BIOMECHANICAL AND STRENGTH FACTORS CONTRIBUTING TO WALKING PERFORMANCE IN PERSONS WITH STROKE

by

Chin-Ah Maria Kim

B.Sc.(PT), McGill University, 1993

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE

REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE STUDIES

(School of Rehabilitation Sciences)

We accept this thesis as conforming to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

April 2001

Department of School of Rehabilitation Sciences

The University of British Columbia
Vancouver, Canada

Date April 24, 2001

ABSTRACT

Purpose: The most often stated goal by persons with stroke is improved walking function, therefore it is important to understand the biomechanical mechanisms of gait and the factors that may improve its performance. The purpose of this study was to 1) describe three-dimensional (3-D) gait patterns, 2) examine the relationship between gait speed and kinematic/kinetic variables and 3) determine the effect of maximal strength training on walking performance, in addition to muscle strength and health-related quality of life, in chronic stroke survivors.

Methods: Twenty individuals with chronic stroke (61.2 ± 8.4 years) were randomized into two six week (three times a week) training programs on the basis of age, sex, and time since onset of stroke (stratified randomization). An optoelectronic system and forceplates were used to generate 3-D kinematic and kinetic profiles during walking. The experimental group undertook maximal concentric isokinetic strength training of the paretic lower limb flexors and extensors with the use of the Kin-Com dynamometer. The control group received passive range of motion exercises using the same instrument. The Mann-Whitney *U* Test was used to compare the changes in score (posttraining-baseline) between the control and experimental groups: composite lower extremity strength score, walking speed (level and stair-walking) and health-related quality of life measure (SF-36). Correlations were used to assess the relationship between gait speed and biomechanical gait variables.

Results: Twenty-five kinematic and twenty-five kinetic patterns were identified across three joints (hip, knee, ankle), three planes and two sides among the 20 participants. Kinetic, more so than kinematic variables were correlated with gait speed, particularly in the sagittal and frontal planes for both the paretic and non-paretic sides. No differences in the change of walking speed or SF-36 scores was found between groups despite the trend ($p = 0.06$) towards greater strength improvement in the experimental group following training.

Conclusions: Strength training of the flexors/extensors of the paretic limb did not result in improvements in walking performance or health-related quality of life. The results of the biomechanical gait analysis suggest that in addition to these muscle groups, training programs that include hip abductors and the non-paretic limb may improve gait performance.

Table of Contents

List of Tables

List of Figures

Acknowledgements

I would like to thank my supervisor, Dr. Janice Eng, for her constant support and guidance throughout my graduate studies. Her constructive advice and encouragement made the process a pleasant learning experience. I would also like to thank my committee members, Dr. Donna McIntyre and Dr. Drew Dawson, for their invaluable input and support. Thanks are also extended to my external committee members, Dr. Carol Richards, Dr. Francine Malouin and Dr. Brad McFadyen, for their time and helpful comments on the thesis on such short notice.

I would like to express my appreciation to our lab engineer, Laurent Mingo, for his technical support in the lab as well as with my home PC. Thanks also to all the other "lab people" including our volunteers, for their assistance with data collection and the training programs. I am particularly grateful to my colleagues, Lisa Inkster and Beth Ott, for the many insightful late night discussions.

A special thanks to my family for their unwavering love and encouragement in whatever endeavors I pursue. I would like to thank Han for his love and for always supporting me (including my decision to move to the other side of the continent) and also for introducing me to the "world of computers". I am also grateful to all my friends for making the past few years in Vancouver a fun experience, and especially to my friend Elliott for helping me with "thesis errands" and for making sure that I got to my defense on time.

Finally, I would like to thank the University of BC Graduate Fellowship, the BC Medical Services Foundation, the Vancouver Unit of the Canadian Physiotherapy Neuroscience Division, the Jane Hudson Award, and especially my sister Yoon-Ah for the much needed financial support. And, last but not least, I will always appreciate the time and commitment of all the twenty participants in the study who made the completion of my thesis possible.

Chapter 1

Introduction

Stroke refers to neurological signs and symptoms resulting from any damage to brain or central nervous system structures caused by abnormalities of the blood supply. A universally accepted definition for stroke is "acute neurologic dysfunction of vascular origin…with symptoms and signs corresponding to the involvement of focal areas of the brain" (World Health Organization, 1989). Stroke is the third leading cause of death and a major cause of disability for those that survive (Mayo et al., 1994). It is estimated that 35 000 Canadians (Mayo et al., 1996) and 500 000 Americans (American Heart Association, 2000) experience a new stroke each year. The incidence increases dramatically with increasing age; it more than doubles in each successive decade for individuals over the age of 55 (American Heart Association, 2000). Improved survival rates of stroke victims and the recent growth in elderly population have led to an increase in the prevalence of stroke (Petrasovits and Nair, 1994), which is estimated to be 300 000 in Canada (Heart and Stroke Foundation, 2000) and 4 500 000 in the United States (American Heart Association, 2000). Consequently, the absolute number of persons living with stroke-related disability is increasing each year.

Disablement has been conceptualized by the World Health Organization (2000) in terms of impairments (problems in body function or structure), activity limitations (difficulties in executing tasks), and participation restrictions (problems with involvement in life situations). Impairments following stroke involve motor, sensory, visuoperceptual, and cognitive systems. Examples of common activity limitations include difficulties with tasks such as rising from a chair, level walking, stair walking, and other activities of daily living.

These impairments and activity limitations may result in significant participation restrictions for the person with stroke (Dombovy et al., 1986; Mayo et al., 1999).

Many studies have examined the course of recovery following stroke. Although the greatest recovery post-stroke occurs within the first three to six months of stroke (Dombovy et al., 1987; Wade & Langton Hewer, 1987), both neurological and functional recovery can occur over a more prolonged period of time (Aguilar, 1969; Andrews et al., 1981; Dombovy et al., 1987; Ferucci et al., 1993; Tangeman et al., 1990). As Speach & Dombovy (1995) pointed out, "functional recovery may occur over the span of a stroke patient's lifetime". More importantly, long term inactivity caused by the lack of functional independence may lead to further declines in function (Wade, 1992). These in turn contribute to a decrease in overall quality of life (Ahlsio et al., 1984). Clearly, it is important to determine the types of training that may enhance recovery and prevent further decline due to inactivity for chronic stroke survivors.

One of the first questions asked by someone after a stroke relates to walking (Mayo et al., 1991), and it has been reported to be the most frequently stated goal by individuals with stroke (Bohannon et al., 1988). Although most stroke survivors regain the ability to walk, only a small percentage (18-25%) attain a walking speed that is required for community activities (Mizrahi et al., 1982; Perry et al., 1995; Wade & Langton-Hewer, 1987). Consequently, understanding the problem of gait in order to address this problem effectively is important in the rehabilitation of stroke survivors. With recent developments in technology, modern gait analysis has provided insight into the biomechanical requirements in able-bodied gait. However, the biomechanical analyses of hemiparetic gait following stroke are still scarce. Thus, the first objective of the present study was to analyze the three-dimensional kinematic and kinetic characteristics of gait in individuals with stroke and to explore the relationship between these characteristics and gait performance. A greater understanding of the mechanisms underlying the gait patterns observed in persons with stroke would help guide assessment and intervention strategies that may be effective in retraining gait.

There are several treatment strategies that are employed by physical therapists to enhance walking ability in the rehabilitation of individuals with stroke. Shumway-Cook & Woollacott (1995) categorize therapeutic strategies into treatments aimed at the impairment level, the strategy level, and the functional level. Examples of treatments aimed at the impairment level are to strengthen weak muscle groups and to enhance or inhibit muscle activity by the use of sensory stimulation techniques. At the strategy level, the goal is to assist the individual develop strategies that are effective in meeting the postural demands of functional tasks (e.g. to facilitate toe clearance using an orthosis around the ankle during gait training). And at the functional level, the focus is on practicing the task in a variety of contexts (e.g. to increase adaptability of gait by experiencing walking on different surfaces) (Shumway-Cook & Woollacott, 1995).

At the impairment level, much emphasis has been directed towards developing therapeutic techniques to alter 'abnormal' muscle tone rather than to improve muscle strength because it was traditionally believed that strength training played only a minor part in neurorehabilitation (Bobath, 1978). Furthermore, resisted movement especially when performed by isolated muscle groups was believed to enhance spasticity and reinforce abnormal movement which was undesirable (Bobath, 1978). More recently, however, there has been increasing evidence that muscle weakness is more disabling than the changes in muscle tone (Burke, 1988; Landau, 1988) and that strength training can have positive effects on function in individuals with cerebral lesions (Damiano et al., 1995; Horvat, 1987; Sharp and Brouwer, 1997).

Muscle weakness, defined as the inability to generate normal levels of muscle force, may occur post-stroke as a result of alterations in the physiology of both the motoneuron and the muscle components of motor units. McComas et al. (1973) reported a reduction in the number of functioning motor units by half between the second and sixth month after stroke and attributed this change to the transsynaptic changes in motoneurons following degeneration of corticospinal fibres. A decrease in motor unit firing rates has also been suggested as a cause of muscle weakness (Rosenfalck & Andreassen, 1980). This observation was supported by Tang & Rymer (1981) who found that paretic muscles exhibited higher levels of EMG activity per unit of muscle force, implying that in order to achieve a given force level, additional motor units have to be recruited since the motor unit firing rate is reduced. Another possible cause of muscle weakness is muscle atrophy. There is evidence that type II or fast twitch fibers are selectively atrophied in individuals with hemiparesis (Dietz et al., 1986). In addition, it is well known that inactivity leads to secondary atrophy (MacDougall et al., 1980; Rutherford et al., 1990) and this may further contribute to muscle weakness post-stroke.

In healthy adults, muscle fiber hypertrophy and neural adaptations have been reported as a result of strength training (Hakkinen & Komi, 1983; Moritani & deVries, 1980; Rutherford & Jones, 1986). It seems reasonable to assume that strength training would also be beneficial in individuals with stroke in order to offset the physiological changes reported and prevent further decline due to disuse. The recovered strength in turn could enhance physical performance. In fact, previous studies on strength training in persons with stroke have shown promising results on functional performance both in the acute and chronic stages of the disease (Engardt et al., 1995; Karimi, 1996; Sharp & Brouwer, 1997; Wilder & Sykes, 1982). However, the benefit of isokinetic strength training alone over other factors of the training programs (e.g. maturation, attention from the therapist, etc) remains unclear. Thus, the second objective of this study was to examine the effectiveness of a strengthening program on muscle strength and walking function as well as health-related quality of life in chronic stroke survivors through a double-blind controlled trial.

The following questions were addressed in the present study:

1. Do the three-dimensional (sagittal, frontal and transverse planes) kinematic (joint angular displacement) and kinetic (joint moment and power) patterns of the hip, knee and ankle joints during gait demonstrate characteristic patterns in persons with chronic stroke?
2. Is gait speed related to the magnitudes of joint angle range, peak joint moment, peak joint power and work over the stride for the hip, knee and ankle joints in the three planes (sagittal, frontal and transverse) during the gait of persons with chronic stroke?
3. Can strength training change the strength of flexors and extensors of the hip, knee and ankle joints in persons with chronic stroke?
4. If so, what are the effects on walking performance (e.g. gait speed, stair climbing speed) and health-related quality of life as measured by the SF-36 in persons with chronic stroke?

References

Aguilar, J.J. (1969). Recovery of motor function after unilateral infarction of the basis pontis. American Journal of Physical Medicine, 48, 279.

Ahlsio, B., Britton, M., Murray, V., Theorell, T. (1984). Disablement and quality of life after stroke. Stroke, 15, 886-890.

American Heart Association (2000). 2001 Heart and Stroke Statistical Update. Dallas, Texas: American Heart Association.

Andrews, K., Brocklehurst, J.C., Richards, B., Laycock, P.J. (1981). The rate of recovery from stroke and its measurement. International Rehabilitation Medicine, 3, 155-161.

Bobath, B. (1978). Adult Hemiplegia: Evaluation and Treatment. London: Heinemann Medical Books Ltd.

Bohannon, R.W., Andrews, A.W., Smith, M.B. (1988). Rehabilitation goals of patients with hemiplegia. International Journal of Rehabilitation Research, 11, 181-183.

Burke, D. (1988). Spasticity as an adaptation to pyramidal tract injury. In S.G. Waxman (Ed.), Advances in Neurology, 47: Functional Recovery in Neurological Disease (pp. 401-423). New York, NY: Raven Press.

Damiano, D.L., Vaughan, C.L., Abel, M.F. (1995). Muscle response to heavy resistance exercise in children with spastic cerebral palsy. Developmental Medicine & Child Neurology, 37, 731-739.

Dietz, V., Ketelsen, U.P., Berger, W., Quintern, J. (1986). Motor unit involvement in spastic paresis: relationship between leg muscle activation and histochemistry. Journal of Neurological Sciences, 75, 89-103.

Dombovy, M.L., Sandok, B.A., Basford, J.R. (1986). Rehabilitation for stroke: A review. Stroke, 17, 363-369.

Dombovy, M.L., Basford, J.R., Whisnant, J.P., Bergstralh, E.J. (1987). Disability and use of rehabilitation services following stroke in Rochester, Minnesota, 1975-1979. Stroke, 18, 830-836.

Engardt, M., Knutsson, E., Jonsson, M., Sternhag, M. (1995). Dynamic muscle strength training in stroke patients: effects on knee extension torque, electromyography activity, and motor function. Archives of Physical Medicine & Rehabilitation, 76, 419-425.

Ferucci, L., Bandinelli, S., Guralnik, J.M., Lamponi, M., Bertini, C., Falchini, M., Baroni, A. (1993). Recovery of functional status after stroke. A postrehabilitation follow-up study. Stroke, 24, 200-205.

Hakkinen, K., Komi, P.V. (1983). Electromyographic changes during strength training and detraining. Medicine & Science in Sports & Exercise, 15, 455-460.

Heart and Stroke Foundation of Canada (2000). Available at: http://www.na.heartandstroke.ca/cgibin/English/Catalog/Public/bR.cgi?320280:::Prevalence%20of%20stroke:::15335083 0077:::300001:310060:320280.

Horvat, M. (1987). Effects of a progressive resistance training program on an individual with spastic cerebral palsy. American Corrective Therapy Journal, 41, 7-11.

Karimi, H. (1996). Isokinetic strength training and its effect on the biomechanics of gait in subjects with hemiparesis as a result of stroke (PhD Dissertation). Kingston (On): Queen's University.

Landau, W.M. (1988). Parables of palsy, pills, and PT pedagogy: a spastic dialectic. Neurology, 38, 1496-1499.

MacDougall, J.D., Elder, G.C.B., Sale, D.G., Moroz, J.R., Sutton, J.R. (1980). Effects of strength training and immobilisation on human muscle fibres. European Journal of Applied Physiology & Occupational Physiology, 43, 25-34.

Mayo, N.E., Chockalingam, A., Reeder, B.A., Phillips, S. (1994). Surveillance for stroke in Canada. Health Reports, 6, 62-72.

Mayo, N.E., Korner-Bitensky, N.A., Becker, R. (1991). Recovery time of independent function post-stroke. American Journal of Physical Medicine & Rehabilitation, 70, 5-12.

Mayo, N.E., Neville, D., Kirkland, S., Ostbye, T., Mustard, C.A., Reeder, B., Joffres, M., Brauer, G., Levy, A.R. (1996). Hospitalization and case-fatality rates for stroke in Canada from 1982 through 1991: The Canadian Collaborative Study Group of Stroke Hospitalizations. Stroke, 27, 1215-1220.

Mayo, N.E., Wood-Dauphinee, S., Ahmed, S., Gordon, C., Higgins, J., McEwen, S., Salbach, N. (1999). Disablement following stroke. Disability & Rehabilitation, 21, 258-268.

McComas, A.J. (1973). Functional changes in motoneurons of hemiparetic muscles. Journal of Neurology, Neurosurgery, and Psychiatry, 36, 183-193.

Mizrahi, J., Susak, Z., Heller, L., Najenson, T. (1982). Variation of time-distance parameters of the stride as related to clinical gait improvement in hemiplegics. Scandinavian Journal of Rehabilitation Medicine, 14, 133-140.

Moritani, T., deVries, H.A. (1980). Potential for gross muscle hypertrophy in older men. Journal of Gerontology, 35, 672-682.

Perry, J., Garrett, M., Gronley, J.K., Mulroy, S.J. (1995). Classification of walking handicap in the stroke population. Stroke, 26, 982-989.

Petrasovits, A., Nair, C. (1994). Epidemiology of stroke in Canada. Health Reports, 6, 39-44.

Rosenfalck, A., Andreassen, S. (1980). Impaired regulation of force and firing patter of single motor units in patients with spasticity. Journal of Neurology, Neurosurgery & Psychiatry, 43, 907-916.

Rutherford, O.M., Jones, D. (1986). The role of learning and coordination in strength training. European Journal of Applied Physiology & Occupational Physiology, 55, 100-105.

Rutherford, O.M., Jones, D.A., Round, J.M. (1990). Long-lasting unilateral muscle wasting and weakness following injury and immobilization. Scandinavian Journal of Rehabilitation Medicine, 22, 33-37.

Sharp, S.A., Brouwer, B.J. (1997). Isokinetic strength training of the hemiparetic knee: Effects on function and spasticity. Archives of Physical Medicine & Rehabilitation, 78, 1231-1236.

Shumway-Cook, A., Woollacott, M. (1995). Motor Control: Theory and Practical Applications. Baltimore: Williams & Wilkins.

Speach, D.P., Dombovy, M.L. (1995). Recovery from stroke: rehabilitation. Bailliere's Clinical Neurology, 4, 317-338.

Tang, A., Rymer, W.Z. (1981). Abnormal force-EMG relations in paretic limbs of hemiparetic human subjects. Journal of Neurology, Neurosurgery & Psychiatry, 44, 690-698.

Tangeman, P.T., Bonaitis, D.A., Williams, A.K. (1990). Rehabilitation and chronic stroke patients: changes in functional performances. Archives of Physical Medicine & Rehabilitation, 71, 876-880.

Wade, D.T. (1992). Stroke: rehabilitation and long-term care. The Lancet, 339, 791-793.

Wade, D.T., Langton-Hewer, R. (1987). Functional abilities after stroke: measurement, natural history, and prognosis. Journal of Neurology, Neurosurgery & Psychiatry, 50, 177-182.

Wilder, P.A., Sykes, J. (1982). Using an isokinetic exercise machine to improve the gait pattern in a hemiplegic patient. Physical Therapy, 62, 1291-1295.

World Health Organization (1989). Stroke-1989. Recommendations on stroke prevention, diagnosis, and therapy: report of the WHO Task Force on Stroke and other Cerebrovascular Disorders. Stroke, 20, 1407-1431.

World Health Organization (2000). International Classification of Functioning, Disability and Health: Prefinal Draft December 2000. Retrieved February 20, 2000 from the World Wide Web: http://www.who.int/icidh

Chapter 2: ABSTRACT

Inter-subject Comparison of Three-dimensional Kinetic and Kinematic Gait Analysis in Persons with Stroke

Purpose: Kinematic and kinetic gait analysis in persons with stroke can help understand the mechanisms underlying the common gait deviations observed in individuals with stroke. In addition, identifying the kinematic and kinetic variables that are related to speed may provide further insight into the gait performance of these individuals. The purpose of this study was to 1) identify predominant kinematic and kinetic gait patterns and 2) examine the relationship between gait speed and magnitudes of the three-dimensional (3-D) joint excursions, moments and powers.

Methods: Twenty individuals with chronic stroke (age: 61.2 ± 8.4 years) were instructed to walk at their self-selected speed. Movement of the body segments, tracked with an optoelectronic system, was combined with ground reaction forces to generate 3-D kinematic and kinetic lower extremity joint profiles. Pearson product correlations were used to assess the relationship between gait speed and selected kinematic and kinetic variables.

Results: Twenty-five kinematic and twenty-five kinetic patterns were identified across three joints (hip, knee, ankle), three planes and two sides among the 20 participants. Kinetic variables, more so than kinematic variables, were highly (r>0.70) correlated with gait speed, particularly the bilateral hip, knee and ankle power, the paretic hip flexor moment and the non-paretic ankle plantarflexor moment variables in the sagittal plane, and bilateral hip power variables in the frontal plane. For some of these variables, correlations were higher for the non-paretic side.

Conclusions: Inter-subject variability is evident in the 3-D kinematic and kinetic gait profiles in persons with stroke. A significant relationship exists between gait speed and variables in

all planes of both the paretic and non-paretic limbs suggesting that intervention aimed at improving function of both limbs and including non-sagittal muscle groups such as hip abductors may improve gait performance.

2.1 INTRODUCTION

The most often stated goal by persons with stroke is improved walking function (Bohannon et al., 1991), therefore retraining of walking is a major goal in the rehabilitation of persons with stroke. Although 65% to 85% of stroke survivors learn to walk independently by six months post stroke (Wade et al., 1987), only 18-25% attain normal walking speed (Mizrahi et al., 1982; Perry et al., 1995; Wade & Langton-Hewer, 1987). As a result, the person's ability to participate in community activities is compromised (Gresham et al., 1979). Stroke results in sensorimotor impairments affecting mainly the side of the body contralateral to the lesion although some minor effects have been recognized on the ipsilateral limbs (Bohannon & Andrews, 1995; Desrosiers et al., 1996; Sjostrom et al., 1980). The gait pattern of persons with stroke, known as hemiparetic gait, is a result of a number of factors. Possible factors contributing to the gait abnormalities observed in individuals with stroke include muscle weakness (Bohannon, 1986; Nadeau et al., 1999; Nakamura et al., 1985), abnormal muscle activation patterns (Knutsson & Richards, 1979), and spasticity, in addition to disturbed sensation, impaired postural control, disturbed cognition, or neglect.

Quantitative gait analysis using computer-assisted motion and force analyses is recognized as a useful tool in the assessment of gait and has been widely used in the analysis of able-bodied gait. However, data on hemiparetic gait following stroke are still scarce especially in regards to the analysis of kinetics (e.g. mechanical joint forces and powers). Understanding the mechanisms underlying the gait patterns seen in persons with stroke would help guide assessment and treatment strategies as well as monitor progress when retraining gait. As the disturbed motor control following stroke depends on the size, location and severity of the lesion along with the manner of compensation employed by each person (Perry, 1969), one might expect to see many variations in the gait patterns displayed by individuals with stroke.

2.1.1 Temporal and 2-D kinematic characteristics of gait in individuals with stroke

Several investigations have documented differences in the temporal, kinematic and kinetic variables between able-bodied and hemiparetic gait. The majority of these studies have been confined to two-dimensional analyses and limited to the assessment of the paretic limb (i.e. the side contralateral to the lesion). Relative to age-matched normal values, hemiparetic gait is generally characterized by a decrease in self-selected speed, cadence, and stride length, and an increase in stance phase duration bilaterally accompanied by an increase in double support duration (Brandstater et al., 1983; Turnbull et al., 1995; von Schroeder et al., 1995).

Two-dimensional kinematic analysis has revealed a general decrease in sagittal joint angles of the paretic lower extremity in individuals with stroke. The following characteristics have been reported relative to values of healthy adults walking at self-selected speeds: 1) at initial contact, there is decreased hip flexion, increased or decreased knee flexion and increased ankle plantarflexion, 2) at toe-off, there is increased hip flexion, decreased knee flexion and decreased ankle plantarflexion, 3) at mid-swing, there is decreased hip flexion, knee flexion and ankle dorsiflexion (Burdett et al., 1988; Lehman et al., 1987; Olney et al., 1991). Inter-subject variability in the sagittal kinematic patterns has been identified by Kramers de Quervain et al. (1996) who described four coupling patterns of knee and ankle motion during the stance phase of hemiparetic gait: 1) the extension thrust pattern (characterized by an extension thrust of the knee with increased plantarflexion at the ankle), 2) the stiff-knee pattern (where the knee remained stiff in flexion and the ankle neutral or in slight plantarflexion), 3) the buckling-knee pattern (characterized by increased knee flexion and dorsiflexion), and 4) the normal knee pattern. On the non-paretic side, Olney et al. (1991) found that joint angles were greater than that of the paretic side, however, values were reduced when compared to reported values of able-bodied persons walking at slow speeds.

2.1.2 2-D kinetic analysis of gait in individuals with stroke

In addition to temporal and kinematic descriptions, the study of the kinetics of gait (joint moments, power and work) can provide further insight into the factors underlying the deficits of hemiparetic gait. Moments of force are the net result of muscular, ligament and friction forces acting to alter the angular rotation of a joint (Winter, 1991). Mechanical power is the product of the joint moment of force and joint angular velocity, and the work performed by a muscle group is calculated by mathematic integration of the power curve. These analyses help understand the role of muscles that produce the movement patterns we observe, i.e. they help determine the motor patterns that are the cause of observed pathological gait profiles (Winter, 1981). Thus for the clinician, kinetic information is of particular interest.

However, kinetic analyses of hemiparetic gait have been scarce. Lehman et al. (1987) compared the knee moments of persons with stroke to those of healthy individuals walking at comparable slow speeds and reported a greater knee flexor moment (although not statistically significant) on the paretic side in six out of seven participants with stroke during gait. The authors (Lehman et al., 1987) suggested that this flexor moment countered the anterior orientation of the vertical force line in relation to the knee joint center. Olney et al. (1991), one of the few groups to assess both the paretic and non-paretic limbs, reported that in the sagittal plane, hemiparetic gait power profiles were similar in shape to normal profiles on both sides but with lower amplitudes compared to reported values of healthy young subjects walking at slow speeds. They found that 40% of the total positive work in the sagittal plane was performed by the paretic limb, thereby challenging the notion that improvement after stroke is only a result of increased use of the non-paretic side.

2.1.3 3-D kinematic and kinetic analysis of gait in individuals with stroke

In normal gait, three-dimensional (3-D) gait analysis has proven to be useful in understanding the mechanisms of gait as a large amount of work is also done in planes other than the sagittal plane. Mechanical work analysis has revealed that although the major portion of work during walking in able-bodied persons was performed in the sagittal plane (82% of the total work), a substantial amount of work is done in the frontal plane especially by the hip (10% of the total work) (Eng & Winter, 1995). It is possible that persons with stroke who often present with generalized weakness and poor balance could show a relative increase in work in planes other than the sagittal when compared to healthy adults.

Few studies have included 3-D analyses of hemiparetic gait and among these, only two studies have explored the kinetic aspect of the analysis. Kuan et al. (1999) reported the following kinematic characteristics in the gait of persons with stroke on their paretic side relative to values of age-matched healthy persons walking at their self-selected speed: 1) an increase in hip abduction and external rotation throughout the gait cycle, 2) a slight increase in knee valgus in mid-stance and reduced knee varus in swing, and 3) increased ankle inversion and outward foot progression angle (abduction) throughout the gait cycle. In addition, Kerrigan et al. (2000) quantified hip hiking and circumduction and found that individuals with stroke had a tendency to elevate the pelvis on the paretic side during swing resulting in increased abduction of the non-paretic hip. Individuals with stroke also circumducted the paretic limb by increasing the frontal thigh angle during swing when compared to healthy controls walking at their self-selected speed (Kerrigan et al., 2000).

To date, no study has extended kinetic analysis of hemiparetic gait in the frontal and transverse planes with the exception of Voigt & Sinkjaer (2000) who explored the frontal hip joint in persons with foot-drop following stroke and Kerrigan et al. (1999) who examined only the non-paretic limb. Kerrigan et al. (1999) found that the moment and power patterns of the non-paretic limb were similar in shape to normal profiles and the peak torques and powers were either reduced or the same compared with controls walking at their self-selected speeds. Voigt & Sinkjaer (2000) found the paretic and non-paretic hips to have similar profiles in the frontal plane, but the paretic hip generated less work. Clearly, the 3-D biomechanical analysis of gait in individuals with stroke warrants further investigation.

