI (kg/m$^2$)</strong></td>
<td>25.9</td>
<td>4.3</td>
</tr>
<tr>
<td><em><em>Physical Activity</em> (MET hr/d)</em>*</td>
<td>13.8</td>
<td>9.7</td>
</tr>
<tr>
<td><strong>Time since stroke (yrs)</strong></td>
<td>7.7</td>
<td>7.4</td>
</tr>
<tr>
<td><strong>Stroke Type (ischemic/hemorrhagic)</strong></td>
<td>10/8</td>
<td></td>
</tr>
<tr>
<td><strong>Paretic side (R/L)</strong></td>
<td>6/12</td>
<td></td>
</tr>
<tr>
<td><strong>Functional Classification†a (class I–V)</strong></td>
<td>II</td>
<td></td>
</tr>
<tr>
<td><strong>Impairment‡a (stage 1–7)</strong></td>
<td>Leg</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Foot</td>
<td>4</td>
</tr>
<tr>
<td><em><em>MAS</em> (0–4; flexors/extensors)</em>*</td>
<td>Ankle</td>
<td>0/1</td>
</tr>
<tr>
<td></td>
<td>Knee</td>
<td>1/0</td>
</tr>
<tr>
<td></td>
<td>Hip</td>
<td>0/0</td>
</tr>
</tbody>
</table>

* Physical Activity Scale for Individuals with Physical Disabilities
† Stroke Functional Classification level (American Heart Association)
‡ Chedoke-McMaster Stroke Assessment Score

a median is reported for the ordinal MAS, Functional Classification, and Impairment scores
**Reliability.** For the paretic lower extremity joint actions, the mean ICCs (SEM) for intrarater reliability were 0.98 (3.4N) for peak concentric torque and 0.96 (7.2N) for peak eccentric torque. For the nonparetic lower extremity joint actions the mean ICCs (SEM) were 0.92 (6.0N) for peak concentric torque and 0.96 (8.4N) for peak eccentric torque \((N = 9)\). As previously mentioned the average concentric and eccentric torque ICCs were slightly lower (range: 0.91-0.96) and therefore only peak torque was used for further analyses (for details see Appendix XV).

**Paretic leg relative torque.** The MANOVA for the paretic lower extremity revealed a significant difference between concentric and eccentric relative torque preservation across ankle, knee, and hip flexion and extension joint actions (Hotelling’s Trace = 1.726, \(P = 0.032\)). Concentric relative torque preservation of paretic muscles (mean = 58%; range = 35-65%) was less than eccentric preservation (mean = 74%; range = 51-82%) (Figure 4-1) and post-hoc tests revealed statistical significance for five out of six joint actions (table 4-2).

**Nonparetic leg relative torque.** The MANOVA for the nonparetic lower extremity revealed a significant difference between concentric and eccentric relative torque preservation across ankle, knee, and hip flexion and extension joint actions (Hotelling’s Trace = 2.271, \(P = 0.012\)). Concentric relative torque preservation of nonparetic muscles (mean = 87%; range = 71-105%) was less than eccentric preservation (mean = 101%; range = 85-109%) (Figure 4-2) and post-hoc tests revealed statistical significance for four out of six joint actions (table 4-2).
Figure 4-1  Relative concentric and eccentric torque preservation for the paretic side of subjects with stroke (expressed as percentage of control torque) for 6 lower extremity joint actions. The error bar represents 1 standard deviation. * indicates concentric preservation significantly different than eccentric preservation ($P \leq 0.05$).
Figure 4-2 Relative concentric and eccentric torque preservation for the nonparetic side of subjects with stroke (expressed as percentage of control torque) for 6 lower extremity joint actions. The error bar represents 1 standard deviation. * indicates concentric preservation significantly different than eccentric preservation $(P < 0.05)$.

Table 4-2 Post-hoc Paired Samples T-test and Wilcoxon Signed Ranks Test for matched pairs $(N = 18)$

<table>
<thead>
<tr>
<th>Joint Action</th>
<th>Paretic side</th>
<th>Nonparetic side</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee Extension</td>
<td>.014*</td>
<td>.679a</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>.008*a</td>
<td>.010*a</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>.199a</td>
<td>.248a</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>.006<em>a</em></td>
<td>.003*</td>
</tr>
<tr>
<td>Ankle Plantarflexion</td>
<td>.002*</td>
<td>.001*a</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>.001*</td>
<td>.001*</td>
</tr>
</tbody>
</table>

* Concentric relative torque preservation significantly different than eccentric preservation at $P \leq 0.05$. a variables did not satisfy assumption of normality and thus the nonparametric Wilcoxon Signed Ranks Test for matched pairs was used.
DISCUSSION

This study demonstrated that eccentric torque production in the paretic leg was consistently less affected by stroke than concentric torque production across all joint actions assessed. This striking result supports the hypothesis that individuals with upper motor neuron lesions, regardless of the etiology, will have an abnormal imbalance between concentric and eccentric torques in paretic limb joints. On average, eccentric torque production was 74% of controls and concentric torque production was 58%; thus concentric torque was reduced an additional 16%. This finding is not as dramatic as the methodologically similar study by Griffin et al. (1994) that reported concentric knee torque to be reduced an additional 26% compared to eccentric knee torque in individuals with amyotrophic and primary lateral sclerosis (ALS and PLS respectively). However, these findings were obtained using isokinetic velocities of 120°/s. When subjects were evaluated at 30°/s, no significant differences were found between concentric and eccentric relative torques. Their results suggest that there was an effect of movement velocity on preservation of eccentric torque. Conversely, our study found significant differences between concentric and eccentric relative torque at 30°/s (no other movement velocities were tested). This discrepancy in findings may be due to differences in disease processes. ALS and PLS are progressive neurological disorders and individuals with chronic stroke are neurologically stable, thus suggesting that different mechanisms may be responsible for the relative preservation of eccentric strength in populations with different upper motor neuron diseases. Furthermore, our study found that the nonparetic leg of individuals with chronic stroke also demonstrated a similar phenomenon, whereby eccentric torque production was relatively preserved (mean = 101% preserved) and concentric torque production was mildly impaired (mean = 87% preserved).

The mechanisms responsible for the relative sparing of eccentric torque post-stroke are unknown and can not be discerned from this work. However, a review of the literature suggests
that several mechanisms may be acting at the same time, and are likely due to both changes in the neuromuscular system and changes in muscle compliance.

Interestingly, in young healthy adults, maximal neural activation occurs during maximal voluntary concentric contractions, but only submaximal neural activation occurs during maximal voluntary eccentric contractions (Westing et al. 1990, 1991). Neural inhibition via the Ib afferent system (golgi tendon organs) likely accounts for this finding and occurs to prevent the muscle from injury that would otherwise be sustained by the extremely high forces generated under maximal activation. Due to primary cerebral damage, it is possible that after a stroke neural inhibition during eccentric contractions does not occur, thus leading to a relative preservation of eccentric force production.

Electromyography (EMG) is commonly used to assess muscle activity. Knutsson et al. (1988, 1997) reported that when individuals with spastic paresis (due to disorders other than stroke) perform eccentric knee contractions, the EMG level in the lengthening agonist increased with increasing movement velocities (30 to 180 °/s). As movement velocity increased during concentric knee contractions, decreasing EMG levels of the shortening agonist occurred with a concurrent increase in EMG in the lengthening antagonist. This was not demonstrated in the healthy control group (Knutsson et al. 1988, 1997). The authors attributed these findings to hyperactivity of spinal reflexes demonstrated in individuals with spasticity, whereby stretch of the agonist during eccentric contractions would facilitate muscle activation, and stretch of the antagonist during concentric contractions would decrease muscle activation in the agonist via reciprocal inhibition. A similar mechanism may have been occurring in the paretic muscles of our subjects. However, there is some debate on whether hyperactivity of the stretch reflex in the lengthening antagonist limits force production of the agonist during voluntary concentric movement in individuals with stroke. Lum et al. (2004) reported increased antagonist coactivation of the brachioradialis, but not the biceps, during active elbow extension in
individuals with stroke, and there was no evidence of increased coactivation of the triceps (antagonist) during active elbow flexion. In addition, Davies et al. (1996) reported no evidence of abnormal antagonist co-contraction during maximal voluntary concentric knee flexion or extension in the paretic or nonparetic legs of individuals with stroke, even at fast isokinetic velocities of 300°/s. However, there was a significant increase in measured torque during passive paretic knee extension that was not accompanied by EMG activity. This latter finding suggests that non-neural mechanisms, such as increased muscle compliance or stiffness, may also be contributing to the discrepancy in concentric and eccentric relative torque levels demonstrated in our study, particularly for the nonparetic side, where hyperactive stretch reflexes do not occur.

During eccentric contractions, increased stiffness in the lengthening agonist muscle may add to the torque produced. During concentric contractions, increased stiffness in the lengthening antagonist muscle may subtract from the torque produced. Evidence of structural changes in the mechanical properties of muscle in individuals with stroke have been reported (Dietz and Berger 1983; Hufschmidt and Mauritz 1985; Lee et al. 1987; Ibrahim et al. 1993; Sinkjaer and Magnussen 1994; Svantesson et al. 2000). Sinkjaer and Magnussen (1994) measured both the reflex and non-reflex mediated stiffness of the paretic ankle plantarflexors in individuals with stroke (mean time after onset = 31 months; range = 2 to 80 months). Total joint stiffness (due to both reflex and non-reflex properties) was first determined by measuring the torque output during a quick stretch of the plantarflexors while the subjects maintained a voluntary contraction at multiple preset levels (0 Nm to a maximum voluntary contraction). Non-reflex stiffness was then determined by repeating the above protocol using electrical stimulation to activate the plantarflexors (thereby suppressing the stretch reflex and eliminating voluntary activation). Subtraction of the two curves provided an estimate of the reflex stiffness (stiffness was calculated by dividing the torque increments by the amplitude of stretch; Nm/°).
From the slope and intercept of the average non-reflex stiffness data, the authors determined average *intrinsic stiffness* (contractile properties of the muscle fibers) and average *passive stiffness* (response from the passive tissues) respectively. They found that intrinsic stiffness did not differ for paretic, nonparetic and control ankle plantarflexors but passive stiffness was increased a remarkable 278% in the paretic leg, and 95% in the nonparetic leg when compared to controls. These bilateral changes in passive stiffness were suggested to be due, in part, to inactivity and changes in collagen tissue. Svantesson et al. (2000) used a similar approach to measure the muscle and tendon stiffness in individuals with chronic stroke and reported higher muscle stiffness in the paretic plantarflexors and higher tendon stiffness in the nonparetic plantarflexors when compared to each other. In addition, there is evidence of increased stiffness at the level of the sarcomere in individuals with spastic paresis (Friden and Lieber 2003). Using muscle biopsies, Friden and Lieber (2003) revealed that sarcomeres were significantly shorter at rest and muscle fibers were twice as stiff under tension when compared to controls. However, some caution should be taken in extrapolating these results to leg muscle in individuals with stroke, as their data represented severely spastic arm muscle in children with cerebral palsy (mean age = 8 years) compared with healthy adults (mean age = 37 years). Thus, the sarcomere adaptations reported may also be due to developmental changes (i.e. response to bone growth) and not spasticity.

In the current study, the Modified Ashworth Scale (MAS) was used to assess muscle tone in our subjects with stroke. The ankle plantarflexors demonstrated the most tone (i.e. increased resistance to passive dorsiflexion), with 13 subjects scoring greater than 0 (0 = no increase in muscle tone). Interestingly, the greatest discrepancy between relative concentric and eccentric torque impairment was found in the antagonist dorsiflexors, whereby eccentric torque production was 19% more preserved than concentric torque production. Furthermore, those who scored 1+ or greater on the MAS (N = 6) had an even higher discrepancy of 35% versus 8% for those with
a score of 1 \((N=7)\), and 7\% for those with a score of 0 \((N=5)\). Thus individuals with more plantarflexion tone demonstrated larger discrepancies between concentric and eccentric strength preservation for ankle dorsiflexion. Passive resistance in the plantarflexors may be limiting the torque production during concentric dorsiflexion without influencing torque production during eccentric dorsiflexion. In other words, during a shortening contraction, the ankle dorsiflexors must work against the increased tone of the passively lengthening ankle plantarflexors. It is important to point out that many of our subjects had a MAS of 0. This is consistent with the literature, whereby Sommerfeld et al. (2004) reported only 28\% of individuals with hemiparesis from stroke had increased spasticity or tone (as measured by the MAS). Despite this, both the paretic and nonparetic limbs displayed a relative preservation of eccentric strength. This suggests that the MAS may not be sensitive enough to detect subtle abnormalities in muscle compliance, as done with the biomechanical techniques of Sinkjaer and Magnussen (1994) or Friden and Lieber (2003).

