Relation between physical exertion and postural stability in hemiparetic participants secondary to stroke

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Abstract

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Tamara Carver

Balance impairment secondary to stroke is an important issue to consider since it significantly increases the risk of falling and can lead to pathological events. The purpose of this study was to determine the effects of physical exertion induced by walking on postural stability in hemiparetic stroke participants. Twelve hemiparetic participants and 12 control participants walked over-ground for a duration of 6 minutes and 18 minutes at their comfortable speed. The control participants walked at a speed that allowed them to maintain the heart rate of their hemiparetic counterpart. Postural stability was measured during double-legged stance, sit-to-stand, and step reaction time tasks before the walk, immediately after the walk, 15 minutes post-walk, and 30 minutes post-walk. Measures of physical exertion during walking were also obtained from cardiorespiratory parameters, time-distance parameters, and subjective scales. The results indicated that physical exertion measures significantly increased when the duration of walk was increased from 6 minutes to 18 minutes in both control and hemiparetic participants. For postural stability measures, increasing the duration of walking led to a significant increase ($p<0.05$) of postural sway in double-legged stance and sit-to-stand for the hemiparetic participants only. This effect on balance of hemiparetic participants was observed immediately after the end of the walk. In conclusion, this study demonstrated that physical exertion can increase postural sway in hemiparetic participants which could possibly lead to an increased risk of falling in these individuals.
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Introduction

**STROKE DEFINITION**

A stroke is a sudden loss of brain function due to an interruption of blood to the brain. One can have an ischemic stroke which occurs when a blood clot narrows the possible blood flow to the brain. This type of stroke occurs 80% of the time and can be defined as either thrombotic or embolic. A blood clot that forms in the artery of the brain is defined as thrombotic, whereas a blood clot that forms somewhere else in the body and then travels to the brain is termed embolic. The remaining 20% of strokes are hemorrhagic and occur when there is a rupture of blood vessels in the brain. This uncontrolled bleeding can be due to an aneurysm or an arteriovenous malformation (Heart and Stroke Foundation of Canada, 2005).

A stroke injures the neurons (brain cells) of the affected area. A stroke can occur in any of the three parts of the brain. When the cerebrum is affected, movement control, sensation, speech, thinking, reasoning, memory, sexual function, and regulation of emotions may be affected (Heart and Stroke Foundation of Canada, 2005). The cerebrum is divided into right and left hemispheres, and the location of the stroke will determine the subsequent effects of the stroke.

If the stroke occurs in the left hemisphere, there may be weakness or paralysis on the right side of the body, difficulty reading, talking, thinking, or doing mathematics. If the stroke occurs in the right hemisphere, the effects may be weakness or paralysis on the left side of the body, vision problems, and problems with spatial perception (Heart and Stroke Foundation of Canada, 2005).
The most uncommon type of stroke occurs in the brain stem and can cause problems with breathing and heart function, body temperature control, balance, and coordination. It is also uncommon to have a stroke in the cerebellum and the effects can be severe. Some of the possible effects of this type of stroke are inability to walk, problems with coordination and balance (ataxia), dizziness, headaches, nausea, and vomiting (Heart and Stroke Foundation of Canada, 2005).

Once a stroke happens, paralysis can occur because of the loss of inputs to the target neurons. According to Krishnan (2003), the resulting paralysis is also due to the maladaptive compensatory mechanisms that occur after the stroke. The target neurons that are innervated by the injured neurons become denervated. Once this occurs, compensatory measures take place to replace the massive loss of synapses of those target neurons. The synapses are then replaced by sprouting synaptic connections from nearby intact neurons. These compensatory measures, instead of being beneficial, are more often maladaptive and functionally detrimental. The motor recovery that may eventually appear can often be incomplete, imperfect and compromised (Krishnan, 2003).

Stroke patients may lose the ability to feel touch, pain, temperature, or limb position. Sensory deficits may also hinder the ability to recognize objects that patients are holding and can even be severe enough to cause loss of recognition of one's own limb. Some stroke patients experience pain, numbness or odd sensations of tingling or prickling in paralyzed or weakened limbs, a condition known as paresthesia ("Post-Stroke Rehabilitation Fact Sheet", National Institute of Neurological Disorders and Stroke NINDS. August 2002). The degeneration of the cortico-spinal tract axons causes hemiplegia or hemiparesis of the contralateral side (Krishnan, 2003). Hemiplegia is
defined as total paralysis of the arm, leg, and trunk on one side of the body; whereas hemiparesis is a weakness on that side of the body. Furthermore, hemiparesis can cause stiffening of the muscles (spasticity) to the affected side thereby creating an altered gait.

**STROKE STATISTICS AND INCIDENCE**

Stroke is the fourth leading cause of death in Canada. Annually, between 40,000 and 50,000 Canadians have a stroke, which is fatal for 16,000 of them. There are in fact about 300,000 Canadians who are living with the effects of stroke (Heart and Stroke Foundation of Canada, 2006). Stroke costs the Canadian economy $2.7 billion a year and the average acute care cost is approximately $27,500 per stroke (Heart and Stroke Foundation of Canada, 2006). In the United States, in 2001, stroke ranks No. 3 among all causes of death, behind diseases of the heart and cancer (CDC/NCHS) (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006). Stroke is also a dominant cause of mortality and morbidity throughout the world (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006). Each year, approximately 15 million people will have a stroke, which will be fatal for 5 million people and permanently disabling for a further 5 million (Mackay & Mensah, 2004). Moreover, based solely on demographic changes in the population of selected countries within the European Union, the World Health Organization predicts a 27% increase in stroke events between the years 2000 and 2025 (Truelsen, Piechowski-Jozwiak, Bonita, Mathers, Bogousslavsky, & Boysen, 2006).

According to the Heart and Stroke Foundation, of every 100 people who have a stroke, approximately 15 will die, 10 will recover completely, 25 will recover with a minor impairment or disability, 40 will be left with a moderate to severe impairment and 10 will be so severely disabled that they will require long-term care (Heart and Stroke
Foundation of Canada, 2005). In addition, the American Heart Association cites stroke as a leading cause of functional impairments, with 20% of survivors requiring institutional care after 3 months and 15% to 30% being permanently disabled (Mackay & Mensah, 2004).

One of the largest longitudinal studies performed in the field of stroke, the Framingham Heart Study, found the following impairments and disabilities 6 months post stroke in ischemic stroke individuals: hemiparesis (50%), inability to walk without some assistance (30%), dependence in activities of daily living (26%), and aphasia which is a loss of the ability to produce and/or comprehend language (19%). In addition, stroke individuals had depressive symptoms (35%) and were institutionalized in a nursing home (28%) (Kelley-Hayes, Beiser, Kase, Scaramucci, D'Agostino, & Wolf, 2003).

In Canada, individuals 65 years and older represented approximately 13% of the population in 2001, and this percentage is expected to increase to nearly 15% by the year 2016, and 19% by the year 2021 (Heart and Stroke Foundation of Canada, 2006). Increased age is the dominant risk factor for heart disease and stroke. As the Canadian population ages, it is to be expected that the number of individuals with heart disease and stroke will increase (Heart and Stroke Foundation of Canada, 2000).

As the population continues to age and stroke incidence increases, there is also an increase in surviving stroke patients due to the reduced mortality rate. Cardiovascular disease death rates have been declining steadily in Canada since the mid-1960s (Heart and Stroke Foundation of Canada, 2000). Although mortality rates have been declining for all cardiovascular disease for both men and women, the actual number of cases of cardiovascular disease has increased. This phenomenon reflects increased survivorship
and an increase in the size of the population over age 65 (Heart and Stroke Foundation of Canada, 2000).