2.1.4 Relationship between walking speed and gait variables

Walking speed has been found to influence the patterns and magnitudes of gait variables such as joint angles, maximum joint moments and powers in able-bodied gait (Andriacchi & Strickland, 1985; Chen et al., 1997; Vardaxis et al., 1998). Similarly, in hemiparetic gait, the degree of the kinematic and kinetic deficits were found to relate to gait speed for both limbs (Knutsson, 1981; Kramers de Quervain et al., 1996; Lehman et al., 1987; Olney et al., 1991). Thus, in order to investigate the kinematic and kinetic factors contributing to gait performance, Olney et al. (1994) correlated sagittal gait variables with gait speed in individuals with stroke and found that the most correlated variables included maximum hip flexion moment, maximum hip and ankle power generation, and minimum (i.e. peak absorption) knee power on the paretic side along with maximum ankle power generation on the non-paretic side. No information is available concerning the association between the frontal or transverse gait variables and gait speed.

A major limitation of previous studies (Kerrigan et al., 1999; Kuan et al., 1999; Olney et al., 1991) of kinematic and kinetic gait profiles in individuals with stroke is that they have interpreted the findings based on a single average profile of all subjects. Following stroke, the type of disturbed motor control may vary, therefore one might expect to see variations in strategies used for walking. For example, Knutsson & Richards (1979) found three types of muscle activation patterns in hemiparetic gait. Information on the different strategies employed by each person is lost when presenting average profiles of all subjects. The lack of information on the 3-D kinematic and kinetic characteristics, particularly of inter-subject strategies, led to the following purposes: 1) identify predominant gait patterns in persons with stroke by quantifying the 3-D kinematic and kinetic characteristics and 2) examine the relationship between gait speed and magnitudes of the 3-D joint excursions, moments and powers.

2.2 METHODS

2.2.1 Participants

The study was approved by the local university's Research Ethics Board and the hospital Research Advisory and Review Committee. Twenty community-dwelling stroke survivors, who had residual unilateral weakness, were recruited on a volunteer basis.

To be included in the study, participants had to be able to walk independently without orthoses for a minimum of 40 metres (with rest intervals) with or without assistive device, have a history of a single cerebrovascular accident of at least six months prior to participating in the study, and be able to follow instructions. Participants were excluded if they had significant musculo-skeletal problems due to conditions other than stroke. Informed consent (Appendix I) was obtained from each participant. Also, a letter was sent to each participant's physician (Appendix II), who was later contacted by phone to ensure the inclusion and exclusion criteria were met prior to the beginning of the study.

Demographic data collected from all participants included age, sex, time since the onset of stroke, the paretic side, degree of resistance to passive movement in the knee extensors and ankle plantarflexors, stage level of the paretic lower extremity and foot on the Chedoke-McMaster Stroke Assessment (Gowland et al., 1995) (Appendix III), use of orthoses and mobility aids. The characteristics of the participants are summarized in Table 1. Resistance to passive movement was evaluated while passively flexing and extending the limb as described by Ashworth (1964) while participants were in a supine position and graded on an ordinal scale from zero to five based on the Modified Ashworth Scale (Bohannon & Smith, 1987) (Appendix IV).

2.2.2 Instrumentation & Procedure

Participants were asked to walk wearing their shoes without the use of an orthosis at their "most comfortable speed" (i.e. self-selected speed) using their usual assistive device along an 8-meter walkway over three force plates. Participants were instructed to walk straight ahead towards the camera and avoid targeting the force plates (Appendix V). Five "appropriate" trials were collected for each limb. A trial was considered "appropriate" if one foot landed on a force plate in its entirety. Rest periods were provided as deemed necessary by the participants.

An optoelectronic sensor (Optotrak, Northern Digital) was used to track the infrared emitting diodes (IREDs) attached to the participant's lower body. In this camera set-up, the error of locating the coordinates of an IRED in space was 0.9 mm in the anterior/posterior (x) direction, 0.45 mm in the up/down (y) direction and 0.45 mm in the medial/lateral (z) direction. IREDs were attached to the participant's pelvis and lower extremities to generate a 3-D model of the lower body (Appendix VI). Three non-collinear markers were used to track each body segment and with the use of custom software, euler angles (an x-y-z rotation sequence) were then calculated. The transformation matrix between the external marker reference system and the principal axes of each body segment was defined by the use of a standing posture of the participant. Joint angles were described as the motion of the distal segment relative to the proximal segment. The 3-D location of the joints were calculated as in Eng & Winter (1995). Data was sampled at 60 Hz and filtered using a fourth-order Butterworth, zero-lag, low-pass cut-off at 6 Hz. Temporal-distance variables (e.g. gait velocity, cadence, stride length) were determined using the distance covered by the markers and the corresponding elapsed time during each gait cycle.

Ground reaction forces were collected using three Bertec force plates embedded in the ground along the plane of progression. Forceplate data were sampled at 600 Hz. A 3-D inverse dynamic solution (Bresler and Frankel, 1950) was performed beginning with the most distal joint to estimate the forces and moments at the joints. Mechanical power of the three joints (hip, knee, and ankle) was calculated from the dot product of the joint angular velocities and joint moments of force transformed in the global reference system (Robertson & Winter, 1980). Power bursts were labeled according to Eng & Winter (1995); the first letter refers to the joint, the number refers to the sequence of the bursts in that joint and the second letter denotes the plane (e.g. A1-S refers to the first ankle burst in the sagittal plane). Work was calculated by the time integration of the power curves over one stride for each power phase. The total muscle work over the stride was the sum of the positive work and the absolute value of the negative work.

Joint angle, moment, and power profiles were normalized on a time base of 60% stance, 40% swing of the gait cycle and were ensemble-averaged across trials for each participant (for each of the three joints of the two limbs). Ensemble-averaging across participants was not performed in order to identify differences in motor patterns.

2.2.3 Data Analysis

Descriptive statistics (mean, range, standard deviation) were calculated for participants' characteristics, temporal-distance variables, joint angle ranges, and maximum joint moments and powers. The frequency of occurrence of the kinematic and kinetic patterns in the three joints of the lower limbs were tabulated. Patterns were determined based on the shape and direction of the curves regardless of magnitudes. To determine the influence of gait speed on patterns, participants were divided into ten "slow" and ten "fast" walkers with the median value separating the two groups. Then the frequency of patterns were compared between the two groups. The correlation between gait variables and gait speed was established using Pearson product moment correlation (r). The significance level selected for this study was $p<0.05$, two-tailed test.

2.3 RESULTS

The mean age of the participants (61 years) (Table 1) was lower than that reported for the general population of individuals with stroke (73 years) (Brown et al., 1996). However, this may be due to the inclusion criteria that all participants had to be able to walk independently. On the American Heart Association Stroke Functional Classification Level (Kelly-Hayes et al., 1998), the stroke severity of the participants in this study would be considered I to II. Temporal and distance characteristics of gait for the groups are presented in Table 2. The mean gait speed of the participants in this study (0.45 m/sec) was much slower than the speed of healthy elderly participants (0.9-1.5 m/sec) in other studies (Bendall et al., 1989; Himann et al., 1988; Murray et al., 1969) and corresponds approximately to the mean velocity of stroke survivors in the medium speed group (mean 0.41 m/sec) in Olney et al.'s (1991) study. The median gait speed value in the present study was 0.41 which was used to separate the participants into "slow" (mean 0.3 ± 0.07m/sec) and "fast" (0.6 ± 0.27 m/sec) walking groups. Only two participants' self-selected speed exceeded 1 m/sec. This reduced speed was due to a decrease in both cadence (74 steps/min) and stride length (0.73 m) when compared with values reported for the healthy elderly (cadence: 100-105 steps/min, stride length: 1-1.4 m) (Himann et al., 1988). Relative to the non-paretic side, there was a decrease in stance time and an increase in swing time accompanied by a greater step length on the paretic side.

Several characteristic kinematic and kinetic patterns were identified. The seven participants who used a cane during the gait assessment were evenly distributed across the two groups (i.e. "slow" and "fast" walkers), cane use did not show any trends in patterns observed. Kinetic analysis was done with 19 participants' data for each limb due to

Table 1: Characteristics of the participants (N = 20)

	Mean	SD	Range
Age (yr)	61.15	8.38	52-82
Mass (kg)	76.24	13.8	52-99.4
Height (cm)	170.64	12.09	143-188
Time since stroke (yr)	4.03	2.56	1.5-10

Type of stroke (Ischemic/Hemorrhagic/Unknown)	11/7/2
Gender (M/F)	14/6
Involved side (Right/Left)	11/9
Dominance (Right/Left)	17/3
Mobility aid (Cane/None)	7/13
Chedoke-McMaster (stage range)	3-6
Modified Ashworth Scale (grade range)	0-2

Table 2: Mean, standard deviation and range of temporal-distance characteristics of gait (N = 20).

Variable	Mean	SD	Range
Speed (m/sec)	0.45	0.25	0.20-1.10
Cadence (steps/min)	74.2	20.6	38.2-111.2
Stride Length (m)	0.71	0.25	0.34-1.30

	Paretic		Non-paretic	
	Mean	SD	Mean	SD
Stance time (sec)	1.13	0.44	1.33	0.51
Swing time (sec)	0.65	0.16	0.41	0.1
Step length (m)	0.37	0.15	0.34	0.12

insufficient forceplate trials for one participant. In the following sections, descriptions of hemiparetic gait characteristics are compared with normal 3-D profiles described in the literature for healthy young adults (Apkarian et al., 1989; Eng & Winter, 1995; Winter, 1991; Winter et al., 1995). Patterns are labeled as "N", "A" or "B" for joint angle and moment patterns. The pattern that most closely resembled the normal pattern described in the literature was labeled as pattern N, patterns A and B are deviations from the normal pattern with A being the more frequently occurring pattern. Power profiles are described in relation to their association with each moment pattern.

2.3.1 Kinematic characteristics

Kinematic patterns were generally more consistent across participants (i.e. low number of identified patterns) in the sagittal plane compared to frontal and transverse planes and more consistent proximally at the hip joint than the knee and ankle joints. The sagittal joint excursion over the stride was greater in the non-paretic limb by 20%, 48% and 39% for the hip, knee and ankle joints respectively, compared to the paretic limb (Table 3). This trend was not evident in the other planes. In contrast, in the frontal ankle joint profiles, the paretic limb showed a greater angle range than the non-paretic limb (5° difference corresponding to a 65% increase). In addition, the frontal knee and transverse hip ranges were similar for the paretic and non-paretic limbs.

As expected, the joint excursion was greatest in the sagittal plane, given that the goal of gait is to move the body forward in the plane of progression. Generally, the joint excursion in the transverse plane was greater than in the frontal plane for each joint. Among joints, there was a tendency for the hip joint to show the greatest range in all three planes except in

Table 3: Mean ± standard deviation of the joint angle ranges* (degrees) over the stride (N = 20).

Plane	Joint	Paretic	Non-paretic
Sagittal	Hip	34.6 ± 19.1	41.1 ± 8.1
	Knee	33.7 ± 21.3	49.8 ± 8.0
	Ankle	17.7 ± 5.1	24.6 ± 8.0
Frontal	Hip	10.7 ± 5.0	12.0 ± 4.8
	Knee	7.8 ± 4.5	8.0 ± 3.9
	Ankle	13.6 ± 6.2	9.0 ± 3.0
Transverse	Hip	17.8 ± 9.5	17.3 ± 8.0
	Knee	10.0 ± 6.2	12.9 ± 6.8
	Ankle	11.2 ± 5.4	13.1 ± 4.8

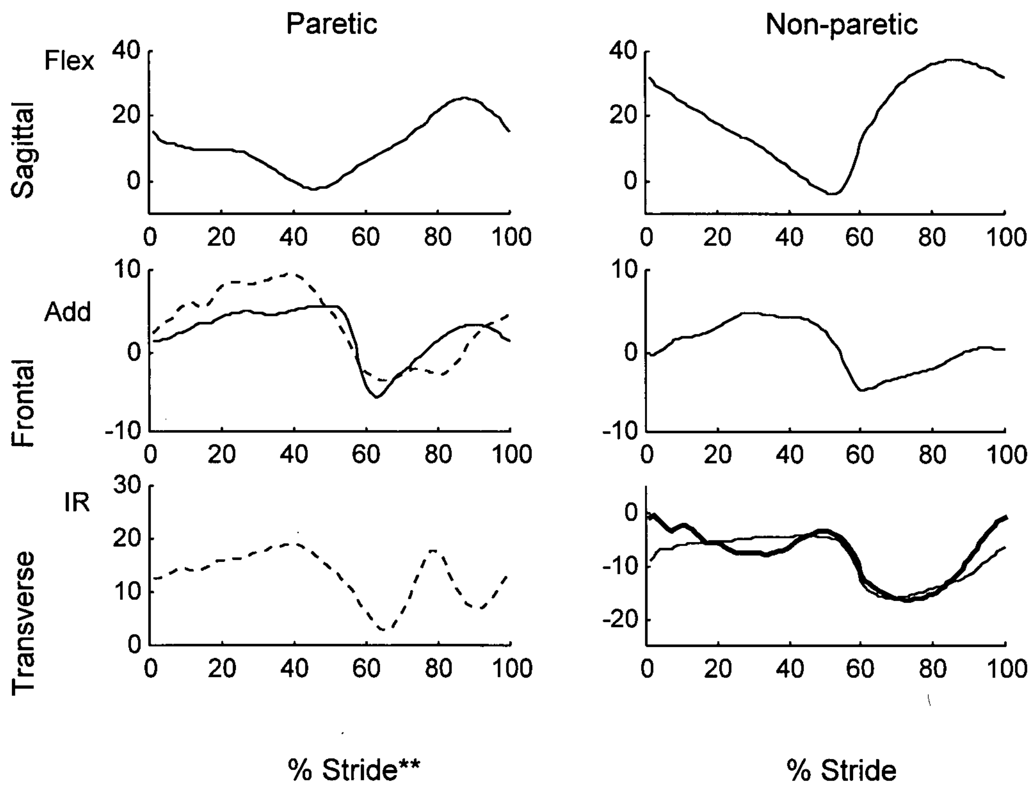
* Joint angle range = maximum - minimum joint angle values

the sagittal plane for the non-paretic side where the knee demonstrated a greater range (Table 3, Figures 1-3).

2.3.1.1 Hip

Representative time-normalized joint angle profiles of both the paretic and non-paretic hips in all three planes are illustrated in Figure 1. The profiles are intra-subject averages (n = 5 trials) which best represented the predominant patterns observed across the 20 participants. In the sagittal plane, both the paretic and non-paretic limbs (20/20 participants) showed a similar joint angle pattern (pattern N) which was similar to the pattern found in able-bodied walking (Winter, 1991): a) During weight acceptance and mid-stance, the hip extended (27° on the paretic side and 36° on the non-paretic side) as the body moved progressively forward of the new leading limb, b) at 45 to 55% of stride, peak extension was reached which coincided with the time when the contralateral foot contacted the ground, c) during propulsion, the hip began to flex in preparation for swing (35° on the paretic side and 41° on the non-paretic side) and d) maximum flexion was reached in mid swing to clear the foot from the ground. Note, although the magnitudes are different between the paretic and non-paretic limbs (Table 3), they are both labeled as "pattern N" since the shape and direction of movement are similar.

In the frontal plane, two patterns were identified for the paretic hip and one for the non-paretic side. Pattern N, identified in both the paretic (8/20) and non-paretic (16/20) hips, resembled the pattern found in able-bodied gait (Kuan et al., 1999; Winter et al., 1995): a) During weight acceptance, the hip adducted slightly (5-6°) as the trunk/pelvis transferred laterally over the leading limb, b) at 40 to 50% of stride, the hip abducted (10-11°) as the



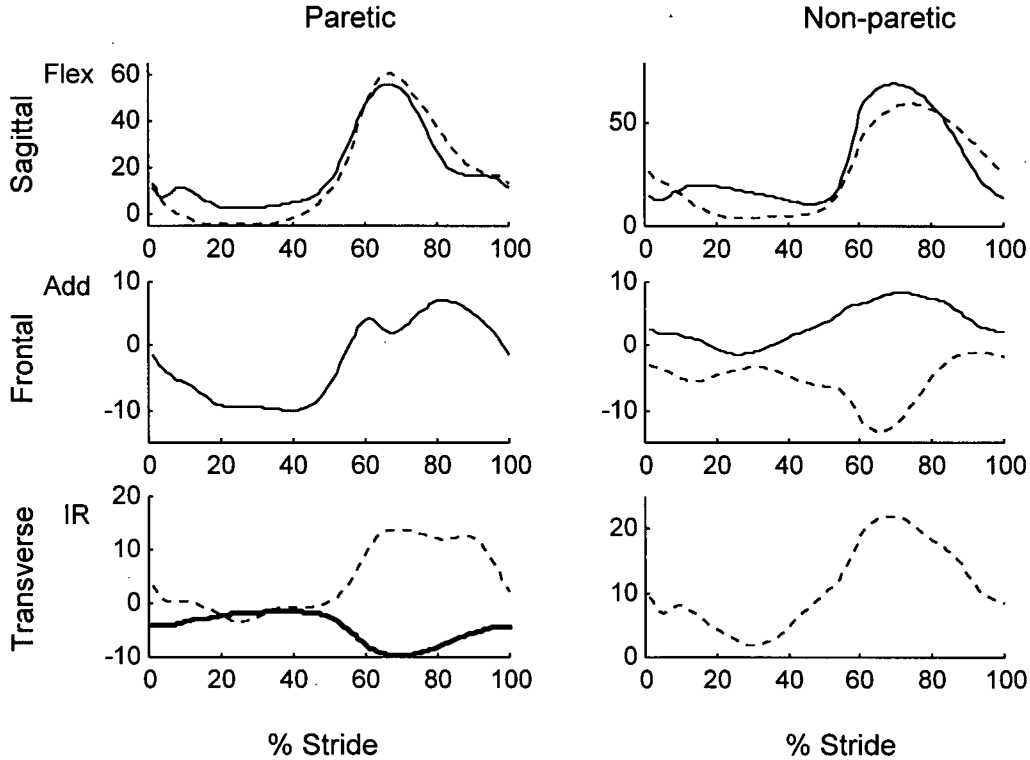
Pattern frequency:

Paretic side					Non-paretic side				
Plane	Pattern	Total*	Slow†	Fast‡	Plane	Pattern	Total*	Slow†	Fast‡
Sagittal	N	20	10	10	Sagittal	N	20	10	10
Frontal	N	8	5	3	Frontal	N	16	7	9
	A	9	3	6		other	4	3	1
	other	3	2	1	Transverse	N	8	3	5
Transverse	A	15	7	8		B	10	4	4
	other	5	3	2		other	2	3	1

Figure 1. Representative hip joint angle (degrees) profiles in the sagittal, frontal, and transverse planes. The profiles are intrasubject averages (n = 5 trials) which best represent the predominant patterns observed across the 20 participants (note: illustrated profiles do not represent profiles from the same participant). Patterns were determined based on the shape of the curves regardless of magnitudes and identified as N (i.e. closest to "normal" patterns), A, or B; each letter representing one pattern found in either or both the paretic and non-paretic sides. Solid line: pattern N; dashed line: pattern A; dark solid line: pattern B. The table above illustrates the occurring frequency (i.e. number of subjects exhibiting the pattern) of each pattern.

Abbreviations: Flexion (Flex), Adduction (Add), Internal Rotation (IR)

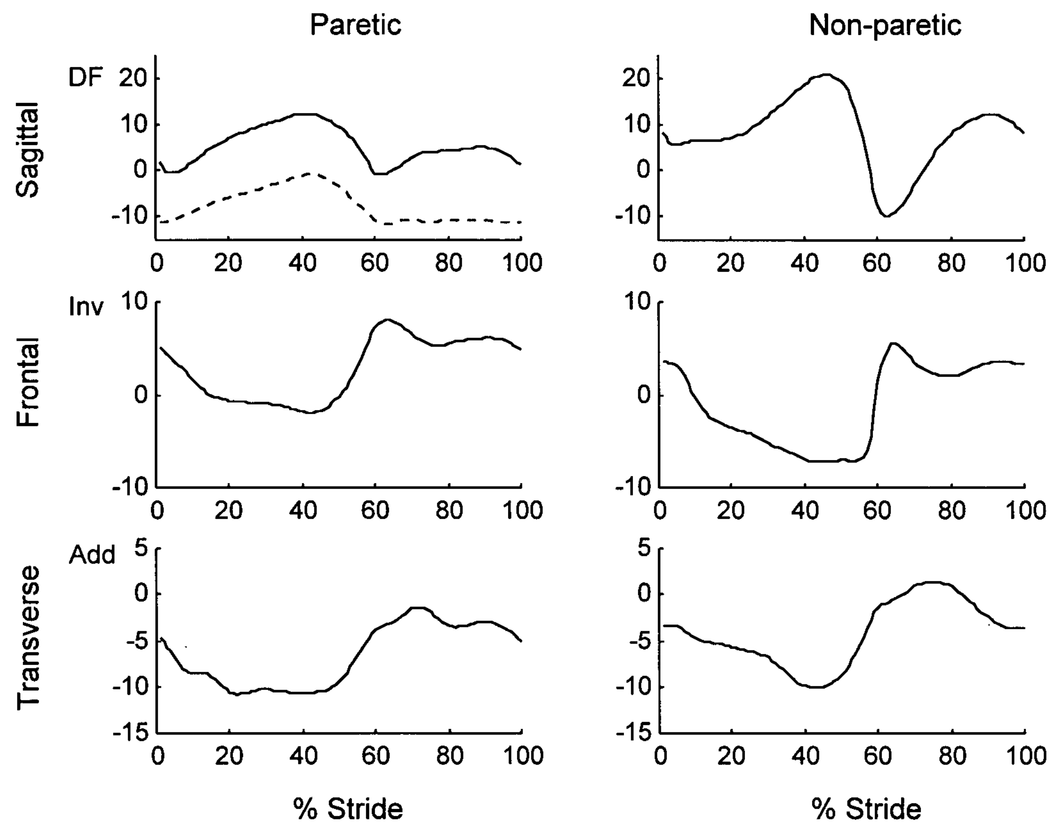
% Stride**: 60% represents toe-off. Total* = total frequency, Slow† = frequency among "slow" walkers (below the median speed), Fast‡ = frequency among "fast" walkers (above the median speed).



Pattern frequency:

Paretic side					Non-paretic side				
Plane	Pattern	Total*	Slow†	Fast‡	Plane	Pattern	Total*	Slow†	Fast‡
Sagittal	N	10	4	6	Sagittal	N	15	6	9
	A	10	6	4		A	5	4	1
Frontal	N	14	5	9	Frontal	N	9	2	7
	other	6	5	1		A	11	8	3
Transverse	A	11	4	7	Transverse	A	17	7	10
	B	5	3	2		other	3	3	0
	other	4	3	1					

Figure 2. Representative knee joint angle (degrees) profiles in the sagittal, frontal, and transverse planes. The profiles are intrasubject averages (n = 5 trials) which best represent the predominant patterns observed across the 20 participants. Solid line: pattern N; dashed line: pattern A; dark solid line: pattern B.
Abbreviations: Flexion (Flex), Adduction (Add), Internal Rotation (IR)
Total* = total frequency, Slow† = frequency among "slow" walkers (below the median speed), Fast‡ = frequency among "fast" walkers (above the median speed).



Pattern frequency:

Paretic side					Non-paretic side				
Plane	Pattern	Total*	Slow†	Fast‡	Plane	Pattern	Total*	Slow†	Fast‡
Sagittal	N	8	5	3	Sagittal	N	20	10	10
	A	12	5	7	Frontal	N	19	10	9
Frontal	N	19	10	9		other	1	0	1
	other	1	0	1	Transverse	N	10**	5	5
Transverse	N	9**	6	3		other	10	5	5
	other	12	4	8					

Figure 3. Representative ankle joint angle (degrees) profiles in the sagittal, frontal, and transverse planes. The profiles are intrasubject averages (n = 5 trials) which best represent the predominant patterns observed across the 20 participants. Solid line: pattern N; dashed line: pattern A.

Abbreviations: Dorsiflexion (DF), Inversion (Inv), Adduction (Add)

Total* = total frequency, Slow† = frequency among "slow" walkers (below the median speed), Fast‡ = frequency among "fast" walkers (above the median speed).

**During propulsion, 15 participants internally rotated their ankle on the paretic side and all 20 on the non-paretic side.

trailing limb moved forward and away from the support limb, c) at toe-off, maximum abduction was reached as the weight was transferred forward and away from the ipsilateral support limb and d) during swing, the hip adducted to bring the swinging limb under the body to optimize the pendular motion of the limb. On the paretic side, another common pattern (pattern A) was identified in 9 participants. Pattern A differed from pattern N during the swing phase in that it showed a plateau in abduction or another abduction peak at 75 to 85% of stride, possibly reflecting hip circumduction to clear the limb given the reduction in the sagittal joint angles. The remaining participants showed highly variable patterns.

In the transverse plane, three common patterns were identified. Pattern N, found on the non-paretic side only (8/20), was similar to the normal pattern: a) From weight acceptance to 45 to 55% of stride, the hip internally rotated by a mean of 6° possibly as a result of the forward progression of the contralateral limb which brings the contralateral side of the pelvis forward. The external rotation motion of the ipsilateral side of the pelvis on the fixed ipsilateral limb would present as hip internal rotation. b) During propulsion, the hip externally rotated by 18° reaching maximum at 70 to 80% of stride. This motion was a result of the advancement of the ipsilateral limb which brings the ipsilateral side of the pelvis forward. Consequently, an internal rotation of the ipsilateral side of the pelvis presents as hip external rotation. Another pattern found on the non-paretic side was pattern B which differed from pattern N during early and mid stance: predominant external rotation (6°) during the first half of stance moving into internal rotation in mid stance (10/20). On the paretic side, the predominant pattern (pattern A) was similar to pattern N during the stance phase but differed in swing where another external rotation peak was found in mid-swing (15/20).

2.3.1.2 Knee

The 3-D representative knee angle profiles are illustrated in Figure 2. In the sagittal plane, two patterns were identified on both the paretic and non-paretic sides. Pattern N was similar to the typical pattern of a) slight knee flexion at weight acceptance as a shock absorber, followed by b) extension (12-13°) during mid stance, then c) a large flexion (33° and 50° on the paretic and non-paretic sides respectively) reaching its peak in early swing to clear the foot. Pattern A (10/20 on the paretic side, 5/20 on the non-paretic side) altered from pattern N during weight acceptance: the typical knee flexion pattern at weight acceptance was replaced by an abrupt knee extension pattern (10/20 and 5/20 on the paretic and non-paretic limbs respectively). This pattern may be similar to the "extension thrust pattern" described by Kramers de Quervain et al. (1996). This extension thrust pattern reported in Kramers de Quervain et al.'s (1996) study was accompanied by a lack of electromyographic activity in the quadriceps, suggesting that a lack of eccentric control of the quadriceps may be the cause of this pattern. Alternatively, this early knee extension could be the result of the limb contacting the ground in an exaggerated flexed knee position and thus further knee flexion is not required. This latter reason may be a more plausible explanation for the occurrence of this pattern on the non-paretic side.