Interestingly, healthy older adults have been shown to have greater amounts of noncontractile content (adipose and connective tissue) within their muscles (Rice et al. 1989; Kent-Braun et al. 2000). They have also been shown to demonstrate relatively preserved eccentric strength with significant reductions in concentric strength when compared to young adults (Vandervoort et al. 1990; Hortobagyi et al. 1995; Porter et al. 1997; Pousson et al. 2001). The abovementioned infiltration of adipose and connective tissue (i.e. collagen) reduces the muscles contractile content while increasing the mechanical stiffness, thus possibly contributing to the preservation of eccentric strength with age. Porter et al. (1997) reported that peak eccentric plantarflexion and dorsiflexion torques in older women were 97\% and 101\% of those torques generated by younger women. Concentric torques, however, were only 74\% and 89\% respectively. The authors used a dynamometer to measure the total passive resistive torque of the ankle plantarflexors and found that older women generated 26\% more passive torque than
younger women. Similar to our results, the authors felt that the increased stiffness found in the ankle plantarflexors of older women may explain the *reduced concentric* torque produced during ankle dorsiflexion, however, it does not explain the *maintenance* of *eccentric* torque with age. This is because the plantarflexion eccentric torque-angle tracings were nearly identical between the old and young groups, even in a plantarflexed position, where resistive torque (i.e. stiffness) was minimal and thus unable to contribute to the torque generated. This latter finding suggests that other unknown mechanisms are at play. Interestingly, compared to concentric contractions, eccentric contractions have greater mechanochemical efficiency (i.e. mechanical power output to ATP synthesis rate) (Ryschon et al. 1997) and require lower levels of muscle activation to generate greater levels of force (Westing et al. 1990; Westing et al. 1991). These advantages of eccentric contractions may contribute to the maintenance of eccentric strength with age. Clearly the mechanisms contributing to this phenomenon in older individuals, as well in individuals with stroke, are complex and multifactorial. Collaborative research efforts examining changes in both the muscle and nervous systems are required.

Lastly, a note regarding muscle fiber type changes in individuals with stroke should be made. Type II fiber atrophy has been demonstrated in both paretic and nonparetic muscles of individuals with stroke, which coincides with an increased percentage of type I fibers (Dattola et al. 1993; Hachisuka et al. 1997). During eccentric contractions, type II fibers have been shown to be selectively activated (Nardone et al. 1989), thus in individuals with stroke, one would expect eccentric strength to be more impaired than concentric strength, but this is opposite to what we found. Therefore our results are not likely due to changes in muscle fiber type, but possibly to the aforementioned changes in the neuromuscular system (neural activation adaptations and hyperactive stretch reflexes), increased mechanical muscle stiffness, and other mechanisms that are yet to be determined.
Limitations. Generalization of these findings can only be made to the lower extremity of individuals with chronic stroke with mild to moderate hemiparesis and muscle tone. Similar studies should be performed during the acute stage and also a comparison between acute and chronic individuals would help discern whether the relative preservation of eccentric strength is due to primary or secondary changes in the neuromuscular system. Also, the application of EMG and testing subjects at both slow and fast isokinetic velocities would have been useful in identifying the mechanisms behind our findings. However, as previously mentioned, individuals with stroke have difficulty achieving isokinetic velocities greater than 30°/s (Eng et al. 2002) and the risk of injury increases as the velocity of eccentric contractions increases (McCully and Faulkner 1986).

Clinical implications and future directions. Our study showed that eccentric strength was less impaired than concentric strength in individuals with stroke, thus clinicians should assess limb strength under both contraction conditions to better quantify muscle performance. Knowledge of maximal concentric and eccentric torque capabilities in this population would allow clinicians to select different relative training loads during both the shortening and lengthening phases of open and closed chain exercises. In other words, due to the relative preservation of eccentric strength in individuals with stroke, care must be taken to avoid underloading of paretic and nonparetic muscles during the eccentric phase of exercises. There is one encouraging previously mentioned study that reported a significant increase in both concentric and eccentric strength of the paretic leg (relative to the nonparetic leg) following maximal isokinetic eccentric training but not maximal isokinetic concentric training (Engardt et al. 1995). In addition, the eccentric group improved weight bearing symmetry during sit to stand but the concentric group did not. It is therefore possible that the higher forces generated during eccentric contractions provided a strong enough stimulus to increase both eccentric and concentric strength, which in turn lead to improvements in functional performance. However,
clinicians should proceed with extreme caution as more research is necessary to elucidate how individuals with stroke will respond to high intensity eccentric training. In particular, delayed onset muscle soreness (DOMS) and the muscle injury associated with eccentric exercise may override its potential training benefits.

Lastly, relative preservation of eccentric strength may allow individuals with stroke who have very weak muscles, such as those with acute hemiparesis, to perform open chain eccentric actions before concentric actions to stimulate muscle activity. Once strength improves, concentric exercises can be introduced. However, as previously mentioned, relative preservation of eccentric strength has not yet been demonstrated in individuals with acute stroke.
Chapter 5: GENERAL DISCUSSION

A better understanding of the impairments that develop post-stroke will lead to evidence-based interventions designed to prevent or reverse these impairments. The work of this thesis has succeeded in newly identifying impairments of muscle performance in both the paretic and nonparetic lower extremities of individuals with chronic stroke. Below is a summary of interesting findings and discussion that connects the three related experiments of this thesis. Included are research directions for the future. To conclude, the clinical implications resulting from this work are presented and treatment strategies are suggested.

SUMMARY.

Paretic leg muscle impairments result from primary damage to supraspinal motor pathways that can worsen over time from disuse.

The paretic leg of individuals with stroke demonstrated a reduction in isometric, concentric, and eccentric torques when compared to control subjects. When averaged across ankle, knee, and hip extension and flexion joint actions, isometric, concentric, and eccentric torques were reduced 40%, 42%, and 26% respectively. Thus isometric and concentric strength was more affected by stroke than eccentric strength. In addition, when compared with controls, the isometric torque-angle curve for paretic knee extension was altered, whereby selective weakness at short muscle lengths was demonstrated (i.e. 15° of knee flexion).

Impairments in paretic leg torque production, regardless of the contraction type, were likely caused by primary damage to the central nervous system causing denervation, decreased motor unit firing rates, and altered motor unit recruitment in individuals with stroke (Rosenfalck and Andreassen 1980; Gemperline et al. 1995). Selective weakness of the paretic knee
extensors at short muscle lengths may also be due to primary mechanisms, whereby unlike healthy individuals, individuals with stroke are unable to achieve the relatively high motor unit firing rates required to achieve fusion of twitches at short lengths.

Relative preservation of eccentric strength found in the paretic leg was attributed to both primary changes in the neuromuscular system and secondary changes in muscle compliance that occurs over time. More specifically, hyperactive stretch reflexes may add to the torque generated when the agonist is lengthening (i.e. during eccentric contractions) but hinder torque production when the agonist is shortening (i.e. during concentric contractions) (Knutsson et al. 1988, 1997). In addition, increased muscle compliance (stiffness) has been reported in the paretic leg of individuals with stroke (Dietz and Berger 1983; Hufschmidt and Mauritz 1985; Lee et al. 1987; Ibrahim et al. 1993; Sinkjaer and Magnussen 1994; Svantesson et al. 2000). This muscle stiffness, which may be due to disuse, would have the same above effect on torque production, thus contributing to the preservation of eccentric strength in paretic leg muscles.

Temporal parameters of muscle performance (times to develop and reduce isometric torque) were also found to be impaired in the paretic leg of individuals with stroke. In addition to the aforementioned reduction in central motor drive, atrophy of fast-twitch fibers (Dattola et al. 1993; Hachisuka et al. 1997) and an increased percentage of slow twitch fibers without atrophy (Dattola et al. 1993) occurs in paretic leg muscles post-stroke and may also contribute to the slower contraction and relaxation times demonstrated.
Nonparetic leg muscle impairments result from secondary compensatory strategies and inactivity.

When averaged across ankle, knee, and hip extension and flexion joint actions, the nonparetic leg of individuals with stroke demonstrated minor reductions in average isometric and concentric torques (11% and 13% respectively), and eccentric torque was not impaired (101%) when compared with control subjects. Thus isometric and concentric strength was mildly affected by stroke and eccentric strength was preserved. However, there was one notable exception; nonparetic ankle plantarflexion torque was markedly reduced for all three contraction types (32% isometrically, 29% concentrically, and 15% eccentrically). Primary damage to the uncrossed ventral corticospinal tract does not explain this finding because these fibers project primarily to axial and proximal limb muscles (Davidoff 1990); thus a relative sparing of distal ankle plantarflexors would be expected. In addition, nonparetic knee extension torque was completely preserved for all three contraction types (101% isometrically, 105% concentrically, and, 107% eccentrically). Instead, these findings may be related to secondary changes in power generation that occur in the nonparetic leg when gait speed is reduced post-stroke. As gait speed becomes slower, nonparetic ankle plantarflexion power has been shown to decrease, while nonparetic knee extension power does not change (Olney et al. 1991). Furthermore, when compared to healthy controls walking at the same reduced speeds, nonparetic ankle plantarflexion power has been shown to be decreased and nonparetic knee extension power increased (Olney and Richards 1995). Thus over time, a reduction in nonparetic ankle plantarflexion strength with concurrent preservation of nonparetic knee extension strength would be expected in individuals with chronic stroke, regardless of the contraction type.

Preservation of eccentric strength in the nonparetic leg muscles with concomitant mild impairments in isometric and concentric leg strength may be due to the increased muscle
compliance (stiffness) that has been demonstrated in the nonparetic legs of individuals with stroke (Sinkjaer and Magnussen 1994; Svantesson et al. 2000). Thus similar to the paretic leg, the stiffness in the agonist would add to the torque produced during a lengthening contraction and assist in the maintenance of eccentric strength. In addition, during a shortening contraction, stiffness in the passively lengthening antagonist would have a negative effect on agonist torque production. It is not known why the nonparetic leg muscles become stiffer after stroke but it is postulated to be from the lower activity levels demonstrated by individuals with stroke. Interestingly, the subjects with stroke in this study had activity scores that were 40% lower than control subjects.

Similar to the paretic leg, temporal parameters of muscle performance (times to develop and reduce isometric torque) were impaired in the nonparetic leg of individuals with stroke. Because functional tasks are often performed slower than normal post-stroke (Hesse et al. 1994; Olney and Richards 1995; von Schroeder et al. 1995; Chou et al. 2003), the nonparetic leg is not required to move quickly. Thus over time, the muscles adapt by becoming “slower”. In other words, due to the bilateral nature of many lower extremity tasks, performance of the nonparetic leg is limited by the impairments of the paretic leg. In addition, atrophy of fast-twitch fibers in the nonparetic leg muscles post-stroke (Hachisuka et al. 1997) may also contribute to the decline in speed of contraction and relaxation.

It is interesting to note that the impairments in the times to develop and reduce isometric torque were much more impaired than torque magnitude in the nonparetic leg of individuals with stroke. For example, nonparetic isometric hip flexion torque was only 4% lower than controls, whereas the time to develop this torque was more than twice as slow as controls (0.25 seconds versus 0.11 seconds respectively). Thus it is possible that the neuronal circuits controlling temporal parameters of torque (times to develop and reduce torque) are different than those that control torque magnitude. Alternatively, the notable impairments in temporal parameters of
torque with only minor reductions in torque magnitude may be due to task-specific muscle adaptations. Individuals with stroke use their nonparetic leg more during functional activities (Winstein et al. 1989; Engardt and Olsson 1992; Eng and Chu 2002; Lomaglio and Eng in press), but they also tend to walk, rise out of a chair, and climb stairs more slowly than normal (Hesse et al. 1994; Olney and Richards 1995; von Schroeder et al. 1995; Chou et al. 2003). Thus over time, nonparetic leg muscles will maintain their strength from the increased use but take longer than normal to contract and relax as a result of slower functional movement times.

Secondary adaptations from increased use during functional activities also explains the non-uniform strength demonstrated across joint range of motion in the nonparetic knee, whereby both extension and flexion joint actions were relatively stronger than controls over a flexed knee position (95° and 105° for extension, and 55°, 75°, and 95° for flexion). Individuals with stroke are known to place more weight through their nonparetic leg during the sit-to-stand task (Engardt and Olsson 1992; Lomaglio and Eng in press). In addition, during sit-to-stand, both quadriceps and hamstring muscle activities peak when the knee is flexed beyond 90° (Millington et al. 1992). Thus over time, the increased use of the nonparetic leg during this frequently performed task may lead to strength gains specifically over flexed knee joint angles.

It should be pointed out that the aforementioned mechanisms of muscle impairments could not be empirically discerned from this work. However evidence from the literature has been presented demonstrating that both primary damage to the central nervous system and secondary changes in the peripheral musculature likely contributed to the impairments identified in the paretic and nonparetic leg muscles of individuals with chronic stroke. To help discern which impairments were primary versus secondary, the experiments of this thesis need to be repeated in individuals with acute stroke. In addition, the use of larger samples in both acute and chronic stroke populations would enable researchers to perform meaningful correlations of impairments with functional task performance. Future research at the basic science level is
needed to further understand how stroke impacts muscle activation, reflex modulation, and muscle physiology.