This translates into a larger number of chronic stroke survivors needing rehabilitation. Many stroke survivors continue to live with residual physical impairments (i.e. reduced mobility, poor balance and muscle weakness), which may lead to physical inactivity and a sedentary lifestyle (Gresham & Dawber, 1975). One of the most important and common problems is related to falls (Baker, O’Neill, & Karpf, 1984).

**Balance Impairment after Stroke**

One of the most critical challenges faced by an individual who has sustained a stroke is recovering the ability to stand and walk. Standing and walking require complex postural control mechanisms (Garland, Willems, Ivanova, & Miller, 2003). Geurts, de Haart, van Nes, & Duysens (2005) suggested that postural control is a major factor in determining the independence of stroke patients. Rapid and optimal improvement of postural control in patients with stroke is, therefore, essential to their independence, social participation, and general health.

Balance control (postural control) is defined as the ability to maintain equilibrium by controlling the center of body mass over its base of support. Balance control is a complex sensory and motor skill necessary for all standing and walking activities (Horak, Henry, & Shumway-Cook, 1997). It requires a combination of static and dynamic balance. Static balance refers to the ability to maintain postural control with very little body movement (steadiness) as well as even symmetry with regard to the weight bearing of each limb (Horak, Henry, & Shumway-Cook, 1997). During the static task of quiet standing, it has been reported that the majority of hemiparetic individuals bear less
weight on the paretic limb (25–43% of body weight) (Eng & Chu, 2002). Dynamic balance, on the other hand, refers to the ability to maintain the center of mass within the body’s limits of stability during a task (Horak, Henry, & Shumway-Cook, 1997).

Following a stroke, hemiparetic patients often present with balance impairment. This impairment is characterized by increased postural sway and an asymmetrical weight distribution between the lower limbs during quiet stance (Niam, Cheung, Sullivan, Kent, & Gu, 1999; Sackley, 1991). Niam, Cheung, Sullivan, Kent, & Gu (1999) showed that this increased postural sway in static balance was associated with impaired ankle joint proprioception in hemiparetic participants. This increase of postural sway has also been shown to correlate with falls in individuals with stroke (Sackley, 1991). Concerning weight-bearing symmetry during quiet stance, Sackley (1991) suggested that it is traditionally regarded as a primary goal in stroke rehabilitation and it has been associated with better motor functioning and greater independence in activities of daily living (ADLs) in the post-acute phase of stroke.

Studies have shown that dynamic balance is impaired in patients with hemiparesis when compared to that of healthy participants. Ikai, Kamikubo, Takehara, Nishi, & Miyano (2003) found a longer response latency and weaker response strength in the paretic side of patients following body perturbation. In contrast, the response latency in the nonparetic side was similar to that observed in healthy participants. The authors suggest that patients with hemiparesis tend to fall easily and that the risk of falls toward the paretic side is high. In fact, femoral neck fractures usually occur on the paretic side in patients with hemiparesis (Ikai, Kamikubo, Takehara, Nishi, & Miyano 2003). Eng & Chu (2002) further stated that impairment of the hemiparetic side during forward weight shifting and sit-to-stand tasks presents a challenge to the motor systems,
which may account for the poor balance associated with stroke patients. Cheng, Wu, Liaw, Wong, & Tang (1998) suggested that the asymmetrical body-weight distribution during a rise from a chair might be a contributing factor to falls in individuals with stroke.

In addition to stroke-related impairments, it is important to note that postural sway during stance can also be affected by natural aging processes (Dickstein & Abulaffio, 2000). Normal aging processes are associated with decreases in vestibular, visual, and somatosensory function (Cohen, Heaton, Congdon, & Jenkins, 1996; Horak, Shupert, & Mirka, 1989) and have been shown to contribute to increases in the unsteadiness and the incidence of falls among the elderly (Peterka & Black, 1990). Furthermore, sensory organization tests demonstrated that age is generally associated with an increased dependence on visual input for balance (Speers, Kuo, & Horak, 2002; Sundermier, Woollacott, Jensen, & Moore, 1996). Dault, de Haart, Geurts, Arts, & Nienhuis (2003) suggested that sagittal plane imbalance in healthy elderly and stroke patients may be largely due to the effects of aging, whereas frontal plane imbalance is much more specific to the postural problems associated with stroke.

**Falling**

In the healthy elderly population, it has been reported that thirty per cent of persons over 65 years of age and 50% of persons over 80 years experience at least one fall each year (Burt & Fingerhut, 1998). More than 90% of hip fractures occur as a result of falls and one-quarter of older people who sustain a hip fracture die within 6 months of the injury (Black, Maki, & Fernie, 1993). Hip fracture survivors experience a 10-15% decrease in life expectancy (Melzer, Benjuya, & Kaplanski, 2004). In the stroke population, the incidence is, unfortunately, even higher and this presents a substantial health problem due to the overwhelming rise in human life expectancy (Lajoie &
Gallagher, 2004). Stroke is considered to be one of the greatest risk factors for falls in older adults. A history of stroke increases risk of falls even years after the initial stroke (Jorgensen, Engstad & Jacobsen, 2002). Fall rates in people with stroke are higher than in the general population of the elderly. Campbell, Reinken, Allan, & Martinez (1981) found that the fall rate to be 34%, increasing to 45% for those between the ages of 80-89. Hyndman, Ashburn, & Stack (2002) found that approximately 50% of hemiparetic participants in the community based study were classed as fallers. Other studies have reported that approximately 75% of individuals with stroke have fallen in the 6 months after discharge from hospital (Forster & Young, 1995; Sackley, 1991). In fact, several recent studies have reported fall incidence rates as high as 73% for stroke victims living in a community (Sackley, 1991; Lamb, Ferrucci, Volapto, Fried, & Guralnik, 2003; Forster & Young, 1995; Langhorne, Stott, Robertson, MacDonald, Jones, McAlpine, Dick, Taylor, & Murray, 2000; Gucuiyener, Ugur, Uzuner, & Ozdemir, 2000; Yates, Lai, Duncan, & Studenski, 2002). Recently, Belgen, Beninato, Sullivan, & Narielwalla (2006) used a cross sectional design study to describe the frequency of falls in the stroke population. Falls were reported by 40% of the participants and multiple falls were reported by 22% of the participants. The consequences of falling include hip fractures, soft tissue injuries, fear of falling, hospitalization, increased immobility, and greater disability.

The potentially disabling consequences of falls, high fall rates in community-dwelling stroke survivors, and number of people living in the community with the effects of stroke, all indicate the need for better understanding of contributing factors to balance and falls in community dwelling people with chronic stroke (Belgen, Beninato, Sullivan, & Narielwalla, 2006) In addition, Harris, Eng, Marigold, Tokuno, & Louis (2005) found that falls occurred frequently during walking; therefore it may be necessary to focus on
reactive balance and environmental interaction when assessing individuals for risk of
falls and when devising fall prevention programs for individuals with chronic stroke.

Identifying fall risk factors is an important first step in maintaining optimal function
and preventing falls. As expected, balance performance during functional tasks has
been identified as a risk factor. Hyndman & Ashburn (2002) used the Berg Balance
Scale (BBS) to assess balance performance in static and dynamic tasks and found a
difference in scores between people who fell more than once and people who did not
fall, but they found no difference in scores between people who did not fall and people
who fell once. Recently, Mackintosh, Hill, Dodd, Goldie, & Culham (2005) evaluated fall
incidences and consequences in 56 stroke participants who were participating in a
rehabilitation program. Participants completed fall diaries for 6 months following their
discharge from rehabilitation. Their balance was assessed using the Berg Balance
Scale. The results showed that 46% of the participants had at least one confirmed fall,
38% fell toward the paretic side, and falls occurred most frequently during walking
(39%). Unfortunately, falls often lead to a loss of confidence and a prolonged restriction
of activity that can contribute to further physical decline (Cumming, Salkeld, Thomas, &
Szonyi, 2000; Ho, Woo, Yuen, Sham, & Chan, 1997; Vellas, Cayla, Bocquet, de Pemille,
& Albarede, 1987).