Reports on frontal plane knee kinematics in healthy adults have been inconsistent in the literature; Apkarian et al. (1989) and Winter et al. (1995) found that their five participants abducted their knee during propulsion whereas Kuan et al. (1999) reported adduction during propulsion. In the present study, a pattern was labeled as N based on the findings of Kuan et al. (1999): a) during weight acceptance, the knee abducted slightly (6° and 4° on the paretic and non-paretic sides respectively), b) during propulsion, the knee adducted (9° and 8°) and

c) reached its maximum in mid swing. The other 6 participants exhibited highly variable patterns on the paretic side. On the non-paretic side, eleven participants displayed a pattern (pattern A) that altered from the former pattern during propulsion and swing: rather than adduction, there was a large abduction component that peaked in early swing similar to the patterns described by Apkarian et al. (1989) and Winter et al. (1995).

In the transverse plane, the most frequently occurring pattern on both sides was pattern A: a) slight external rotation (6°-7°) at weight acceptance, followed by b) a large internal rotation (12°-14°) during propulsion until early swing. Another pattern was identified in 5 participants on their paretic knee (pattern B) which showed reversed motions from pattern A: a) slight internal rotation (3°) at weight acceptance rather than external rotation, followed by b) external rotation (6°) during propulsion instead of the internal rotation motion found in pattern A. Variation in patterns have also been reported in healthy adults; Apkarian et al. (1989) reported different patterns in each one of their three subjects. Hence, there does not appear to be consensus on an existing pattern N reported in the literature.

2.3.1.3 Ankle

The 3-D representative ankle angle profiles are illustrated in Figure 3. In the sagittal plane, the typical ankle joint angle pattern found in able-bodied gait (Winter, 1991) was identified on both the paretic and non-paretic sides (pattern N): a) small plantarflexion at weight acceptance to lower the foot to the floor, b) dorsiflexion in mid stance as the leg rotates over the foot, c) plantarflexion during propulsion, followed by d) dorsiflexion in swing to clear the foot. Pattern A, identified on the paretic side, deviated from the normal pattern during weight acceptance: there was an absence of the normal plantarflexion component at weight acceptance (12/20) possibly as a result of forefoot landing (i.e. plantarflexed position). In addition, these participants did not dorsiflex their ankle during swing to clear the foot from the ground.

In the frontal plane, as in normal profiles, both the paretic (19/20) and non-paretic (19/20) ankles a) everted (7-10°) at weight acceptance as a loading response until mid to late stance as the weight transferred over the ipsilateral planted foot. b) The motion reversed into inversion (9-14°) during propulsion and c) reached maximum inversion at toe-off or early swing (pattern N).

During weight acceptance and mid stance in the transverse plane, 9 and 10 participants exhibited a pattern of a) abduction (6-7° toe-out) in the paretic and non-paretic ankles respectively (pattern N), while the rest showed highly variable patterns. b) During propulsion, the ankle (15/20 and 20/20 on the paretic and non-paretic sides respectively) moved into adduction (by 11-12°) possibly as a result of the heel lifting off from the floor and moving laterally on the planted forefoot. c) Peak adduction occurred at 60-70% of stride. This pattern of abduction followed by adduction is similar to the normal profiles reported by Apkarian et al. (1989) and Winter et al. (1995).

2.3.2 Joint moments & powers

The magnitudes of the joint moments and powers were generally lower on the paretic side when compared to the non-paretic side in all three planes; 11 of the 15 and 17 of the 18 of the peak means for the moments and powers, respectively, were greater for the non-paretic side. Of the mean comparisons in which the paretic side was greater, the peak knee flexor moment presented the greatest difference as the paretic side was 150% of the non-paretic value (Tables 4, Figures 4, 6, 8). Generally, the sagittal paretic mean moments were 20-50% of the non-paretic side. In addition, in the frontal plane the mean hip abductor moment for the paretic side was 73% of the value for the non-paretic side. Transverse plane moments were very small; less than 0.07 Nm/kg on the paretic side and less than 0.15 Nm/kg on the non-paretic side. The largest moment on the non-paretic side was by the ankle plantarflexors while on the paretic side it was by the hip abductors.

For power variables, the largest generation burst on the paretic side was found at the hip corresponding to H3-S phase during pull-off. On the non-paretic side, the largest generation was at the ankle (A2-S) during push-off. The largest absorption burst was at the knee in the sagittal plane for both lower extremities. In general, peak powers were decreased on the paretic side compared to the non-paretic side especially the sagittal ankle generation (mean power for the paretic side was 24% of the non-paretic side), the sagittal knee absorption (42%) and the frontal hip generation (55%) (Table 5, Figures 5, 7, 9). On average, transverse plane powers were very small; less than 0.06 W/kg on the paretic side and less than 0.09 W/kg on the non-paretic side.

2.3.2.1 Hip

Representative 3-D hip moment and power patterns are illustrated in Figures 4 and 5. In the sagittal plane, the moment pattern was similar to that of normal gait (Eng & Winter, 1995) for both the paretic and non-paretic limbs. There was a) an extensor moment during the first half of stance to provide a "push-from-behind" as the trunk moved forward, b) a

Table 4: Mean ± standard deviation of moment variables (Nm/kg) over the stride (N = 19).

Plane	Moment variable	Paretic	Non-paretic
Sagittal	Maximum hip extension	0.33 ± 0.17	0.60 ± 0.25
	Maximum hip flexion	0.32 ± 0.21	0.41 ± 0.23
	Maximum knee extension	0.24 ± 0.26	0.44 ± 0.26
	Maximum knee flexion	0.30 ± 0.20	0.20 ± 0.12
	Maximum ankle plantarflexion	0.64 ± 0.22	1.10 ± 0.25
Frontal	Maximum hip abduction	0.68 ± 0.23	0.93 ± 0.23
	Maximum knee abduction	0.24 ± 0.17	0.30 ± 0.15
	Maximum knee adduction	0.07 ± 0.07	0.05 ± 0.04
	Maximum ankle inversion	0.17 ± 0.13	0.17 ± 0.11
Transverse	Maximum hip IR	0.06 ± 0.04	0.10 ± 0.05
	Maximum hip ER	0.07 ± 0.03	0.15 ± 0.06
	Maximum knee IR	0.05 ± 0.03	0.06 ± 0.04
	Maximum knee ER	0.04 ± 0.02	0.09 ± 0.04
	Maximum ankle abduction	0.05 ± 0.05	0.11 ± 0.04
	Maximum ankle adduction	0.04 ± 0.04	0.03 ± 0.02

Abbreviations: Internal Rotation (IR), External Rotation (ER)

Table 5: Mean ± standard deviation of power variables (W/kg) over the stride (N = 19).

Plane	Power variable	Paretic	Non-paretic
Sagittal	Maximum hip gen	0.50 ± 0.64	0.61 ± 0.27
	Maximum hip abs	0.19 ± 0.16	0.25 ± 0.16
	Maximum knee gen	0.19 ± 0.15	0.30 ± 0.21
	Maximum knee abs	0.31 ± 0.37	0.73 ± 0.53
	Maximum ankle gen	0.40 ± 0.43	1.64 ± 1.19
	Maximum ankle abs	0.21 ± 0.15	0.43 ± 0.26
Frontal	Maximum hip gen	0.22 ± 0.28	0.40 ± 0.32
	Maximum hip abs	0.19 ± 0.15	0.28 ± 0.26
	Maximum knee gen	0.07 ± 0.07	0.07 ± 0.06
	Maximum knee abs	0.05 ± 0.05	0.08 ± 0.08
	Maximum ankle gen	0.11 ± 0.16	0.13 ± 0.16
	Maximum ankle abs	0.05 ± 0.04	0.08 ± 0.13
Transverse	Maximum hip gen	0.05 ± 0.05	0.06 ± 0.05
	Maximum hip abs	0.06 ± 0.05	0.09 ± 0.10
	Maximum knee gen	0.01 ± 0.01	0.05 ± 0.07
	Maximum knee abs	0.02 ± 0.05	0.03 ± 0.04
	Maximum ankle gen	0.01 ± 0.01	0.02 ± 0.01
	Maximum ankle abs	0.02 ± 0.02	0.03 ± 0.02

Abbreviations: Generation (gen), Absorption (abs)

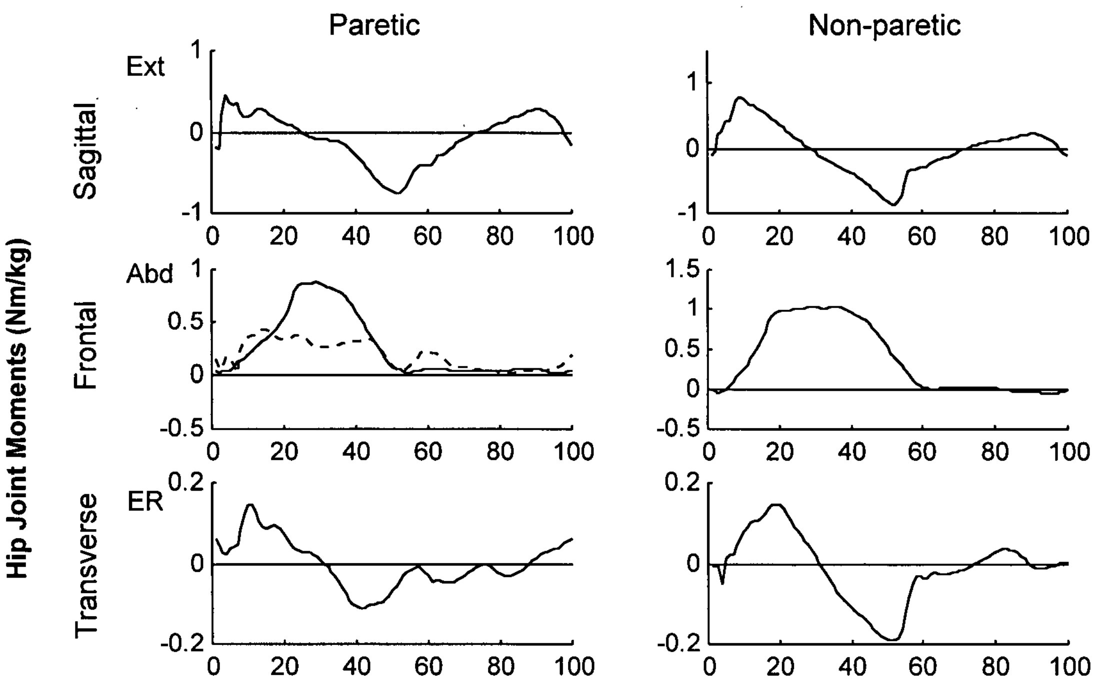


Figure 4

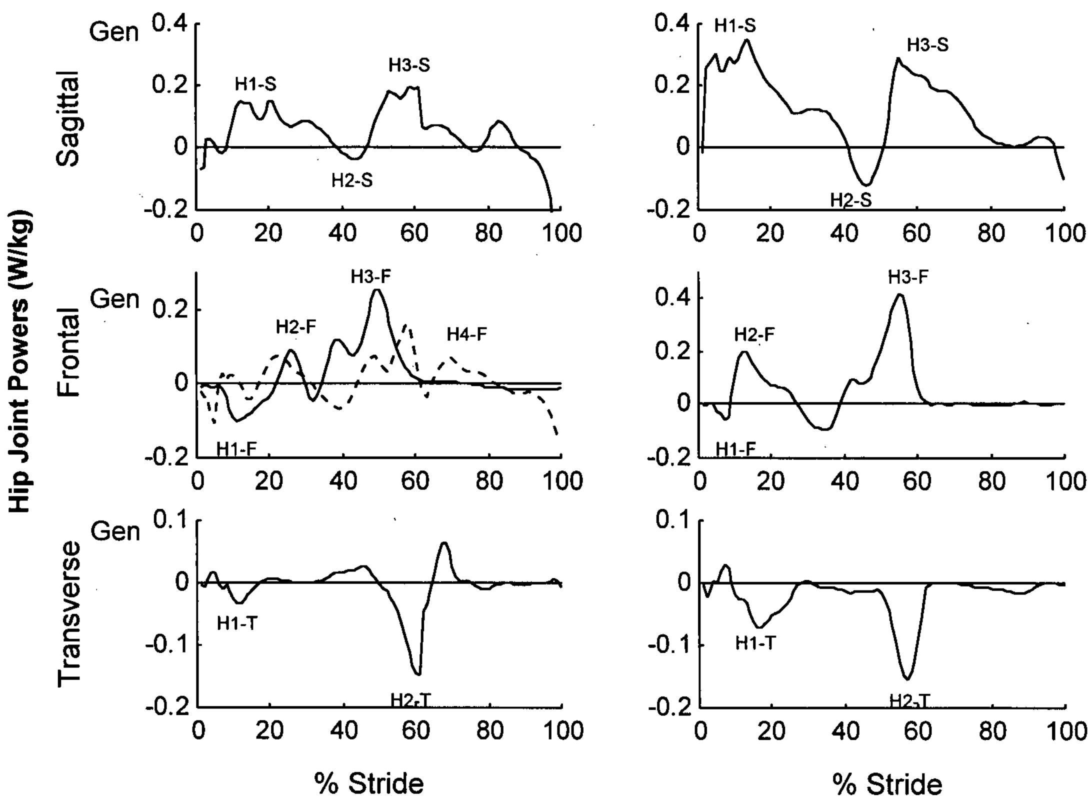


Figure 5

Pattern frequency of moments and powers:

Paretic side					Non-paretic side				
Plane	Pattern	Total*	Slow†	Fast‡	Plane	Pattern	Total*	Slow†	Fast‡
Sagittal	N	19	10	9	Sagittal	N	19	9	10
Frontal	N	7	5	2		other	1	1	0
	A	13	5	8	Frontal	N	20	10	10
Transverse	N	20	9	10	Transverse	N	19	9	10
	other	1	1	0		other	1	1	0

Abbreviations: Extension (Ext), Abduction (Abd), External Rotation (ER)
Total* = total frequency, Slow† = frequency among "slow" walkers (below the median speed), Fast‡ = frequency among "fast" walkers (above the median speed).
Note: N = 19 (paretic side data on 10 "slow" & 9 "fast" walkers, and non-paretic side data on 9 "slow" & 10 "fast" walkers) for kinetic variables.

Figures 4 and 5. Representative hip moment (Nm/kg) and power (W/kg) profiles in the sagittal, frontal, and transverse planes. The profiles are intrasubject averages (n = 5 trials) which best represent the predominant patterns observed across the 19 participants. Solid line: pattern N; dashed line: pattern A.

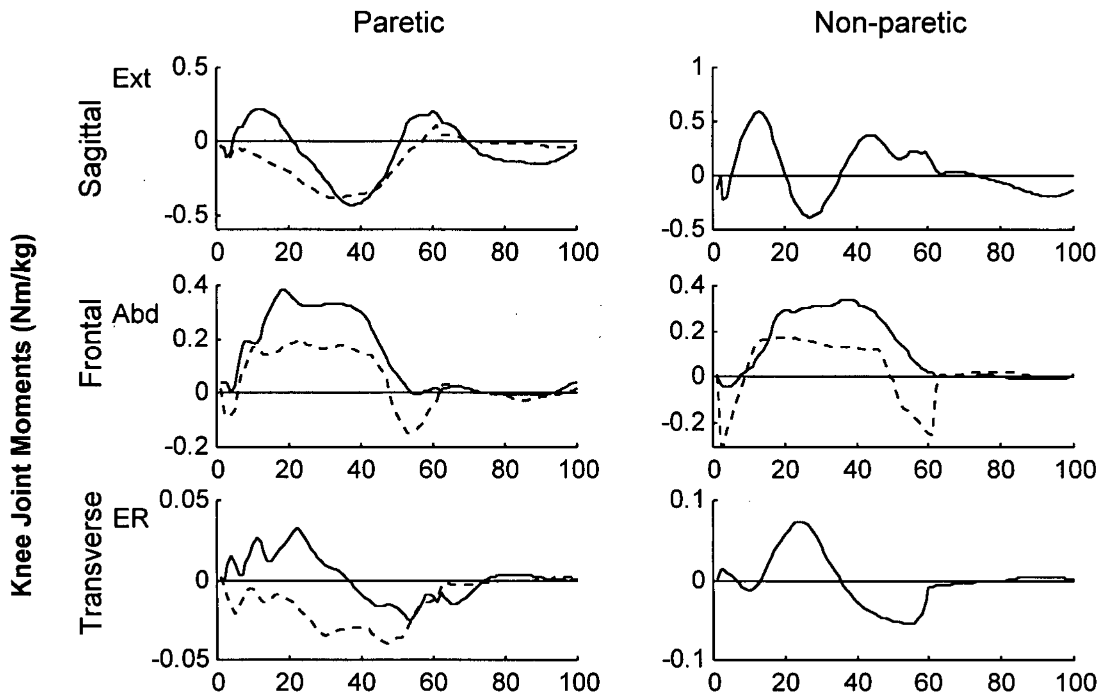


Figure 6

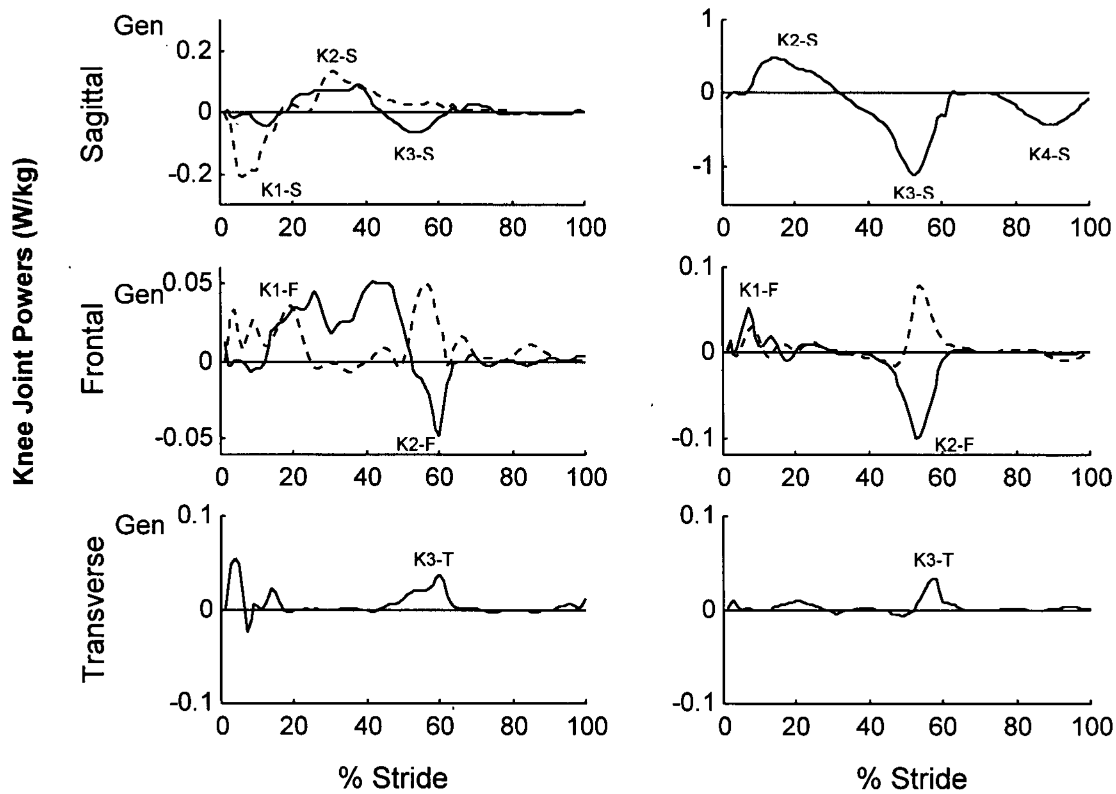


Figure 7

Pattern frequency of moments and powers:

Paretic side					Non-paretic side				
Plane	Pattern	Total*	Slow†	Fast‡	Plane	Pattern	Total*	Slow†	Fast‡
Sagittal	N	8	4	4	Sagittal	N	16	9	7
	A	11	6	5		other	3	0	3
Frontal	N	7	5	2	Frontal	N	13	5	8
	A	8	3	5		A	6	4	2
	other	4	2	2	Transverse	N	18	8	10
Transverse	N	8	6	2		other	1	1	0
	A	10	4	6					
	other	1	0	1					

Abbreviations: Extension (Ext), Abduction (Abd), External Rotation (ER)
Total* = total frequency, Slow† = frequency among "slow" walkers (below the median speed), Fast‡ = frequency among "fast" walkers (above the median speed).

Figures 6 and 7. Representative knee moment (Nm/kg) and power (W/kg) profiles in the sagittal, frontal, and transverse planes. The profiles are intrasubject averages (n = 5 trials) which best represent the predominant patterns observed across the 19 participants. Solid line: pattern N; dashed line: pattern A.

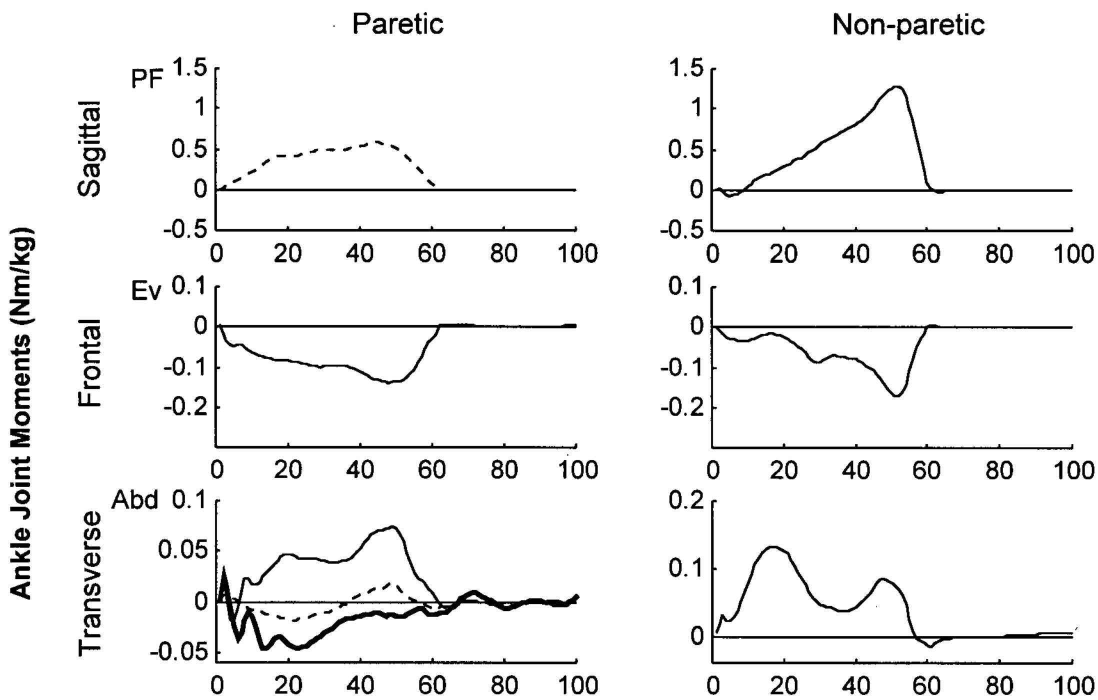


Figure 8

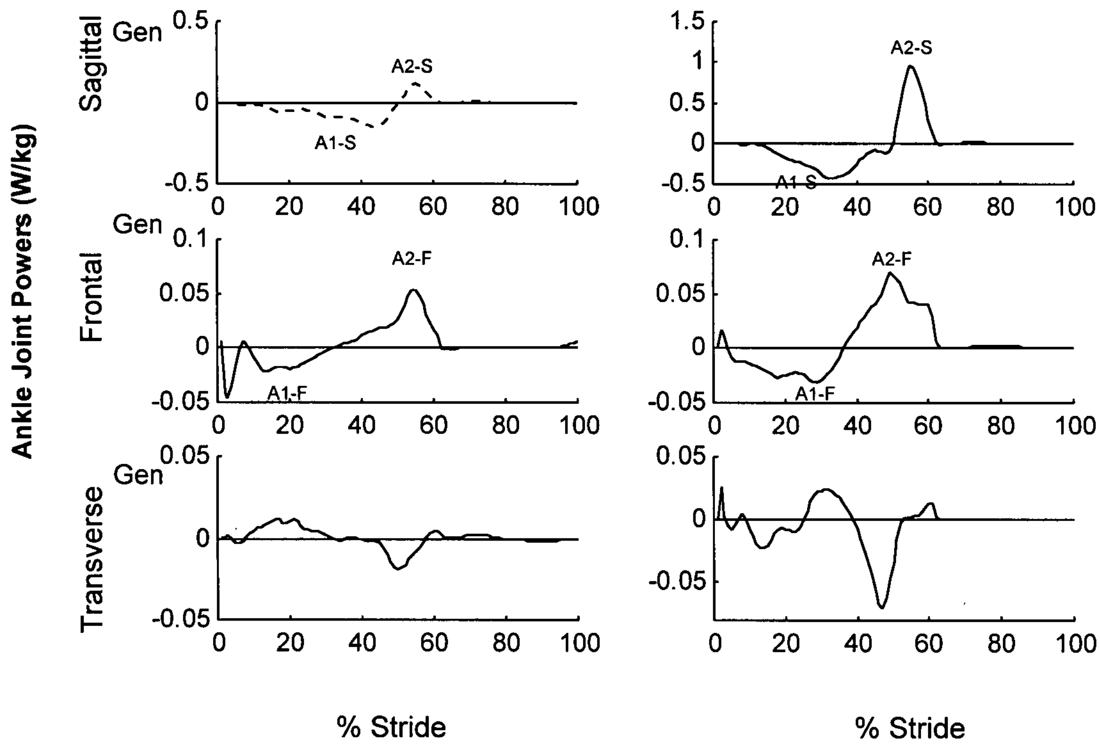


Figure 9

Pattern frequency of moments and powers:

Paretic side					Non-paretic side				
Plane	Pattern	Total*	Slow†	Fast‡	Plane	Pattern	Total*	Slow†	Fast‡
Sagittal	A	19	10	9	Sagittal	N	19	9	10
Frontal	N	19	10	9	Frontal	N	19	9	10
Transverse	N	8	6	2	Transverse	N	17	9	8
	A	6	2	4		other		0	2
	B	3	1	2					
	other	2	1	1					

Abbreviations: Plantarflexion (PF), Eversion (Ev), Abduction (Abd)
Total* = total frequency, Slow† = frequency among "slow" walkers (below the median speed), Fast‡ = frequency among "fast" walkers (above the median speed).

Figures 8 and 9. Representative ankle moment (Nm/kg) and power (W/kg) profiles in the sagittal, frontal, and transverse planes. The profiles are intrasubject averages (n = 5 trials) which best represent the predominant patterns observed across the 19 participants. Solid line: pattern N; dashed line: pattern A, dark solid line: pattern B.

reversal into a flexor moment in mid stance to control the backward rotating thigh and then to provide pull-off of the lower limb in swing, and c) a slight extensor moment in terminal swing for backward acceleration of the thigh prior to initial contact (pattern N).

The associated power profiles demonstrate that the three sagittal plane power phases seen in able-bodied gait were also found in the gait of individuals with stroke but with a decrease in amplitude especially on the paretic side (Table 5). Note that power profiles are described in relation to their association with each moment pattern (i.e. moment pattern N, A, or B). There was a) an H1-S generation phase by the hip extensors during the first half of stance, which normally contributes to forward propulsion, and was relatively low on the paretic side, b) an H2-S absorption during mid stance that normally controls thigh extension as the center of mass moves forward of the support limb which was either low or absent on the paretic side possibly as a consequence of the lack of hip extension during the first half of stance, and c) an H3-S generation phase by hip flexors during pull-off but generally lower in amplitude compared to normal values.