CLINICAL IMPLICATIONS AND TREATMENT RECOMMENDATIONS

Individuals with stroke demonstrate a huge amount of variability in their clinical presentation. Lesion size and location, as well as duration post-stroke, will influence the clinician’s assessment and treatment approach. Thus the following recommendations will be most appropriately applied to individuals with chronic stroke with mild to moderate disability. However, since some of the identified impairments from this study are likely due to secondary changes and compensatory mechanisms, it would not be unreasonable to employ these recommendations as a preventative measure in individuals with subacute or acute stroke.

Firstly, clinicians should be aware that the so-called nonparetic leg is also impacted by stroke and should therefore exercise caution when using it as a reference leg for comparison. Due to the non-uniformity of muscle strength demonstrated across joint range of motion in both the paretic and nonparetic legs of individuals with stroke, clinicians may wish to assess strength at multiple joint angles and not only at mid-range, as is done with the conventional manual muscle test. In addition, a thorough evaluation of maximal isometric, concentric, and eccentric torque production is recommended. Because eccentric torque appears to be relatively less impaired than concentric and isometric torque, care should be taken when designing exercises to ensure that the appropriate amount of resistance is used to avoid underloading the target muscle during the lengthening phase of movement. However, clinicians should proceed with caution until further research can ascertain whether the muscle soreness and muscle injury associated with eccentric training outweigh its potential benefits. Since this study has provided evidence of significant weakness in the nonparetic ankle plantarflexors post-stroke (possibly due to compensatory gait strategies), a careful evaluation of this muscle group is recommended along
side an observational gait analysis. Encouragement of symmetrical weight bearing during functional activities should also be emphasized to help prevent or reverse weakness in the paretic leg from disuse, and prevent or reverse compensatory strength gains in the nonparetic leg from overuse. Because the paretic knee extensors were shown to demonstrate exaggerated weakness at short muscle lengths, resistance training of the quadriceps near terminal knee extension (i.e. repeated sit-to-stand from an elevated surface using only the paretic leg to lift the body) may lead to improvements in functional performance. Lastly, to address impairments in temporal muscle performance, both functional and conventional strength training exercises designed to improve the speed of contraction and relaxation should be explored.

In closing, this thesis has taken one small step towards answering a few of the many unanswered questions regarding muscle performance post-stroke. The clinical implications and evidence based recommendations gleamed from this work need to be tested under the scrutiny of randomized clinical trials to ensure that individuals with stroke are being managed in the most effective manner possible.
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Appendix I

The Folstein Mini-Mental Status Examination (MMSE)  
(screening tool)

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?  

“Spell out the word WORLD”; “now spell it backwards”. If troubles or errors occur, ask “Please count from 100 subtracting 7 every time”. Score both. Subject gets the best score of the two. If 5/5 for DLROW, do not score 100-7.

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 or say a complete sentence of your choice. (Item is scored only for writing the sentence, must have subject and verb. Misspelling and poor grammar is acceptable).

Give the patient the geometric design page and ask:

30. Could you please copy this drawing?
Appendix II

Physical Activity Readiness Questionnaire (PAR Q)
(screening tool)

1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
   - Yes
   - No

2. Do you feel pain in your chest or heart when you do physical activity?
   - Yes
   - No

3. In the past year, have you had chest or heart pain when you were not doing physical activity?
   - Yes
   - No

4. Do you lose your balance because of dizziness or do you ever lose consciousness?
   - Yes
   - No

5. Do you have a bone or joint problem, such as arthritis, that could be made worse by a change in physical activity?
   - Yes
   - No

6. Do you know if your blood pressure is controlled?
   - Yes
   - No
   - Don't know

7. Do you know of any other reason why you should not do physical activity?
   - Yes
   - No
Appendix III

Functional Comorbidity Index
(screening tool)

If you have ever been diagnosed with or had any of the following conditions or diseases, please circle them:

1. Arthritis
2. Osteoporosis
3. Asthma
4. Angina (chest pain or heart pain)
5. Neurological Disease (such as Multiple Sclerosis or Parkinson's)
6. Visual Impairment (cataract, near-sighted, far-sighted, glaucoma, etc)
7. Scoliosis
8. Congestive Heart Failure
9. Ulcers (Peptic Ulcer Disease, Gastro-Esophageal Reflux Disease)
10. Diabetes
11. Depression
12. Dementia (such as Alzheimer's)
13. Phlebitis: inflammation of the veins, typically in legs, but can be anywhere
14. Anxiety
15. COPD (Chronic Obstructive Pulmonary Disease; Emphysema, chronic bronchitis)
16. Hypertension
17. Hearing Impairment
18. Stroke or Transient Ischemic Attack
19. Liver Disease
20. Kidney Disease
21. Obesity
22. Paget's Disease (bone disorder causing increased and irregular formation of bone)
23. Cancer of any kind, if yes, please specify:
24. Hyperlipidemia (high cholesterol)
25. Family History of Heart disease or heart attack (Immediate Male relatives before age 55 and Immediate Female relatives before age 65)
26. Sedentary Lifestyle

Total Score: ___
INTRODUCTION
You are being invited to participate in this study because you have had one stroke, are 50 years of age or older and can walk at least 10 metres on your own (with or without a cane or walker), and we would like to investigate your muscle strength and mobility (ability to move/get around).

Your Participation is VOLUNTARY
• Your participation is entirely voluntary, so it is up to you to decide whether or not to take part in this study. Before you decide, it is important for you to understand what the research involves. This consent form will tell you about the study, why the research is being done, what will happen to you during the study and the possible benefits, risks, and discomforts.
• If you wish to participate, you will be asked to sign this form. If you decide to take part in this study, you are still free to withdraw at any time and without giving any reasons for your decision.
• If you do not wish to participate, you do not have to provide any reason for your decision not to participate nor will you lose the benefit of any medical care to which you are entitled or are presently receiving.
• Please take time to read the following information carefully and to discuss it with your family, friends, and doctor before you decide.

Who is conducting the study?
Dr. Janice Eng and Melanie Lomaglio are conducting the study in conjunction with the University of British Columbia, School of Rehabilitation Sciences. The study will take place at the GF Strong Rehab Centre in Vancouver, British Columbia.

BACKGROUND
Leg muscle weakness is a common problem following stroke and it is often associated with a decreased ability to perform everyday activities such as rising from a chair, climbing stairs, and walking quickly. In order for rehab clinicians to develop better muscle strengthening exercise programs, the reasons why your muscles are weak need to be better understood and explained.
WHAT IS THE PURPOSE OF THE STUDY?
The purpose of this research project is to examine the muscle function in both your stronger and weaker legs, as well as compare this function to people who have not had a stroke and are of similar age to you. By muscle function we mean, how fast and by how much are you able to tighten and relax your muscles?

WHO CAN PARTICIPATE IN THE STUDY?
If you meet the following criteria you may be eligible to participate in this study:
• Must have had only one stroke, at least one year ago
• Must be 50 years of age or older
• Must be able to walk by yourself for at least 10 metres with or without your cane/walker
• Must be able to tolerate two hours of activity with frequent and regular rest breaks
• Must be able to understand and follow instructions in English
• Must be able to actively move the ankle, knee, and hip joints in all directions
• Must not have any severe muscle or joint stiffness in your legs

WHO SHOULD NOT PARTICIPATE IN THE STUDY?
If you have any of the following conditions you are NOT eligible to participate in the study.
• Heart disease such as heart failure, chest pains, or an irregular heart beat
• Lung/chest disease, uncontrolled high blood pressure, injuries to muscles, bones, ligaments, tendons, or joints
• Increased pain with activities such as walking or getting out of a chair
• Unable to verbally speak with the investigators when asked questions
• Weakness or loss of sensation in both legs

WHAT DOES THE STUDY INVOLVE?
This study will take place at the Rehabilitation Research Laboratory at GF Strong Rehab Centre (4255 Laurel Street, Vancouver, BC). Thirty subjects living with stroke and thirty healthy control subjects will be recruited on a volunteer basis for this study. This study does not involve any physiotherapy treatment; it will, however, involve research procedures not normally done during routine rehabilitation care.

Time commitment
You will be requested to participate in two testing sessions, lasting two and a half hours each on non-consecutive days, over a three-week period. This means that you will be required to designate a total of five hours of your time to this research study, not including the time it takes to travel to and from the rehabilitation centre.

Study Overview
Your muscle strength will be tested under two different conditions, on two different days (see below for a detailed explanation). In addition, your walking speed will be determined and your time to go up and down four steps will be measured. You will also be required to fill out four questionnaires so that we can determine if you have any other health-related problems, if you need help at home or in the community, and whether or not you are active or sedentary. These questionnaires will each take between 5 and 10 minutes to complete.
If you agree to take part in this study, the procedures and visits you can expect will include the following:

Day 1:
You will initially be required to answer a few questions to ensure to us that you will be able to understand and follow all of the instructions that will be given during the research study. You will also be asked general questions about your current health status to ensure that you are safe to exercise. You will then have your legs assessed to determine if they are too stiff/tight to participate in the study. We will also examine how much you can actively move your ankle, knee, and hip. Following these quick questionnaires and assessments (5-10 minutes each) you will begin the muscle strength testing. All of the strength testing will be performed from a seated position. You will perform multiple static muscle contractions using your ankle, knee, and hip muscles. This means you will push or pull as hard as you can with your leg against a fixed lever for 3 seconds at a time (therefore your leg will not move but your muscles will tighten). Both of your legs will be tested for comparison. You will be given lots of rest breaks between your efforts and your blood pressure and heart rate will be closely monitored. The strength testing will last approximately 120 minutes including the frequent rest breaks. Total test time for day 1: not more than two and a half hours.

Day 2:
You will be required to walk by yourself for 10 metres at a comfortable pace so that we can determine your walking speed. You can use your cane or walker if needed. You will be asked to repeat this three times. We will also time how long it takes you to go up and down four regular height steps. You will be allowed to use the handrail. This should take approximately 15 minutes to complete. You will then begin more seated strength testing for the ankle, knee, and hip muscles of both legs. This time you will push or pull as hard as you can with your leg against a moving lever (therefore your leg will move). Again, plenty of rest breaks will be provided and your heart rate and blood pressure will be monitored. The strength testing will last approximately 100 minutes including the frequent rest breaks. Before you leave, you will be asked to complete three questionnaires, each taking 5-20 minutes to complete. The first questionnaire will ask you about any other health-related problems which you may have in addition to your stroke. The second questionnaire will ask you about how much assistance, if any, you need in your home and in the community. The last questionnaire will help us determine how active or sedentary your lifestyle is. The results of these questionnaires are kept confidential and will be used to describe the subjects that participated in our study. Total test time for day 2: not more than two and a half hours.

Possible Side Effects and Harms of Participating
There is a slight chance that you may feel tired or experience some muscle soreness one to three days after the testing sessions.

What are the Benefits of participating in this study?
There are no direct benefits to you for participating in this study. It is hoped that the information gained from this research will contribute to the understanding of leg muscle function in individuals who have had a stroke. This in turn will allow more effective treatment interventions to be developed. In addition, you will receive a short summary of your leg muscle strength after the data has been analyzed (this may take several months).
What Happens if I decide to Withdraw my Consent to Participate?

- Your participation in this research is entirely voluntary. You may withdraw from this study at any time. If you decide to enter the study and to withdraw at any time in the future, there will be no penalty or loss of benefits to which you are otherwise entitled, and your future medical care will not be affected.
- The study investigators may decide to discontinue the study at any time, or withdraw you from the study at any time, if they feel that it is in your best interests.
- If you choose to enter the study and then decide to withdraw at a later time, all data collected about you during your enrollment in the study will be retained for analysis. By law, this data cannot be destroyed.

What if Something Goes Wrong?

*You do not waive any of your legal rights to compensation by signing this consent form.* In case of a serious medical event, please report to an emergency room and inform them that you are participating in a research study and the following person can then be contacted for further information: Dr. Janice Eng at telephone number 604-714-4108.

After the Study is Completed

As previously mentioned, you will receive a short summary of your leg muscle strength after the data has been analyzed (this may take several months).

What will the study cost me?

You may incur personal travel expenses by participating in this study. In order to defray the costs of transportation and to compensate you for your time you will receive $50.00 after each session for a total of $100.00.

Will my taking part in this study be kept confidential?

Your confidentiality will be respected. No information that discloses your identity will be released or published without your specific consent to the disclosure. However, research records and medical records identifying you may be inspected in the presence of the Investigator or his or her designate by representatives of Health Canada and the UBC Research Ethics Board for the purpose of monitoring the research. However, no records which identify you by name or initials will be allowed to leave the Investigators’ offices.

Who do I contact if I have questions about the study during my participation?

If you have any questions or desire further information with respect to this study or during participation, you can contact Melanie Lomaglio (study co-ordinator) at 604-714-4108.

Who do I contact if I have questions or concerns about my rights as a subject during the study?