In fact, when researching the risk factors for falls in the stroke population, several
factors have been identified. We have already discussed increased postural sway as a
risk factor (Sackley, 1991). In addition, Mayo, Korner-Bitensky, & Kaizer (1990) found
that fallers had slower response times than non-fallers when evaluating the influence of
simple motor response time using visual stimuli. Also, Cheng, Liaw, Wong, Tang, Lee, &
Lin (1998) found that the significantly lower rate of rise in force and greater postural
sway while rising/sitting down may be useful in identifying stroke patients who are at risk of falling.

In summary, chronic hemiparetic stroke individuals are at a higher risk of falling and this may be due to balance impairments associated with stroke effects.

**Fitness Level after Stroke**

The post-stroke effects and subsequent disabilities perpetuate a vicious cycle of physical deconditioning in individuals who have sustained a stroke. This phenomenon is characterized by a decrease in exercise tolerance and physical fitness which is attributed to the loss of mobility and the reduction of cardiovascular capabilities (Figoni, 1995; Michael, Allen, & Macko, 2005; Macko, DeSouza, Tretter, Silver, Smith, Anderson, Tomoyasu, Gorman, & Dengel, 1997; Potempa, Braun, Tinknell, & Popovich, 1996; Macko, Ivey, Forrester, Hanley, Sorkin, Katz, Silver, & Goldberg, 2005). This deconditioning increases cardiovascular risk in chronic stroke patients. The use of exercise interventions to improve fitness and cardiovascular health after stroke is a novel approach in breaking the deconditioning cycle. It is evident that increased ambulation and improved cardiovascular fitness in the chronic hemiparetic stroke population is necessary. What all too frequently occurs, however, is that stroke patients complete a rehabilitation program and then fall back into the sedentary habits which may have contributed to the stroke in the first place. This makes regular activities of daily living more difficult to achieve and can, subsequently, affect patients physically and psychologically, thus perpetuating this deconditioning cycle (Michael, Allen, & Macko, 2005).
A stroke survivor has a 20% chance of having another stroke within 2 years (Heart and Stroke Foundation of Canada, 2005). This is a very important statistic as studies have shown that physical inactivity is a risk factor for stroke (Heart and Stroke Foundation of Canada, 2005). Since many hemiparetic participants become inactive, they are at even higher risk due to factors such as hypertension (Heart and Stroke Foundation of Canada, 2005).

The vicious cycle of deconditioning is exacerbated by the decreased ability to walk and the post stroke effect of reduced balance control, and poor cardiovascular fitness. In fact, Ivey, Hafer-Macko, & Macko (2006) suggest that patients with the poorest balance have the lowest ambulatory activity levels. In addition, Michael, Allen, & Macko (2005) used a descriptive correlational study with 28 men and 22 women who were at least 6 months post stroke and found that hemiparetic participants had a low level of ambulatory activity compared to controls and that their inactivity was related to mobility deficits. The authors also suggested that balance related inactivity might be an important factor in deconditioning.

Studies on the energy requirements of walking reported that the energy cost of walking of hemiparetic participants was 1.5 to 2 times greater than healthy controls and that the energy expenditure was elevated by 55–100% in hemiparetic participants compared with healthy controls (Gerston & Orr, 1971; Ivey, Hafer-Macko, & Macko, 2006; Corcoran, Jebson, Brengelman, & Simons, 1970). Zamparo, Francescato, De Luca, Lovati, & di Prampero (1995) also found that the difference in energy cost of level walking between hemiplegic participants and healthy controls was even increased at lower speeds. Among other possible factors contributing to this increase of energy
expenditure, the level of spasticity following stroke has been correlated with the energy
cost of walking (Bard, 1963; Zamparo, 1998).

Hemiparetic stroke participants were also observed as being unable to maintain
their self-selected speed comfortably, indicating that poor endurance limited their
functional mobility (Ivey, Hafer-Macko, & Macko, 2006). In a study performed by Mol &
Baker (1991), elderly stroke participants demonstrated substantial activity intolerance
including dyspnea, progressive slowing, and worsened motor dexterity during a 50 yard
self selected speed ambulation task. This raises the issue of walking safety as well as
the limits of tolerance for chronic stroke patients. In fact, these effects of body
deconditioning have a significant impact on the ability to perform activities of daily living.
This results in decreased independence and the continuation of deconditioning. The
activity most affected by stroke is walking (Skilbeck, Wade, Langton Hewer, & Wood,
1983) with as many as 80% of individuals initially losing this ability (Jorgensen,
Nakayama, Raaschou, & Olsen, 1995). It has been noted that people returning home
after rehabilitation walk at average speeds that are insufficient to cross the street safely,
or even to walk safely in the community (Jorgensen, Nakayama, Raaschou, & Olsen,
1995).

In terms of cardio-pulmonary capacity, it has been shown that the VO₂ peak
range needed to perform ADLs is between 3-5 METS (1 MET is equal to the resting
consumption value of 3.5 ml/kg/min) (Ivey, Hafer-Macko, & Macko, 2006). Ivey, Hafer-
Macko, & Macko (2006) showed that the VO₂ peaks of stroke patients tested while
walking on a treadmill were in the middle of this range representing about half of that of
sedentary age-matched controls. This may indicate that stroke participants must work to
almost complete exhaustion in order to achieve average tasks for ADL due to the
deconditioning. Furthermore, the upper range of ADL is nearly impossible for them to achieve.

Physical deconditioning along with age-associated declines in fitness and muscle mass can further contribute to activity intolerance, therefore compromising patients’ capacity to meet the high-energy demands of hemiparetic gait (Macko, Smith, Dobrovolny, Sorkin, Goldberg, & Silver KH, 2001). Cress & Meyer (2003) found that a peak VO₂ of 20 ml/kg/min was needed for independent living for adults aged 65-97 years. Unfortunately, individuals who have sustained a stroke have VO₂ levels of 14ml/kg/min (Ivey, Hafer-Macko, & Macko, 2006; Eng, Dawson, & Chu, 2004; MacKay-Lyons & Makrides, 2004; Fujitani, Ishikawa, Akai, & Kakurai, 1999). Other behavioural factors contributing to the loss of cardiovascular fitness of hemiparetic participants include the lack of regular physical activity (Ryan, Dobrovolny, Smith, Silver, & Macko, 2000; Newman, Haggerty, Kritchevsky, Nevitt, & Simonsick, 2003). It has been recently reported that the ambulatory activity profiles of hemiparetic participants were particularly low (2837 steps/day) when compared with sedentary older adults (5000-6000 steps/day) (Michael, Allen, & Macko, 2005). This low level of physical activity likely results in a poor fitness level.