In the frontal plane, there was a hip abductor moment throughout stance for both the paretic and non-paretic limbs (pattern N) to balance the body mass medial to the stance hip as in normal gait (Eng & Winter, 1995). The hip abductors a) absorbed energy (H1-F) to control the degree of pelvic drop on the contralateral side during weight acceptance, b) generated energy (H2-F) to raise the pelvis on the contralateral side during single support to prevent a Trendelenburg sign and c) generated energy (H3-F) during,propulsion to transfer the pelvis/trunk over the new leading limb. In addition, 12 of 19 participants showed an additional abductor moment at toe-off into early swing on the paretic hip (pattern A).This additional abductor moment was associated with a positive power burst (labeled as H4-F) possibly as a result of the hip abductors attempting to clear the foot from the ground to compensate for the insufficient hip and knee flexion and ankle dorsiflexion.

Most participants exhibited a similar hip moment pattern on both sides in the transverse plane (pattern N): a) an initial external rotator moment during the first half of stance followed by b) an internal rotator moment similar to the pattern found in able-bodied gait described by Eng & Winter (1995). The associated transverse plane power profiles also resembled the normal profile (Eng & Winter, 1995): a) a small absorption phase occurred early in stance (H1-T) as a result of the external rotators controlling the hip internal rotation which accompanied forward motion of the contralateral side of the pelvis and b) a larger absorption burst occurred during push-off (H2-T) when the internal rotators eccentrically controlled hip external rotation caused by the weight transference over to the contralateral limb while the ipsilateral foot was planted on the floor.

2.3.2.2 Knee

Representative 3-D knee moment and power profiles are illustrated in Figures 6 and 7. The patterns found in able-bodied gait as reported by Eng & Winter (1995) were also found in our participants in all planes with the exception of a few characteristics. The sagittal knee moment pattern found in able-bodied gait consists of a) extensor moment at weight acceptance to provide shock absorption and then to assist knee extension as the center of mass is moved forward, b) flexor moment in mid-stance reversing into extensor moment during propulsion to control the collapsing knee resulting from the propulsive forces of ankle push-off and c) flexor moment in the latter half of swing to decelerate the swinging limb.

This normal pattern was exhibited by 16 participants on their non-paretic side, however only eight participants showed that same pattern on their paretic knee (pattern N).

The power profile associated with this knee moment pattern (pattern N) showed the typical a) K1-S absorption burst from the extensors at weight acceptance for shock absorption, b) K2-S (extensor) generation in mid-stance to increase the height of the center of mass as it moves forward and facilitate foot clearance on the contralateral side, c) K3-S (extensor) absorption during propulsion to control the collapsing knee caused by the ankle push-off and hip pull-off forces and d) K4-S (flexor) absorption in late swing to decelerate the swinging limb. The K1-S and K4-S absorption bursts were low or absent in many on both sides suggesting a lack in shock absorption during weight acceptance and lack of energy absorption from the swinging limb prior to initial contact.

On the paretic side, the most common knee moment pattern was a flexor pattern throughout most of stance (pattern A) (11/19). This pattern was also associated with the kinematic knee extension thrust pattern in eight participants. These individuals extended (or hyperextended) their knee at weight acceptance, during which time there was a flexor moment. Therefore a) the corresponding K1-S power phase represented the negative work done by the flexors attempting to eccentrically control the abrupt knee extension, b) the K2-S phase occurred later in stance than commonly seen in able-bodied gait and represented the positive work done by the flexors (instead of extensors as commonly seen in normal gait) attempting to flex the extended knee in preparation for swing, and c) the K3-S (extensor absorption burst) was absent in this group possibly due to the weak push-off by the ankle plantarflexors and pull-off by the hip flexors which normally would collapse the knee. Note that although the kinematic knee extension thrust pattern was also observed on the non-paretic knee (5/20), the associated kinetic patterns differed from the patterns found on the paretic side, i.e. the knee extension was the result of the positive power (K2-S) generated by the knee extensors, not flexors.

In the frontal plane, as in able-bodied gait (Eng & Winter, 1995), there was an abductor moment throughout stance on both sides (pattern N) which countered the gravitational adductor stress caused by the center of mass medial to the ipsilateral knee via passive structures (e.g. iliotibial band, tensor fascia latae or lateral ligaments) resulting in a) K1-F generation phase in early stance and b) K2-F absorption phase during propulsion. In addition, a knee adductor moment just prior to toe-off (pattern A) was found in 8 and 6 participants on their paretic and non-paretic limbs respectively which resulted in a power generation phase during propulsion.

Similar to able-bodied knee transverse moment profiles, participants in this study also exhibited a) an external rotator moment for the first half of stance followed by b) an internal rotator moment in the latter half of stance on both the paretic and non-paretic sides (pattern N). Another common transverse moment pattern found on the paretic knee was an internal rotator moment pattern throughout stance (pattern A). Transverse knee powers were low and highly variable on both sides. One consistent characteristic was a push-off generation burst (labeled as K3-T) by the internal rotators as found by Allard et al. (1996) in able-bodied persons.

2.3.2.3 Ankle

Ankle moment patterns were relatively consistent across participants in the sagittal and frontal planes (Figure 8). The dominant plantarflexor moment in stance found in able-bodied gait (Eng & Winter, 1995) was also identified in the gait of all our participants on both sides. However, the small dorsiflexor moment that normally helps lower the foot at initial contact was only present on the non-paretic side (pattern N), while the plantarflexor moment was initiated early in stance in the paretic limb (pattern A). The associated power profiles (Figure 9) displayed both the a) negative A1-S burst as a result of eccentric work from the plantarflexors controlling the forward rotation of the leg over the foot and b) positive A2-S burst corresponding to the push-off power provided by the concentric work of the plantarflexors. These power phases were exhibited by both the paretic and non-paretic limbs, however, the amplitude of the positive A2-S burst by the plantarflexors during propulsion was low especially on the paretic side (70% lower compared to the non-paretic side) (Table 5).

The frontal ankle moment pattern consisted of an invertor moment throughout stance for both the paretic and non-paretic limbs (pattern N). This pattern was similar to the pattern reported by Allard et al. (1996) and Apkarian et al. (1989) in able-bodied gait, as opposed to the evertor or evertor-invertor-evertor patterns described by others (Eng & Winter, 1995; Kadaba et al., 1989, McKinnon & Winter, 1993). The frontal power profiles found in most of our participants (18 and 13 participants for the paretic and non-paretic sides respectively) resembled the pattern found by Allard et al. (1996) in healthy young adults: a) an absorption burst (A1-F) in early stance as the invertors eccentrically controlled eversion of the foot as a loading response followed by b) a generation burst (A2-F) resulting from the invertors concentrically contracting to assist with propulsion. As propulsion on the paretic side often occurs after the weight is transferred to the non-paretic side in persons with stroke, it is possible that the invertors generated the A2-F power burst in an attempt to lift the foot and compensate for the weak dorsiflexors.

Patterns were variable in the transverse plane for the ankle. Pattern N, similar to the normal pattern (Eng & Winter, 1995), was exhibited by 17 participants on their non-paretic ankle and by eight participants on their paretic side: abductor moment through most of stance. This pattern was associated with a consistent absorption burst (not labeled) by the abductors during push-off. Power phases in able-bodied gait have also been reported as low and highly variable (Eng & Winter, 1995). Two other moment patterns were identified in the paretic ankle: an adductor moment early in stance followed by an abductor moment in late stance (pattern A) and an adductor pattern throughout stance (pattern B).

2.3.3 Mechanical work

Relative to reported normal self-paced gait values (Eng & Winter, 1995), there was a decrease in the total work done by both limbs (Table 6-7). Sagittal work was much reduced relative to normal values (25% and 75% of normal values on the paretic and non-paretic sides respectively). Although to a lesser extent, the work done in the frontal plane was also reduced on the paretic side. These reductions in work were expected as the mean gait speed of our participants was slow compared to that of healthy adults. In the transverse, however, work values were comparable between sides and to normal values.

The paretic limb performed 33% of the total work (across all 3 joints and 3 planes) as opposed to 67% done by the non-paretic side. The main difference in the amount of work done between the two limbs was in the sagittal plane (50% less on the paretic side), and least in the transverse plane. There was a marked reduction of positive work done by the sagittal

Table 6. Mean and standard deviation of work (J/kg) over the stride (N = 19).

Paretic		Sagittal		Frontal		Transverse		Total		
		gen	abs	gen	abs	gen	abs	gen	abs	Total
Hip	Mean	**0.143**	**0.037**	**0.051**	**0.045**	**0.010**	**0.015**	**0.205**	**0.097**	**0.302**
	SD	0.110	0.042	0.038	0.032	0.007	0.012			
Knee	Mean	**0.046**	**0.074**	**0.016**	**0.008**	**0.004**	**0.004**	**0.066**	**0.086**	**0.152**
	SD	0.032	0.071	0.011	0.008	0.003	0.005			
Ankle	Mean	**0.054**	**0.069**	**0.018**	**0.015**	**0.004**	**0.004**	**0.076**	**0.088**	**0.164**
	SD	0.044	0.033	0.025	0.010	0.004	0.003			
Total		**0.423**		**0.154**		**0.041**				

Non-paretic		Sagittal		Frontal		Transverse		Total		
		gen	abs	gen	abs	gen	abs	gen	abs	Total
Hip	Mean	**0.228**	**0.060**	**0.103**	**0.064**	**0.012**	**0.020**	**0.343**	**0.144**	**0.488**
	SD	0.088	0.051	0.053	0.049	0.007	0.015			
Knee	Mean	**0.066**	**0.189**	**0.015**	**0.018**	**0.011**	**0.003**	**0.092**	**0.211**	**0.302**
	SD	0.059	0.109	0.020	0.019	0.008	0.003			
Ankle	Mean	**0.235**	**0.141**	**0.021**	**0.017**	**0.005**	**0.006**	**0.261**	**0.164**	**0.425**
	SD	0.150	0.061	0.020	0.015	0.004	0.005			
Total		**0.919**		**0.239**		**0.057**				

Normal values from Eng & Winter (1995) at mean gait speed of 1.6 m/sec

		Sagittal		Frontal		Transverse		Total		
		gen	abs	gen	abs	gen	abs	gen	abs	Total
Hip	Mean	**0.48**	**0.14**	**0.092**	**0.10**	**0.009**	**0.022**	**0.58**	**0.26**	**0.84**
	SD									
Knee	Mean	**0.11**	**0.35**	**0.035**	**0.026**	**0.008**	**0.015**	**0.15**	**0.39**	**0.54**
	SD									
Ankle	Mean	**0.42**	**0.10**	**0.015**	**0.014**	**0.005**	**0.006**	**0.44**	**0.12**	**0.56**
	SD									
Total		**1.6**		**0.282**		**0.065**				

Abbreviations: Generation (gen), Absorption (abs)

Table 7. Percentage work (%) of the total lower limb work over one stride (N = 19).

Paretic		Sagittal		Frontal		Transverse		Total		
		gen	abs	gen	abs	gen	abs	gen	abs	Total
Hip	Mean	**21.8**	**5.5**	**8.2**	**7.6**	**1.8**	**2.3**	**31.8**	**15.5**	**47.3**
	SD	9.5	5.7	3.3	4.2	1.1	1.4			
Knee	Mean	**7.8**	**10.7**	**2.8**	**1.4**	**0.7**	**0.7**	**11.2**	**12.8**	**24.0**
	SD	4.9	4.7	1.5	1.1	0.6	0.9			
Ankle	Mean	**8.6**	**12.8**	**3.1**	**2.7**	**0.7**	**0.8**	**12.4**	**16.3**	**28.7**
	SD	6.3	7.2	4.0	1.9	0.6	0.7			
Total		**67.2**		**25.8**		**7.0**				

Non-paretic		Sagittal		Frontal		Transverse		Total		
		gen	abs	gen	abs	gen	abs	gen	abs	Total
Hip	Mean	**19.4**	**5.1**	**9.0**	**5.3**	**1.1**	**1.5**	**29.5**	**11.9**	**41.4**
	SD	5.7	4.4	4.5	3.0	0.7	1.0			
Knee	Mean	**5.8**	**14.8**	**1.2**	**1.5**	**0.9**	**0.3**	**7.8**	**16.6**	**24.4**
	SD	5.1	5.6	1.5	1.6	0.6	0.3			
Ankle	Mean	**18.5**	**11.6**	**1.8**	**1.3**	**0.4**	**0.6**	**20.7**	**13.4**	**34.2**
	SD	5.2	3.6	1.7	0.7	0.4	0.4			
Total		**75.2**		**20.1**		**4.8**				

Normal values from Eng & Winter (1995) at mean gait speed of 1.6 m/sec

		Sagittal		Frontal		Transverse		Total		
		gen	abs	gen	abs	gen	abs	gen	abs	Total
Hip	Mean	**24.7**	**7**	**5**	**5.0**	**0.5**	**1.1**	**30**	**13.4**	**43.3**
	SD									
Knee	Mean	**5.6**	**18.0**	**1.8**	**1.3**	**0.4**	**0.8**	**7.7**	**20**	**27.8**
	SD									
Ankle	Mean	**21**	**5.2**	**0.8**	**0.7**	**0.3**	**0.3**	**23**	**6**	**28.9**
	SD									
Total		**82**		**15**		**3**				

Abbreviations: Generation (gen), Absorption (abs)

ankle and negative work by the sagittal knee on the paretic side suggesting that the major deficits are the lack of push-off generation by the ankle plantarflexors and lack of eccentric control by the knee extensors.

2.3.4 Correlations between speed and gait variables

There was a general trend for the faster walkers to exhibit the kinematic and kinetic patterns closest to the ones found in able-bodied gait. However, this trend was not seen in the frontal hip profiles where the second pattern (prolonged abduction into swing) was more predominant among "fast" walkers (Figures 1 and 4).

Correlational analysis revealed that magnitudes of joint excursions, maximum moments and powers on the paretic side are most closely related to gait speed in the plane of progression, i.e. the sagittal plane, than in the frontal or transverse planes (Tables 8-10). On the paretic side, among kinematic variables, sagittal and transverse hip and knee joint angle ranges were significantly correlated with speed (r = 0.50-0.62) with the strongest correlation at the sagittal hip (r = 0.62). Whereas on the non-paretic side, the sagittal ankle together with frontal hip and knee angle ranges were correlated (r = 0.64-0.74) with the strongest correlation at the frontal knee. Only two of the fourteen moment variables measured correlated with gait speed on the paretic side: maximum hip flexor (r = 0.83) and maximum hip external rotator (r = 0.70) moments. On the non-paretic side, however, maximum hip extensor and flexor moments as well as hip and knee internal rotator moments and ankle plantarflexor moment were related to speed (r = 0.63-0.78).

Power variables were more closely related to speed than joint angle or moment variables. All variables except maximum knee power generation on the non-paretic side were

Table 8: Pearson product moment correlation (r) between gait speed (m/sec) and joint angle range (degrees) over the stride (N = 20).

Plane	Joint	Paretic	Non-paretic
Sagittal	Hip range	0.62**	0.15
	Knee range	0.52*	0.30
	Ankle range	0.32	0.65**
Frontal	Hip range	0.41	0.64**
	Knee range	0.34	0.74**
	Ankle range	-0.21	0.43
Transverse	Hip range	0.59**	0.37
	Knee range	0.50*	0.14
	Ankle range	0.23	0.32

** Correlation is significant at the 0.01 level (2-tailed).
*Correlation is significant at the 0.05 level (2-tailed).

Table 9: Pearson product moment correlation (r) between gait speed (m/sec) and moment variables (Nm/kg) over the stride (N = 19).

Plane	Moment variable	Paretic	Non-paretic
Sagittal	Maximum hip extension	0.07	0.65**
	Maximum hip flexion	0.83**	0.63**
	Maximum knee extension	0.04	0.25
	Maximum knee flexion	0.09	0.14
	Maximum ankle plantarflexion	0.44	0.78**
Frontal	Maximum hip abduction	-0.01	-0.02
	Maximum knee abduction	-0.23	0.17
	Maximum knee adduction	-0.04	-0.13
	Maximum ankle inversion	0.09	0.24
Transverse	Maximum hip IR	0.08	0.69**
	Maximum hip ER	0.70**	0.39
	Maximum knee IR	-0.13	0.74**
	Maximum knee ER	-0.24	0.22
	Maximum ankle abduction	-0.31	0.28

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
Abbreviations: Internal Rotation (IR), External Rotation (ER)

Table 10: Pearson product moment correlation (r) between gait speed (m/sec) and power variables (W/kg) over the stride (N = 19).

Plane	Power variable	Paretic	Non-paretic
Sagittal	Maximum hip gen	0.88**	0.75**
	Maximum hip abs	0.46*	0.47*
	Maximum knee gen	0.49*	-0.01
	Maximum knee abs	0.85**	0.91**
	Maximum ankle gen	0.71**	0.95**
	Maximum ankle abs	0.85**	0.83**
Frontal	Maximum hip gen	0.81**	0.83**
	Maximum hip abs	0.58**	0.51*
	Maximum knee gen	0.33	0.29
	Maximum knee abs	0.11	0.25
	Maximum ankle gen	0.41	0.44
	Maximum ankle abs	0.16	0.76**
Transverse	Maximum hip gen	-0.02	0.19
	Maximum hip abs	0.63**	0.74**
	Maximum knee gen	0.58**	0.21
	Maximum knee abs	-0.10	0.13
	Maximum ankle gen	-0.10	0.29
	Maximum ankle abs	-0.06	-0.09

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
Abbreviations: Generation (gen), Absorption (abs)

significantly correlated with speed on both sides in the sagittal plane (r = 0.46-0.95). Interestingly, the strongest correlation was at the hip (generation) on the paretic side (r = 0.88) and at the ankle (generation) on the non-paretic side (r = 0.95). Frontal hip power variables were associated with speed on both sides (r = 0.51-0.83) as was the maximum ankle power absorption on the non-paretic side (r = 0.76). In the transverse plane, the maximum hip power absorption was related on both sides (r = 0.63-0.74) as well as the maximum knee power generation on the paretic side (r = 0.58). Contrary to expectations, when comparing the two sides of the body, correlations were generally higher on the non-paretic limb than on the paretic limb.

2.4 DISCUSSION

The purpose of this study was to identify typical patterns in the gait of individuals with stroke and explore the relationship between these patterns and gait speed. In addition, the correlation between gait speed and the magnitudes of selected gait variables was examined. Although stereotyped patterns were identified, inter-subject variability in patterns was evident in all planes for both lower extremities.

2.4.1 Inter-subject variability in patterns

Several kinematic and kinetic patterns were identified across participants in this study. Inter-subject variability was greater in the kinetic profiles than the kinematic profiles on the paretic side where 16 different moment patterns were identified in the three joints across the three planes. The number of kinematic patterns identified in the two limbs were similar (12 and 13 patterns in the non-paretic and paretic limbs respectively, across the three planes), however, a greater number of kinetic patterns were identified on the paretic side compared to the non-paretic side. Given that kinetic patterns are the cause of kinematic outcomes, our results suggest that some stroke survivors may use different strategies to achieve similar observable outcomes. For example, at the knee in the sagittal plane, two different kinetic patterns on the paretic (flexor moment) and non-paretic (extensor moment) sides were associated with the same kinematic pattern (knee extension thrust pattern). This emphasizes the importance of kinetic analysis in understanding the underlying mechanisms of the kinematic observations, which may be important in determining treatment approaches for gait training.

As expected, the number of different identifiable patterns was lowest in the plane of progression, i.e. the sagittal plane, and greatest in the transverse plane. Inter-subject variability in the frontal and transverse planes seems to also exist in able-bodied gait. For example, in the transverse plane, Eng & Winter (1995) reported a hip external rotator moment in the first half of stance followed by an internal rotator moment in the latter half of stance while Kadaba et al. (1989) found the opposite. Given that these previous studies generally reported a single pattern across all subjects, it is unlikely that different populations of healthy young subjects could contrast so starkly in their walking pattern. It is likely that some of the differences in the patterns reported between studies may be due to methodological differences in the link segment model, i.e. differences in the mathematical calculation of the 3-D joint angles. The specific order of the sequential rotations, the projections of the principal axes on the segments and the exact location of the joint centre may all lead to variability in the results between studies (Eng & Winter, 1995; Fioretti et al., 1997). Therefore, comparisons of gait patterns in individuals with stroke should ideally be made with data collected within the same laboratory with able-bodied persons as controls. In our study, the inter-subject pattern variability was not due to the link segment model differences as the same model and kinematic and kinetic algorithms were used for all participants.

The clinical significance of the variability in patterns found in persons with stroke is that treatment approaches should be individualized depending on the specific deficits found in each individual. Other studies have also identified different kinematic (Kramers de Quervain, 1996) and electromyographic (Knutsson & Richards, 1979) gait patterns within a single group of persons with stroke. Kramers de Quervain (1996) described four coupling patterns of knee and ankle motion during stance and Knutsson & Richards (1979) described three types of abnormal muscle activation patterns during gait following stroke. Depending on the type of disturbed motor control presented by the individual person, the choice of therapeutic procedures should vary to address the specific aspects of the locomotor disturbance. Another implication of the inter-subject variability found in the gait patterns is that when presenting gait profiles of individuals with stroke, the profiles should not be averaged across subjects as valuable information regarding different strategies used may be lost.

2.4.2 Correlation between gait speed and kinematic/kinetic variables

Self-selected gait speed has been recognized as a valid measure of gait performance, sensitive enough to reflect both functional and physiological changes (Richards et al., 1995) and reliable for studying locomotor performance (Olney et al., 1979). Gait speed is also positively related to the muscle strength of the paretic and non-paretic lower limbs in individuals with stroke (Bohannon, 1986; Bohannon &Walsh, 1992; Nadeau et al., 1999; Kim & Eng, in review). Identifying the variables that closely relate to gait speed may provide a focus of intervention aimed at improving gait performance.

Among the selected variables, power variables related more closely to gait speed than joint angle or moment variables suggesting that power variables are more indicative of gait performance in persons with stroke. Supporting our findings, Olney et al. (1994) also reported higher correlations for power variables than joint angle or moment variables in the gait of individuals with stroke. In able-bodied gait, joint angles particularly of the sagittal hip motion have been shown to strongly correlate with speed (Crowinshield et al., 1978) as increasing the flexion/extension angle would increase the step length and thereby increase gait speed. In hemiparetic gait however, this relationship between speed and hip range is only moderate on the paretic side ($r = 0.62$ in the present study, $r = 0.32$-0.61 in Olney et al., 1994) and insignificant on the non-paretic side possibly due to differences in patterns and motor strategies between individuals. For example, the pattern of prolonged hip abduction in swing (hip circumduction) was more predominant among faster walkers suggesting that they are able to walk faster with compensatory mechanisms rather than by increasing the sagittal hip angle as would normally occur in able-bodied gait. Thus, our results suggest that among persons with stroke, faster walkers may use different motor strategies compared to slower walkers.

In general, variables were more significantly related to speed in the sagittal plane than in the frontal or transverse planes. The high correlation coefficients found in the sagittal plane concur with the results by Olney et al. (1994) and emphasize the importance of muscle function in this plane. For example, in our study, the important role of hip flexors during pull-off is reflected by the high correlations between speed and a) maximum hip power (generation) on both sides, b) maximum hip flexor moment on both sides, and c) hip range on the paretic side. Similarly, the strong correlations found between ankle powers and speed on both sides confirm the importance of ankle plantarflexors to generate push-off as well as to absorb power during the stance phase of gait when the stance leg normally rotates over the foot.

Consistent with Olney et al.'s (1994) findings, the maximum knee absorption power (K3-S), which represents the work of the quadriceps to eccentrically control the flexing knee during propulsion, was highly correlated with gait speed on both sides. Our results indicate that the slower walkers did not require this eccentric control from the quadriceps as the weak ankle push-off /hip pull-off mechanism did not result in the forces that normally would collapse the knee. Interestingly, the maximum knee generation power (K2-S) was significantly, although moderately, correlated with speed on the paretic side. Given that eleven of our participants displayed a flexor moment during the occurrence of K2-S, the significant association between peak K2-S and speed suggests that the ability to actively flex the knee from the extended (or hyperextended) position during mid or late stance in preparation for swing may be an important compensatory mechanism in some individuals with stroke. Consistent with this finding, Kim & Eng (in review) found that the strength of the knee flexors measured by the maximum isokinetic torque was significantly correlated with gait speed.

In the frontal plane, the importance of the hip abductors in controlling the lateral pelvic displacement is reflected by the significant correlations found between maximum frontal hip powers and speed on both sides. The eccentric (H1-F) control of the lateral pelvic drop and concentric (H3-F) work to raise the pelvis on the contralateral side by the ipsilateral abductors is likely important in increasing the contralateral step length and consequently, the gait speed.

2.4.3 Major deviations from normal profiles

Except for a few variables, magnitudes were lower on the paretic side compared to that of the non-paretic side. Although this difference was more predominant in the sagittal plane, it was also evident in the other planes. Two thirds of the total sagittal work and 60% of the total frontal plane work was done by the non-paretic side. These findings suggest that the non-paretic limb performs a larger proportion of the work necessary for forward progression and also for body support and balance in the frontal plane. Contrary to our findings, Olney et al. (1991) found that 40% of the total positive work in the sagittal plane was performed by the paretic limb in stroke survivors walking at speeds that were comparable to those of our participants. Differences in findings may be reflective of differences in the level of motor impairment between participants in the two studies. However, no impairment measures were included in Olney et al.'s (1991) study, therefore this assumption cannot be confirmed.

On the paretic side, the major kinematic deviations included a general decrease in sagittal joint angle range in all three joints, decreased ankle dorsiflexion and prolonged hip abduction during swing, decreased dorsiflexion at initial contact and lack of knee flexion during weight acceptance compared to the non-paretic side. The reduced sagittal joint angle range was accompanied by a decrease in joint moments, power and work at the hip, knee and ankle except for knee flexor moment which was greater on the paretic side when compared to the non-paretic limb. In the frontal plane, prolonged hip abduction in swing was associated with a fourth power burst (H4-F) not normally found in able-bodied gait.

These movement pattern deviations may be the direct or compensatory result of one or several sensorimotor impairments following stroke. Possible causes include muscle weakness, abnormal muscle activation patterns, contractures, spasticity, disturbed sensation and poor balance (Dettman et al., 1987; Knutsson & Richards, 1979; Lehman et al., 1987; Morris et al., 1992; Peat, 1976; Perry, 1992; Simon et al., 1978). For example, weakness in the hip flexors and ankle plantarflexors would limit the amount of pull-off/push-off force during propulsion reflected by the decrease in H3-S and A2-S power bursts, and hip flexor and ankle plantarflexor moments. Similarly, weakness in the ankle dorsiflexors or plantarflexor contractures could limit ankle dorsiflexion in swing which in turn limits foot clearance. The prolonged hip abduction in swing identified in half of our participants may be a compensation for this lack of dorsiflexion and the insufficient flexion at the hip and knee required to clear the ground. Also, impaired balance and/or weakness in the hip abductors may limit the ability to weight shift laterally (Goldie et al., 1996) and this may be reflected by the reduced hip abductor moment.

Another common deviation identified was a lack of knee flexion during weight acceptance. The motor impairment associated with this deviation may be weakness in the knee extensors which would normally control the knee flexion as a shock-absorbing mechanism during the loading response. Without this shock-absorbing mechanism the knee may thrust into extension and cause hyperextension of the knee. Spasticity of the ankle plantarflexors, although low in our participants, may also cause the knee to hyperextend during stance. These individuals also displayed a positive power burst (K2-S) later in stance than normally seen in able-bodied gait and represented the work performed by the knee flexors attempting to flex the hyperextended knee in preparation for swing. The knee flexor work observed in these participants during stance was likely a compensatory mechanism for the knee hyperextension or alternatively, the lack of push-off by the ankle plantarflexors.