If you have any concerns about your rights as a research subject and/or your experiences while participating in this study, contact the ’Research Subject Information Line in the University of British Columbia Office of Research Services’ at 604-822-8598.
SUBJECT CONSENT TO PARTICIPATE
This is not a contract and I understand that I do not give up any legal rights by signing it. By signing the form I am indicating that:

- I have read and understood the subject information and consent form.
- I have had sufficient time to consider the information provided and to ask for advice if necessary.
- I have had the opportunity to ask questions and have had satisfactory responses to my questions.
- I understand that all the information collected will be kept confidential and that the result will only be used for scientific objectives.
- I understand that my participation in this study is voluntary and I am completely free to refuse to participate or to withdraw from this study at any time without changing in any way the quality of care that I receive.
- I understand that I am not waiving any of my legal rights as a result of signing this consent form.
- I have read this form and I freely consent to participate in this study.
- I have been told that I will receive a dated and signed copy of this form.

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Appendix V

CONTROL SUBJECT INFORMATION AND CONSENT FORM

Leg muscle properties in individuals with chronic stroke

Principal Investigator:
Dr. Janice Eng, PhD PT/OT
School of Rehabilitation Sciences, UBC
Rehab Research Lab, GF Strong Rehab Centre
Phone: 604-714-4108

Study coordinator:
Melanie Lomaglio, BScPT, MSc candidate
School of Rehabilitation Sciences, UBC
Rehab Research Lab, GF Strong Rehab
Phone: 604-714-4108

INTRODUCTION
You are being invited to participate in this study because we are seeking healthy control subjects to use for comparison in our investigation of muscle strength and mobility in individuals with stroke. You need to be 50 years of age or older, and able to walk at least 10 metres by yourself and have no known heart, lung, or joint disease.

Your Participation is VOLUNTARY
• Your participation is entirely voluntary, so it is up to you to decide whether or not to take part in this study. Before you decide, it is important for you to understand what the research involves. This consent form will tell you about the study, why the research is being done, what will happen to you during the study and the possible benefits, risks, and discomforts.
• If you wish to participate, you will be asked to sign this form. If you decide to take part in this study, you are still free to withdraw at any time and without giving any reasons for your decision.
• If you do not wish to participate, you do not have to provide any reason for your decision not to participate nor will you lose the benefit of any medical care to which you are entitled or are presently receiving.
• Please take time to read the following information carefully and to discuss it with your family, friends, and doctor before you decide.

Who is conducting the study?
Dr. Janice Eng and Melanie Lomaglio are conducting the study in conjunction with the University of British Columbia, School of Rehabilitation Sciences. The study will take place at the GF Strong Rehab Centre in Vancouver, British Columbia.

BACKGROUND
Leg muscle weakness is a common problem following stroke and it is often associated with a decreased ability to perform everyday activities such as rising from a chair, climbing stairs, and walking quickly. In order for rehab clinicians to develop better muscle strengthening interventions, the reasons as to why this weakness occurs needs to be better understood and explained.
WHAT IS THE PURPOSE OF THE STUDY?
The purpose of this research project is to examine the muscle function in both the stronger and weaker legs of individuals with stroke, and compare this function to similar aged people like you, who have not had a stroke. By muscle function we mean, how fast and by how much are you able to tighten and relax your muscles?

WHO CAN PARTICIPATE IN THE STUDY?
If you meet the following criteria you may be eligible to participate in this study:
• Must be 50 years of age or older
• Must not have any history of stroke or heart disease such as heart failure, chest pains, or an irregular heart beat
• Must not have any lung/chest disease, uncontrolled high blood pressure, injuries to muscles, bones, ligaments, tendons, or joints
• Must be able to walk by yourself for at least 10 metres
• Must be able to tolerate two hours of activity with frequent and regular rest breaks
• Must not have dementia
• Must be able to understand and follow instructions in English
• Must be able to actively move the ankle, knee, and hip joints in all directions

WHAT DOES THE STUDY INVOLVE?
This study will take place at the Rehabilitation Research Laboratory at G.F. Strong Rehab Centre (4255 Laurel Street, Vancouver, BC). Thirty subjects living with stroke and thirty healthy control subjects will be recruited on a volunteer basis for this study. This study does not involve any physiotherapy treatment; it will, however, involve research procedures not normally done during routine rehabilitation care.

Time commitment
You will be requested to participate in two testing sessions, lasting two and a half hours each on non-consecutive days, over a three-week period. This means that you will be required to designate a total of five hours of your time to this research study, not including the time it takes to travel to and from the rehabilitation centre.

Study Overview
Your muscle strength will be tested under two different conditions, on two different days (see below for a detailed explanation). In addition, your walking speed will be determined and your time to go up and down four steps will be measured. You will also be required to fill out three questionnaires so that we can determine if you have any other health-related problems and whether or not you are active or sedentary. These questionnaires will each take between 5 and 10 minutes to complete.
If you agree to take part in this study, the procedures and visits you can expect will include the following:

**Day 1:**
You will initially be required to answer a few questions to ensure to us that you will be able to understand and follow all of the instructions that will be given during the research study. You will also be asked general questions about your current health status to ensure that you are safe to exercise. Following the quick assessment and questionnaire (5-10 minutes each) you will begin the muscle strength testing. All of the strength testing will be performed from a seated position. You will perform multiple *static* muscle contractions using your ankle, knee, and hip muscles. This means you will push or pull as **hard** as you can with your leg against a fixed lever for 4 seconds at a time (therefore your leg will not move but your muscles will tighten). Both of your legs will be tested for comparison. You will be given lots of rest breaks between your efforts and your blood pressure and heart rate will be closely monitored. The strength testing will last approximately 120 minutes including the frequent rest breaks. Total test time for day 1: not more than two and a half hours.

**Day 2:**
You will be required to walk by yourself for 10 metres at a comfortable pace so that we can determine your walking speed. You will be asked to repeat this three times. We will also time how long it takes you to go up and down four regular height steps. You will be allowed to use the handrail. This should take approximately 15 minutes to complete. You will then begin more seated strength testing for the ankle, knee, and hip muscles of both legs. This time you will push or pull as **hard** as you can with your leg against a moving lever (therefore your leg will move). Again, plenty of rest breaks will be provided and your heart rate and blood pressure will be monitored. The strength testing will last approximately 100 minutes including the frequent rest breaks. Before you leave, you will be asked to complete two questionnaires, each taking 5-10 minutes to complete. The first questionnaire will ask you about any health-related problems which you may have. The second questionnaire will help us determine how active or sedentary your lifestyle is. The results of these questionnaires are kept confidential and will be used to describe both the subjects with stroke and the healthy control subjects participating in the study. Total test time for day 2: not more than two and a half hours.

**Possible Side Effects and Harms of Participating**
There is a slight chance that you may feel tired or experience some muscle soreness one to three days after the testing sessions.

**What are the Benefits of participating in this study?**
There are no direct benefits to you for participating in this study. It is hoped that the information gained from this research will contribute to the understanding of leg muscle function in individuals who have had a stroke. This in turn will allow more effective treatment interventions to be developed. In addition, you will receive a short summary of your leg muscle strength after the data has been analyzed (this may take several months).
What Happens if I decide to Withdraw my Consent to Participate?

- Your participation in this research is entirely voluntary. You may withdraw from this study at any time. If you decide to enter the study and to withdraw at any time in the future, there will be no penalty or loss of benefits to which you are otherwise entitled, and your future medical care will not be affected.
- The study investigators may decide to discontinue the study at any time, or withdraw you from the study at any time, if they feel that it is in your best interests.
- If you choose to enter the study and then decide to withdraw at a later time, all data collected about you during your enrollment in the study will be retained for analysis. By law, this data cannot be destroyed.

What if Something Goes Wrong?

*You do not waive any of your legal rights to compensation by signing this consent form.* In case of a serious medical event, please report to an emergency room and inform them that you are participating in a research study and the following person can then be contacted for further information: Dr. Janice Eng at telephone number 604-714-4108.

After the Study is Completed

As previously mentioned, you will receive a short summary of your leg muscle strength after the data has been analyzed (this may take several months).

What will the study cost me?

You may incur personal travel expenses by participating in this study. In order to defray the costs of transportation and to compensate you for your time you will receive $50.00 after each session for a total of $100.00.

Will my taking part in this study be kept confidential?

Your confidentiality will be respected. No information that discloses your identity will be released or published without your specific consent to the disclosure. However, research records and medical records identifying you may be inspected in the presence of the Investigator or his or her designate by representatives of Health Canada and the UBC Research Ethics Board for the purpose of monitoring the research. However, no records which identify you by name or initials will be allowed to leave the Investigators’ offices.

Who do I contact if I have questions about the study during my participation?

If you have any questions or desire further information with respect to this study or during participation, you can contact Melanie Lomaglio (study coordinator) at 604-714-4108.

Who do I contact if I have questions or concerns about my rights as a control subject during the study?

If you have any concerns about your rights as a research subject and/or your experiences while participating in this study, contact the “Research Subject Information Line in the University of British Columbia Office of Research Services” at 604-822-8598.
CONTROL SUBJECT CONSENT TO PARTICIPATE
This is not a contract and I understand that I do not give up any legal rights by signing it. By signing the form I am indicating that:

- I have read and understood the control subject information and consent form.
- I have had sufficient time to consider the information provided and to ask for advice if necessary.
- I have had the opportunity to ask questions and have had satisfactory responses to my questions.
- I understand that all the information collected will be kept confidential and that the result will only be used for scientific objectives.
- I understand that my participation in this study is voluntary and I am completely free to refuse to participate or to withdraw from this study at any time without changing in any way the quality of care that I receive.
- I understand that I am not waiving any of my legal rights as a result of signing this consent form.
- I have read this form and I freely consent to participate in this study.
- I have been told that I will receive a dated and signed copy of this form.

<table>
<thead>
<tr>
<th>Printed name of control subject</th>
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<th>Date</th>
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<table>
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<tr>
<th>Printed name of principal investigator/designated representative</th>
<th>Signature of principal investigator/designated representative</th>
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Appendix VI

Chedoke-McMaster Stroke Assessment - Score form

Impairment inventory: stage of recovery of leg and foot

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

LEG

1. ___ not yet Stage 2

2. Crook lying ___ resistance to passive hip/knee flexion
   ___ facilitated hip flexion
   ___ facilitated extension

3. ___ abduction: adduction to neutral
   ___ hip flexion to 90 degrees
   ___ full extension

4. ___ hip flexion to 90 degrees then extension synergy
   Sitting ___ bridging hip with equal weightbearing
   ___ knee flexion beyond 100 degrees

5. Crook lying ___ extension synergy, then flexion synergy
   Sit ___ raise thigh off bed
   Stand ___ hip extension with knee flexion

6. Sit ___ extension synergy, then flexion synergy
   ___ full range internal rotation
   Stand ___ trace a pattern: forward, side, back, return

7. Stand ___ unsupported: rapid high stepping 10 x in 5 sec
   ___ unsupported: trace a pattern quickly; forward, side, back, reverse
   ___ on weak leg with support: hop on weak leg

___ STAGE OF LEG
FOOT

1. ___ not yet Stage 2

2. Crook lying ___ resistance to passive dorsiflexion
   ___ facilitated dorsiflexion or toe extension
   ___ facilitated plantarflexion

3. Supine ___ plantarflexion > ½ range
   Sit ___ some dorsiflexion
   ___ extension of toes

4. ___ some eversion
   ___ inversion
   ___ legs crossed: dorsiflexion, then plantarflexion

5. ___ legs crossed: toe extension with ankle plantarflexion
   ___ sitting with knee extended: ankle plantarflexion, then dorsiflexion
   Stand ___ heel on floor: eversion

6. ___ heel on floor: tap foot 5 x in 5 sec
   ___ foot off floor: foot circumduction
   ___ knee straight, heel off floor: eversion

7. ___ heel touching forward, then toe touching behind, repeat 5 x in 10 sec
   ___ foot off floor: circumduction quickly, reverse
   ___ up on toes, then back on heels 5 x

___ STAGE OF FOOT

Appendix VII
American Heart Association Outcome Classification Score

Questions:

BADL (Basic Activities of Daily Living)

Do you require assistance with (please check):

- Dressing
- Bathing
- Climbing stairs
- Feeding
- Toileting
- Grooming
- Sphincter control (continence i.e. are you able to control your bowel and bladder and have no accidents?)

If independent in all areas, proceed to IADL
If less than 3 areas checked, level III
If 3 or more areas checked, level IV
If 5 or more areas checked, level V

IADL (Instrumental Activities of Daily Living)

Do you require assistance with (please check):

- Shopping
- Using transportation
- Preparing meals
- Handling finances
- Home maintenance
- Taking medications
- Community access
- Leisure

If Yes, Level II

Do you have anything that you used to do but are not able to do now after the stroke? Yes / No

If Yes, Level II
If No, Level I
American Heart Association Outcome Classification Score

I = Independent in BADL and IADL activities and tasks required of roles patient had before the 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 medications.