The decrease in cardiovascular fitness post-stroke can be caused by several factors. It has been found that there are changes in skeletal muscle that could propagate disability and contribute to low fitness levels; these include gross muscular atrophy (Ryan, Dobrovolny, Smith, Silver, & Macko, 2002), fiber phenotype shifts (De Deyne, Hafer-Macko, Ivey, Ryan, & Macko, 2004), and associated insulin resistance (Kernan, Inzucchi, Viscoli, Brass, Bravata, Shulman, McVeety & Horwitz, 2003). Moreover, the decrease of the central neural drive (Ivey, Macko, Ryan, & Hafer-Macko, 2005; Sheperd,
2001), blow flow (Landin, Hagenfeldt, Saltin, & Wahren, 1977; Ivey, Gardner, Dobrovolny, & Macko, 2004), and muscle mass (Ryan, Dobrovolny, Smith, Silver, & Macko, 2000; Ryan, Dobrovolny, Smith, Silver, & Macko, 2002) of the paretic limb has been closely associated with the poor fitness of hemiparetic participants. Paretic muscles primarily recruit glycolytic Type II fibers to initiate contraction, whereas nonparetic muscles activate Type I fibers (De Deyne, Hafer-Macko, Ivey, Ryan, & Macko, 2004). This modification of recruitment patterns can result in diminished capacity for oxidative metabolism and, eventually, decreased exercise endurance. The proportion of the fast myosin heavy chain has also been shown to be inversely associated with severity of gait deficit in the paretic leg of chronic stroke individuals, indicating a possible contribution of muscle phenotype to the disability of stroke (De Deyne, Hafer-Macko, Ivey, Ryan, & Macko, 2004). Ryan, Dobrovolny, Smith, Silver, & Macko (2002) used a cross sectional design to study 60 chronic hemiparetic stroke patients and found that lean tissue mass was lower and fat deposition within the muscle was higher in the hemiparetic limb. The authors suggested that these abnormalities may contribute to functional disability and increase the risk of secondary stroke due to their association with insulin resistance.

In summary, chronic hemiparetic stroke individuals have cardiovascular and muscular deconditioning that increases the energy cost of walking. This may be a contributor to the early onset of fatigue in this population.

**Effect of Physical Exertion on Balance**

The factors that could potentially cause a decrease in balance performance after physical exertion focus on both central and local means of fatigue. Central, or whole-body fatigue, occurs after endurance / aerobic exercises such as walking, biking, or
Whole body fatigue refers to a decrease in the central nervous system output to the muscles and likely includes factors responsible for the sense of effort in addition to the alterations in motor pathways (Potempa, Lopez, Braun, Szidon, Fogg, & Tincknell, 1995). Local fatigue is caused by repetitive contractions of one or a small number of muscles such as the plantar flexors. Localized muscle fatigue is induced by a decrease in the metabolic substrates available for muscle contraction, such as adenosine triphosphate, creatine phosphate, and glycogen. There is also an increase in metabolites, including lactic acid, resulting in an inability to maintain a desired muscular force output (Potempa, Lopez, Braun, Szidon, Fogg, & Tincknell, 1995). Since balance depends upon the central nervous system and on the three sensory systems (visual, vestibular, and somatosensory), alterations in central nervous system ability due to fatigue will likely affect one’s ability to maintain balance (Shumway-Cook & Woollacott, 2001). Recent cross-sectional studies indicated that nearly 70% of stroke participants self-report fatigue as a problem affecting function, irrespective of the time since they sustained their stroke (Ingles, Eskes, & Phillips, 1999).

Corbeil, Blouin, Begin, Nougier, & Teasdale (2003) performed a study that examined the effects of fatigue on balance in healthy males. The participants were asked to perform repeated plantar flexion of both legs at 50-75% of their maximal workload while sitting. Following the exercise protocol, participants exhibited an increased postural sway characterized by faster center of pressure (COP) velocity and greater mean and median frequency of COP displacements. The authors suggested that fatigue places higher demands on the postural control system by increasing the frequency of actions needed to regulate the upright stance. These actions would be associated with discrete control of the postural oscillations required to compensate the motor and/or sensory deficiencies induced by peripheral muscular fatigue. The altered
force production by the fatigued muscles would require an increase in the frequency of
the corrections in order to avoid greater displacements of the COP (Corbeil, Blouin,
Begin, Nougier, & Teasdale, 2003).

How localized muscle fatigue may affect the control of posture is not clear.
Muscle fatigue is defined as a decrease in the force generating capacity of the muscles.
The mechanisms that cause this reduction in force must be addressed peripherally and
centrally with regard to the nervous system. Within the initial minutes after exercise,
fatigue is due to metabolic factors such as the resynthesis of phosphocreatine (Baker,
Kostov, Miller, & Weiner, 1993). The second stage is related to impairment of the
excitation-contraction coupling and lasts longer (Moussavi, Carson, Boska, Weiner, &
Miller, 1989). The decrease in muscle force leads to a decrease in working capacity and
represents an "internal perturbation" to the motor system. It can therefore produce
impairment in motor coordination and possibly in postural control. At the peripheral level,
pre- and postsynaptic mechanisms and sites are potentially implicated, including a
failure in the transmission of the neural signal or a failure of the muscle to respond to
neural excitation. At the central level, fatigue may induce a failure of excitation of the
motoneurons caused by changes in the nervous system (supraspinal, segmental, and
sensory feedback). The origin of the changes in motoneuron firing has been attributed to
the intrinsic properties of the motoneurons, recurrent inhibition (Renshaw cell), reflex
inhibition or disfacilitation, and changes in descending drive to the motoneuron pool
(Dietz, 1992).

With regard to whole-body fatigue, Nardone, Haggerty, Kritchevsky, Nevitt, &
Simonsick (1998) performed a study examining the effect of physical exertion on
balance in young healthy adults. The participants performed 25 minutes (85% maximum
predicted heart rate) of running on a treadmill. The COP was assessed before and after the exercise and the results showed a significant increase in body sway when comparing pre and post measures. The postural sway increased the most in the initial minutes after having completed the exercise. After 15 minutes post-exercise, full recovery to baseline COP measures was achieved. It should be noted, however, that the study consisted of a small sample group (n=8). Also, the participants reported being dizzy after having finished the exercise. However, to wait out the dizziness, the participants were given a 3 minute rest period before measuring COP. This could have affected the results in terms of timing of recovery. The authors stated that dizziness itself was not the consequence of post-exercise orthostatic hypotension or vasovagal response, since blood pressure and heart rate increased after the exercise.

In summary, balance seems to be affected by a generalized fatigue induced by strenuous aerobic physical exercise and/or local fatigue of the plantar flexors. The effects of fatigue are characterized by increased postural sway due to the reduction in muscle force, failure of excitation of the motoneurons, and possible alterations of the three sensory systems (visual, vestibular, and somatosensory).

Rationale and Objective

Studies have shown that physical exertion can cause a state of postural instability in healthy individuals (Corbeil, Blouin, Begin, Nougier, & Teasdale, 2003; Nardone, Tarantola, Galante, & Schieppati, 1998). A common effect of stroke is balance impairment (Geurts, de Haart, van Nes, & Duysens, 2005; Eng & Chu, 2002; Niam, Cheung, Sullivan, Kent, & Gu, 1999; Sackley, 1991; Ikai, Kamikubo, Takehara, Nishi, & Miyano, 2003; Cheng, Liaw, Wong, Tang, & Lee, 1998; Dault, de Haart, Geurts, Arts, & Nienhuis, 2003). This impairment in postural stability increases the risk of falling and
may cause injuries such as hip fractures, thereby further reducing a stroke person's ability to perform activities of daily living and to achieve independence (Sackley, 1991; Melzer, Benjuya, & Kaplanski, 2004; Jorgensen, Engstad & Jacobsen, 2002; Campbell, Reinken, Allan, & Martinez, 1981; Hyndman & Ashburn, 2002; Forster & Young, 1995; Belgen, Beninato, Sullivan, & Narielwalla, 2006; Harris, Eng, Marigold, Tokuno & Louis, 2005; Mackintosh, Hill, Dodd, Goldie, & Culham, 2005; Cumming, Salkeld, Thomas, & Szonyi, 2000; Ho, Woo, Yuen, Sham, & Chan, 1997; Vellas, Cayla, Bocquet, de Pemille, & Albareda, 1987; Rapport, Webster, Flemming, Lindberg, Godlewska, Brees, & Abadee, 1993; Mayo, Korner-Bitensky, & Kaizer, 1990; Cheng, Liaw, Wong, Tang, Lee, & Lin, 1998). Furthermore, chronic hemiparetic individuals are often in a state of physical deconditioning. This state is caused by a cycle that starts with poor initial pre-stroke fitness levels that are exacerbated by the impact of the stroke; namely the reduced ability to walk due to impaired gait and reduced balance control. This leads to increased sedentary behaviour that continues the cycle and can lead to further deconditioning (Figoni, 1995; Michael, Allen, & Macko, 2005; Macko, DeSouza, Tretter, Silver, Smith, Anderson, Tomoyasu, Gorman, & Dengel, 1997; Potempa, Braun, Tinknell, & Popovich, 1996; Macko, Ivey, Forrester, Hanley, Sorkin, Katzel, Silver, & Goldberg, 2005).