2.4.4 Contribution of the non-paretic limb

It has been suggested that persons with stroke may use their non-paretic limb to compensate for the deficits on the paretic side and thus improve gait performance (McDowell & Louis, 1971). However, the high correlations found between gait speed and selected gait variables of both the paretic and non-paretic limbs suggest that individuals with stroke are not able to fully compensate with the non-paretic limb and increase their gait speed.

Given that the correlations were in many cases higher on the non-paretic side, it does suggest that the performance of the non-paretic limb itself is an important contributing factor of speed. In addition, gait deviations were also identified on the non-paretic side (e.g. the knee extension thrust pattern). A plausible explanation for the high correlations and gait deviations found on the non-paretic limb may be that motor impairments are also apparent in the non-paretic limb. This hypothesis is supported by previous reports that weakness is bilateral following stroke (Bohannon & Andrews, 1995; Sjostrom et al., 1980) and that the muscle strength of the non-paretic limb is highly correlated with gait speed (Kim & Eng, in review; Suzuki et al., 1990). In addition, the ability to transfer the body weight onto the non-paretic leg has been shown to be decreased in individuals with stroke compared to healthy controls (Goldie et al., 1996). Thus, the function of the non-paretic limb should not be overlooked in the assessment and planning of intervention techniques aimed at improving gait performance.

2.4.5 Contribution of the non-sagittal planes

Although sagittal plane analyses of gait provide useful information regarding the mechanisms underlying the appearance of gait in persons with stroke (Olney et al., 1991), analyses of other planes should not be neglected as they may provide valuable information concerning the strategies used by each individual to accomplish the task of walking. A number of kinematic and kinetic deviations from the normal profiles have been identified in the frontal and transverse planes for both the paretic and non-paretic limbs, possibly as a result of motor deficits in the lower limb (e.g. decreased frontal hip power) or compensations for the deficits (e.g. hip circumduction).

The contribution of the muscle work in the frontal plane is an important one as the hip abductors/adductors are dominant in controlling the medio-lateral balance of the whole body about the base of support (Jian et al., 1993; McKinnon & Winter, 1993). Our results indicate that although the deficits of the paretic limb relative to the non-paretic limb are larger in the sagittal plane, the deficits are also apparent in the frontal plane in hemiparetic gait following stroke. When compared to normal values (Eng & Winter, 1995), the work performed in the frontal plane by the non-paretic limb was of comparable amount, but the work done by the paretic limb was only about 60% of the normal values. These findings suggest that the ability to control medio-lateral balance is essential in order to gain the ability to walk. In addition, the frontal hip power was strongly correlated with gait speed indicating that an improved frontal balance control may improve gait performance.

2.4.6 Clinical implications

The results of this study indicate that 3-D analysis of gait in persons with stroke may be a useful tool in identifying deficits and compensatory actions which may help guide treatment approaches aimed at improving gait performance. Particularly, the analysis of the kinetic patterns is important in identifying the underlying mechanisms of the kinematic deviations we observe.

The inter-subject variability in the kinematic and kinetic patterns identified in this study implies that individuals with stroke may use different strategies to achieve the goal of walking. Thus treatment approaches need to be individualized depending on the type of motor strategy used by each individual. Olney et al. (1988) presented some guidelines for treatment of gait in individuals with stroke based on the biomechanical analysis of each person and discussed the advantages and limitations of this type of intervention. Experimental studies are needed to assess the effects of this suggested treatment approach.

Correlational analyses revealed some important gait variables that closely relate to self-selected gait speed which is recognized as a good indicator of overall gait performance. The results suggest that treatment interventions directed at increasing the sagittal hip, knee and ankle power and frontal hip power, e.g. concentric strengthening of the hip flexors and ankle plantarflexors and concentric/eccentric strengthening of the hip abductors, may be of particular benefit during the gait training of stroke survivors. The contribution of the non-paretic limb in the performance of walking in stroke survivors is noteworthy. The strong correlations found between the non-paretic gait variables and speed imply that interventions aimed at improving the function of the non-paretic limb may lead to improvements in gait performance. However, since correlation studies do not infer causation, further research is needed to confirm these hypotheses.

2.4.7 Limitations

An important limitation of this study is the small sample size which limits the generalizability of the results to the general stroke population. In addition, the small sample size may have limited the strength of the associations between gait speed and selected gait variables.

Another limitation of the study is that comparisons of gait patterns were made with normal data on young healthy persons walking at their self-selected speeds reported in the literature. Given that gait profiles can be influenced by age and gait speed (Crowinshield et al., 1978), it would be ideal to analyze the gait profiles presented by our participants relative to the gait of healthy elderly controls walking at speeds that are comparable to those of our participants. However, no normal data is currently available on the 3-D kinematics and kinetics of able-bodied gait in the elderly. In addition, forcing a very slow speed on a healthy person's gait could result in gait patterns that may deviate from the normal profiles.

Correlational analyses included only the kinematic and kinetic variables of gait without taking into account other factors that may affect gait speed. For example, faster walking may require greater balance control due to the higher acceleration of the top heavy head, arms and trunk mass balanced over the hip joint (Winter, 1995).

Gait assessment was done in a gait laboratory and not in a natural environment. Karimi (1996) found that the speed at which their participants walked in the gait laboratory differed from that obtained in a more natural setting. It is possible that the unfamiliar environment of the laboratory affected the gait pattern and speed of our participants. However, participants were instructed to walk in a natural manner and practice trials were incorporated before the actual testing.

References

Allard, P., Lachance, R., Aissaoui, R., Duhaime, M. (1996). Simultaneous bilateral 3-D able-bodied gait. Human Movement Science, 15, 327-346.

Andriacchi, T.P., Strickland, A.B. (1985). Gait analysis as a tool to assess joint kinetics. In N. Berme, A.E. Engin, K.M. Correia Da Silva (Eds.), Biomechanics of Normal and Pathological Human Articulating Joints. NATO SI Series (p. 83-102). Martinus Nijhoff: Dordrecht.

Apkarian, J., Naumann, S., Cairns, B. (1989). A three-dimensional kinematic and dynamic model of the lower limb. Journal of Biomechanics, 22, 143-155.

Ashworth, B. (1964). Preliminary trial of carisoprodal in multiple sclerosis. Practitioner, 192, 540-2.

Bendall, M.J., Bassey, E.J., Pearson, M.B. (1989). Factors affecting walking speed of elderly people. Age Ageing, 18, 327-332.

Bohannon, R.W. (1986). Strength of lower limb related to gait velocity and cadence in stroke patients. Physiotherapy Canada, 38, 204-206.

Bohannon RW, Andrews AW. (1995). Limb muscle strength is impaired bilaterally after stroke. Journal of Physical Therapy Science,7, 1-7.

Bohannon, R.W., Smith, M.B. (1987). Interrater reliability of a modified Ashworth scale of muscle spasticity. Physical Therapy, 67, 206-7.

Bohannon, R.W., Walsh, S. (1992). Nature, reliability, and predictive value of muscle performance measures in patients with hemiparesis following stroke. Archives of Physical Medicine and Rehabilitation, 73, 721-5.

Bohannon, R.W., Horton, M.G., Wikholm, J.B. (1991). Importance of four variables of walking to patients with stroke. International Journal of Rehabilitation Research, 14, 246-50.

Brandstater, M.E., de Bruin, H., Gowland, C., Clark, B.M. (1983). Hemiplegic gait: analysis of temporal variables. Archives of Physical Medicine and Rehabilitation, 64, 583-7.

Bresler, B., Frankel, J. (1950). The forces and moments in the leg during level walking. Transactions American Society of Mechanical Engineering, 72, 27-36.

Brown, R.D., Whisnant, J.P., Sicks, J.D., O'Fallon, W.M., Wiebers, D.O. (1996). Stroke incidence, prevalence, and survival: secular trends in Rochester, Minnesota, through 1989. Stroke, 27, 373-380.

Burdett, R.G., Borello-France, D., Blatchly, C., Poptter, C. (1988). Gait comparison of subjects with hemiplegia walking unbraced, with ankle-foot orthoses, and with Air-Stirrup brace. Physical Therapy, 68, 1197-1203.

Chen, I.H., Kuo, K.N., Andriacchi, T.P. (1997). The influence of walking speed on mechanical joint power during gait. Gait and Posture, 6, 171-176.

Crowinshield, R.D., Brand, R.A., Johnston, R.C. (1978). The effects of walking velocity and age on hip kinematics and kinetics. Clinical Orthopaedics and Related Research, 132, 140-144.

Dettmann, M.A., Linder, M.T., Sepic, S.B. (1987). Relationships among walking performance, postural stability, and functional assessments of the hemiplegic patient. American Journal of Physical Medicine, 66, 77-90.

Eng, J.J., Winter, D.A. (1995). Kinetic analysis of the lower limb during walking: what information can be gained from a three-dimensional model? Journal of Biomechanics, 28, 753-758.

Fioretti, S., Cappozzo, A., Lucchetti, L. (1997). Joint kinematics. In P. Allard, A. Cappozzo, A. Lundberg & C. Vaughan (Eds.), Three-dimensional Analysis of Human Locomotion (pp.173-189). New York, NY: John Wiley & Sons.

Goldie, P.A., Matyas, T.A., Evans, O.M., Galea, M., Bach, T.M. (1996). Maximum voluntary weight-bearing by the affected and unaffected legs in standing following stroke. Clinical Biomechanics, 11, 333-342.

Gowland, C., VanHullenaar, S., Torresin, W., Moreland, J., Vanspall, B., Barrecca, S., Ward, M., Huijbregts, M., Stratford, P., Barclay-Goddard, R. (1995). Chedoke-McMaster Stroke Assessment: Development, Validation and Administration Manual. Hamilton: Chedoke-McMaster Hospitals and McMaster University.

Gresham, G.E., Phillips, T.F., Wolf, P.A., McNamara, P.M., Kannel, W.B., Dawber, T.R. (1979). Epidemiologic profile of long-term stroke disability: the Framingham study. Archives of Physical Medicine and Rehabilitation, 60, 487-491.

Himann, J.E., Cunningham, D.A., Rechnitzer, P.A., Paterson, D.H. (1988). Age-related changes in speed of walking. Medicine & Science in Sports & Exercise, 20, 161-166.

Jian, Y., Winter, D.A., Ishac, M.G., Gilchrist, L. (1993). Trajectory of the body COG and COP during initiation and termination of gait. Gait and Posture, 1, 9-22.

Kadaba, M.P., Ramakrishnan, H.K., Wootten, M.E., Gainey, J., Gorton, G., Cochran, G.V.B. (1989). Repeatability of kinematic, kinetic and electromyographic data in normal adult gait. Journal of Orthopaedic Research, 7, 849-860.

Karimi, H. (1996). Isokinetic strength training and its effect on the biomechanics of gait in subjects with hemiparesis as a result of stroke (PhD Dissertation). Kingston, Ontario: Queen's University.

Kelly-Hayes M, Robertson JT, Broderick JP, Duncan PW, Hershey LA, Roth EJ, Thies WH, Trombly CA. (1998). The American Heart Association Stroke Outcome Classification. Stroke, 29,1274-1280.

Kerrigan, D.C., Frates, E.P., Rogan, S., Riley, P.O. (1999). Spastic paretic stiff-legged gait: biomechanics of the unaffected limb. American Journal of Physical Medicine and Rehabilitation, 78, 354-360.

Kerrigan, D.C., Frates, E.P., Rogan, S., Riley, P.O. (2000). Hip hiking and circumduction: quantitative definitions. American Journal of Physical Medicine and Rehabilitation, 79, 247-252.

Kim, C.M., Eng, J.J. (in review). Non-paretic, in addition to paretic lower extremity muscle strength correlates with locomotor performance after stroke.

Knutsson, E. (1981). Gait control in hemiparesis. Scandinavian Journal of Rehabilitation Medicine, 13, 101-108.

Knutsson, E., Richards, C. (1979). Different types of disturbed motor control in gait of hemiparetic patients. Brain, 102, 405-30.

Simon, S.R., Deutsch, S.D., Nuzzo, R.M., Mansour, M.J., Jackson, J.L., Koskinen, M., Rosenthal, R.K. (1978). Genu recurvatum in spastic cerebral palsy: Report on findings by gait analysis. Journal of Bone & Joint Surgery, 60, 882-894.

Kramers de Quervain, I.A., Simon, S.R., Leurgans, S., Pease, W.S., McAllister, D. (1996). The Journal of Bone and Joint Surgery, 78-A, 1506-14.

Kuan, T-S, Tsou, J-Y, Su, F-C. (1999). Hemiplegic gait of stroke patients: the effect of using a cane. Archives of Physical Medicine and Rehabilitation, 80, 777-784.

Lehmann, J.F., Condon, S.M., Price, R., deLateur, B.L. (1987). Gait abnormalities in hemiplegia: their correction by ankle-foot orthoses. Archives of Physical Medicine and Rehabilitation, 68, 763-771.

McDowell, F., Louis, S. (1971). Improvement in motor performance in paretic and paralyzed extremities following nonembolic cerebral infarction. Stroke, 2, 395-399.

McKinnon, C.D., Winter, D.A. (1993). Control of whole body balance in the frontal plane during human walking. Journal of Biomechanics, 26, 633-644.

Mizrahi, J., Susak, Z., Heller, L., Najenson, T. (1982). Variation of time-distance parameters of the stride as related to clinical gait improvement in hemiplegics. Scandinavian Journal of Rehabilitation Medicine, 14, 133-140.

Morris, M.E., Matyas, T.A., Bach, T.M., Goldie, P.A. (1992). Electrogoniometric feedback: its effect on genu recurvatum in stroke. Archives of Physical Medicine and Rehabilitation, 73, 1147-1154.

Murray, M.P., Kory, R.C., Clarkson, B.H. (1969). Walking patterns in healthy old men. Journal of Gerontology, 24, 169-178.

Murray, M.P., Mollinger, L.A., Gardner, G.M., Sepic, S.B. (1984). Kinematic and EMG patterns during slow, free, and fast walking. Journal of Orthopaedic Research, 2, 272-280.

Nadeau, S., Arsenault, A.B., Gravel, D., Bourbonnais, D. (1999). Analysis of the clinical factors determining natural and maximal gait speeds in adults with a stroke. American Journal of Physical Medicine Rehabilitation, 78, 123-130.

Nakamura, R., Hosokawa, T., Tsuji, I. (1985). Relationship of muscle strength for knee extension to walking capacity in patients with spastic hemiparesis. Tohoku Journal of Experimental Medicine, 145, 335-340.

Olney, S.J., Elkin, N.D., Lowe, P.J., Symington, D.O. (1979). An ambulation profile for clinical gait evaluation. Physiotherapy Canada, 31, 85-90.

Olney, S.J., Griffin, M.P., Monga, T.N., McBride, I.D. (1991). Work and power in gait of stroke patients. Archives of Physical Medicine and Rehabilitation, 72, 309-314.

Olney, S.J., Griffin, M.P., McBride, I.D. (1994). Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. Physical Therapy, 74, 872-885.

Peat, M., Dubo, H.I.C., Winter, D.A., Quanbury, A.O., Steinke, T., Graham, R. (1976). Electromyographic analysis of gait: hemiplegic locomotion. Archives of Physical Medicine and Rehabilitation, 57, 421-425.

Perry, J. (1969). The mechanics of walking in hemiplegia. Clinical Orthopaedics and Related Research, 63, 23-31.

Perry, J. (1992). Gait Analysis: Normal and Pathological Function. Thorofare: SLACK.

Perry, J., Garrett, M., Gronley, J.K., Mulroy, S.J. (1995). Classification of walking handicap in the stroke population. Stroke, 26, 982-989.

Richards, C.L., Malouin, F., Dumas, F., Tardif, D. (1995). Gait velocity as an outcome measure of locomotor recovery after stroke. In R. Craik & C. Oatis (Eds.), Gait analysis: Theory and applications (pp. 355-364). St. Louis: C.V. Mosby, Inc.

Robertson, D.G.E., Winter, D.A. (1980). Mecahnical enerfy generation, absorption and transfer amongst segments during walking. Journal of Biomechanics, 13, 845-854.

Sjostrom, M., Fugl-Meyer, A.R., Nordin, G., Wahlby, L. (1980). Post-stroke hemiplegia; crural muscle strength and structure. Scandinavian Journal of Rehabilitation Medicine, Supp 7, 53-67.

Suzuki, K., Nakamura, R., Yamada, Y., Handa, T. (1990). Determinants of maximum speed in hemiparetic stroke patients. Tokohu Journal of Experimental Medicine, 162(4), 337-344.

Turnbull, G.I., Charteris, J., Wall, J.C. (1995). A comparison of the range of walking speeds between normal and hemiplegic subjects. Scandinavian Journal of Rehabilitation Medicine, 27, 175-182.

Vardaxis, V.G., Allard, P., Lachance, R., Duhaime, M. (1998). Classification of able-bodied gait using 3-D muscle powers. Human Movement Science, 17, 121-136.

Voigt, M., Sinkjaer, T. (2000). Kinematic and kinetic analysis of the walking pattern in hemiplegic patients with foot-drop using a peroneal nerve stimulator. Clinical Biomechanics, 15, 340-351.

von Schroeder, H.P., Coutts, R.D., Lyden, P.D., Billings Jr., E., Nickel, V.L. (1995). Gait parameters following stroke: a practical assessment. Journal of Rehabilitation Research and Development, 32(1), 25-31.

Wade, D.T., Langton-Hewer, R. (1987). Functional abilities after stroke: measurement, natural history, and prognosis. Journal of Neurology, Neurosurgery & Psychiatry, 50, 177-182.

Wade, D.T., Wood, V.A., Heller, A., Maggs, J., Hewer, R.L. (1987). Walking after stroke: measurement and recovery over the first three months. Scandinavian Journal of Rehabilitation Medicine, 19, 25-30.

Winter, D.A. (1981). Use of kinetic analyses in the diagnostics of pathological gait. Physiotherapy Canada, 33, 209-214.

Winter, D.A. (1991). The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological. Waterloo, Ontario: University of Waterloo Press.

Winter, D.A. (1995). A.B.C. (Anatomy, Biomechanics and Control) of Balance During Standing and Walking. Waterloo, Ontario: Waterloo Biomechanics.

Winter, D.A., Eng, J.J., Ishac, M.G. (1995). A Review of kinetic parameters in human walking. In R.L. Craik & C.A. Oatis (Eds.), Gait Analysis: Theory and Application (pp. 252-270). St. Louis, Missouri: Mosby-Year Book, Inc.

Chapter 3: ABSTRACT

Effects of Strength Training on Walking and Health-Related Quality of Life: A Double-blind Controlled Clinical Trial

Purpose: A common motor impairment following stroke is muscle weakness which has been shown to relate to walking performance. The purpose of this study was to determine the effect of maximal isokinetic strength training on walking performance, in addition to muscle strength and health-related quality of life, in chronic stroke survivors.

Methods: Twenty participants (61.2 ± 8.4 years) with chronic stroke were randomized into two groups on the basis of age, sex, and time since onset of stroke (stratified randomization). Both groups participated in a training program three times a week for six weeks. The experimental group undertook maximal concentric isokinetic strength training of the lower extremity flexors and extensors whereas the control group received passive range of motion exercises of the paretic lower limb with the use of the Kin-Com dynamometer. The Mann-Whitney *U* Test was used to compare the changes in score (posttraining-baseline) between the control and experimental groups for the following outcome measures: composite lower extremity strength score, walking speed (level and stair-walking) and health-related quality of life measure (SF-36 physical and mental health scales).

Results: Although there was an overall training effect on strength and walking speed for both groups, no differences in the improvement of walking speed were detected between groups despite the trend ($p = 0.06$) towards greater strength improvement in the experimental group. No changes in SF-36 scores were found in either group.

Conclusions: Despite previous reports of high correlations between strength and walking performance, intervention aimed at increasing strength did not induce improvements in walking. The results of this study stress the importance of controlled clinical trials in

determining the effect of specific treatment approaches. Strength training in conjunction with other task-related training may be indicated.

3.1 INTRODUCTION

Stroke, defined as an "acute neurologic dysfunction of vascular origin…with symptoms and signs corresponding to the involvement of focal areas of the brain" (World Health Organization, 1989), is one of the leading causes of disability. In Canada, it is estimated that 35 000 persons experience a stroke each year (Mayo et al., 1996). Although the incidence is decreasing, the prevalence of stroke in Canadian society has increased due to the growth in elderly population and improved survival rates of stroke victims (Petrasovits and Nair, 1994). A large proportion of these stroke survivors is left with significant impairments and disabilities (Dombovy et al., 1986; Mayo et al., 1999).

Functional abilities such as level-walking and stair-walking are often compromised following stroke and are generally described as slow and asymmetric (Bohannon & Walsh, 1991; Hesse et al., 1994; Turnbull et al., 1995; von Schroeder et al., 1995). Several studies have reported mean ranges for walking speeds of stroke survivors to be between 0.3 to 0.8 m/s for self-selected speed (Brandstater et al., 1983; Nadeau et al., 1999; Roth et al., 1997; vonSchroeder et al., 1995) and 0.7 to 1.1 m/s for maximum safe speed (Nadeau et al., 1999; Nakamura et al., 1985; Suzuki et al., 1999). The walking speed required to cross a street within the time allowed by a traffic signal was found to be 1.2 m/s (Lerner-Frankiel et al., 1986). That is, even individuals among the highest functioning group of stroke survivors will be limited in community activities. In addition, walking up stairs has also been reported as slow following stroke (45 to 51 stairs/min) (Sharp & Brouwer, 1997; Texeira-Salmela et al., 1999) compared to healthy adults (114 stairs/min) (Olney et al., 1979).

In a study on rehabilitation goals, Bohannon et al. (1988) found that walking function was the most frequently mentioned goal by individuals with stroke and sometimes it was the only one mentioned. For this reason, gait retraining is commonly the primary focus in the rehabilitation of stroke survivors. Despite this effort to regain optimal walking function during the first six months of rehabilitation, many stroke survivors are left with significant limitations in community activities (Duncan, 1994; Gresham et al., 1979) possibly due to the slowness of their walking or alternatively their lack of endurance (Mayo et al., 1999). Given the important role of walking to independence and the continuing problem of gait in the chronic stroke survivor, it is important to determine the type of training that is most effective in addressing this important aspect of mobility.

Besides a decline in activity level, post-stroke survivors also report a decrease in quality of life (Ahlsio et al., 1984). Depression, anxiety, physical dependency and inability to return to work are some of the factors contributing to diminished quality of life after stroke (Ahlsio et al., 1984; Niemi et al., 1988; Viitanen et al., 1988). Ahlsio et al. (1984) reported that impairment of motor function and walking often limited opportunities for leisure or social functions in stroke survivors, thereby affecting overall quality of life. It has been suggested that interventions aimed at improving function may lead to improved quality of life in individuals with stroke (Teixeira-Salmela et al., 1999).

A common motor impairment following stroke is muscle weakness (Adams et al., 1990; Canning et al., 1999; Chan, 1986), possibly as a result of a decrease in number of motor units (McComas et al., 1973), disrupted recruitment order of motor units (Grimby & Hannerz, 1973), decrease in motor unit firing rates (Rosenfalck & Andreassen, 1980), or muscle atrophy following disuse (McComas, 1994). Characteristics of muscle strength following stroke include a reduction in isometric and isokinetic torque generation (Andrews, 2000; Bohannon & Smith, 1987a; Nadeau et al., 1997; Sharp and Brouwer, 1997) in addition to a slowness to generate torque (Canning et al., 1999). The severity of isometric and isokinetic torque reduction in stroke survivors has been shown to relate to the performance of several functional tasks such as transfers (Bohannon, 1988), standing (Bohannon, 1987a), level-walking (Bohannon, 1986 & 1987b; Nadeau et al., 1997; Nakamura et al., 1985; Nakamura et al., 1988) and stair-walking (Bohannon & Walsh, 1991) suggesting that strength training could lead to improved functional performance.

In the past, strength testing and training in persons with spasticity have been controversial issues. Bobath (1978) advocated that decreased muscle power was not due to weakness but to the opposition of spastic antagonists. In addition, muscle strengthening was not recommended as it was thought to increase spasticity and reinforce abnormal movement (Bobath, 1978). Recent studies, however, have shown that strength testing can be done reliably in individuals with spasticity (Bohannon & Andrews, 1987; Eng et al., in press; Tripp & Harris, 1991) and that strength training is not associated with increases in spasticity (Damiano et al., 1995; Horvat, 1987; Sharp and Brouwer, 1997).

Several promising studies have suggested that lower extremity strength training can improve functional performance in addition to self-perceived health in individuals with stroke (Dean et al., 2000; Duncan et al., 1998; Engardt et al., 1995; Karimi, 1996; Sharp & Brouwer, 1997; Teixeira-Salmela et al., 1999; Wilder & Sykes, 1982). Many of these studies have combined strength training with other types of training; only a few have investigated the functional gains achieved by strength training alone. Maximal isokinetic strengthening of the knee musculature has been found to significantly improve sit-to-stand and walking performance in stroke survivors (Engardt et al., 1995; Sharp & Brouwer, 1997). Furthermore, combined bilateral isokinetic training using the kinetron (Wilder & Sykes, 1982) as well as training of isolated flexor and extensor muscle groups of the paretic lower extremity (Karimi, 1996) have also been reported to improve walking performance.

These studies suggest that strength training can be done with positive effects on function in persons with stroke. However, they could be criticized for their study design. The lack of a control group in these studies poses some validity questions. The reported changes in performance could be attributed to several factors other than strengthening, e.g., joint mobility, maturation effects, testing effects, or simply the attention from a therapist two to three times a week. One study that has included a control group (Glasser, 1986) found improvements in gait performance in all participants but no significant differences between the two groups. The sample for the study was a group of acute stroke survivors, therefore, the control group was receiving therapeutic exercises and gait training while the experimental group received the same treatment in addition to isokinetic strengthening of bilateral lower extremities using a kinetron. Although a control group was included, the type of design used provided a comparison between two intervention strategies for gait training rather than a direct assessment of strength training effects on gait.

In the present study, because we targeted chronic stroke survivors, we were able to institute a double-blind controlled trial where the control group did not receive treatment other than to minimize the effects of variables that might threaten the internal validity of the study. Apart from the added resistance in the experimental group, the two groups followed the same activities, i.e. they both attended the exercise sessions three times a week for six weeks, undertook the same warm-up and cool-down routine, and experienced the same joint mobility through a specified range of motion for three sets of ten repetitions in each joint of the paretic lower limb.

The purpose of the study was to determine the effect of six weeks of maximal isokinetic strength training on strength, walking (level-walking and stair-walking), and health-related quality of life in chronic stroke survivors. The following null hypotheses were tested: Following a 6-week training program, there will be no differences between the experimental and control groups for change scores (post-training – baseline scores) of 1) lower extremity muscle (hip, knee and ankle flexors and extensors) strength as measured by the isokinetic dynamometer, 2) walking performance as measured by the speed of level and stair walking and 3) health-related quality of life as measured by the Short Form 36 (SF-36) physical and mental health summary scales.

3.2 METHODS

3.2.1 Participants

The study was approved by the local university's Research Ethics Board and the hospital Research Advisory and Review Committee. Twenty community-dwelling stroke survivors, who had residual unilateral weakness, were recruited on a volunteer basis. Participants were recruited in various ways: recruitment notices (Appendix VII) were posted in all community centres and hospitals of the Lower Mainland, letters were sent to previous patients at the local Rehab Centre (within the past 5 years) inviting them to participate (Appendix VIII), and introductory presentations were given by the investigator to members of seven different stroke clubs in the Lower Mainland.