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 (> or = 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 (> or = 5 areas) and IADL. Patient is unable to live alone safely and requires full-time care.

BADL = basic activities of daily living. Feeding, grooming, dressing, bathing, toileting, transferring from place to place, sphincter control, mobility

Independence in BADL could enable the patient to live at home with help from family or community providers for meals or other household tasks as needed.

IADL = Instrumental activities of daily living. More complex tasks of daily living. Needed to maintain independence in the home and community and include shopping, using transportation, telephoning, preparing meals, handling finances and maintaining a household. Independence in these activities enables the patient to be discharged home without being dependent on others. Other instrumental activities of daily living that affect quality of life are work skills, religious activities, and leisure-time and recreational activities.

## Appendix VIII

### Modified Ashworth Scale

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

Appendix IX

Physical Activity Scale for Persons with Physical Disabilities

Leisure Time Activity

1. During the past 7 days how often did you engage in stationary activities such as reading, watching TV, computer games, or doing handcrafts?
   a) never (go to question #2)
   b) seldom (1 – 2 days)
   c) sometimes (3 – 4 days)
   d) often (5 – 7 days)

   What were these activities? ________________________________

   On average, how many hours per day did you spend in these stationary activities?
   a) less than 1 hour
   b) 1 but less than 2 hours
   c) 2 – 4 hours
   d) more than 4 hours

2. During the past 7 days, how often did you walk, wheel, push outside your home other than specifically for exercise. For example, getting to work or class, walking the dog, shopping, or other errands?
   a) never (go to question #3)
   b) seldom (1 – 2 days)
   c) sometimes (3 – 4 days)
   d) often (5 – 7 days)

   On average, how many hours per day did you spend walking, wheeling or pushing outside your home?
   a) less than 1 hour
   b) 1 but less than 2 hours
   c) 2 to 4 hours
   d) more than 4 hours

3. During the past 7 days, how often did you engage in light sport or recreational activities such as bowling, golf with a cart, hunting or fishing, darts, billiards or pool, therapeutic exercise (physical or occupational therapy, stretching, use of a standing frame) or other similar activities?
   a) never (go to question #4)
   b) seldom (1 – 2 days)
   c) sometimes (3 – 4 days)
   d) often (5 – 7 days)

   What were these activities? ________________________________
On average, how many hours per day did you spend in these light sport or recreational activities?

a) less than 1 hour  
b) 1 but less than 2 hours  
c) 2 ~ 4 hours  
d) more than 4 hours

4. During the past 7 days, how often did you engage in moderate sport and recreational activities such as double tennis, softball, golf without a cart, ballroom dancing, wheeling or pushing for pleasure or other similar activities?

a) never (go to question #5)  
b) seldom (1 ~ 2 days)  
c) sometimes (3 ~ 4 days)  
d) often (5 ~ 7 days)

What were these activities? ____________________________

On average, how many hours per day did you spend in these moderate sport and recreational activities?

a) less than 1 hour  
b) 1 but less than 2 hours  
c) 2 ~ 4 hours  
d) more than 4 hours

5. During the past 7 days, how often did you engage in strenuous sport and recreational activities such as jogging, wheelchair racing (training), off-road pushing, swimming, aerobic dance, arm cranking, cycling (hand or leg), singles tennis, rugby, basketball, walking with crutches and braces, or other similar activities?

a) never (go to question #6)  
b) seldom (1 ~ 2 days)  
c) sometimes (3 ~ 4 days)  
d) often (5 ~ 7 days)

What were these activities? ____________________________

On average, how many hours per day did you spend in these strenuous sport or recreational activities?

a) less than 1 hour  
b) 1 but less than 2 hours  
c) 2 ~ 4 hours  
d) more than 4 hours

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6. During the past 7 days, how often did you do any *exercise specifically to increase muscle strength and endurance* such as lifting weights, push-ups, pull-ups, dips, or wheelchair push-ups, etc?

a) never (go to question #7)

b) seldom (1 – 2 days)

c) sometimes (3 – 4 days)

d) often (5 – 7 days)

What were these activities? ______________________________________________________

On average, how many hours per day did you spend in these *exercises to increase muscle strength and endurance*?

a) less than 1 hour

b) 1 but less than 2 hours

c) 2 – 4 hours

d) more than 4 hours

**Household Activity**

7. During the past 7 days, how often have you done any *light housework*, such as dusting, sweeping floors or washing dishes?

a) never (go to question #8)

b) seldom (1 – 2 days)

c) sometimes (3 – 4 days)

d) often (5 – 7 days)

On average, how many hours per day did you spend doing *light housework*?

a) less than 1 hour

b) 1 but less than 2 hours

c) 2 – 4 hours

d) more than 4 hours

8. During the past 7 days, how often have you done any *heavy housework or chores* such as vacuuming, scrubbing floors, washing windows, or walls, etc?

a) never (go to question #9)

b) seldom (1 – 2 days)

c) sometimes (3 – 4 days)

d) often (5 – 7 days)

On average, how many hours per day did you spend doing *heavy housework or chores*?

a) less than 1 hour

b) 1 but less than 2 hours

c) 2 – 4 hours

d) more than 4 hours
9. During the past 7 days, how often have you done home repairs like carpentry, painting, furniture refinishing, electrical work, etc?
   a) never (go to question #10)
   b) seldom (1 ~ 2 days)
   c) sometimes (3 ~ 4 days)
   d) often (5 ~ 7 days)

   On average, how many hours per day did you spend doing home repairs?
   a) less than 1 hour
   b) 1 but less than 2 hours
   c) 2 ~ 4 hours
   d) more than 4 hours

10. During the past 7 days, how often have you done lawn work or yard care including mowing, leaf or snow removal, tree or bush trimming, or wood chopping, etc?
   a) never (go to question #11)
   b) seldom (1 ~ 2 days)
   c) sometimes (3 ~ 4 days)
   d) often (5 ~ 7 days)

   On average, how many hours per day did you spend doing lawn work?
   a) less than 1 hour
   b) 1 but less than 2 hours
   c) 2 ~ 4 hours
   d) more than 4 hours

11. During the past 7 days, how often have you outdoor gardening?
   a) never (go to question #12)
   b) seldom (1 ~ 2 days)
   c) sometimes (3 ~ 4 days)
   d) often (5 ~ 7 days)

   On average, how many hours per day did you spend doing outdoor gardening?
   a) less than 1 hour
   b) 1 but less than 2 hours
   c) 2 ~ 4 hours
   d) more than 4 hours

12. During the past 7 days, how often have you care for another person, such as children, a dependent spouse, or another adult?
   a) never (go to question #13)
   b) seldom (1 ~ 2 days)
   c) sometimes (3 ~ 4 days)
   d) often (5 ~ 7 days)
On average, how many hours per day did you spend caring for another person?

a) less than 1 hour  
b) 1 but less than 2 hours  
c) 2 ~ 4 hours  
d) more than 4 hours

**Work-Related Activity**

13. During the past 7 days, how often have you worked for pay or as a volunteer? Exclude work that mainly involved sitting with slight arm movement such as light office work, computer work, light assembly line work, driving bus or van, etc.)

a) never (go to END)  
b) seldom (1 ~ 2 days)  
c) sometimes (3 ~ 4 days)  
d) often (5 ~ 7 days)

On average, how many hours per day did you spend working for pay or as a volunteer?

a) less than 1 hour  
b) 1 but less than 4 hours  
c) 5 but less than 8 hours  
d) 8 hours or more

Appendix X

Subject positioning and stabilization

**Hip.** Using a KinCom dynamometer, hip torques were tested in a semi-reclined position (40° from the horizontal). Subjects had their backs supported and a pillow was placed under the head for comfort. A pad attached to the seat supported the contralateral thigh. Three straps stabilized the trunk and pelvis (two criss-crossing over the chest and one over the anterior-superior iliac spines). The greater trochanter was aligned with the axis of rotation. The cuff of the force transducer was placed three fingerbreadths proximal to the popliteal space. Subjects were required to keep their hands resting in their laps throughout the testing.
Knee. Using a KinCom dynamometer, knee torques were tested at a 90° hip angle. Thus subjects were seated and their backs were supported. Three straps stabilized the trunk and pelvis (two criss-crossing over the chest and one just distal to the anterior-superior iliac spines). A rigid clamp placed over the distal thigh musculature of the test leg was used for additional stabilization. The lateral femoral condyle was aligned with the dynamometer’s axis of rotation. The cuff of the force transducer was placed three fingerbreadths proximal to the medial malleolus. Subjects were required to keep their hands resting in their laps throughout the testing.
Ankle. Using a KinCom dynamometer, ankle torques were tested at a 90° hip and knee angle. Thus subjects were seated and their backs were supported. One strap was used to stabilize the pelvis. The foot was secured in a rigid metal ankle attachment provided by the manufacturer and the lateral malleolus was aligned with the dynamometer's axis of rotation. Subjects were required to keep their hands resting in their laps throughout the testing.
Appendix XI

Muscle soreness assessment

Self-administered 24-48 hours after Day 1 protocol (maximal isometric torque testing at the ankle, knee, and hip joints)

Prior to this task, sit and rest in a chair for 5 minutes.

Circle the one number that describes how much **muscle soreness you feel** as you stand up from the chair (without using your arms), walk a few (3) metres, then turn around and walk back to the chair and sit down again (again without using your arms)

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
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<tr>
<td>No pain</td>
<td>Pain as bad as you can imagine</td>
<td></td>
<td></td>
<td></td>
<td></td>
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Circle the one number that describes how, during the past 24 hours, **pain has interfered** with your:

**Walking ability**

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<th>3</th>
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<th>7</th>
<th>8</th>
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<th>10</th>
</tr>
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<tbody>
<tr>
<td>does not interfere</td>
<td>completely interferes</td>
<td></td>
<td></td>
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Circle the one number that describes how, during the past 24 hours, **pain has interfered** in with your:

**General Activity:**

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<th>7</th>
<th>8</th>
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<th>10</th>
</tr>
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<td>completely interferes</td>
<td></td>
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Appendix XII

Results of muscle soreness assessment

There was very little muscle soreness following maximal isometric torque testing for both individuals with stroke and healthy controls. The results of the pain questionnaire were not reported in the body of the thesis and are presented here for interest only.

Visual analogue of pain (0 = no pain to 10 = worst pain imaginable)

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<th>CONTROL GROUP</th>
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<td>N = 17</td>
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Visual analogue of pain interfering with walking ability (0 = does not interfere to 10 completely interferes)

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</table>
Visual analogue of pain interfering with activity level (0 = does not interfere to 10 completely interferes)

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<th>CONTROL GROUP</th>
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<tbody>
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Appendix XIII

Test-retest intrarater reliability for mean peak isometric torque, time to develop torque, and time to reduce torque

Intraclass correlations, ICC(3,3), Standard Error of Measurements (in Newton-meters), and P Values from the F Test for Mean Peak Isometric Torque (N = 9)

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<th>Nonparetic Leg</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td>P values from F test</td>
<td>ICC</td>
<td>P values from F test</td>
</tr>
<tr>
<td>Ankle plantarflexion</td>
<td>0.90 (8.3)</td>
<td>0.053</td>
<td>0.22 (13.9)</td>
<td>0.027</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.89 (5.8)</td>
<td>0.124</td>
<td>0.97 (2.6)</td>
<td>0.642</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.93 (4.0)</td>
<td>0.320</td>
<td>0.97 (3.3)</td>
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<tr>
<td>Knee Extension</td>
<td>0.98 (4.7)</td>
<td>0.463</td>
<td>0.98 (6.3)</td>
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<tr>
<td>Hip Extension</td>
<td>0.91 (11.0)</td>
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<td>0.97 (8.5)</td>
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<tr>
<td>Hip Flexion</td>
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<td>0.724</td>
<td>0.99 (3.1)</td>
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<tr>
<td>MEAN</td>
<td><strong>0.93 (6.3)</strong></td>
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<td><strong>0.85 (6.3)</strong></td>
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Intraclass correlations, ICC(3,3), Standard Error of Measurements (in seconds), and P Values from the F Test for Mean Time to Develop Isometric Torque (N = 9)

<table>
<thead>
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<th>Nonparetic Leg</th>
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<td></td>
<td>ICC</td>
<td>P values from F test</td>
<td>ICC</td>
<td>P values from F test</td>
</tr>
<tr>
<td>Ankle plantarflexion</td>
<td>0.94 (0.063)</td>
<td>0.567</td>
<td>0.72 (0.097)</td>
<td>0.0336</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.59 (0.164)</td>
<td>0.292</td>
<td>0.87 (0.028)</td>
<td>0.017</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.89 (0.071)</td>
<td>0.589</td>
<td>0.62 (0.088)</td>
<td>0.899</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.83 (0.077)</td>
<td>0.037</td>
<td>0.96 (0.029)</td>
<td>0.482</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.74 (0.081)</td>
<td>0.348</td>
<td>0.61 (0.141)</td>
<td>0.236</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.83 (0.067)</td>
<td>0.017</td>
<td>0.78 (0.088)</td>
<td>0.986</td>
</tr>
<tr>
<td>MEAN</td>
<td>0.80 (0.087)</td>
<td></td>
<td>0.76 (0.079)</td>
<td></td>
</tr>
</tbody>
</table>

Intraclass correlations, ICC(3,3), Standard Error of Measurements (in seconds), and P Values from the F Test for Mean Time to Reduce Isometric Torque (N = 9)

<table>
<thead>
<tr>
<th></th>
<th>Paretic Leg</th>
<th></th>
<th>Nonparetic Leg</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td>P values from F test</td>
<td>ICC</td>
<td>P values from F test</td>
</tr>
<tr>
<td>Ankle plantarflexion</td>
<td>0.73 (0.097)</td>
<td>0.210</td>
<td>0.76 (0.120)</td>
<td>0.078</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.77 (0.103)</td>
<td>0.744</td>
<td>0.88 (0.093)</td>
<td>0.527</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.93 (0.104)</td>
<td>0.027</td>
<td>0.56 (0.117)</td>
<td>0.772</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.84 (0.075)</td>
<td>0.586</td>
<td>0.63 (0.101)</td>
<td>0.822</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.36 (0.147)</td>
<td>0.391</td>
<td>0.58 (0.130)</td>
<td>0.338</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.38 (0.231)</td>
<td>0.042</td>
<td>0.73 (0.219)</td>
<td>0.927</td>
</tr>
<tr>
<td>MEAN</td>
<td>0.67 (0.126)</td>
<td></td>
<td>0.69 (0.130)</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Standard error of measurement in parentheses.
Bolded text reported in the body of the thesis.
Note: For times to develop and reduce isometric torque, ICCs below 0.70 were examined for outliers using visual inspection of the relevant scatterplots. In each case one outlier was removed and new ICC tables were generated. For each outlier removed there was a substantial learning effect in that the subject was faster at time two than at time one (these adjusted mean values were reported in the body of the thesis).