As a result of the deconditioning of the muscles and of poor cardiovascular fitness, chronic hemiparetic stroke individuals have less endurance to accomplish activities of daily living. For example, tasks such as walking across a street in a community setting can be difficult and can, therefore, greatly affect their independence (Michael, Allen, & Macko, 2005; Ivey, Hafer-Macko, & Macko, 2006; Gerston & Orr, 1971; Corcoran, Jebson, Brengelman, & Simons, 1970; Zamparo, Francescato, De Luca, Lovati, & di Prampero, 1995; Bard, 1963; Mol & Baker, 1991; Skilbeck, Wade, Langton Hewer, & Wood, 1983; Jorgensen, Nakayama, Raaschou, & Olsen, 1995;
Macko, Smith, Dobrovolny, Sorkin, Goldberg, & Silver, 2001; Cress & Meyer, 2003; Eng, Dawson, & Chu, 2004; MacKay-Lyons & Makrides, 2004; Fujitani, Ishikawa, Akai, & Kakurai, 1999; Ryan, Dobrovolny, Smith, Silver, & Macko, 2000; Newman, Tarantola, Galante, & Schieppati, 2003). Unfortunately, postural instability associated with fatigue will likely be attained more rapidly for stroke individuals than for healthy individuals. This raises the issue of safety and risk of falling after chronic hemiparetic stroke individuals perform a bout of aerobic exercise as they will have fatigued more rapidly. Furthermore, it has been shown that the time course recovery for initial postural stability of young healthy individuals is about 15 minutes (Nardone, Tarantola, Galante, & Schieppati, 1998). Since hemiparetic participants present with poor fitness levels, this time course of balance recovery could be even longer.

The main objective of this study was to determine the effects of physical exertion induced by over-ground walking on the postural stability of chronic hemiparetic participants. The results from the hemiparetic participants were compared to those of healthy control participants. The specific objective was to determine the effects of walking duration (6 min vs. 18 min) at comfortable speed on postural sway and step reaction time. The effects were measured in both groups before the walks, immediately after having completed the timed walks, 15 minutes post-exercise, and 30 minutes post-exercise to establish the time course of recovery.

It was hypothesized that physical exertion would cause a decrease of postural stability and an increase in step reaction time in hemiparetic participants compared to healthy controls. Specifically, that following the 6 minute walk, hemiparetic participants would show a decrease of postural stability and an increase in step reaction time whereas healthy control participants would not. Also, that walking over a longer period of
time (18 min vs. 6 min) would lead to a greater decrease of postural stability and an increase in step reaction time in hemiparetic participants compared to healthy controls. It was further hypothesized that walking over a longer period of time (18 min vs. 6 min) would increase the time course of balance recovery in hemiparetic participants compared to healthy controls.
Methods

The study was conducted at the Constance-Lethbridge Rehabilitation Centre in Montreal, Quebec, Canada. The Centre is a research site for the Centre for Interdisciplinary Research in Rehabilitation of Greater Montreal (CRIR). The study was approved by the CRIR ethics committee and the Human Research Ethics committee of Concordia University.

Participants

The recruitment for the study took place through the Constance-Lethbridge Rehabilitation Center. Twelve chronic hemiparetic participants and 12 healthy control participants between the ages of 48 and 72 years were recruited. The healthy control group was matched for age and sex. Pre-experimental power analysis was based on pilot data from three hemiparetic participants and two healthy control participants. The center of pressure (COP) mediolateral velocity was selected to calculate the sample size. This variable is sensitive to changes in double-legged stance performance after the completion of the 6-min walk at comfortable speed. According to Cohen’s equations (Cohen, 1988), a sample size of 12 participants per group was needed to reach an alpha level of 0.05 and a power of 80% (Appendix A).

The participants in the study signed an informed consent form prior to taking part in the study. To be included in the study, stroke participants had to fulfill these inclusion criteria: 1) present hemiparesis secondary to a stroke; 2) be a minimum of six months post stroke; 3) Chedoke-McMaster Stroke Assessment (Gowland, Van Hullenaar, Torresin, Moreland, Vanspall, Barreca, Ward, Huijbregts, Stratford, & Barclay-Goddard, 1995) score between 3 and 6; 4) be able to walk six minutes with or without assistive
devices (such as canes) and; 5) have completed the modified Par-Q form by the Cummings Jewish Center for Seniors Physical Activity Questionnaire (Appendix B). The inclusion criteria for control participants were that they were age and sex matched. The exclusion criteria were filtered with the use of the above mentioned modified PAR-Q which provided the medical history, current condition, and activity profile of the individual. They include medical conditions such as; 1) uncontrolled high blood pressure; 2) heart condition (arrhythmia); 3) a history of a heart condition (such as a myocardial infarction); 4) any other condition that would have severely limited participation in the physical exertion protocol as stipulated by the American College of Sports Medicine's guidelines (2005) for exercise testing and the American Thoracic Society guidelines (2002) for the 6 Minute Walk Test (6-MWT) (Guyatt, Sullivan, Thompson, Fallen, Pugsley, Taylor, & Berman, 1985); and 5) neurological conditions not related to stroke (e.g., Parkinson's disease) or severe musculoskeletal conditions that would alter postural stability.

**Experimental Procedures**

The overall research design looked at the effect of walking duration and walking speed on postural stability of hemiparetic participants. During the study, results were obtained for both parameters. The present research project focused on the duration aspect. Therefore, when describing the experimental procedures, on the days that the participants walked a duration of 6 minutes at comfortable speed, they also walked a duration of 6 minutes at their fastest safe speed after a half hour break.

Each hemiparetic participant was randomized as to whether they performed the 6 minute walk on their first visit or on their second visit. For each hemiparetic participant, their age and sex matched control participant followed the same randomization. If the
hemiparetic participant performed the 6 minute walk on the first testing day, then the
control participant did the same. Therefore, on their second testing day, they performed
the 18 minute walk. This ensured that they followed the same testing procedure.