The inclusion criteria were as follows: 1) have a history of a single cerebrovascular accident of at least six months prior to participating in the study, 2) able to walk independently for a minimum of 40 metres (with rest intervals) with or without assistive device, 3) achieve a minimum of stage 3 for the leg and foot on the Chedoke-McMaster Stroke Assessment, 4) have an activity tolerance of 45 minutes with rest intervals, and 5) not participating in any formal therapy program.

Participants were excluded if they: 1) had comprehensive aphasia, 2) were not medically stable (i.e., have uncontrolled hypertension, arrhythmia, congestive heart failure, or unstable cardiovascular status), or 3) had significant musculo-skeletal problems due to conditions other than stroke. Informed consent (Appendix I) was obtained from each participant. Also, a letter (Appendix II) was sent to the each participant's physician, who was later contacted by phone to ensure the inclusion and exclusion criteria were met prior to the beginning of the study.

Demographic data collected from all participants included age, sex, time since the onset of stroke, type of stroke, the affected (paretic) side, degree of resistance to passive movement in the knee extensors and ankle plantarflexors, stage level on the Chedoke-McMaster Stroke Assessment for the foot and leg (Gowland et al., 1995) (Appendix III), and use of mobility aids. The characteristics of each participant and of each group are presented in Tables 1 and 2 respectively. Resistance to passive movement was evaluated while passively flexing and extending the limb as described by Ashworth (1964) while participants were in a supine position and graded on an ordinal scale from zero to five based on the Modified Ashworth Scale (MAS) (Bohannon & Smith, 1987b). See Appendix IV for description of the MAS.

3.2.2 Design

For group assignment, stratified randomization was chosen based on three confounders that could potentially impact the response to treatment (Domholdt, 1993; Tate et al., 1999). Participants were stratified on the basis of sex (male, female), age (low: 50-59 years, high: 60 years or above), and the time since onset of stroke (low: 6 months-2 years, high: above 2 years) before randomly assigning them to the treatment and control groups. Participants had no knowledge of which training program they were involved; they were aware that they were involved in one of the two different "leg training" programs (see consent form in Appendix I). One researcher undertook all randomization and stratification procedures and training sessions, while another researcher undertook all assessment evaluations without knowledge of the participants' grouping in order to achieve researcher blinding.

A control group was included to control for effects due to maturation, testing, attention from a therapist, stretching exercises during warm-up/cool-down, and joint mobility during exercises. A double-blind design was chosen to control effects due to participant expectations and researcher expectations.

3.2.3 Outcome Measures

The outcome measures (measures of lower extremity muscle strength, level-walking and stair-walking performance, and health-related quality of life) were undertaken 2-4 days before and 2-4 days after the intervention for both groups.

3.2.3.1 Isokinetic Strength of Lower Extremities

The Kin-Com Isokinetic Dynamometer (Chattanooga Group Inc., TN) was used to measure the strength of hip flexors/extensors, knee flexors/extensors, and ankle dorsiflexors/ plantarflexors bilaterally. These muscle groups were selected because of their important role in walking; Eng & Winter (1995) reported that the flexion/extension moments of the hip, knee and ankle accounted for 82% of the total work over a stride as opposed to 15% and 3% in the frontal and transverse planes respectively. The Kin-Com has been shown to be accurate for position, velocity, and force (Farrell & Richards, 1986; Mayhew et al., 1994). The calibration of the instrument was tested prior to the study with known weights and was accurate to within ±1 N. All participants had a practice session 2-4 days before the actual testing day to reduce the learning effect as recommended by Eng et al. (in press).

An angular velocity of 60°/s was initiated for the isokinetic strength assessment. This velocity was selected because the majority of our participants had difficulty generating faster movements. If a participant was not able to achieve an angular velocity of 60°/s, 30°/s was used for that specific joint for both limbs during all testing and training sessions.

Preloads were determined during the practice cycles for each participant, joint and direction of motion, and were set at a minimum of 50% of the peak torque values. Three submaximal cycles and one maximal cycle were completed as practice on the Kin-Com as per Kramer's (1990) protocol. Participants were asked to "push or pull as hard as possible" throughout their available range of motion (as assessed by their active range on the paretic side) for four to six repetitions so that consistency of the force-angle profile was found across three repetitions. The testing range of motion was consistent for all testing within each participant. Body positioning and stabilization is documented in Appendix IX. This protocol has been shown to be reliable in stroke survivors with intraclass correlation coefficients > 0.88 for average torque values (Eng et al., in press).

The three torque-angle curves of each set of contractions were ensembled to obtain a mean curve and average torque over the range was extracted from this single curve. Since this torque value is derived from three trials, it is most correctly denoted as "mean average torque", however, the simpler term of "average torque" is used in this paper. All torque values were corrected for the effect of gravity on the lower extremity segment and the effect of gravity on the cuff of the dynamometer. This gravity-correction procedure has been shown to be accurate (Finucane et al., 1994). Average torque values were normalized to body mass (Nm/kg).

3.2.3.2 Level-walking Performance

Instrumentation and data collection procedures for gait assessment have been described in chapter 2. Temporal variables used in this study as outcome measures include: gait speed at self-selected and maximal speeds, and cadence and stride length at self-selected speeds. Gait speed has been recognized as an indicator of gait performance (Andriacchi et al., 1977), sensitive enough to reflect physiological and functional changes (Richards et al., 1995) and has been shown to be a reliable measure (Olney et al., 1979). To understand the kinematic and kinetic mechanisms underlying the changes in the temporal measures, angle ranges, maximum moments, maximum powers and total work performed during gait at self-selected speeds were also included. Only the sagittal plane variables of the paretic lower limb were analyzed as they were the most relevant to the muscle groups addressed in the training program. Furthermore, these variables were selected because of their significant correlations with gait speed in stroke survivors (see chapter 2).

3.2.3.3 Stair-walking Performance

Participants were asked to climb up four 18cm-steps at their "most comfortable speed" (i.e. self-selected speed) employing their usual pattern of foot placement and hand support and then "safely as fast as possible" (i.e. maximum speed). The average time of ascent over two trials was calculated for each testing condition (i.e. self-paced and maximum pace) and converted to stairs per minute. This protocol has been described elsewhere (Olney et al., 1979) and has been shown to be reliable with a reliability coefficient of 0.90 with healthy adults.

3.2.3.4 Health-related Quality of Life

Health-related quality of life (HRQoL) was measured with the use of the SF-36 physical (PCS) and mental (MCS) health summary scales (Ware et al., 1994). The PCS and MCS were developed from the SF-36 health survey (Ware et al., 1993) as summary measures of the physical and mental health. The SF-36 measures eight health attributes: physical functioning, role limitations due to physical health, role limitations due to emotional problems, energy and fatigue, emotional well-being, social functioning, pain, and general health (Appendix X). These eight subscales were aggregated into two summary measures (PCS and MCS) based on a factor analysis of subscale scores in a U.S. general population sample (Ware et al., 1994). In this study, the two summary scales were used as outcome measure for HRQoL. Both the SF-36 PCS and MCS have been shown to be reliable in general populations as well as in patient subgroups with a reliability coefficient (Cronbach's coefficient alpha) ranging from 0.84 to 0.94 (Ware et al., 1994). The SF-36 survey has also been shown to be a valid measure of physical and mental health after stroke (Anderson et al., 1996).

3.2.4 Intervention

3.2.4.1 Experimental Group

The training program consisted of three 45-minute-sessions per week for six consecutive weeks for a total of 18 sessions. Each session began with a five-minute gentle warm-up, followed by five minutes of mild stretching exercises of the paretic lower extremity (Appendix XI). Strength training was done with the use of the Kin-Com dynamometer. Three sets of ten repetitions of maximal effort concentric hip flexion/extension, knee flexion/extension, and ankle dorsiflexion/plantarflexion of the paretic limb were performed for approximately 30 minutes. Strength training was done in the same positions, same angular velocities and through the same range of motion as the testing protocol. Rest breaks were provided as deemed necessary by the participants. The training program ended with a 5-minute cool-down consisting of stretching exercises. During the training sessions, heart rate and blood pressure were continuously monitored and recorded.

3.2.4.2 Control Group

The control group followed the same program as the experimental group except that the isokinetic exercises were replaced by passive range of motion exercises with the use of the Kin-Com dynamometer. The exercise protocol was consistent with the testing protocol (i.e. same positioning, angular velocities and range of motion). Participants were instructed to relax the limb as it was moved into flexion and extension by the dynamometer. Three sets of ten repetitions were performed for each joint of the paretic lower extremity.

3.2.5 Data Analysis

Descriptive statistics were performed for participants' characteristics and all outcome measures at baseline and post-training for each group. A composite score of percent strength improvement for the paretic and non-paretic limbs was calculated for each participant. The percent strength improvement ($[a-b]/b$ where a is the post-training torque measure and b is the baseline torque measure) was calculated for each of the six muscle groups and then summated to provide an overall composite lower extremity percent strength improvement for each participant. Percent improvement for each muscle group, as opposed to absolute change in average torque values, was used to account for differences in relative force-generating capacity of different muscle groups as well as differences in baseline status between participants. A mean composite strength improvement score was calculated each for the control group and the experimental group. Due to the small sample size, a nonparametric test was used for comparisons between groups. The Mann-Whitney *U* Test for two independent samples was used to compare differences between the control and experimental groups for a) baseline outcome measures and b) change scores (or composite scores in the case of strength changes) for each outcome measure. In addition, the kinematic (joint angles) and kinetic (joint moments, powers and work) variables of gait were compared for the pooled group ($N = 20$) between baseline and post-training measures using the Wilcoxon Signed Rank Test for two related samples. A significant level of $p < 0.05$ (two-tailed) was selected for all statistical tests.

3.3 RESULTS

3.3.1 Baseline Performance

3.3.1.1 Participant characteristics

Twenty participants, ten in each group, completed the training program. All participants attended all 18 sessions of training (make-up training sessions were held within the same week of missed sessions in case of statutory holidays) and none dropped out from the study. The characteristics of each participant are presented in Table 1. No significant differences (p>0.05) in participant demographics (age, time since the onset of stroke, height, mass) were found between the experimental and control groups (Table 2). Levels of increased muscle tone and recovery stage, as measured by the MAS and the Chedoke-McMaster Stroke Assessment respectively, were also of comparable ranges in the two groups (Table 2).

3.3.1.2 Muscle strength

The joint range of motion used during the isokinetic tests was similar in the two groups with a mean of 54° for the hip, 60° for the knee, and 23° for the ankle (Table 3). All twenty participants were able to complete the isokinetic strength test at an angular velocity of 60°/s bilaterally at the hip. Six participants (three participants in each group) could not complete the test at 60°/s at the knee and were tested bilaterally at 30°/s. At the ankle, only two participants (one participant in each group) completed the test at 60°/s while the remaining eighteen were tested at 30°/s bilaterally.

Table 1: Characteristics of each participant (N = 20).

Code	Group	Sex	Age (yr)	Time since stroke (yr)	Paretic Side	Height (cm)	Mass (kg)	MAS		CM-Stage		Mobility Aid
								Ankle	Knee	Foot	Leg	
SG02	E	M	60	2	Right	188	86.3	1	1+	3	4	quad cane
SG03	E	F	52	9	Right	153	64	1	1+	3	5	none
SG04	E	M	60	9	Left	178	81	1	1	5	6	none
SG05	E	M	82	10	Left	171.5	72.8	1	2	4	5	none
SG06	E	F	59	1.5	Left	158	67.3	1+	1	4	5	none
SG07	E	M	53	5	Left	159	58.5	1+	0	3	5	std cane
SG08	C	F	52	3.5	Right	178	85	1	1	3	5	none
SG09	C	M	55	4	Right	164.5	78.5	0	1	3	4	none
SG10	C	M	65	2	Right	164	81.6	1	0	3	5	std cane
SG11	C	M	59	3.5	Right	178	84.5	0	0	3	5	none
SG13	C	M	62	5	Left	177	99.4	1+	0	5	6	none
SG14	C	F	69	3	Left	161.5	59	2	1+	3	4	std cane
SG15	E	M	55	1.5	Left	182	93.8	1	0	3	3	std cane
SG16	E	M	54	4.5	Left	165	69.6	1+	0	3	5	none
SG18	E	M	57	3.5	Right	184	84.3	1+	0	3	4	none
SG19	C	M	58	1.5	Left	181	96	1+	0	3	3	std cane
SG20	C	F	76	2.5	Right	164	54	1	0	4	5	std cane
SG21	C	M	55	2	Right	183	75	1	0	3	5	none
SG22	C	M	68	4.5	Right	180.3	82.1	1	0	3	5	none
SG24	E	F	72	3	Right	143	52	1	0	5	5	none

Abbreviations: Control (C), Experimental (E), Female (F), Male (M), Modified Ashworth Scale (MAS), Chedoke-McMaster stage (CM-stage), Standard cane (std cane).

Table 2: Characteristics of the participants in the experimental and control groups (N = 10 per group).

	Experimental		Control	
	Mean	SD	Mean	SD
Age (yrs)†	60.4	9.5	61.9	7.5
Mass (kg)†	73.0	13.2	79.5	14.3
Height (cm)†	168.2	14.9	173.1	8.5
Time since stroke (yrs)†	4.90	3.28	3.15	1.16

	Experimental	Control
Type of stroke (Ischemic/Hemorrhagic/ Unspecified)	6/4/0	5/3/2
Gender (M/F.)	7/3	7/3
Paretic side (Right/Left)	4/6	7/3
Dominance (Right/Left)	9/1	8/2
Mobility aid (Cane/None)	3/7	4/6
Chedoke-McMaster (stage range)	3-6	3-6
Modified Ashworth Scale (grade range)	0-2	0-2

† Mann-Whitney *U* Test result was not significant (p>0.3) between the experimental and control groups

Table 3: Mean angle values for each movement (N = 10 per group).

	Ankle	Knee	Hip
Start position for extension/plantarflexion motion = End position for flexion/dorsiflexion motion	E: 10.0° (3.3) C: 9.8° (0.6)	E: 74.3° (5.0) C: 77.0° (3.5)	E: 93.0° (6.7) C: 95.0° (5.8)
End position for extension/plantarflexion motion = Start position for flexion/dorsiflexion motion	E: 33.0° (3.5) C: 32.5° (3.5)	E: 16.4° (3.3) C: 14.5° (1.6)	E: 39.3° (2.5) C: 40.0° (2.4)
Range of motion	E: 23.0° (5.4) C: 22.7° (3.4)	E: 57.9° (6.4) C: 62.5° (4.2)	E: 53.7° (8.0) C: 55.0° (5.8)

Hip: 0° = neutral position (pelvis and thigh segment aligned) with positive value indicating increasing flexion; Knee: 0° = neutral position (thigh and shank segment aligned) with positive value indicating increasing flexion; Ankle: 0°= neutral position (foot segment perpendicular to shank segment) with positive value indicating increasing plantarflexion. Standard deviation in parentheses.

Abbreviations: Experimental group (E), Control group (C)

There were no significant differences (p>0.05) in any of the baseline average torque values between the two groups (Table 4). As expected, average torque values were lower on the paretic side compared to the non-paretic side in all participants (Figure 1). The strength of the paretic limb relative to that of the non-paretic limb (i.e. paretic/non-paretic ratio) ranged between 0.19-0.78 in the experimental group and 0.23-0.89 in the control group. Generally, distal muscle groups were more affected (i.e. greater deficit relative to the non-paretic side) than proximal muscle groups and flexors more affected than extensors except at the ankle joint where the plantarflexors were more affected than the dorsiflexors.

3.3.1.3 Level-walking, Stair-walking, and HRQoL

All twenty participants were community ambulators; three and four participants in the experimental and control groups respectively used canes for walking. For stair climbing, all participants but one used the handrail on the non-paretic side for assistance and five and two participants in the experimental and control groups respectively used a "step to" pattern instead of a "step through" pattern to ascend the stairs.

There were no significant differences (p>0.05) in baseline measures of walking performance between the two groups (Table 5) indicating a similarity in functional level across groups. Gait speed was slow for both groups (0.45-0.46 m/s) compared to reported values for the healthy elderly (1.2-1.5 m/s) (Himann et al., 1988; Murray et al., 1969), with a 57% and 51% increase from self-selected to maximal speed walking in the experimental and control groups respectively. Similarly, stair climbing speed increased by 32% and 38% from self-selected to maximal speeds in the experimental and control groups respectively (Figure 2). The low speeds of level-walking in all participants were accompanied by low values in

Table 4: Baseline average torque (Nm/kg) of each muscle group in the experimental and control groups (N = 10 per group) and the *p* value for test of baseline differences.

	Experimental Group	Control Group	*p* value
Hip Extension			
Paretic	0.67 ± 0.46	0.87 ± 0.49	0.33
Non-paretic	0.79 ± 0.42	0.94 ± 0.32	0.36
Hip Flexion			
Paretic	0.45 ± 0.20	0.47 ± 0.16	0.88
Non-paretic	0.71 ± 0.28	0.75 ± 0.16	0.82
Knee Extension			
Paretic	0.45 ± 0.31	0.64 ± 0.32	0.08
Non-paretic	1.08 ± 0.36	1.27 ± 0.45	0.26
Knee Flexion			
Paretic	0.11 ± 0.11	0.14 ± 0.13	0.71
Non-paretic	0.48 ± 0.22	0.52 ± 0.19	0.55
Ankle Plantarflexion			
Paretic	0.17 ± 0.15	0.20 ± 0.14	0.36
Non-paretic	0.96 ± 0.34	0.91 ± 0.24	0.55
Ankle Dorsiflexion			
Paretic	0.15 ± 0.15	0.15 ± 0.12	0.65
Non-paretic	0.50 ± 0.16	0.48 ± 0.09	0.71

Values reported as mean ± one standard deviation
Comparisons between the experimental and control groups are by Mann-Whitney *U* Test

Table 5: Walking performance and SF-36 scores at baseline and post-training in the experimental and control groups (N = 10 per group).

	Baseline†	Post-training
Self-selected gait speed (m/sec)		
Experimental	0.45 ± 0.25	0.49 ± 0.20
Control	0.46 ± 0.25	0.55 ± 0.29
Maximal gait speed (m/sec)		
Experimental	0.70 ± 0.37	0.74 ± 0.33
Control	0.68 ± 0.34	0.75 ± 0.36
Cadence (steps/min)		
Experimental	79.21 ± 19.49	81.73 ± 18.53
Control	68.65 ± 21.40	74.25 ± 21.45
Stride length (m)		
Experimental	0.68 ± 0.30	0.72 ± 0.23
Control	0.77 ± 0.20	0.84 ± 0.22
Self-selected stair climbing speed (stairs/sec)		
Experimental	0.65 ± 0.27	0.65 ± 0.21
Control	0.61 ± 0.19	0.69 ± 0.22
Maximal stair climbing speed (stairs/sec)		
Experimental	0.84 ± 0.31	0.87 ± 0.30
Control	0.82 ± 0.21	0.90 ± 0.26
SF-36 (physical health)		
Experimental	38.6 ± 6.7	39.9 ± 8.1
Control	40.6 ± 7.0	39.8 ± 7.5
SF-36 (mental health)		
Experimental	50.1 ± 13.4	51.8 ± 13.4
Control	55.6 ± 7.3	54.5 ± 9.6

Values reported as mean ± one standard deviation

† No significant (p>0.2) differences between the experimental and control groups in baseline measures (comparisons were made by Mann-Whitney *U* Test)

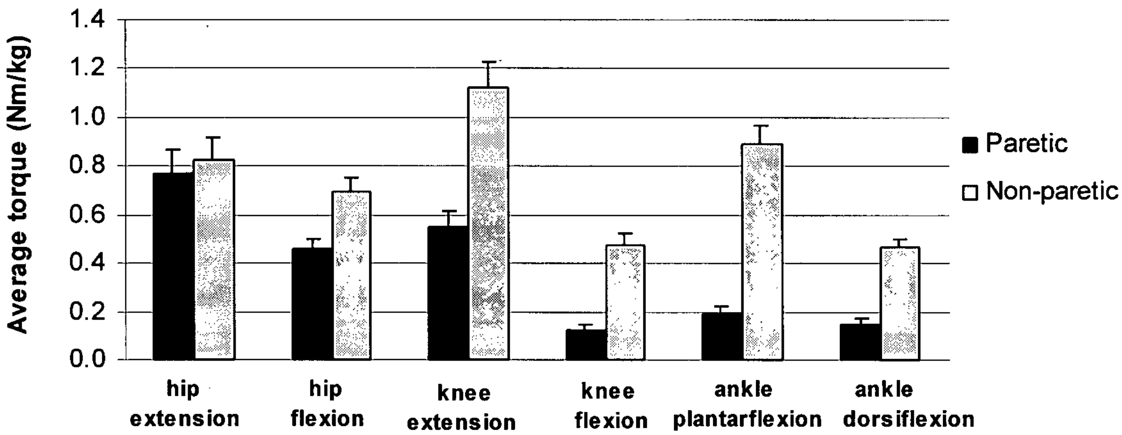


Figure 1. Mean and one standard error of baseline average torque values (Nm/kg) of the paretic and non-paretic lower extremities for all participants (N = 20).

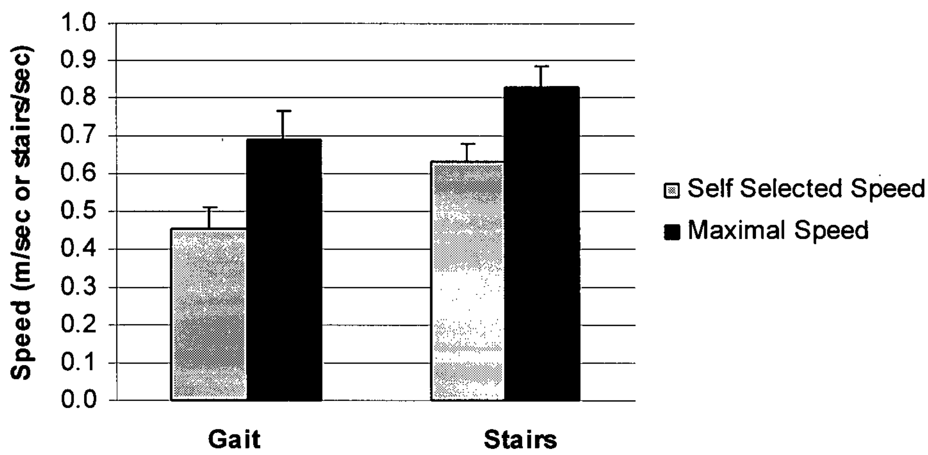


Figure 2. Mean and one standard error of baseline measures of gait speed (m/sec) and stair climbing speed (stairs/sec) for all participants (N = 20).

angle ranges, power variables and work done at all the three joints of the paretic lower limb (Table 6) relative to reported values in the healthy elderly walking at self-selected speeds (Winter, 1991).

No significant differences were found in SF-36 PCS or MCS scores between groups at baseline (Table 5). In the experimental group, 7 and 5 participants reported lower than norm-referenced values based on the general U.S. population of age 65 and over (PCS = 41.3, MCS = 51.8) (Ware, 1995), whereas in the control group, 5 and 3 participants reported lower values than expected in the PCS and MCS respectively.

3.3.2 Training Effects

3.3.2.1 Muscle Strength

For both the experimental and control groups, the means for the individual muscle groups were higher post-training than at baseline except for the hip extensors. Differences in composite scores of strength improvement between groups did not reach statistical significance at the 0.05 level for the paretic and non-paretic limbs; however, there was a trend for the experimental group to show greater improvements than the control group on the paretic side ($p = 0.06$) (Table 7). In the experimental group, average torque changes in the paretic limb represented 7% to 155% improvements in strength whereas in the control group, strength improvements were in the range of 1% to 58%. The experimental group showed greater improvements in strength over the control group in all of the muscle groups tested. Figure 3 summarizes mean percentage changes in average torque of each muscle group on the paretic side in the experimental and control groups.

Table 6: Mean joint angle range (degrees), moment (Nm/kg), power (W/kg) and work (J/kg) over the stride on the paretic lower limb at baseline and post-training in all participants (N = 20).

Variable	Baseline	Post-training	*p* value
Hip range	34.6 ± 19.1	37.1 ± 14.2	0.29
Knee range	33.7 ± 21.3	36.0 ± 17.4	0.07
Ankle range	17.7 ± 5.1	21.3 ± 6.5	0.02*
Max. hip ext moment	0.33 ± 0.17	0.41 ± 0.19	0.13
Max. hip flex moment	0.32 ± 0.21	0.37 ± 0.24	0.12
Max. knee ext moment	0.29 ± 0.21	0.26 ± 0.24	0.51
Max. knee flex moment	0.22 ± 0.25	0.24 ± 0.21	0.67
Max. ankle pf moment	0.64 ± 0.22	0.69 ± 0.21	0.24
Max. hip power gen	0.50 ± 0.64	0.62 ± 0.62	0.01*
Max. hip power abs	0.19 ± 0.16	0.21 ± 0.19	0.39
Max. knee power gen	0.19 ± 0.15	0.24 ± 0.16	0.08
Max. knee power abs	0.31 ± 0.37	0.41 ± 0.49	0.02*
Max. ankle power gen	0.40 ± 0.43	0.48 ± 0.47	0.08
Max. ankle power abs	0.21 ± 0.15	0.26 ± 0.16	0.11
Total hip work†	0.18 ± 0.13	0.23 ± 0.16	0.00**
Total knee work	0.12 ± 0.09	0.13 ± 0.09	0.38
Total ankle work	0.12 ± 0.07	0.13 ± 0.06	0.40

Values reported as mean ± one standard deviation

† Total work is the sum of the positive and negative work done at the joint in the sagittal plane.

Comparisons between baseline and post-training measures are by Wilcoxon Signed Rank Test

* indicates significance at $p<0.05$

** indicates significance at $p<0.01$

Abbreviations: Maximum (max), Extension (ext), Flexion (flex), Plantarflexion (pf), Generation (gen), Absorption (abs)

Table 7: Composite scores* of strength improvement in the experimental and control groups (N = 10 per group) and the associated *p* value for test of differences.

Side	Experimental Group	Control Group	*p* value
Paretic	507 ± 559	142 ± 193	0.06
Non-paretic	57 ± 89	23 ± 70	0.41

* Composite scores calculated as the sum of the percent improvement ([posttest-pretest]/pretest) of all six muscle groups.
Values reported as mean ± one standard deviation
Comparisons between the scores of the experimental and control groups are by Mann-Whitney *U* Test

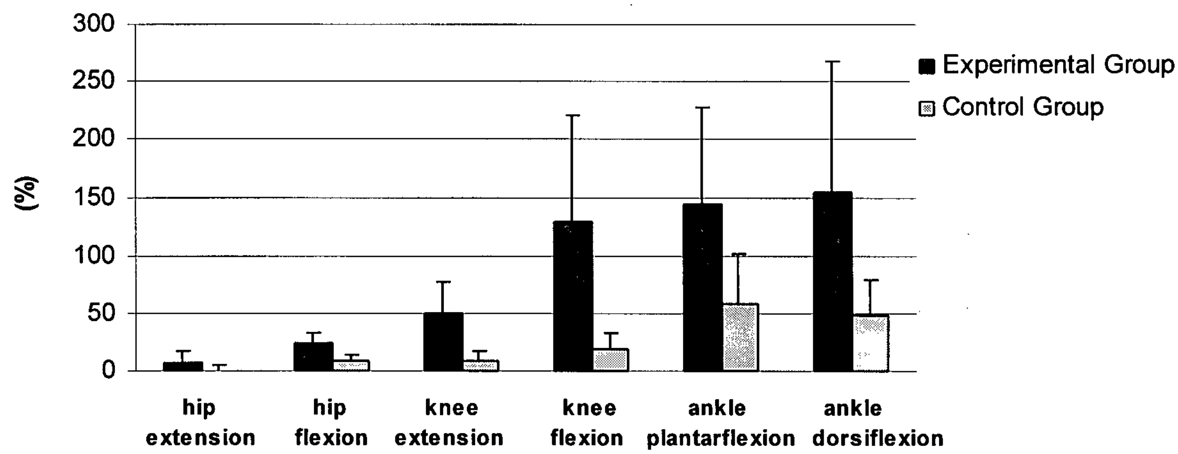


Figure 3: Mean and one standard error of percentage change in average torque (Nm/kg) of each muscle group on the paretic side in the experimental and control groups (N = 10 per group) following 6 weeks of training.