Intraclass correlations, ICC(3,3), Standard Error of Measurements (in seconds), and P Values from the F Test for Mean Time to Develop Isometric Torque (N = 8)

<table>
<thead>
<tr>
<th></th>
<th>Paretic Leg ICC</th>
<th>P values from F test</th>
<th>Nonparetic Leg ICC</th>
<th>P values from F test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle plantarflexion</td>
<td>0.94 (0.063)</td>
<td>0.567</td>
<td>0.72 (0.097)</td>
<td>0.0336</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.67 (0.148)</td>
<td>0.544</td>
<td>0.87 (0.028)</td>
<td>0.017</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.89 (0.071)</td>
<td>0.589</td>
<td>0.72 (0.074)</td>
<td>0.452</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.83 (0.077)</td>
<td>0.037</td>
<td>0.96 (0.029)</td>
<td>0.482</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.74 (0.081)</td>
<td>0.348</td>
<td>0.76 (0.079)</td>
<td>0.492</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.83 (0.067)</td>
<td>0.017</td>
<td>0.78 (0.088)</td>
<td>0.986</td>
</tr>
<tr>
<td>MEAN</td>
<td>0.82 (0.085)</td>
<td>0.80 (0.066)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Intraclass correlations, ICC(3,3), Standard Error of Measurements (in seconds), and P Values from the F Test for Mean Time to Reduce Isometric Torque (N = 8)

<table>
<thead>
<tr>
<th></th>
<th>Paretic Leg ICC</th>
<th>P values from F test</th>
<th>Nonparetic Leg ICC</th>
<th>P values from F test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle plantarflexion</td>
<td>0.73 (.097)</td>
<td>0.210</td>
<td>0.76 (.120)</td>
<td>0.078</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.77 (.103)</td>
<td>0.744</td>
<td>0.88 (.093)</td>
<td>0.527</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.93 (.104)</td>
<td>0.027</td>
<td>0.72 (.084)</td>
<td>0.621</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.84 (.075)</td>
<td>0.586</td>
<td>0.86 (.059)</td>
<td>0.167</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.86 (.191)</td>
<td>0.997</td>
<td>0.69 (.106)</td>
<td>0.673</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.74 (.135)</td>
<td>0.051</td>
<td>0.73 (.219)</td>
<td>0.927</td>
</tr>
<tr>
<td>MEAN</td>
<td>0.81 (.118)</td>
<td>0.77 (.114)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Standard error of measurement in parentheses.
Appendix XIV

Test-retest intrarater reliability for isometric torque-angle curves

Intraclass correlations ICC(3,1), Standard Error of Measurements (in percentage), and $P$ Values from the F Test for Isometric Torque-angle Curves ($N=9$)

<table>
<thead>
<tr>
<th></th>
<th>Paretic Leg</th>
<th>Nonparetic Leg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td>$P$ values from F test</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.82 (0.085)</td>
<td>0.166</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.65 (0.112)</td>
<td>0.290</td>
</tr>
</tbody>
</table>

NOTE: Standard error of measurement in parentheses.
Appendix XV

Test-retest intrarater reliability for mean peak and mean average concentric and eccentric torques

Intraclass correlations, ICC(3,3), Standard Error of Measurements (in Newton-meters) and *P* Values from the F Test for Mean Peak Concentric Torque (*N* = 9)

<table>
<thead>
<tr>
<th></th>
<th>Paretic Leg</th>
<th></th>
<th>Nonparetic Leg</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td><em>P</em> values from F test</td>
<td>ICC</td>
<td><em>P</em> values from F test</td>
</tr>
<tr>
<td>Ankle plantarflexion</td>
<td>0.96 (5.1)</td>
<td>0.383</td>
<td>0.73 (11.0)</td>
<td>0.742</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.99 (1.8)</td>
<td>0.043</td>
<td>0.93 (3.3)</td>
<td>0.917</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.97 (2.3)</td>
<td>0.946</td>
<td>0.97 (2.9)</td>
<td>0.815</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.97 (4.4)</td>
<td>0.147</td>
<td>0.97 (6.9)</td>
<td>0.380</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.98 (4.6)</td>
<td>0.630</td>
<td>0.98 (5.6)</td>
<td>0.004</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.98 (2.2)</td>
<td>0.226</td>
<td>0.95 (6.3)</td>
<td>0.855</td>
</tr>
<tr>
<td>MEAN</td>
<td><strong>0.98 (3.4)</strong></td>
<td></td>
<td><strong>0.92 (6.0)</strong></td>
<td></td>
</tr>
</tbody>
</table>

Intraclass correlations, ICC(3,3), Standard Error of Measurements (in Newton-meters) and *P* Values from the F Test for Mean Peak Eccentric Torque (*N* = 9)

<table>
<thead>
<tr>
<th></th>
<th>Paretic Leg</th>
<th></th>
<th>Nonparetic Leg</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td><em>P</em> values from F test</td>
<td>ICC</td>
<td><em>P</em> values from F test</td>
</tr>
<tr>
<td>Ankle plantarflexion</td>
<td>0.94 (10.6)</td>
<td>0.734</td>
<td>0.90 (11.2)</td>
<td>0.402</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.98 (3.2)</td>
<td>0.037</td>
<td>0.98 (3.1)</td>
<td>0.480</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.95 (5.4)</td>
<td>0.235</td>
<td>0.93 (10.0)</td>
<td>0.163</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.95 (7.7)</td>
<td>0.805</td>
<td>0.97 (10.5)</td>
<td>0.514</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.96 (11.4)</td>
<td>0.249</td>
<td>0.98 (11.0)</td>
<td>0.019</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.98 (4.6)</td>
<td>0.909</td>
<td>0.99 (4.3)</td>
<td>0.042</td>
</tr>
<tr>
<td>MEAN</td>
<td><strong>0.96 (7.2)</strong></td>
<td></td>
<td><strong>0.96 (8.4)</strong></td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Standard error of measurement in parentheses.
Bolded text reported in the body of the thesis.
Intraclass correlations, ICC(3,3), Standard Error of Measurements (in Newton-meters) and $P$ Values from the $F$ Test for Mean Average Concentric Torque ($N = 9$)

<table>
<thead>
<tr>
<th></th>
<th>Paretic Leg</th>
<th>$P$ values from F test</th>
<th>Nonparetic Leg</th>
<th>$P$ values from F test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td></td>
<td>ICC</td>
<td></td>
</tr>
<tr>
<td>Ankle plantarflexion</td>
<td>0.96 (4.2)</td>
<td>0.283</td>
<td>0.58 (11.0)</td>
<td>0.674</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.98 (2.3)</td>
<td>0.018</td>
<td>0.95 (2.5)</td>
<td>0.653</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.97 (1.7)</td>
<td>0.367</td>
<td>0.99 (1.4)</td>
<td>0.807</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.97 (3.4)</td>
<td>0.247</td>
<td>0.98 (4.8)</td>
<td>0.228</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.87 (8.9)</td>
<td>0.802</td>
<td>0.97 (5.9)</td>
<td>0.009</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.97 (2.3)</td>
<td>0.781</td>
<td>0.98 (3.0)</td>
<td>0.435</td>
</tr>
<tr>
<td>MEAN</td>
<td>0.95 (3.8)</td>
<td></td>
<td>0.91 (4.8)</td>
<td></td>
</tr>
</tbody>
</table>

Intraclass correlations, ICC(3,3), Standard Error of Measurements (in Newton-meters) and $P$ Values from the $F$ Test for Mean Average Eccentric Torque ($N = 9$)

<table>
<thead>
<tr>
<th></th>
<th>Paretic Leg</th>
<th>$P$ values from F test</th>
<th>Nonparetic Leg</th>
<th>$P$ values from F test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td></td>
<td>ICC</td>
<td></td>
</tr>
<tr>
<td>Ankle plantarflexion</td>
<td>0.94 (6.7)</td>
<td>0.628</td>
<td>0.74 (10.0)</td>
<td>0.438</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.98 (2.9)</td>
<td>0.041</td>
<td>0.98 (2.5)</td>
<td>0.756</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.99 (2.0)</td>
<td>0.393</td>
<td>0.98 (3.6)</td>
<td>0.221</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>0.99 (2.8)</td>
<td>0.518</td>
<td>0.97 (7.4)</td>
<td>0.316</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.91 (11.1)</td>
<td>0.349</td>
<td>0.99 (5.4)</td>
<td>0.007</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.97 (4.0)</td>
<td>0.935</td>
<td>0.99 (3.2)</td>
<td>0.085</td>
</tr>
<tr>
<td>MEAN</td>
<td>0.96 (4.9)</td>
<td></td>
<td>0.94 (5.35)</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Standard error of measurement in parentheses.
Appendix XVI

Test-retest intrarater reliability for functional measures

Intraclass correlations ICC(3,3), Standard Error of Measurements (in seconds), and \( P \) values from the F Test for Functional Measures (\( N=9 \))

<table>
<thead>
<tr>
<th></th>
<th>ICC</th>
<th>( P ) values from F test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ambulation time – Fast paced</td>
<td>0.99 (0.11)</td>
<td>0.786</td>
</tr>
<tr>
<td>Ambulation time – Self paced</td>
<td>0.98 (0.18)</td>
<td>0.645</td>
</tr>
<tr>
<td>Time to ascend four steps</td>
<td>0.94 (0.34)</td>
<td>0.340</td>
</tr>
<tr>
<td>Time to descend steps</td>
<td>0.97 (0.34)</td>
<td>0.796</td>
</tr>
<tr>
<td>MEAN</td>
<td>0.97 (0.24)</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Standard error of measurement in parentheses.
Ambulation time was recorded over the middle 4-metre section of a 10-metre walkway.
Appendix XVII

Post-hoc results for Relative and Absolute knee extension and flexion torque-angle curves (paretic and nonparetic legs of individuals with stroke)

Note: Only the statistical results for relative knee extension and flexion torque-angle curves were reported in the body of the thesis (Chapter 3). A statistical analysis of the absolute knee extension and flexion torque-angle curves were performed and reported here for interest only.

Post-hoc Independent Samples T - tests for Relative Knee Extension Torques (N = 19)

<table>
<thead>
<tr>
<th>Joint Angle</th>
<th>Paretic - Control</th>
<th>Nonparetic - Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>15°</td>
<td>0.002*</td>
<td>0.142</td>
</tr>
<tr>
<td>35°</td>
<td>0.472</td>
<td>0.529</td>
</tr>
<tr>
<td>55°</td>
<td>0.224</td>
<td>0.633</td>
</tr>
<tr>
<td>75°</td>
<td>0.328</td>
<td>0.188</td>
</tr>
<tr>
<td>95°</td>
<td>0.943</td>
<td>0.015*</td>
</tr>
<tr>
<td>105°</td>
<td>0.361</td>
<td>0.004*</td>
</tr>
</tbody>
</table>

* significant at \( P \leq 0.05 \)

Note: The ANOVA revealed a significant interaction between group (paretic, nonparetic, and control) and joint angle (6 angles) (\( P = 0.001 \)).