Hemiparetic participants

The hemiparetic participants (Table 1) had 3 visits to the laboratory. During their
first visit, hemiparetic participants performed the following impairments and functional
performance measures: Berg Balance Scale (Berg, Wood-Dauphinee, Williams, &
Gayton, 1989), Timed-Up-and-Go (Podsiadlo & Richardson, 1991), Human Activity
Profile (physical activity status questionnaire) (Fix & Daughton, 1988) and the lower-
extremity component (leg and foot) of the Chedoke-McMaster Stroke Assessment
(Gowland, Van Hullenaar, Torresin, Moreland, Vanspall, Barreca, Ward, Huijbregts,
Stratford, & Barclay-Goddard, 1995). The hemiparetic participants performed the test
using their regular assistive device if needed. These clinical tests served to establish the
severity of the hemiparesis and the functional level of hemiparetic participants. During
the same session, hemiparetic participants were asked to perform a practice trial of 6-
minute walking. This walking trial was performed in an empty corridor (50 meters long) at
the participant’s own comfortable walking speed. During this trial, the participants wore
the Cosmed system in order to familiarize themselves with the mask. In addition, their
maximal speed was measured by timing their walk over a 10m distance. During the
second and third visit to the laboratory, hemiparetic participants were asked to perform
either the 6 minute walk or the 18 minute walk depending on the randomization results.
On the day of their 6 minute walk, participants performed two 6-min trials of overground
walking: 1) at comfortable speed and 2) at the fastest safe speed. To minimize fatigue
build-up due to repeated tests, participants were given a 30-minute break between the 2
<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59.9</td>
<td>8.1</td>
<td>48 - 72</td>
</tr>
<tr>
<td>Time since stroke (years)</td>
<td>8.1</td>
<td>4.0</td>
<td>4 - 15</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>79.0</td>
<td>14.4</td>
<td>56 - 97</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165.0</td>
<td>9.5</td>
<td>149 - 186</td>
</tr>
<tr>
<td>Functional Performance Measures:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chedoke Score (1-7)</td>
<td>4.4</td>
<td>1.24</td>
<td>3 - 6</td>
</tr>
<tr>
<td>Berg Balance Score / 56</td>
<td>45.5</td>
<td>8.9</td>
<td>29 - 56</td>
</tr>
<tr>
<td>TUG (sec)</td>
<td>23.2</td>
<td>17.5</td>
<td>10.2 - 60.1</td>
</tr>
</tbody>
</table>

| N                                |       |       |             |
| Type of stroke                    |       |       |             |
| Ischemic / Hemorrhagic            | 7 / 5 |       |             |
| Sex                              |       |       |             |
| Male / Female                     | 5 / 7 |       |             |
| Chronic conditions                |       |       |             |
| Hypertension                      | 8     |       |             |
| Arthritis                         | 1     |       |             |
| Epilepsy                          | 1     |       |             |
| Diabetes                          | 4     |       |             |
| Hemiparetic side                  |       |       |             |
| Right / Left                      | 6 / 6 |       |             |
| Assistive device used             |       |       |             |
| Cane                              | 4     |       |             |
| Quad-stick                        | 3     |       |             |
| Stick                             | 2     |       |             |
| Ankle-foot Orthotic               | 3     |       |             |

Abbreviations: TUG, Timed-Up-and-Go

*Table 1. Profile of the Hemiparetic Participants*
walking tests. Postural stability assessment was performed before and after the walking trials. On the day of the 18 minute walk, participants were asked to walk at their self-selected comfortable speed. As stated above, postural stability assessment was performed before and after the 18-minute walk trial.

Control participants

The control participants had 2 visits to the laboratory. Prior to the actual testing, the participant’s physical measurements of height and weight were taken. In addition, their maximal speed\(^\text{A}\) was measured by timing them over a 10-m distance. Following a break, the walking trial was performed in an empty corridor (50 meters long) during which they maintained the exact average heart rate of their hemiparetic counterpart. During their first visit to the laboratory, control participants performed either the 6 minute walk or the 18 minute depending on what their matched hemiparetic participant had been randomized to do. Postural stability assessment was performed before and after the walking test.

Functional Performance Measures

*Berg Balance Scale (BBS)*

The BBS measures static and dynamic balance. It is composed of 14 tasks, graded on a 5-point scale for a total of 56, that require the participant to perform various balance tasks such as maintaining a static position, changing the orientation of the center of mass with respect to base of support, and diminishing base of support. The BBS has been established as a valid and reliable tool for measuring functional balance

\(^\text{A}\) The maximal speed is equivalent to the fastest secure speed of participants.
in patients who present with a history cerebrovascular accident. Its reliability in patients with acute stroke is excellent (intraclass correlation coefficient of 0.99) (Berg, Wood-Dauphinee, Williams, & Gayton, 1989).

**Chedoke-McMaster Stroke Assessment Impairment Inventory**

Stages of recovery of the leg, foot, and postural control were assessed by using the Chedoke-McMaster Stroke Assessment (CMSA) Impairment Inventory. The CMSA provides an indication of the level of impairment of motor control at admission in the study. High intrarater (r range, .94-.98) and interrater (r range, .85-.96) reliability have been reported for the CMSA Impairment Inventory items (Gowland, Van Hullenaar, Torresin, Moreland, Vanspall, Barreca, Ward, Huijbregts, Stratford, & Barclay-Goddard, 1995).

**Timed-Up-and-Go Test (TUG)**

The TUG was used to assess functional mobility where “functional mobility” refers to balance and gait maneuvers used in daily activities. The TUG has been shown to have good interrater (ICC .99) and intrarater (ICC .99) reliability (Cohen, Heaton, Congdon, & Jenkins, 1996; Podsiadlo & Richardson, 1991). The participant sat in a chair with arms, with their back against the chair and feet on the floor. The time it takes for the participant to rise, walk 3m at their usual pace, turn around, and walk back to the chair and sit down was timed using a stopwatch. If needed, ambulatory assistive devices were allowed (Podsiadlo & Richardson, 1991).

**Physical Exertion Protocol**

For the present study, the time duration of 6 minutes was selected based on scientific standards described in the literature. As previously discussed, this research
The traditional 6-MWT (Guyatt, Sullivan, Thompson, Fallen, Pugsley, Taylor, & Berman, 1985), the goal of which is to cover as much ground as possible in 6 min, is commonly used in stroke rehabilitation to measure walking endurance and exercise capacity (Eng & Chu, 2002; Dean, Richards, & Malouin, 2000; Leroux, 2005; Salbach, Mayo, Wood-Dauphinee, Hanley, Richards, & Cote, 2004; Olney, Nymark, Brouwer, Culham, Day, Heard, Henderson, & Parvataneni, 2006). This walking test has been shown to reflect an exercise intensity ranging from 63 to 77% of age-predicted heart rate maximum value (Eng & Chu, 2002; Eng, Dawson, & Chu, 2004) and to bring stroke participants to a level of 70% of the VO2max. This value is above the anaerobic threshold and is high enough to initiate lactate production (Knuttgen & Saltin, 1972). On the other hand, the time duration of 18 minutes was chosen to parallel the average time that individuals with stroke usually need to perform outside activities of daily living (Lerner-Frankiel, Vargas, Brown, Krusell, & Shoneberger, 1986).

The hemiparetic participants were asked to walk from one end of the corridor to the other (marked by cones) at a comfortable speed during the allotted time. For safety purposes, participants were accompanied by two exercise specialists during their walk. One specialist noted the distance covered by the participant at each minute. For the control participants, one exercise specialist monitored the heart rate to maintain the exact heart rate as their matched hemiparetic participant. The exercise specialist would ask the control participant to either slow down or increase their speed in order to match the heart rate.
**Measurements**

Postural stability measures were the main outcome measures of this study. In addition to postural stability, measures of physical exertion during walking were obtained from the cardiorespiratory parameters, time-distance parameters and subjective scales. The effects of physical exertion on postural stability were quantified before (pre-test) and immediately after the walk (post-test 1). To establish the time course of recovery, postural stability testing was performed at 15 minutes post-exercise (post-test 2) and 30 minutes post-exercise (post-test 3). Participants were asked to sit and rest between each post-test evaluation.