3.3.2.2 Walking Performance

There were no group differences in change scores for any of the walking performance measures (gait speed, cadence, stride length, and stair climbing speed) between baseline and post-training (Table 8). No changes in hand placement or step patterns were observed in either group during stair ascent except for one participant in the experimental group, who changed from a "step to" to a "step through" pattern.

Pooling data across groups revealed an increase in the mean of all measured temporal variables of walking (level and stair walking) post-training (6% to 17% improvements) (Figure 4). As differences in change scores of level-walking performance between groups were not significant, the two groups' data were pooled for the analysis of changes in kinematic and kinetic variables of gait following training. The improvement in temporal walking performance of all participants was accompanied by a significant ($p<0.05$) increase in ankle range, maximum hip power generation, maximum knee power absorption, and total hip work during gait (Table 6). When examining the power curves, the maximum hip power occurred during the pull-off phase of gait and corresponded to the H3 phase generated by the hip flexors. The maximum power absorbed by the knee occurred during propulsion and corresponded to the K3 phase absorbed by the knee extensors controlling the collapse of the knee that resulted from the ankle push-off and hip pull-off forces. The increase in ankle range was likely a result of the increase ($p = 0.08$) in ankle power generated during push-off (A2).

Table 8: Mean change scores in walking performance and SF-36 for the experimental and control groups (N = 10 per group) and the *p* values for test of differences in change score.

	Experimental Group	Control Group	*p* value
Self-selected gait speed (m/sec)	0.04 ± 0.13	0.09 ± 0.07	0.29
Maximal gait speed (m/sec)	0.05 ± 0.09	0.07 ± 0.08	0.65
Cadence (steps/min)	2.53 ± 6.59	5.60 ± 5.50	0.26
Stride length (m)	0.03 ± 0.13	0.07 ± 0.11	0.41
Self-selected stair climbing speed (stairs/sec)	0.00 ± 0.12	0.08 ± 0.06	0.17
Maximal stair climbing speed (stairs/sec)	0.03 ± 0.08	0.08 ± 0.10	0.26
SF-36 (physical health)	0.74 ± 7.15	-0.73 ± 5.81	0.55
SF-36 (mental health)	1.73 ± 7.34	-1.07 ± 10.13	0.60

Values reported as mean ± one standard deviation
Comparisons between the experimental and control groups are by Mann-Whitney *U* Test

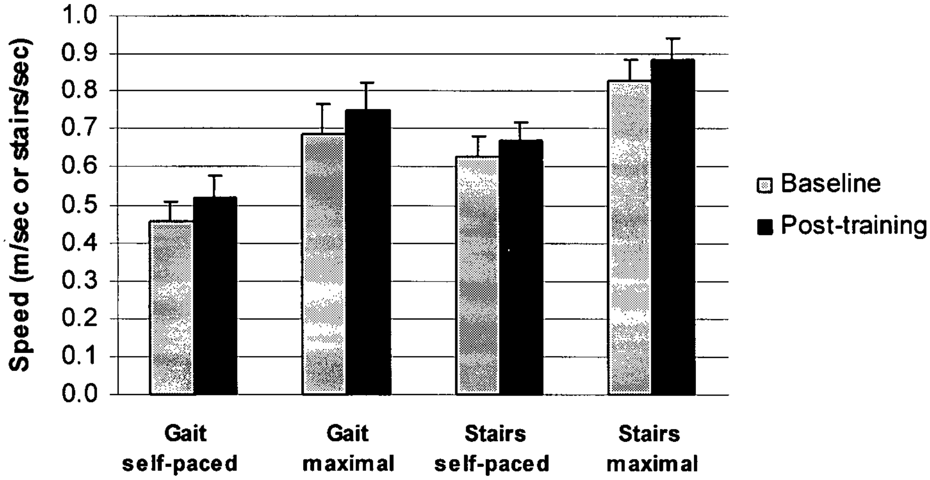


Figure 4: Mean and one standard error of baseline and post-training measures of gait speed (m/sec) and stair climbing speed (stairs/sec) for all participants (N = 20).

3.3.2.3 HRQoL

There were no significant differences in the change scores of both the physical and mental health components of the SF-36 between the two groups (Table 8). The reported values post-training remained very close to baseline values in both groups (Table 5, Figure 5).

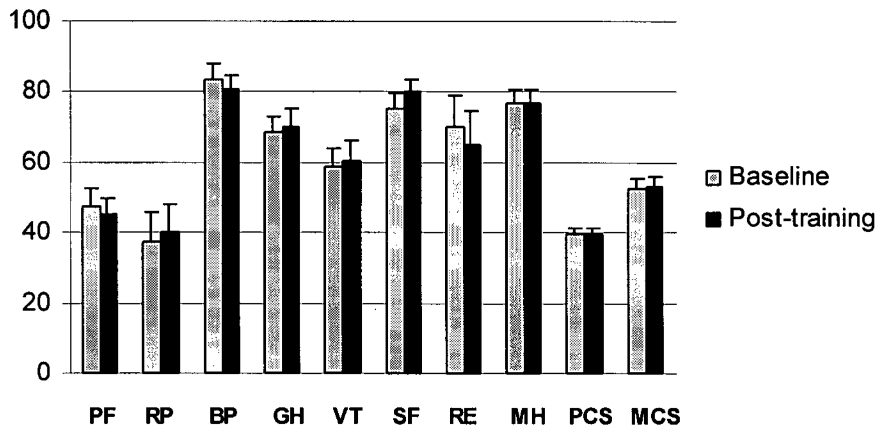


Figure 5: Mean and one standard error of baseline and post-training measures of SF-36 scores (including the eight subscales and summary scores) for all participants (N = 20). *Abbreviations*: Physical functioning (PF), Role-Physical (RP), Bodily pain (BP), General health (GH), Vitality (VT), Social functioning (SF), Role-Emotional (RE), Mental health (MH), Physical component summary scale (PCS), Mental component summary scale (MCS).

3.4 DISCUSSION

This is the first double-blind controlled trial to directly assess the effects of isokinetic strength training in stroke survivors. The main finding of this study was that there was a trend for the experimental group to show greater strength improvements than the control group following six weeks of strength training, however, no group differences were found over time in walking function or HRQoL in this group of chronic stroke survivors. In the following sections, training effects are described as "overall effects" (i.e. effects of training across both groups) and "strength training effects" (i.e. effects of training in the experimental group relative to the control group).

3.4.1 Overall Training Effects

There was an overall improvement in strength and walking function post-training in all participants regardless of grouping. Observed improvements in muscle strength in all participants grouped together ranged between 4% to 101% on the paretic side which exceed the learning effects (<10%) reported by Eng et al (in press) from test-retest measures of strength. Given that our participants had a practice session prior to the baseline test and the high reliability of average torque measures reported in stroke survivors (Eng et al., in press; Tripp & Harris, 1991), the improvements observed in this study were likely training effects rather than testing effects. Note that on the non-paretic side however, changes in strength post-training (4-13%) were within the learning effect range as reported by Eng et al. (in press). Improvements in walking performance (i.e. gait and stair climbing speed) ranged between 6% to 17% across the two groups. These gains are comparable to, if not greater than, gains reported in other studies of isokinetic strength training in stroke survivors (Engardt et al., 1995; Karimi, 1996; Sharp & Brouwer, 1997).

As the mean values in strength and walking performance for both the experimental and control groups were higher post-training compared to baseline, the possibility that simply attending and participating in a structured program three times a week is enough to induce strength and functional changes cannot be ruled out from the present study. Involvement of a third group consisting of only baseline and post-training testing without a regular program would have been necessary to discount this hypothesis. For functional changes, this hypothesis seems reasonable as participants were required to get out of their homes and travel to the rehabilitation centre three times a week for six weeks which may have increased their walking activity. However, this possibility seems unlikely in the case of strength changes since only the paretic side, and not the non-paretic side, showed improvements post-training. Consequently, the training programs of both the experimental and control groups seem to have influenced the results in strength. What aspects of the training program resulted in strength improvements for the paretic limb? Certainly the experimental group benefited from the isokinetic strength training. However, for the control group, perhaps the passive range of motion and 5 to 10 minutes of stretching exercises had an effect on their ability to generate force. It is possible that six weeks of passive lower limb movements enhanced learning of the motor task involved in isokinetic strength testing. Passive movements in acute stroke survivors have been shown to elicit brain activation patterns that were similar to the patterns seen during active movements in individuals with stroke who had substantial motor recovery (Nelles et al., 1999). In addition, by watching the limb being moved by the dynamometer, participants in the control group may have been mentally rehearsing the action of flexing and extending the limb. Mental activity may be related to motor activity. Decety et al. (1994) investigated brain activity during mental practice and suggested that mental tasks may involve neural mechanisms which are involved in planning and programming thereby improving motor tasks.

The temporal, kinematic and kinetic changes in gait following training suggest that the main mechanism adopted on the paretic side to increase the walking speed was to increase the power generated by the hip flexors during pull-off (H3) and thus increase the stride length. This is in accordance with the improvements seen in the paretic hip flexor strength post-training and also with the strong correlation found between maximum hip power generation and speed as reported in chapter 2 and by others (Olney et al., 1991). Concomitant with increases in ankle plantarflexor strength, the ankle push-off power (A2) also improved post-training although the value did not reach significance ($p = 0.08$). The significant increase in knee power absorption (K3) was likely a result of the increase in hip and ankle power during propulsion causing the knee to collapse. Karimi (1996) has also investigated the biomechanical changes and found that following eight weeks of isokinetic strength training, persons with stroke were able to increase the power generated by the ankle during push-off (A2) and increase gait speed. The increase in H3 was more moderate in their study as it was above normal values even before training (Karimi, 1996). Our results along with that of Karimi's (1996) suggest that training aimed at increasing strength may induce stroke survivors to use their improved strength to walk more efficiently to some extent. However, as discussed in the next section, the amount of strength gained from the training was not directly associated with improvements in gait performance as the smaller strength improvements in the control group still led to similar improvements in gait performance compared to the experimental group.

No changes were noted in the physical and mental health components of the SF-36 following the six weeks of training. One study that has used this particular quality of life measure reported that among the 8 subscales of the SF-36, only the physical functioning scale showed a trend towards improvement following a home-based physical exercise program in persons with stroke (Duncan et al., 1998). The physical functioning subscale includes ten items: vigorous activities, moderate activities, lifting or carrying groceries, climbing one flight of stairs, bending, kneeling or stooping, walking several blocks, walking one block, and bathing or dressing (Appendix X). Given that the training program in the present study was also focused on physical exercise, the physical components of the SF-36 survey were expected to show improvements. However, no improvements in physical functioning scores were identified following training (Figure 5) despite the improvements found in walking performance. Compared to participants in our study, the more acute stroke survivors in Duncan et al.'s (1998) study reported lower physical functioning scores at baseline while their post-training scores were comparable to the baseline scores reported in the present study. Our findings suggest that the SF-36 may not be a sensitive measure of change of perceived function in chronic stroke survivors who can perform most of the activities addressed in the physical functioning subscale except for the more vigorous activities.

Another reason for the lack of change in SF-36 scores may be due to the relatively high average scores reported by our participants at baseline (PCS = 39.6 compared to norm-referenced value of 41.3, MCS = 52.8 compared to norm-referenced 51.8). Other studies have also reported high SF-36 scores (except for physical functioning) among chronic stroke survivors compared with controls (Hackett et al., 2000; Mayo et al., 1999; Rodriguez et al., 1996) which suggests that individuals with stroke may adjust well to their disability years after the injury.

3.4.2 Strength Training Effects

The experimental group that received six weeks of isokinetic strength training showed a trend ($p = 0.06$) towards greater improvements in strength than the control group that received a passive range of motion program. A composite score was used to compare strength improvements between groups as it provides a representation of overall lower extremity strength improvements rather than the changes in scores of each individual muscle group. In fact, although individual muscle groups showed increases in strength, further post-hoc analyses showed that differences between groups in individual muscle strength improvements were not statistically significant likely due to the small sample size and the large variability in the data. Differences in composite scores, however, did show a trend towards greater strength improvements in the experimental group relative to the control group.

Only three other studies have investigated the effects of isokinetic strength training of isolated muscle groups in stroke survivors. Engardt et al. (1995) and Sharp & Brouwer (1997) have studied the paretic knee musculature and reported strength improvements in the range of 17% to 38% following six weeks of training. One study (Karimi, 1996) included all the flexor/extensor muscle groups of the paretic lower extremity in the training program and found 22% to 54% increases in strength following eight weeks of training. Relative strength improvements found in the present study (7-155%) were higher than the improvements reported in the above-mentioned studies measured at comparable angular velocities. The greater strength improvements found in this study may reflect the lower baseline strength status of our participants compared to other studies. Although direct comparisons are difficult to make due to differences in protocol used, relative strength measures (paretic/non-paretic) at baseline reveal greater strength deficits in this study. The paretic lower extremity strength relative to the non-paretic side ranged from 20-78% (in the six muscle groups) and 25-41% (in knee flexors and extensors) in this study while others reported 62-86% (in flexors and extensors of the three joints) (Karimi, 1996) and 40-71% (in knee flexors and extensors) (Engardt et al., 1995; Sharp & Brouwer, 1997). As percentage changes are calculated based on baseline status, lower baseline strength measures will tend to show greater improvement.

Our results along with that of others (Engardt et al., 1995; Karimi, 1996; Sharp & Brouwer, 1997) suggest that muscle strength can be improved in stroke survivors following isokinetic strength training. Whether the improvements in muscle performance gained following training are due to muscle property changes or neural changes cannot be determined from this study. Following strength training, increases in muscle size have been reported in healthy young adults as early as three to five weeks of training (Moritani & de Vries, 1979). Furthermore, it has been suggested that atrophied muscles may respond more rapidly to training (Jones & Rutherford, 1987). Given that stroke survivors are generally inactive due to functional limitations which in turn may lead to muscle atrophy, it is possible that muscle hypertrophy contributed to the strength improvements seen in this study.

On the other hand, it appears that neural changes can occur before hypertrophy during training (Hakkinen & Komi, 1983; Moritani & deVries, 1979) and it may be the dominant factor contributing to training-induced strength improvements in the elderly (Moritani & deVries, 1980). During the course of eight weeks progressive strength training, Moritani & deVries (1980) found that young men showed strength improvements due to neural factors only at the initial stage with hypertrophy becoming the dominant factor after the first 4 weeks. However, neural factors played a dominant role throughout the training in older men (Moritani & deVries, 1980). Possible mechanisms of neural adaptation following training include increased activation of prime movers as a result of improved motor unit recruitment or firing rates, and appropriate changes in the activation of synergists and antagonists leading to improved skill and coordination (Rutherford & Jones, 1986; Sale, 1988). Considering the relatively short training period in this study (six weeks) and the age of our participants, it is reasonable to speculate that neural factors contributed to the strength improvements observed to a greater extent than possible changes in muscle properties.

Although the experimental group showed greater improvements in strength than the control group, this trend was not seen in other outcome measures indicating that six weeks of isokinetic strength training alone does not induce changes in walking performance or health-related quality of life. The strength gained from the training may not have been large enough to make a difference in function in the experimental group relative to the control group. Furthermore, one cannot eliminate the possibility that a longer training period could have induced significant differences in functional gains between groups.

Another explanation for the lack of functional improvement may be around the issue of specificity of training. First, although our training protocol considered some of the aspects of the specificity principle (e.g. the type of resistance training selected was dynamic, as opposed to static, to simulate dynamic activities such as walking), strengthening was not done within the context of a functional task. Response to strength training has been reported to be very specific to the training itself, i.e. training effects are specific to the movement, velocity and angles used in the training which reduces the potential for carryover to other tasks (Rutherford, 1988). This is particularly important in short-term training programs when neural factors, rather than muscle mass, are altered as a result of training. Second, practice of functional tasks was not part of the training program in this study. As the purpose of the study was to assess the effects of strength training, practice of tasks was purposely not included in the program. The results, however, suggest that strength training alone may not be effective in inducing improved functional performance. That is, in order to better perform a task, one may need to not only restore the strength required for a task, but also practice using the restored strength within the context of the task. Task-related gait training using intensive treadmill walking (with or without support) has been recently shown to improve gait performance in both acute (Hassid et al., 1997; Malouin et al., 1992; Richards et al., 1993; Visintin et al., 1998) and chronic (Macko et al., 1997) stroke survivors. However, further assessment of this type of intervention is needed, particularly on the retention of these effects, the carryover into other functional tasks and the effects on quality of life. A study which incorporated both strength training and practice of tasks in individuals with stroke was initiated by Dean et al. (2000). The preliminary results of this type of training program were promising with improvements in the experimental group over the control group. However, no more than five subjects were included in each group. Further investigation using this type of treatment approach is warranted.

Finally, despite the reported strong correlations between muscle strength of the paretic lower limb and function (Kim & Eng, in review; Nadeau et al., 1999; Nakamura et al., 1985), strengthening the paretic leg alone may not be sufficient for functional improvement. Factors other than the strength of the paretic limb, such as balance, sensation and non-paretic limb strength, have also been shown to be associated with functional performance (Bohannon, 1987b; Bohannon & Walsh, 1991; Kim & Eng, in review; Nadeau et al., 1999).

The results of our study stress the importance of including a control group in clinical trials. Other studies that have reported functional gains following isokinetic strength training in stroke survivors found gains (6-12% in gait speed) (Engardt et al., 1995; Karimi, 1996; Sharp & Brouwer, 1997) that were comparable to our findings (11-15% in the experimental group). Without a control group, however, those gains cannot be solely attributed to the strength training itself.

3.4.3 Limitations

The major limitation of this study is the small sample size which affects the generalizability of the findings to the stroke population. In addition, the variability (as measured by the standard deviations) in strength and functional performance measures between participants was also large. These factors reduce the power to detect a difference in change scores between groups which in turn increase the probability of making a type II error. However, a nonparametric test (i.e. Mann Whitney *U* Test) was used, which takes into consideration the small sample size and the non-normal distribution of the data (including the variability). In fact, some argue that computing standard deviations for nonparametric data is not meaningful (Rosner, 1986).

To reduce the probability of making a type II error, clinical trials involving stroke survivors should ideally include large sample sizes as a large variability in performance is expected given the various types of disturbed motor control and recovery levels following stroke. However, due to time, recruitment and financial constraints, this may be difficult to achieve.

3.4.4 Future Directions

Further studies with a larger sample size are needed to confirm the results of this study. In addition, the role of strength training in the acute stage of stroke, i.e. early in rehabilitation, needs to be explored. To better understand the effects of strength training in stroke survivors, research investigating the actual mechanisms of strength gains (e.g. neural versus muscle adaptations) following a strength training program is indicated. The optimal frequency and duration of strengthening programs need to be defined along with the long-term effects of these programs.

The results of the present study suggest that strength training should be done in a task-related manner and in conjunction with practice of tasks in order to be of benefit. The impact of such training programs on mobility and overall quality of life need to be further explored with controlled study designs. Recently, there has been increasing evidence suggesting that stroke survivors can show functional improvements even years after the onset of injury (Dean et al., 2000; Rodriguez et al., 1996; Tangeman et al., 1990; Teixeira-Salmela et al., 1999). If this is the case, it is important to determine the type of intervention strategies that are most effective in maximizing outcome.

References

Adams, R.W., Gandevia, S.C., Skuse, N.F. (1990). The distribution of muscle weakness in upper motoneuron lesions affecting the lower limb. Brain, 113, 1459-1476.

Ahlsio, B., Britton, M., Murray, V., Theorell, T. (1984). Disablement and quality of life after stroke. Stroke, 15, 886-890.

Anderson, C., Laubscher, S., Burns, R. (1996). Validation of the short-form 36 (SF-36) health survey questionnaire among stroke patients. Stroke, 27, 1812-1816.

Andrews, A.W. (2000). Distribution of muscle strength impairments following stroke. Clinical Rehabilitation, 14, 79-87.

Andriacchi, T., Ogle, J., Galante, J. (1977). Walking speed as a basis for normal and abnormal gait parameters. Journal of Biomechanics, 10, 261-268.

Ashworth, B. (1964). Preliminary trial of carisoprodal in multiple sclerosis. Practitioner, 192, 540-542.

Bobath, B. (1978). Adult Hemiplegia: Evaluation and Treatment. London: Heinemann Medical Books Ltd.

Bohannon, R.W. (1986). Strength of lower limb related to gait velocity and cadence in stroke patients. Physiotherapy Canada, 38, 204-206.

Bohannon, R.W. (1987a). The relationship between static standing capacity and lower limb static strength in hemiparetic stroke patients. Clinical Rehabilitation, 1, 287-291.

Bohannon, R.W. (1987b). Gait performance of hemiparetic stroke patients: Selected variables. Archives of Physical Medicine and Rehabilitation, 68, 777-781.

Bohannon, R.W. (1988). Determinants of transfer capacity in patients with hemiparesis. Physiotherapy Canada, 40, 236-239.

Bohannon, R.W., Andrews, A.W. (1987). Interrater reliability of hand-held dynamometry. Physical Therapy, 67, 931-33.

Bohannon, R.W., Andrews, A.W., Smith, M.B. (1988). Rehabilitation goals of patients with hemiplegia. International Journal of Rehabilitation Research, 11, 181-183.

Bohannon, R.W., Smith, M.B. (1987a). Upper extremity strength deficits in hemiplegic stroke patients: relationship between admission and discharge assessment and time since onset. Archives of Physical Medicine and Rehabilitation, 68, 155-157.

Bohannon, R.W., Smith, M.B. (1987b). Interrater reliability of a modified Ashworth scale of muscle spasticity. Physical Therapy, 67, 206-207.

Bohannon, R.W., Walsh, S. (1991). Association of paretic lower extremity muscle strength and standing balance with stair-climbing ability in patients with stroke. Journal of Stroke and Cerebrovascular Disease, 1, 129-133.

Brandstater, M.E., de Bruin, H., Gowland, C., Clark, B.M. (1983). Hemiplegic gait: analysis of temporal variables. Archives of Physical Medicine and Rehabilitation, 64, 583-587.

Canning, C.G., Ada, L., O'Dwyer, N. (1999). Slowness to develop force contributes to weakness after stroke. Archives of Physical Medicine and Rehabilitation, 80, 66-70.

Chan, C.W.Y. (1986). Motor and sensory deficits following a stroke: relevance to a comprehensive evaluation. Physiotherapy Canada, 38, 29-34.

Damiano, D.L., Vaughan, C.L., Abel, M.F. (1995). Muscle response to heavy resistance exercise in children with spastic cerebral palsy. Developmental Medicine and Child Neurology, 37, 731-739.

Dean, C.M., Richards, C.L., Malouin, F. (2000). Task-related circuit training improves performance of locomotor tasks in chronic stroke: a randomized, controlled pilot trial. Archives of Physical Medicine and Rehabilitation, 81, 409-417.

Decety, J., Perani, D., Jeannerod, M., et al. (1994). Mapping motor representations with positron emission tomography. Nature, 371, 600-602.

Dombovy, M.L., Sandok, B.A., Basford, J.R. (1986). Rehabilitation for stroke: A review. Stroke, 17, 363-369.

Duncan, P.W. (1994). Stroke disability. Physical Therapy, 74, 399-407.

Duncan, P., Richards, L., Wallace, D., Stoker-Yates, J., Pohl, P., Lucjies, C., Ogle, A., Studenski, S. (1998). A randomized, controlled pilot study of a home-based exercise program for individuals with mild and moderate stroke. Stroke, 29, 2055-2060.

Eng, J.J., Kim, C.M., MacIntyre, D.L. (in press). Reliability of lower extremity strength measures in persons with chronic stroke. Archives of Physical Medicine and Rehabilitation.

Eng, J.J., Winter, D.A. (1995). Kinetic analysis of the lower limb during walking: what information can be gained from a three-dimensional model? Journal of Biomechanics, 28, 753-758.

Engardt, M., Knutsson, E., Jonsson, M., Sternhag, M. (1995). Dynamic muscle strength training in stroke patients: effects on knee extension torque, electromyography activity, and motor function. Archives of Physical Medicine and Rehabilitation, 76, 419-425.

Farrell, M., Richards, J.G. (1986). Analysis of the reliability and validity of the kinetic communicator exercise device. Medicine and Science in Sports Exercise, 18, 44-49.

Finucane, S.D.G., Mayhew, T.P., Rothstein, J.M. (1994). Evaluation of the gravity-correction feature of a Kin-Com isokinetic dynamometer. Physical Therapy, 74, 1125-1133.

Glasser, L. (1986). Effects of isokinetic training on the rate of movement during ambulation in hemiparetic patients. Physical Therapy, 66, 673-676.

Gowland, C., VanHullenaar, S., Torresin, W., Moreland, J., Vanspall, B., Barrecca, S., Ward, M., Huijbregts, M., Stratford, P., Barclay-Goddard, R. (1995). Chedoke-McMaster Stroke Assessment: Development, Validation and Administration Manual. Hamilton: Chedoke-McMaster Hospitals and McMaster University.

Gresham, G.E., Phillips, T.F., Wolf, P.A., McNamara, P.M., Kannel, W.B., Dawber, T.R. (1979). Epidemiologic profile of long-term stroke disability: the Framingham study. Archives of Physical Medicine and Rehabilitation, 60, 487-491.

Grimby, L., Hannerz, J. (1973). Tonic and phasic recruitment order of motor units in man under normal and pathological conditions. In J.E. Desmedt (Ed.), New Developments in Electromyography and Clinical Neurophysiology, vol. 3 (pp. 225-233). New York: S. Karger.

Hackett, M.L., Duncan, J.R., Anderson, C.S., Broad, J.B., Bonita, R. (2000). Health-related quality of life among long-term survivors of stroke: results from the Auckland stroke study, 1991-1992. Stroke, 31, 440-447.

Hakkinen, K., Komi, P.V. (1983). Electromyographic changes during strength training and detraining. Medicine & Science in Sports & Exercise, 15, 455-460.

Hassid, E., Rose, D., Commisarow, J., Guttry, M., Dobkin, B.H. (1997). Improved gait symmetry in hemiparetic stroke patients induced during body weight-supported treadmill stepping. Journal of Neurological Rehabilitation, 11, 21-26.

Hesse, S., Janke, M., Bertelt, C., Schreiner, C., Lucke, D., Mauritz, K. (1994). Gait outcome in ambulatory hemiparetic patients after a 4-week comprehensive rehabilitation program and prognostic factors. Stroke, 25, 1999-2004.

Himann, J.E., Cunningham, D.A., Rechnitzer, P.A., Paterson, D.H. (1988). Age-related changes in speed of walking. Medicine and Science in Sports and Exercise, 20, 161-166.