Post-hoc Independent Samples T - tests for Relative Knee Flexion Torques (N = 19)

<table>
<thead>
<tr>
<th>Joint Angle</th>
<th>Paretic - Control</th>
<th>Nonparetic - Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>15°</td>
<td>0.389</td>
<td>0.044*</td>
</tr>
<tr>
<td>35°</td>
<td>0.641</td>
<td>0.159</td>
</tr>
<tr>
<td>55°</td>
<td>0.429</td>
<td>0.050*</td>
</tr>
<tr>
<td>75°</td>
<td>0.584</td>
<td>0.014*</td>
</tr>
<tr>
<td>95°</td>
<td>0.560</td>
<td>0.016*</td>
</tr>
<tr>
<td>105°</td>
<td>0.504</td>
<td>0.057</td>
</tr>
</tbody>
</table>

* significant at \( P \leq 0.05 \)

Note: The ANOVA revealed a significant interaction between group (paretic, nonparetic, and control) and joint angle (6 angles) (\( P = 0.041 \)).

Refer to figures 3-3 and 3-4 for paretic and nonparetic relative knee extension torque-angle curves respectively. Refer to figures 3-7 and 3-8 for relative knee flexion torque-angle curves respectively (results section of Chapter 3).
Post-hoc Independent Samples T-tests for *Absolute* Knee Extension Torques (N = 19)

<table>
<thead>
<tr>
<th>Joint Angle</th>
<th>Paretic – Control</th>
<th>Nonparetic – Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>15°</td>
<td>0.001*</td>
<td>0.299</td>
</tr>
<tr>
<td>35°</td>
<td>0.001*</td>
<td>0.765</td>
</tr>
<tr>
<td>55°</td>
<td>0.003*</td>
<td>0.866</td>
</tr>
<tr>
<td>75°</td>
<td>0.001*</td>
<td>0.959</td>
</tr>
<tr>
<td>95°</td>
<td>0.001*</td>
<td>0.478</td>
</tr>
<tr>
<td>105°</td>
<td>0.001*</td>
<td>0.317</td>
</tr>
</tbody>
</table>

* significant at P ≤ 0.05

Note: The ANOVA revealed a significant interaction between group (paretic, nonparetic, and control) and joint angle (6 angles) (P = 0.014).

Not surprisingly, the paretic knee torque of individuals with stroke was significantly lower than controls at every joint angle tested for both extension and flexion joint actions (thus demonstrating significant weakness across joint range of motion).

Post-hoc Independent Samples T-tests for *Absolute* Knee Flexion Torques (N = 19)

<table>
<thead>
<tr>
<th>Joint Angle</th>
<th>Paretic – Control</th>
<th>Nonparetic – Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>15°</td>
<td>0.001*</td>
<td>0.108</td>
</tr>
<tr>
<td>35°</td>
<td>0.001*</td>
<td>0.358</td>
</tr>
<tr>
<td>55°</td>
<td>0.001*</td>
<td>0.386</td>
</tr>
<tr>
<td>75°</td>
<td>0.001*</td>
<td>0.674</td>
</tr>
<tr>
<td>95°</td>
<td>0.001*</td>
<td>0.994</td>
</tr>
<tr>
<td>105°</td>
<td>0.001*</td>
<td>0.978</td>
</tr>
</tbody>
</table>

* significant at P ≤ 0.05

Note: The ANOVA revealed a significant interaction between group (paretic, nonparetic, and control) and joint angle (6 angles) (P = 0.001).

The nonparetic knee torque of individuals with stroke was not significantly different than controls at any joint angle for both extension and flexion joint actions (thus, nonparetic knee strength was preserved across joint range of motion).

Refer to figures 3-1 and 3-2 for paretic and nonparetic *absolute* knee extension torque-angle curves respectively. Refer to figures 3-5 and 3-6 for *absolute* knee flexion torque-angle curves respectively (results section of Chapter 3).
Appendix XVIII

Functional Correlations (isometric torque, times to develop and reduce torque)

The following correlations were performed for interest only and are not reported in the body of the thesis. Future research with a larger sample is needed to extend these preliminary results.

Spearman rank correlations (Rho) between functional measures and paretic leg isometric torque values ($N = 19$)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>-0.160</td>
<td>-0.218</td>
<td>-0.168</td>
<td>-0.154</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td><strong>-0.486</strong></td>
<td><strong>-0.523</strong></td>
<td><strong>-0.513</strong></td>
<td>0.274</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td><strong>-0.678</strong></td>
<td><strong>-0.786</strong></td>
<td><strong>-0.753</strong></td>
<td><strong>-0.709</strong></td>
</tr>
<tr>
<td>Knee Extension</td>
<td><strong>-0.551</strong></td>
<td><strong>-0.542</strong></td>
<td><strong>-0.679</strong></td>
<td><strong>-0.746</strong></td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.189</td>
<td>-0.261</td>
<td>-0.263</td>
<td>-0.426</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>-0.225</td>
<td>-0.188</td>
<td>-0.249</td>
<td>-0.288</td>
</tr>
</tbody>
</table>

* Significant at $P < 0.05$, ** Significant at $P < 0.01$

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used

Spearman rank correlations (Rho) between functional measures and paretic leg time to develop torque (s) ($N = 19$)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>0.236</td>
<td>0.112</td>
<td>0.154</td>
<td>0.055</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>-0.355</td>
<td>-0.221</td>
<td>-0.305</td>
<td>-0.431</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.071</td>
<td>0.104</td>
<td>0.173</td>
<td>0.269</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.240</td>
<td>-0.077</td>
<td>-0.012</td>
<td>0.002</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.067</td>
<td>0.012</td>
<td>0.044</td>
<td>0.121</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.032</td>
<td>0.161</td>
<td>-0.008</td>
<td>0.168</td>
</tr>
</tbody>
</table>

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used

Spearman rank correlations (Rho) between functional measures and paretic leg time to reduce torque (s) ($N = 19$)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>0.343</td>
<td>0.446</td>
<td>0.250</td>
<td>0.273</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>0.365</td>
<td>0.249</td>
<td>0.156</td>
<td>-0.032</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td><strong>0.481</strong></td>
<td>0.423</td>
<td>0.308</td>
<td>0.193</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.045</td>
<td>-0.094</td>
<td>-0.223</td>
<td>-0.053</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>0.175</td>
<td>0.256</td>
<td>0.086</td>
<td>0.211</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>-0.014</td>
<td>-0.193</td>
<td>-0.192</td>
<td>-0.265</td>
</tr>
</tbody>
</table>

* Significant at $P < 0.05$

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used
Spearman rank correlations (Rho) between functional measures and nonparetic leg isometric torque values \((N = 19)\)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>-0.068</td>
<td>-0.242</td>
<td>-0.359</td>
<td>-0.370</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>-0.412</td>
<td>-0.416</td>
<td>-0.479*</td>
<td>-0.349</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>-0.384</td>
<td>-0.528*</td>
<td>-0.559*</td>
<td>-0.574*</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.404</td>
<td>-0.319</td>
<td>-0.428</td>
<td>-0.465*</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.351</td>
<td>-0.360</td>
<td>-0.424</td>
<td>-0.568*</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>0.091</td>
<td>0.081</td>
<td>-0.038</td>
<td>-0.142</td>
</tr>
</tbody>
</table>

* Significant at \(P < 0.05\)

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used

Spearman rank correlations (Rho) between functional measures and nonparetic leg time to develop torque \((s)\) \((N = 19)\)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>0.243</td>
<td>0.212</td>
<td>0.164</td>
<td>0.222</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>-0.289</td>
<td>-0.255</td>
<td>-0.437</td>
<td>-0.265</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>-0.054</td>
<td>-0.065</td>
<td>0.033</td>
<td>0.254</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.054</td>
<td>0.078</td>
<td>0.101</td>
<td>0.112</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.413</td>
<td>-0.280</td>
<td>-0.425</td>
<td>-0.104</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>-0.029</td>
<td>0.126</td>
<td>0.053</td>
<td>0.305</td>
</tr>
</tbody>
</table>

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used

Spearman rank correlations (Rho) between functional measures and nonparetic leg time to reduce torque \((s)\) \((N = 19)\)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>0.040</td>
<td>-0.112</td>
<td>-0.049</td>
<td>-0.054</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>-0.113</td>
<td>-0.239</td>
<td>-0.192</td>
<td>-0.188</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>0.185</td>
<td>0.057</td>
<td>0.223</td>
<td>0.297</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.052</td>
<td>-0.143</td>
<td>-0.180</td>
<td>-0.137</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.149</td>
<td>-0.243</td>
<td>-0.103</td>
<td>-0.079</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>-0.065</td>
<td>-0.162</td>
<td>-0.081</td>
<td>0.019</td>
</tr>
</tbody>
</table>

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used
Appendix XIX

Functional Correlations (concentric and eccentric torque)

The following correlations were performed for interest only and are not reported in the body of the thesis. Future research with a larger sample is needed to extend these preliminary results.

Spearman rank correlations \((Rho)\) between functional measures and paretic leg concentric torque \((N = 18)\)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>-0.761**</td>
<td>-0.709**</td>
<td>-0.704**</td>
<td>-0.531*</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>-0.771**</td>
<td>-0.761**</td>
<td>-0.719**</td>
<td>-0.562*</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>-0.845*</td>
<td>-0.655*</td>
<td>-0.808*</td>
<td>-0.633*</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.622**</td>
<td>-0.562*</td>
<td>-0.592**</td>
<td>-0.655**</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.569*</td>
<td>-0.467</td>
<td>-0.662**</td>
<td>-0.695**</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>-0.476*</td>
<td>-0.416</td>
<td>-0.471*</td>
<td>-0.575*</td>
</tr>
</tbody>
</table>

* Significant at \(P < 0.05\), ** Significant at \(P < 0.01\)

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used

Spearman rank correlations \((Rho)\) between functional measures and paretic leg eccentric torque \((N = 18)\)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>-0.507*</td>
<td>-0.391</td>
<td>-0.542**</td>
<td>-0.422</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>-0.734**</td>
<td>-0.573*</td>
<td>-0.776**</td>
<td>-0.521*</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>-0.670**</td>
<td>-0.567*</td>
<td>-0.770**</td>
<td>-0.607**</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.418</td>
<td>-0.408</td>
<td>-0.495*</td>
<td>-0.608**</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.309</td>
<td>-0.302</td>
<td>-0.407</td>
<td>-0.579**</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>-0.129</td>
<td>-0.044</td>
<td>-0.148</td>
<td>-0.263</td>
</tr>
</tbody>
</table>

* Significant at \(P < 0.05\), ** Significant at \(P < 0.01\)

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used
Spearman rank correlations (Rho) between functional measures and nonparetic leg concentric torque ($N = 18$)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>-0.544*</td>
<td>-0.406</td>
<td>-0.485*</td>
<td>-0.575*</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>-0.633**</td>
<td>-0.649**</td>
<td>-0.690**</td>
<td>-0.540*</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>-0.608**</td>
<td>-0.501*</td>
<td>-0.576*</td>
<td>-0.581*</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.344</td>
<td>-0.212</td>
<td>-0.322</td>
<td>-0.265</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.337</td>
<td>-0.284</td>
<td>-0.443</td>
<td>-0.474*</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>-0.259</td>
<td>0.125</td>
<td>-0.259</td>
<td>-0.354</td>
</tr>
</tbody>
</table>

* Significant at $P < 0.05$, ** Significant at $P < 0.01$

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used.