**Postural Stability Measures**

Postural stability was measured during the following functional tasks: Task 1) double-legged stance (Piirtola & Era, 2006) for 30 s; Task 2) step reaction time (Marigold, Eng, Dawson, Inglis, Harris, & Gyfadaldr, 2005; Brauer, Burns, & Galley, 2000); Task 3) sit-to-stand (Cheng, Liaw, Wong, Tang, Lee, & Lin, 1998) from a chair. Task 1 was chosen to measure postural stability under conditions of static balance whereas tasks 2 and 3 assessed the lower-limb speed of motion and force exertion under dynamic balance conditions. When performing the double-legged stance task, participants were instructed to maintain a steady posture and a horizontal gaze while fixating on a red dot on the wall. For the step reaction time, participants were instructed to step forward as fast as possible after hearing an auditory cue. The same procedure was repeated bilaterally. For the sit-to-stand task, participants were seated on an armless and backless chair adjusted to their lower-leg length and were instructed to rise from the chair at their maximal speed and stand 3 seconds and sit back using a standardized symmetric foot position (15° of dorsiflexion) (Roy, Nadeau, Gravel,
Malouin, McFadyen, & Piotte, 2006; Leroux, Pinet, & Nadeau, 2006). Participants performed one trial of task 1 and three trials of tasks 2 and 3 (Leroux, Pinet, & Nadeau, 2006). In the event of unsuccessful trials, participants were allowed an additional trial to complete the task. The task order presented above was kept constant for all test sessions. During tasks 1, 2 and 3, COP measurements were recorded using the Matscan system (Tekscan, Inc., USA). The Matscan is a pressure sensing floor mat (43.2 cm x 36.8 cm) consisting of 2288 sensors to measure the applied pressures at a sampling rate of 40 Hz. This technology has shown to be accurate (Hsiao, Guan, & Weatherly, 2002), reliable (Pinet, Boucher, & Leroux, 2003), and sensitive in detecting changes in postural stability of stroke participants (Leroux, Pinet, & Nadeau, 2006).

Physical Exertion Measures

A portable metabolic measurement system (Cosmed k4b2, Italy) was used to measure the cardiorespiratory responses (VO2 consumption, heart rate, respiratory rate, and energy cost) to exercise during each walking trial. These parameters served to characterize the effects of walking for various durations on the participants’ physical exertion. This system accurately measures oxygen consumption and carbon dioxide production on a breath-by-breath basis at both rest and maximal exercise capacity (McLaughlin, King, Howley, Bassett, & Ainsworth, 2001). The test-retest reliability of the cardiorespiratory variables measured by the Cosmed k4b2 system has been established for the 6-min walk test in healthy elderly participants (Kervio, Carre, & Ville, 2003). This system has also shown to be well tolerated by hemiparetic participants performing the conventional 6-min walk test (Leroux, 2006). When performing walking trials, the participants wore a soft mask and a harness that contains the Cosmed k4b2 device, a small battery pack, a heart rate monitor and a temperature receiver, for a total weight of
800 g. In order to familiarize the participants with the portable metabolic system, the device was used during their trial run on their first visit to the laboratory.

In addition to their cardiorespiratory responses, the participants’ physical exertion was measured using these variables: walking speed, walking distance, rating of perceived exertion (RPE) and the visual analog scale (VAS). Participants were asked to rate their level of RPE on the 6-20 point Borg Scale (Borg, 1982) every 2 minutes and during the last 10 seconds of the walk. This scale correlates closely with several variables, including oxygen uptake, heart rate and lactate production in older adults (Shigematsu, Ueno, Nakagaichi, Nho, & Tanaka, 2004). Participants also filled out a 10-cm VAS at the end of the walk to measure their level of fatigue. The extreme left of the line will represent “No fatigue” and the extreme right “Worst possible fatigue” (Banthia, Malcarne, Roesch, Ko, Greenbergs, Varni, & Sadler, 2006). Participants were asked to cross the VAS line at the point where it best represented their state of fatigue as well as to specify their type of fatigue (general or lower limb).

**Quantification and Analyses**

**Postural Stability Measures**

For double-legged stance, the peak-to-peak and standard deviation of the COP displacement were calculated over 30 seconds and reported as “peak-to-peak” and the “variability” in the anteroposterior (AP) and mediolateral (ML) axes. For sit-to-stand, the peak-to-peak of the COP displacement was calculated during the period corresponding to the rising phase: from onset of movement to standing as measured by the peak of vertical ground reaction forces (Leroux, Pinet, & Nadeau, 2006). Included in postural stability measures, were postural reactions which were measured using the step reaction
time. The time between the auditory cue and the moment when the vertical force on the Matscan reached 0 was calculated for each trial and each lower limb. Three trials per test were averaged for statistical analyses.

Physical Exertion Measures

Cardiorespiratory responses (VO₂ consumption, heart rate, respiratory rate, energy cost) and walking speed data of each participant were reported on a minute-by-minute basis and a mean profile was established for the walking trials. To assess the effect of the walking conditions on physical exertion, the VO₂ consumption in the steady-state period, the walking distance covered by the participants, RPE (6-20), and VAS (0-10) were used as first outcomes.

Statistical Analyses

The statistical model served to determine whether walking a longer duration produces a greater destabilizing effect on postural stability than performing the walk for a shorter duration in stroke and healthy control participants. The analyses were done using SPSS 15.0. Variables on postural stability were analyzed using Generalized Estimating Equations (GEE) with one between-subject factor (hemiparetic vs. control) and two within-subject or repeated factors (walking duration and measurement repetition). For the first objective (immediate effect of exertion), GEE were performed on the data from the baseline and post-test 1 only between the two groups. For the second objective (recovery pattern), GEE were performed on the data from all four measurement points. We chose to test the complete factorial model to assess the presence of factor interactions. The significance level for GEE analyses was set at \( p < 0.05 \) for each one of the model components. The GEE procedure extends the generalized linear model to allow for analysis of repeated measurements or other
correlated observations, and does not assume multivariate normality. If an interaction term was found significant, appropriate sub-analyses were performed to determine where the differential effect was. When significant differences were found, all pairwise contrasts (with a Bonferroni adjustment) were used to determine where the differences were. In the sub-analyses, the overall level of significance was set at $p < 0.05$ for each dependent variable.

Variables on physical exertion, on the other hand, were analyzed using a 2-way (group x walking condition) repeated-measure ANOVA for each experiment. When significant differences were found ($p < 0.05$), pairwise comparisons were made using the Tukey test. These statistical tests served to establish whether walking over a longer duration (18 minutes) had a higher energy demand on the body than performing the 6-minute walk in stroke and healthy control participants.
Results

**Physical Exertion**

Table 2 shows the effects of walking duration on physical exertion parameters. In general, the results revealed higher values for the physical exertion variables of both hemiparetic and control participants when performing the 18 minute walk versus the 6 minute walk. Statistical analyses revealed a significant main effect due to walking duration (6 min vs. 18 min) for heart rate, RPE and VAS. These variables showed significantly larger values ($p < 0.05$) during the 18 minute walk vs. 6 minute walk for both groups. In contrast, the minor changes in oxygen uptake did not produce a significant change due to walking duration in both groups of participants. A significant main effect due to group (hemiparetic vs. control) was, however, found for the oxygen uptake. The oxygen uptake was significantly larger ($p < 0.05$) for the control group at each walking duration.