Horvat, M. (1987). Effects of a progressive resistance training program on an individual with spastic cerebral palsy. American Corrective Therapy Journal, 41, 7-11.

Jones, D.A., Rutherford, O.M. (1987). Human muscle strength training: the effects of three different regimes and the nature of the resultant changes. Journal of Physiology, 391, 1-11.

Karimi, H. (1996). Isokinetic strength training and its effect on the biomechanics of gait in subjects with hemiparesis as a result of stroke (PhD Dissertation). Kingston (On): Queen's University.

Kim, C.M., Eng, J.J. (in review). Non-paretic, in addition to paretic lower extremity muscle strength correlates with locomotor performance after stroke.

Kramer, J.F. (1990). Reliability of knee extensor and flexor torques during continuous concentric-eccentric cycles. Archives of Physical Medicine and Rehabilitation, 71, 460-464.

Lerner-Frankiel, M., Vargus, S., Brown, M., Krusell, L., Schoneberger, W. (1986). Functional community ambulation: what are your criteria? Clinical Management in Physical Therapy, 6, 12-15.

Macko, R.F., DeSouza, C.A., Tretter, L.D., Silver, K.H., Smith, G.V., Anderson, P.A., Tomoyasu, N., Gorman, P., Dengel, D.R. (1997). Treadmill aerobic exercise training reduces the energy expenditure and cardiovascular demands of hemiparetic gait in chronic stroke patients. Stroke, 28, 326-330.

Malouin, F., Potvin, M., Prevost, J., Richards, C.L., Wood-Dauphinee, S. (1992). Use of an intensive task-oriented gait training program in a series of patients with acute cerebrovascular accidents. Physical Therapy,72, 781-793.

Mayhew, T.P., Rothstein, J.M., Finucane, S.D.G., Lamb, R.L. (1994). Performance characteristics of the Kin-Com dynamometer. Physical Therapy, 74, 1047-1054.

Mayo, N.E., Neville, D., Kirkland, S., Ostbye, T., Mustard, C.A., Reeder, B., Joffres, M., Brauer, G., Levy, A.R. (1996). Hospitalization and case-fatality rates for stroke in Canada from 1982 through 1991: The Canadian Collaborative Study Group of Stroke Hospitalizations. Stroke, 27, 1215-1220.

Mayo, N.E., Wood-Dauphinee, S., Ahmed, S., Gordon, C., Higgins, J., McEwen, S., Salbach, N. (1999). Disablement following stroke. Disability & Rehabilitation, 21, 258-268.

McComas, A.J. (1973). Functional changes in motoneurons of hemiparetic muscles. Journal of Neurology, Neurosurgery, and Psychiatry, 36, 183-193.

McComas, A.J. (1994). Human neuromuscular adaptations that accompany changes in activity. Medicine and Science in Sports and Exercise, 26, 1498-1509.

Moritani, T., deVries, H.A. (1979). Neural factors versus hypertrophy in the time course of muscle strength gain. American Journal of Physical Medicine, 58, 115-30.

Moritani, T., deVries, H.A. (1980). Potential for gross muscle hypertrophy in older men. Journal of Gerontology, 35, 672-82.

Murray, M.P., kory, R., Clarkson, B.H. (1969). Walking patterns in healthy old men. Journal of Gerontology, 24, 169-178.

Nadeau, S., Arsenault, A.B., Gravel, D., Bourbonnais, D. (1999). Analysis of the clinical factors determining natural and maximal gait speeds in adults with a stroke. American Journal of Physical Medicine and Rehabilitation, 78, 123-130.

Nadeau, S., Gravel, D., Arsenault, A.B., Bourbonnais, D., Goyette, M. (1997). Dynamometric assessment of the plantarflexors in hemiparetic subjects: Relations between muscular, gait and clinical parameters. Scandinavian Journal of Rehabilitation Medicine, 29, 137-146.

Nakamura, R., Hosokawa, T., Tsuji, I. (1985). Relationship of muscle strength for knee extension to walking capacity in patients with spastic hemiparesis. Tohoku Journal of Experimental Medicine, 145, 335-340.

Nakamura, R., Watanabe, S., Handa, T., Morohash, I. (1988). The relationship between walking speed and muscle strength for knee extension in hemiparetic stroke patients: a follow-up study. Tohoku Journal of Experimental Medicine, 154, 111-113.

Nelles, G., Spiekermann, G., Jueptner, M., Leonhardt, G., Muller, S., Gerhard, H., Diener, C. (1999). Reorganization of sensory and motor systems in hemiplegic stroke patients: a positron emission tomography study. Stroke, 30, 1510-1516.

Niemi, M.L., Laaksonen, R., Kotila, M., Waltimo, O. (1988). Quality of life 4 years after stroke. Stroke, 19, 1101-1107.

Olney, S.J., Elkin, N.D., Lowe, P.J., Symington, D.O. (1979). An ambulation profile for clinical gait evaluation. Physiotherapy Canada, 31, 85-90.

Olney, S.J., Griffin, M.P., Monga, T.N., McBride, I.D. (1991). Work and power in gait of stroke patients. Archives of Physical Medicine and Rehabilitation, 72, 309-314.

Petrasovits, A., Nair, C. (1994). Epidemiology of stroke in Canada. Health Reports, 6, 39-44.

Richards, C.L., Malouin, F., Dumas, F., Tardif, D. (1995). Gait velocity as an outcome measure of locomotor recovery after stroke. In R. Craik & C. Oatis (Eds.), Gait analysis: Theory and applications (pp. 355-364). St. Louis, Missouri: C.V. Mosby, Inc.

Richards, C.L., Malouin, F., Wood-Dauphinee, S., Williams, J.I., Bouchard, J.P., Brunet, D. (1993). Task-specific physical therapy for optimization of gait recovery in acute stroke patients. Archives of Physical Medicine and Rehabilitation, 74, 612-620.

Rodriguez, A.A., Black, P.O., Kile, K.A., Sherman, J., Stellberg, B., McCormick, J., Roszkowski, J., Swiggum, E. (1996). Gait training efficacy using a home-based practice model in chronic hemiplegia. Archives of Physical Medicine and Rehabilitation, 77, 801-805.

Rosenfalck, A., Andreassen, S. (1980). Impaired regulation of force and firing patter of single motor units in patients with spasticity. Journal of Neurology, Neurosurgery & Psychiatry, 43, 907-916.

Rosner, B. (1986). Fundamentals of Biostatistics (2nd ed.). Boston, MA: Duxbury Press.

Roth, E.J., Merbitz, K., Dugan, S.A., Suh, W.W. (1997). Hemiplegic gait: relationships between walking speed and other temporal parameters. American Journal of Physical Medicine and Rehabilitation, 76, 128-133.

Rutherford, O.M. (1988). Muscular coordination and strength training: implications for injury rehabilitation. Sports Medicine, 5, 196-202.

Rutherford, O.M., Jones, D. (1986). The role of learning and coordination in strength training. European Journal of Applied Physiology & Occupational Physiology, 55, 100-105.

Sale, D.G. (1988) Neural adaptation to resistance training. Medical Science and Sports Exercise, 20, S135-S145.

Sharp, S.A., Brouwer, B.J. (1997). Isokinetic strength training of the hemiparetic knee: Effects on function and spasticity. Archives of Physical Medicine and Rehabilitation, 78, 1231-1236.

Suzuki, K., Imada, G., Iwaya, T., Handa, T., Kurogo, H. (1999). Determinants and predictors of the maximum walking speed during computer-assisted gait training in hemiparetic stroke patients. Archives of Physical Medicine and Rehabilitation, 80, 179-182.

Tangeman, P.T., Bonaitis, D.A., Williams, A.K. (1990). Rehabilitation and chronic stroke patients: changes in functional performances. Archives of Physical Medicine & Rehabilitation, 71, 876-880.

Teixeira-Salmela, L.F., Olney, S.J., Nadeau, S., Brouwer, B. (1999). Muscle strengthening and physical conditioning to reduce impairment and disability on chronic stroke survivors. Archives of Physical Medicine and Rehabilitation, 80, 211-218.

Tripp, E.J., Harris, S.R. (1991). Test-retest reliability of isokinetic knee extension and flexion torque measurements in persons with spastic hemiparesis. Physical Therapy, 71, 390-396.

Turnbull, G.I., Charteris, J., Wall, J.C. (1995). A comparison of the range of walking speeds between normal and hemiplegic subjects. Scandinavian Journal of Rehabilitation Medicine, 27, 175-182.

Viitanen, M., Fugl-Meyer, K.S., Bernspang, B., Fugl-Meyer, A.R. (1988). Life satisfaction in long-term survivors after stroke. Scandinavian Journal of Rehabilitation Medicine, 20, 17-24.

Visintin, M., Barbeau, H., Korner-Bitensky, N., Mayo, N.E. (1998). A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. Stroke, 29, 1122-1128.

von Schroeder, H.P., Coutts, R.D., Lyden, P.D., Billings Jr., E., Nickel, V.L. (1995). Gait parameters following stroke: a practical assessment. Journal of Rehabilitation Research and Development, 32, 25-31.

Ware, J.E.Jr., Kosinski, M., Gandec, B. (1993). SF-36 Health Survey: Manual and Interpretation Guide. Boston, MA: The Health Institute, New England Medical Center.

Ware, J.E.Jr., Kosinski, M., Keller, S.D. (1994). SF-36 Physical and Mental Health Summary Scales: A User's Manual. Boston, MA: The Health Institute, New England Medical Center.

Wilder, P.A., Sykes, J. (1982). Using an isokinetic exercise machine to improve the gait pattern in a hemiplegic patient. Physical Therapy, 62, 1291-1295.

Winter, D.A. (1991). The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological. Waterloo, Ontario: University of Waterloo Press.

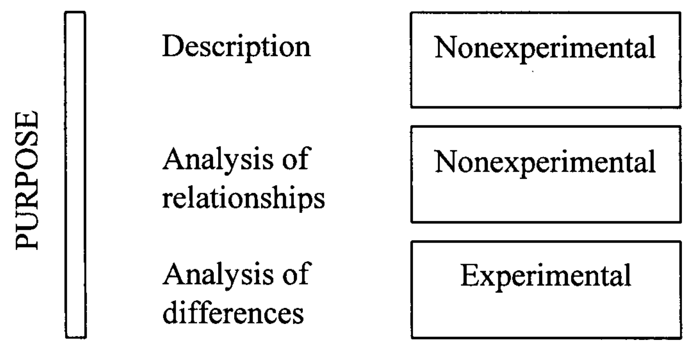
World Health Organization (1989). Stroke-1989. Recommendations on stroke prevention, diagnosis, and therapy: report of the WHO Task Force on Stroke and other Cerebrovascular Disorders. Stroke, 20, 1407-1431.

Chapter 4

General Conclusions

Although most stroke survivors regain the ability to walk, only 25% of them attain normal walking speed (Wade & Langton-Hewer, 1987) leaving many limited in community activities despite the intensive training during the first six months of rehabilitation. It is clearly important to investigate the factors that may be the cause of this gait disturbance and evaluate the potential effects of treatment approaches aimed at improving these factors on the gait performance of stroke survivors.

The investigation of the factors contributing to gait performance can be done with different types of research designs as illustrated in this matrix of research types modified from Domholdt (1993).



In the present study, all three types of research designs were used. First, descriptive analysis of the three-dimensional kinematic and kinetic profiles was used to understand the mechanisms underlying the problem of gait in stroke survivors. Second, the relationships between gait speed and selected kinematic/kinetic gait variables were analyzed to determine the biomechanical factors contributing to gait performance. These nonexperimental research designs help guide assessment and intervention strategies that may be effective in retraining gait. However, experimental studies are needed to confirm the hypotheses formed based on the nonexperimental investigations. Therefore, the third type of investigation used in this study was an analysis of differences through a double-blind experimental trial to determine whether strength training induces improvement in the gait performance of individuals with stroke. Note that in the present study, the data for all the investigations (i.e. chapters 2 and 3) were collected simultaneously. Therefore, the design of the treatment program (in chapter 3) was not based on the results of the biomechanical analyses (in chapter 2). A better approach would have been to first investigate the function of muscle groups important in gait and then design the intervention based on those results.

Results of previous investigations (Bohannon, 1986; Bohannon &Walsh, 1992; Kim & Eng, in review; Nadeau et al., 1999; Suzuki et al., 1990) have shown strong correlations between walking (level and stair-walking) speed and muscle strength in stroke survivors. Consistent with these previous findings, the strong correlations found in our study between gait speed and kinetic variables emphasized the importance of muscle function in the gait performance of persons with stroke. However, since correlational studies do not infer causation, it cannot be assumed that treatment aimed at increasing muscle strength, thus the ability to generate power during gait, will increase gait speed.

In fact, the results of the double-blind controlled trial in this study indicate that isokinetic strength training of the flexors/extensors of the hip, knee and ankle does not improve walking performance in persons with stroke despite the trend (p =0.06) towards improvement in strength following training. These findings suggest that other factors contributing to walking performance need to be addressed. The results of our correlational analysis revealed that in addition to the sagittal plane variables, gait variables in other planes were also important particularly the frontal hip power variables. In addition, variables for the non-paretic limb were also strongly correlated with speed. Given these findings, inclusion of the hip abductors and the muscle groups of the non-paretic limb in the strengthening program may have resulted in more positive findings. In addition, eccentric strengthening was not included in the training.

This is the first double-blind controlled trial to directly assess the effects of isokinetic strength training of the paretic lower limb in stroke survivors. Although rehabilitation programs do not normally address only one aspect of training (e.g. strength training), this study was an attempt to quantify the contribution of strength training to walking performance in individuals with stroke. Isokinetic strength training seems effective in improving strength, therefore, the inclusion of isokinetic strengthening in training programs is indicated. However, our results suggest that strength training should be done in a task-related manner and in conjunction with practice of functional tasks in order to be of benefit in functional performance. This type of training has been investigated more recently with positive effects on function in stroke survivors (Dean et al., 2000; Richards et al., 1993).

References

Bohannon, R.W. (1986). Strength of lower limb related to gait velocity and cadence in stroke patients. Physiotherapy Canada, 38, 204-206.

Bohannon, R.W., Walsh, S. (1992). Nature, reliability, and predictive value of muscle performance measures in patients with hemiparesis following stroke. Archives of Physical Medicine and Rehabilitation, 73, 721-5.

Dean, C.M., Richards, C.L., Malouin, F. (2000). Task-related circuit training improves performance of locomotor tasks in chronic stroke: a randomized, controlled pilot trial. Archives of Physical Medicine and Rehabilitation, 81, 409-417.

Domholdt, E. (Ed.). (1993). Research problems and questions (pp.48). Physical Therapy Research: Principles and Applications. Philadelphia: W.B. Saunders Company.

Kim, C.M., Eng, J.J. (in review). Non-paretic, in addition to paretic lower extremity muscle strength correlates with locomotor performance after stroke.

Nadeau, S., Arsenault, A.B., Gravel, D., Bourbonnais, D. (1999). Analysis of the clinical factors determining natural and maximal gait speeds in adults with a stroke. American Journal of Physical Medicine and Rehabilitation, 78, 123-130.

Richards, C.L., Malouin, F., Wood-Dauphinee, S., Williams, J.I., Bouchard, J.P., Brunet, D. (1993). Task-specific physical therapy for optimization of gait recovery in acute stroke patients. Archives of Physical Medicine and Rehabilitation, 74, 612-620.

Suzuki, K., Nakamura, R., Yamada, Y., Handa, T. (1990). Determinants of maximum speed in hemiparetic stroke patients. Tohoku Journal of Experimental Medicine, 162, 337-344.

Wade, D.T., Langton-Hewer, R. (1987). Functional abilities after stroke: measurement, natural history, and prognosis. Journal of Neurology, Neurosurgery & Psychiatry, 50, 177-182.

Appendix III

Chedoke-McMaster Stroke Assessment: 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 Xs. For "standing" test items, light support may be provided but weight bearing through the hand is not allowed. Shoes and socks off.

		LEG			FOOT
1		☐ not yet Stage 2	1		☐ not yet Stage 2
2	Crook lying	☐ resistance to passive hip or knee flexion	2	Crook lying	☐ resistance to passive dorsiflexion
		☐ facilitated hip flexion			☐ facilitated dorsiflexion or toe extension
		☐ facilitated extension			☐ facilitated plantarflexion
3		☐ abduction: adduction to neutral	3	Supine	☐ plantarflexion > ½ range
		☐ hip flexion to 90°		Sit	☐ some dorsiflexion
		☐ full extension			☐ extension of toes
4		☐ hip flexion to 90° then extension synergy	4		☐ some eversion
		☐ bridging hip with equal weightbearing			☐ inversion
	Sit	☐ knee flexion beyond 100°			☐ legs crossed: dorsiflexion, then plantarflexion
5	Crook lying	☐ extension synergy, then flexion synergy	5		☐ legs crossed: toe extension with ankle plantarflexion
	Sit	☐ raise thigh off bed			☐ sitting with knee extended: ankle plantarflexion, then dorsiflexion
	Stand	☐ hip extension with knee flexion		Stand	☐ heel on floor: eversion
6	Sit	☐ lift foot off floor 5 x in 5 sec.	6		☐ heel on floor: tap foot 5 x in 5 sec
	Stand	☐ full range internal rotation			☐ foot off floor: foot circumduction
		☐ trace a pattern: forward, side, back, return			☐ knee straight, heel off floor: eversion
7	Stand	☐ unsupported: rapid high stepping 10 x in 5 sec	7		☐ heel touching forward, then toe touching behind, repeat 5 x in 10 sec
		☐ unsupported: trace a pattern quickly; forward, side, back, reverse			☐ foot off floor: circumduction quickly, reverse
		☐ on weak leg with support: hop on weak leg			☐ up on toes, then back on heels 5 x
		☐ **STAGE OF LEG**			☐ **STAGE OF FOOT**

From: Gowland, C., VanHullenaar, S., Torresin, W., Moreland, J., Vanspall, B., Barrecca, S., Ward, M., Huijbregts, M., Stratford, P., Barclay-Goddard, R. (1995). Chedoke-McMaster Stroke Assessment: Development, Validation and Administration Manual. Hamilton: Chedoke-McMaster Hospitals and McMaster University.

Appendix IV

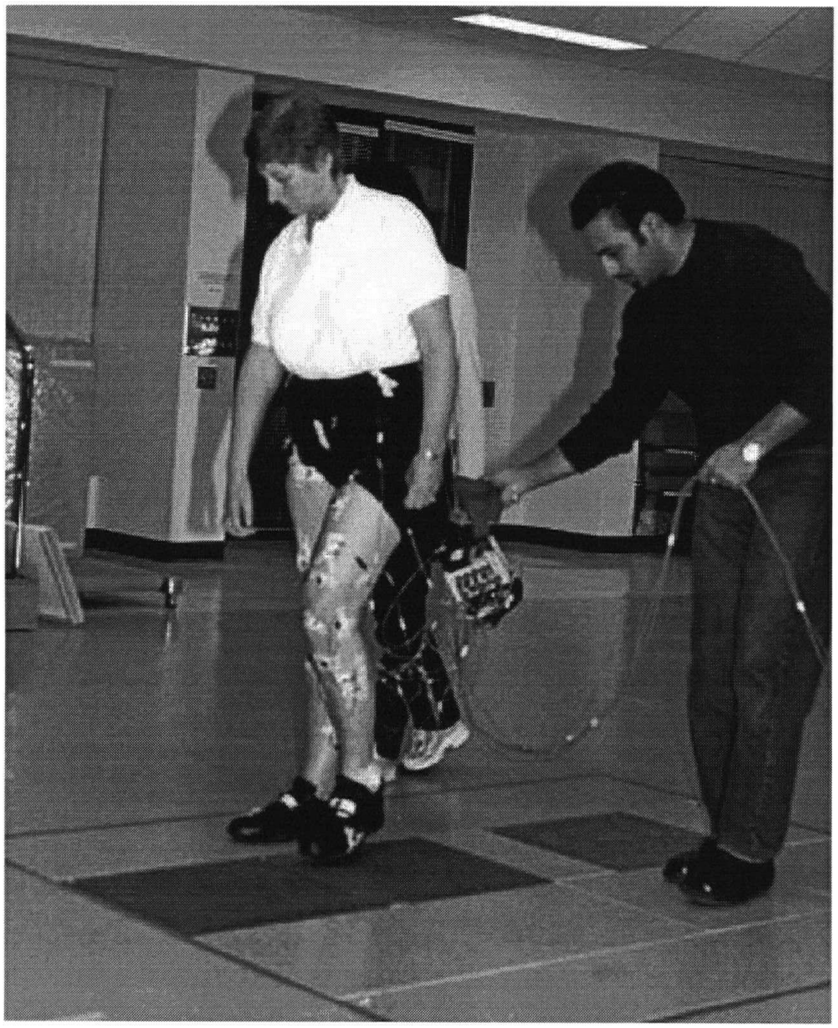
Modified Ashworth Scale

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

From: Bohannon, R.W., Smith, M.B. (1987). Interrater reliability of a modified Ashworth scale of muscle spasticity. Physical Therapy, 67, 206-207.

Appendix V

Gait Assessment Set-up



Appendix VI

Marker Placement sites

1 Right metatarsal (head of 5th metatarsal)
2 Right dorsum (midpoint of metatarsals and ankle joint)
3 Right lateral malleolus

4 Right midshank (anterior aspect of shank, midpoint of ankle and knee)
5 Right knee joint (lateral)
6 Right lower thigh (anterior aspect of thigh, ~10cm above knee)

7 Right upper thigh (anterior aspect of thigh, ~10cm below hip)
8 Right anterior iliac crest
9 Right anterior superior iliac spine (ASIS)

10 Left ASIS
11 Left anterior iliac crest
12 Left upper thigh

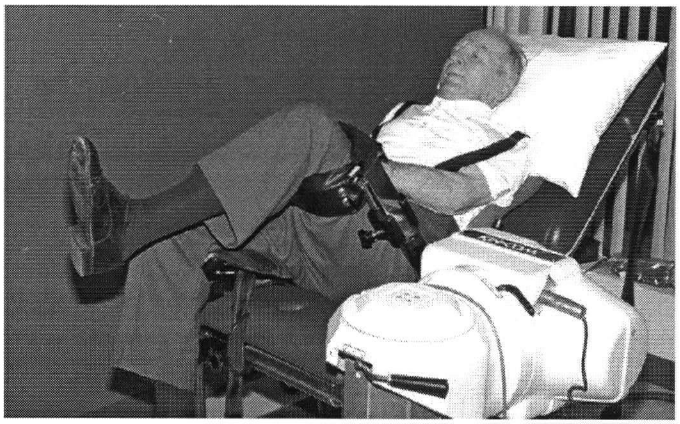
13 Left metatarsal
14 Left dorsum
15 Left lateral malleolus

16 Left midshank
17 Left knee joint
18 Left lower thigh

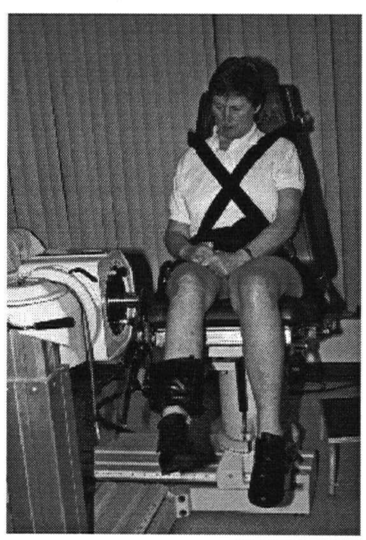
Appendix IX

Participant positioning and stabilization during strength testing and training

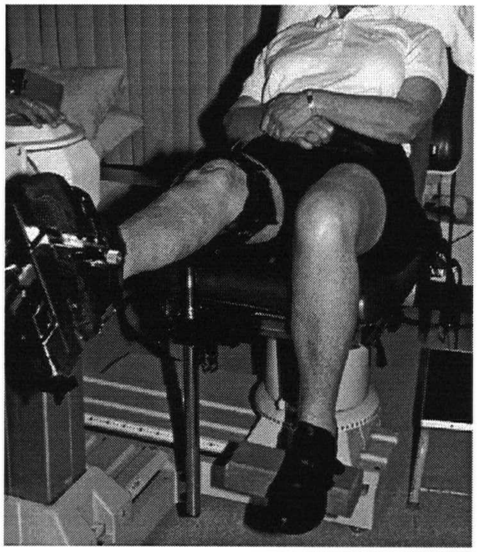
Hip: Participants were in a semi-reclined (30° angle from horizontal) position, with the pelvis fixed by a strap and the back supported. The contralateral thigh was supported by a pad attached to the seat. The greater trochanter of the leg being tested was aligned with the Kin-Com system's rotation axis. The cuff of the force transducer was placed three fingerbreadths proximal to the popliteal fossa.



Knee: Participants were seated with the back support set at a 90° sitting angle. Large straps were applied horizontally across the pelvis and diagonally across the trunk to minimize body movement during testing. Participants were asked to place their hands on their lap. The lateral femoral condyle was aligned with the dynamometer's rotation axis. The cuff of the force transducer was placed three fingerbreadths proximal to the lateral malleoli.



Ankle: Participants were in a semi-reclined (45°) position with the back supported. The leg being tested was placed on a pad attached to the seat which allowed the knee to flex stlightly. The foot was tightly fixed in the metal boot attached to the Kin-Com dynamometer with the lateral malleolus aligned with the dynamometer's rotation axis. The contralateral foot rested on a foot support attached to the seat.



Appendix X

SF-36

Cs	No of Items	No of Levels	Meaning of Scores: Low	High
PF	10	21	Limited a lot in performing all physical activities including bathing or dressing due to health	Performs all types of physical activities including the most vigorous without limitations due to health
R-P	4	5	Problems with work or other daily activities as a result of physical health	No problems with work or other daily activities as a result of physical health
BP	2	11	Very severe and extremely limiting pain	No pain or limitations due to pain
GH	5	21	Evaluates personal health as poor and believes it is likely to get worse	Evaluates personal health as excellent
VT	4	21	Feels tired and worn out all of the time	Feels full of pep and energy all of the time
SF	2	9	Extreme and frequent interference with normal social activities due to physical or emotional problems	Performs normal social activities without interference due to physical or emotional problems
R-E	3	4	Problems with work or other daily activities as a result of emotional problems	No problems with work or other daily activities as a result of emotional problems
MH	5	26	Feelings of nervousness and depression all of the time	Feels peaceful, happy, and calm all of the time
RHT	1	5	Believes general health is much better now than one year ago	Believes general health is much worse now than one year ago

Abbreviations: Concepts (Cs), Physical Functioning (PF), Role-Physical (R-P), Bodily Pain (BP), General Health (GH), Vitality (VT), Social Functioning (SF), Role-Emotional (R-E), Mental Health (MH), Reported Health Transition (RHT).

Adapted from: Ware, J.E., Sherbourne, C.D. (1992). The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. Medical Care, 30, 473-483.

Appendix XI

Warm-up and Cool-down

Warm-up:

- Sitting- * Bilateral arm raises (non-paretic arm supporting the paretic arm) x 10 reps
  * Alternating knee raises x 5 reps each side
  * Alternating knee extensions x 5 reps each side
  * Circular ankle movements x 5 reps each side

- Walking or marching- for 2-3 minutes with the usual assistive device

- Stretching-* Stretching of the quadriceps, hamstrings, rectus femoris, and gluteus maximus of the paretic limb was performed by the investigator with the participant in supine position x 10 sec. each
  * Participants were instructed to do their own calf stretches on a step x 10 sec.

Cool-down:

- Stretching as above.