Spearman rank correlations (Rho) between functional measures and nonparetic leg eccentric torque ($N = 18$)

<table>
<thead>
<tr>
<th></th>
<th>Gait time fast-paced</th>
<th>Gait time self-paced</th>
<th>Time up stairs</th>
<th>Time down stairs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Plantarflexion</td>
<td>-0.478*</td>
<td>-0.317</td>
<td>-0.574*</td>
<td>-0.608**</td>
</tr>
<tr>
<td>Ankle Dorsiflexion</td>
<td>-0.453</td>
<td>-0.385</td>
<td>-0.503*</td>
<td>-0.385</td>
</tr>
<tr>
<td>Knee Flexion</td>
<td>-0.315</td>
<td>-0.234</td>
<td>-0.399</td>
<td>-0.525*</td>
</tr>
<tr>
<td>Knee Extension</td>
<td>-0.352</td>
<td>-0.307</td>
<td>-0.464</td>
<td>-0.430</td>
</tr>
<tr>
<td>Hip Extension</td>
<td>-0.115</td>
<td>-0.075</td>
<td>-0.262</td>
<td>-0.395</td>
</tr>
<tr>
<td>Hip Flexion</td>
<td>-0.079</td>
<td>-0.044</td>
<td>-0.234</td>
<td>-0.323</td>
</tr>
</tbody>
</table>

* Significant at $P < 0.05$, ** Significant at $P < 0.01$

Note: Variables did not satisfy assumption of normality and thus the nonparametric Spearman rank correlation was used.
### Appendix XX

**Key articles examining mechanical muscle properties in stroke (in chronological order)**

**Note:** Abbreviations: Upper extremity (UE), Lower extremity (LE), Range of motion (ROM), Maximal voluntary contraction (MVC), Electromyography (EMG), Paretic (P), Nonparetic (NP)

<table>
<thead>
<tr>
<th>Author</th>
<th>Subjects and extremity tested</th>
<th>Task/Analysis</th>
<th>Results/primary findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lum et al., 2004</td>
<td>14 chronic stroke Bilateral UE</td>
<td>Isometric and isokinetic concentric (30, 75, and 120°/s) MVC’s of P and NP elbow flexion and extension Passive torque during elbow flexion and extension cycles Measurements: Peak torque Passive joint stiffness EMG Isokinetic torque-angle curves</td>
<td>P elbow flexion torque decreased 44% as shortening velocity increased from 30 – 120°/s. NP elbow flexion torque decreased 9%. Thus the rate of decline for P arm torque was greater than for NP arm torque. This was not demonstrated for P elbow extension. Passive stiffness was not different between P and NP arms. With increasing movement velocity, both P flexion and extension torque-angle curves became progressively more different than NP torque-angle curves. No abnormal EMG activity was present during P elbow flexion. Increased antagonist activity was present during P elbow extension.</td>
</tr>
<tr>
<td>Ada et al., 2003</td>
<td>22 chronic stroke (4 with contracture and 8 without) 11 healthy controls P UE</td>
<td>Isometric MVC’s of P arm; elbow flexion and extension (every 20° from 0-120°) Measurements: Torque-angle curves Peak torque</td>
<td>Stroke torque-angle curves were different from controls (even after the curves were rescaled for peak torque and ROM). Stroke curves demonstrated relative weakness when the flexors and extensors were working at shortened lengths.</td>
</tr>
<tr>
<td>Author</td>
<td>Subjects and extremity tested</td>
<td>Task/Analysis</td>
<td>Results/primary findings</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-------------------------------</td>
<td>---------------</td>
<td>----------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Andrews and Bohannon, 2003</td>
<td>50 acute stroke</td>
<td>Isometric MVC's of P and NP arms and legs (7 joint actions) measured at admission and discharge to in-patient rehab</td>
<td>At discharge, muscle torque had increased in 7 of 7 joint actions on the P side and 4 of 7 on the NP side, however, all joint actions on both sides were still less than data from healthy individuals. Muscle torque is impaired bilaterally soon after stroke and improves with rehab.</td>
</tr>
<tr>
<td></td>
<td>Bilateral UE and LE</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Retrospective analysis)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Koo et al., 2003</td>
<td>10 chronic stroke 5 healthy controls</td>
<td>Isometric MVC's of P arm; elbow flexion and extension (every 15° from 15-120°)</td>
<td>Stroke curves demonstrated relative weakness when the flexors and extensors were working at shortened lengths (although flexion not significant). EMG of the control flexors (brachioradialis only) was significantly higher at short muscle lengths then at long lengths (brachioradialis receives more descending input then biceps because of it's mechanical advantage i.e. larger moment arm). EMG of the hemiparetic extensors was significantly lower at short muscle lengths then at long lengths (thus reduced central drive).</td>
</tr>
<tr>
<td></td>
<td>P UE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>McCrea et al., 2003</td>
<td>20 chronic stroke 10 healthy controls</td>
<td>Isometric MVC's of P and NP arms; 8 UE joint actions</td>
<td>Peak torque and time to generate and reduce torque impaired in the P arm (53%, 61%, and 22% respectively compared to control). Peak torque and time to generate torque also impaired in NP arm (15% and 22% respectively compared to control). Time to reduce torque not significantly different than control.</td>
</tr>
<tr>
<td>Author</td>
<td>Subjects and extremity tested</td>
<td>Task/Analysis</td>
<td>Results/primary findings</td>
</tr>
<tr>
<td>------------------------</td>
<td>-----------------------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| Chae et al., 2002      | 26 chronic stroke (22-85 years old, mild to severe impairment)                                  | Isometric MVC’s of the P and NP wrist extensors and flexors EMG activity Measurements: Delay of initiation and termination of muscle contraction Motor impairment Disability | The P arm had significantly longer initiation and termination of contraction times (approximately 140ms and 795ms longer, respectively).  
Delay in initiation and termination of muscle contraction relates with disability and UE motor impairment.  
Delay in initiation and termination of muscle contraction not affected by stroke type, level or side.                                                                 |
<table>
<thead>
<tr>
<th>Author</th>
<th>Subjects and extremity tested</th>
<th>Task/Analysis</th>
<th>Results/primary findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Harris et al., 2001</td>
<td>10 acute stroke</td>
<td>Isometric non-voluntary twitch tension and MVC’s (with twitch interpolation)</td>
<td>Stroke group nonvoluntary peak torque dropped from 7.6 to 5.9kg (No change for control group). Stroke group voluntary peak torque dropped from 12 to 8kg (No change for control group). Stroke quadriceps activation level was 59% at time 1 and 2. Control quadriceps activation level was 92% at time 1 and 2. The NP quadriceps muscle develops weakness during the first week after stroke, thus it is a secondary complication and may be related to inactivity and poor nutrition.</td>
</tr>
<tr>
<td></td>
<td>10 controls</td>
<td>of the NP knee extensors Measurements (tested within 48 hours post stroke and again one week later): Nonvoluntary peak torque Voluntary peak torque Activation level</td>
<td></td>
</tr>
<tr>
<td></td>
<td>NP LE</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Prospective analysis)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Newham and Hsiao, 2001</td>
<td>12 acute stroke</td>
<td>Isometric MVC’s of the P and NP knee extensors and flexors EMG, femoral nerve twitch superimposition Measurements (tested at 21 days, then again at 1, 2, 3, and 6 months post stroke): Peak torque Activation level (extension only) Spasticity co-activation index</td>
<td>P quads and hamstrings had lower peak torque than the NP side and controls until 3 months post stroke; no difference between NP and controls at any time point. The P quadriceps activation level improved to that of the NP side by one-month post stroke, however no further improvements were made (control activation levels were 93%, P and NP were 60-75%). Coactivation in antagonist the same for all groups. Thus strength is impaired only on the P side and improves over time. Activation failure present bilaterally and does not improve over time. Activation failure cannot be explained by cocontraction of the antagonist.</td>
</tr>
<tr>
<td></td>
<td>20 controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bilateral LE</td>
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<td>(Prospective analysis)</td>
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| Canning et al., 1999 | 10 subacute stroke 10 controls P UE (Prospective) | Isometric MVC's of the P elbow flexors and extensors Measurements (tested at 6 and 25 weeks post stroke): Peak torque Time to 90% peak torque                                                                 | At 6 weeks, the stroke group was weaker and took two to three times longer to generate torque than the controls.  
At 25 weeks, the stroke group was no longer different than the controls, thus recovery of these variables occurs.  
The relationship between peak torque and time to develop torque was mostly non-significant for both stroke and control groups, thus peak torque and the rate of torque development may be controlled by independent mechanisms. |
| Sunnerhagen et al., 1999 | 16 chronic stroke Bilateral LE | Isometric and isokinetic (60 and 180 degrees/s) MVC's of the P and NP knee extensors and flexors Measurements  
Peak torque  
Isometric and isokinetic endurance  
Fiber type classification (n = 9)  
Muscle area | Peak torque was lower on the P side (except knee flexion at 60 degrees/sec was the same).  
There were no differences in any of the endurance tests.  
Fiber type was the same for P and NP legs (the P side had a lower % of type 2 and higher % of type 1 fibers, but not significant).  
Capillary density was lower in the P leg compared to NP leg.  
Total muscle area was the same for P and NP legs (there was a sarcopenia effect, i.e. lower muscle and higher adipose tissue, in the P leg, but not significant). |
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<td>Nadeau et al., 1997</td>
<td>16 chronic stroke (mean age 44 years with near normal PROM)</td>
<td>Isometric, isotonic and isokinetic (concentric at 30, 90, and 180 degrees/s)</td>
<td>Torque at 30 degrees/s tended to be higher than 90 and 180. Torque-angle curve was curvilinear and peak torque occurred at the beginning of the movement (i.e. in dorsiflexion). Rate of torque development, peak torque and power moderately to highly interrelated. None of the muscle parameters were related to gait measures.</td>
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<td>P LE</td>
<td>MVC’s of P ankle plantar flexors Measurement: Peak torque, Isokinetic torque-angle curve, Power, Rate of torque development</td>
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<td>Davies et al., 1996</td>
<td>12 subacute to chronic stroke (mild spasticity) 12 controls</td>
<td>Isometric and isokinetic (concentric at 30, 100, 200, 300 degrees/s) MVC’s of P and NP knee flexors and extensors and passive isokinetic movement Antagonist EMG Measurement: Peak torque 10m walk Antagonist muscle activity</td>
<td>The P and NP legs generated relatively less torque than the control legs at slower angular velocities. The majority of P and NP legs were unable to generate torque at 300 degrees/s; controls achieved all speeds. There was no difference between the NP and control legs for isometric peak torque. Maximal P knee extension angular velocity correlated with gait speed. P leg generated more passive torque, however no EMG activity recorded (thus tone is mechanical not reflexive).</td>
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<td>Bilateral LE</td>
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| Sinkjaer and Magnussen, 1994 | 8 subacute to chronic stroke, 1 chronic tumor 8 controls           | The mechanical response to a quick stretch was measured during an isometric contraction (both active and with electrical stimulation) of the ankle plantarflexors | Passive stiffness (a property of the passive tissues) was increased 278% in the P leg, and 95% in the NP leg when compared to controls.  
Intrinsic stiffness (a property of the muscle fibers) did not differ for the P, NP, and control ankle plantarflexors.  
Reflex stiffness (stretch-reflex activity) did not differ for the P, NP, and control ankle plantarflexors.  
The P plantarflexion MVC was significantly reduced 77% and the NP plantarflexion MVC was significantly reduced 35% when compared with controls. However, average maximal amplitude of the muscle twitch did not differ between the P, NP, and control legs (thus contractile properties in P muscle preserved). |
|                        | Bilateral LE                                                       | **Measurements:** Non-reflex (passive and intrinsic) and reflex stiffness of ankle plantarflexors  
Peak torque – MVC  
Muscle twitch amplitude |                                                                                                                                                                                                                             |
| Bohannon and Walsh, 1992 | 14 acute to chronic stroke  
Bilateral LE                                                              | Isometric MVC’s of P and NP knee extensors  
**Measurements:**  
Pea-k torque  
Time to peak and 90% peak torque  
Gait speed | P leg time to peak torque and 90% peak torque was 13% and 24% slower than NP leg, respectively.  
Time to peak torque was highly variable with reliability at 0.67.  
Gait speed highly related to NP leg’s time to peak torque ($r = -0.74$) and over 74% of the variance in gait speed was explained by NP time to peak torque combined with P leg peak torque. |
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<td>Tsuji and Nakamura, 1987</td>
<td>14 subacute stroke 8 controls Bilateral LE</td>
<td>Isometric MVC’s of P knee extensors EMG <strong>Measurements:</strong> Tension lag time (from EMG onset to rise in tension) Contraction time (from rise in tension to max tension) Maximum force Rate of force development</td>
<td>Tension lag time, contraction time, and rate slower on the P side compared to NP and control legs (authors propose this is due to an increase in type 1 fibers and decreased firing rate). NP leg was not different than the control leg on any measures. Rate was correlated to maximum force in the P, NP, and control legs ($r = 0.85-0.96$).</td>
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<td>Bohannon, 1987</td>
<td>27 acute to subacute stroke Bilateral LE</td>
<td>Isokinetic MVC’s at 30, 60, 120, and 180 degrees/s <strong>Measurements:</strong> Peak torque</td>
<td>As angular velocity increased, mean peak torque decreased in both P and NP legs; the amount of decrease was the same in both legs.</td>
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<td>Colebach and Gandevia, 1989</td>
<td>16 subacute to chronic stroke (mild to severe impairment) 14 controls Bilateral UE</td>
<td>Isometric MVC’s of the P and NP arms; 12 joint actions <strong>Measurements:</strong> Peak torque</td>
<td>The P arm demonstrated more distal than proximal muscle weakness and the NP arm demonstrated more proximal than distal weakness when compared to controls (this affect was attributed to the corticospinal crossed (more distal input) and uncrossed (more proximal input) tracts.</td>
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<td>Nakamura et al., 1985</td>
<td>11 acute to subacute stroke Bilateral LE</td>
<td>Isometric (at 30, 60, and 90 degrees) and isokinetic (concentric at 30, 90, and 180 degrees/s) MVC's of the P and NP knee extensors Measurements: Peak torque Gait speed and rate</td>
<td>The P leg showed a trend for lower isokinetic torques at faster angular velocities when compared to the NP legs. Isometrically, lower torques were produced as the knee was brought closer to extension; this effect was more remarkable on the P side. Walking speed correlated with isokinetic torque at fast angular velocities but not with isometric torque for the P leg (Thus, speed and torque may be controlled by separate mechanisms).</td>
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