**Postural Stability**

Figure 1 shows AP peak-to-peak during double-legged stance for both groups and for both walking durations. When examining the immediate effect of exertion, Table 3A shows a main effect due to group ($Wald \chi^2_{1df} = 5.1, p = 0.02$). Figure 1 indicates that the hemiparetic group had a higher AP peak-to-peak compared to the control group (2.9 cm ± 0.2 for hemiparetic group vs. 2.4 cm ± 0.1 for control group).
### Table 2. Effects of Walking Duration on Physical Exertion Parameters

<table>
<thead>
<tr>
<th>Variables</th>
<th>Hemiparetic Participants</th>
<th>Control Participants</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6-Minute Walk</td>
<td>18-Minute Walk</td>
</tr>
<tr>
<td>Walking Speed&lt;sup&gt;†&lt;/sup&gt; (m/s)</td>
<td>0.84 ± 0.28</td>
<td>0.87 ± 0.30</td>
</tr>
<tr>
<td>Walking Distance (m)</td>
<td>300.68 ± 99.77</td>
<td>963.27 ± 319.78</td>
</tr>
<tr>
<td>Oxygen Uptake&lt;sup&gt;‡&lt;/sup&gt; (ml/kg/min)</td>
<td>9.7 ± 1.9</td>
<td>10.1 ± 3.1</td>
</tr>
<tr>
<td>Heart Rate&lt;sup&gt;‡&lt;/sup&gt; (beats/min)</td>
<td>99.4 ± 14.4</td>
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Abbreviations. RPE, rating of perceived exertion; VAS, visual analog scale.  
<sup>†</sup> data averaged over the steady-state period (min 3 to 18).
Figure 1. Average (mean ± 1 SD) antero-posterior peak-to-peak center of pressure displacement during double-legged stance for the 6-minute (A) and 18-minute (B) walk in control and hemiparetic participants. The mean center of pressure displacement was measured before the walk (baseline), immediately after the walk (post 1), 15 minutes post-walk (post 2), and 30 minutes post-walk (post 3).
### A. Tests of Model Effects

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### B. Tests of Model Effects

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*Table 3.* Generalized Estimating Equations for the immediate effect of exertion (A) and recuperation pattern (B) with one between-subject factor (hemiparetic participants vs. control participants) and two within-subject or repeated factors (walking duration and measurement points) for antero-posterior peak-to-peak center of pressure displacement during double-legged stance.
With regards to the recuperation pattern after exertion, the AP peak-to-peak after double-legged stance did not show a significant interaction. Table 3B shows that there was a main effect due to group on AP peak-to-peak (Wald $\chi^2_{1,df} = 4.3$, $p = 0.04$). Figure 1 shows that the hemiparetic group had a somewhat higher AP peak-to-peak compared to the control group (2.8 cm ± 0.10 for hemiparetic group vs. 2.3 cm ± 0.2 for control group). The measurement point had a significant effect on AP peak-to-peak (Wald $\chi^2_{3,df} = 24.6$, $p < 0.001$). Post hoc tests indicate that there was a significant decrease in AP peak-to-peak between post-tests 1 and 2 for both groups after both walking durations.

The profile of ML peak-to-peak during the double-legged stance task can be seen in Figure 2. Table 4A shows that the ML peak-to-peak had a significant triple interaction for the immediate effect of exertion (Wald $\chi^2_{1,df} = 13.1$, $p < 0.001$). Two sub-analyses were first performed: one on the baseline data and one on the post-test 1 data. At baseline, the groups showed significant differences in ML peak-to-peak (Wald $\chi^2_{1,df} = 9.4$, $p = 0.002$), where the hemiparetic group had a larger ML peak-to-peak than the control group (2.1 cm ± 0.1 vs. 1.5 cm ± 0.1). At post-test 1, there is a significant interaction of group by duration of the ML peak-to-peak (Wald $\chi^2_{1,df} = 7.7$, $p = 0.005$). Further sub-analyses indicated that the hemiparetic group had a significantly higher ML peak-to-peak compared to the control group (2.9 cm ± 0.2 vs. 1.7 cm ± 0.2) after the 18 minute walk (Wald $\chi^2_{1,df} = 20.5$, $p < 0.001$) which is not the case after the 6 minute walk (Wald $\chi^2_{1,df} = 1.3$, $p = 0.261$). Furthermore, the hemiparetic group had a significantly higher ML peak-to-peak after the 18 minute walk compared to after the 6 minute walk (2.9 cm ± 0.2 vs. 1.7 cm ± 0.1, Wald $\chi^2_{1,df} = 15.2$, $p < 0.001$).
Figure 2. Average (mean + or - 1 SD) medio-lateral peak-to-peak center of pressure displacement during double-legged stance for the 6-minute (A) and 18-minute (B) walk in control and hemiparetic participants. The mean center of pressure displacement was measured before the walk (baseline), immediately after the walk (post 1), 15 minutes post-walk (post 2), and 30 minutes post-walk (post 3).
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Dependent Variable: ML peak  
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Dependent Variable: ML peak  
Model: (Intercept), duration, group, test, duration * group * test, duration * group, duration * test, group * test

*Table 4.* Generalized Estimating Equations for the immediate effect of exertion (A) and recuperation pattern (B) with one between-subject factor (hemiparetic participants vs. control participants) and two within-subject or repeated factors (walking duration and measurement points) for medio-lateral peak-to-peak center of pressure displacement during double-legged stance.
When examining the recuperation pattern after exertion, Table 4B shows that the ML peak-to-peak after double-legged stance had a significant triple interaction ($Wald \chi^2_{3df} = 20.2, p < 0.001$). Subsequent sub-analyses on each of the groups separately indicated that there was a significant duration of walk by measurement point interaction for the hemiparetic group ($Wald \chi^2_{3df} = 35.1, p < 0.001$) but not for the control group. For the hemiparetic group, after the 6 minute walk, there was a significant decrease of ML peak-to-peak after the post-test 2 compared to the baseline ($Wald \chi^2_{1df} = 9.2, p = 0.007$) whereas a significant decrease occurred between post-test 1 and post-test 2 after the 18 minute walk ($Wald \chi^2_{3df} = 26.3, p < 0.001$).

Figure 3 shows AP variability during double-legged stance for both groups and for both walking durations. When examining the immediate effect of exertion in Table 5A, the AP variability showed a significant interaction between duration of walk and measurement point ($Wald \chi^2_{1df} = 7.7, p = 0.005$). Sub-analyses (one on the baseline data and one on the post-test 1 data) indicated that the duration of the walk had a significant effect on AP variability at post-test 1 but not at baseline for both groups. Both groups had significantly higher AP variability after the 18 minute walk compared to after the 6 minute walk at post-test 1 (0.62 ± 0.057 vs. 0.54 ± 0.04, $Wald \chi^2_{1df} = 4.6, p = 0.033$). With regards to the recuperation pattern, Table 5B shows that the AP variability after double-legged stance had a significant interaction between duration of walk and measurement point ($Wald \chi^2_{3df} = 8.2, p = 0.042$). Subsequent sub-analyses revealed that this interaction reflected the lack of parallelism between the patterns of recuperation at post-test 1 only.
Figure 3. Average (mean ± 1 SD) antero-posterior variability center of pressure displacement during double-legged stance for the 6-minute (A) and 18-minute (B) walk in control and hemiparetic participants. The mean center of pressure displacement was measured before the walk (baseline), immediately after the walk (post 1), 15 minutes post-walk (post 2), and 30 minutes post-walk (post 3).
Table 5. Generalized Estimating Equations for the immediate effect of exertion (A) and recuperation pattern (B) with one between-subject factor (hemiparetic participants vs. control participants) and two within-subject or repeated factors (walking duration and measurement points) for antero-posterior variability center of pressure displacement during double-legged stance.
The ML variability during double-legged stance for both groups and for both walking durations is illustrated in Figure 4. Table 6A shows a significant interaction in ML variability for the immediate effect of exertion between duration of walk and measurement point ($Wald \chi^2_{1df} = 7.7, p = 0.006$). Sub-analyses (one on the baseline data and one on the post-test 1 data) indicated that the duration of the walk had a significant effect on ML variability at post-test 1 but not at baseline for both groups. When both groups are averaged together, there was a significantly higher ML variability after the 18 minute walk compared to after the 6 minute walk at post-test 1 ($0.435 \pm 0.032$ vs. $0.323 \pm 0.021$, $Wald \chi^2_{1df} = 8.4, p = 0.004$). Moreover, the hemiparetic group had a significantly higher ML variability than the control group at post-test 1 ($0.453 \pm 0.027$ vs. $0.305 \pm 0.025$, $Wald \chi^2_{1df} = 16.7, p < 0.001$).

While examining the recuperation pattern after exertion (see Table 6B), the ML variability after double-legged stance showed a significant interaction between duration of walk and measurement point ($Wald \chi^2_{3df} = 13.0, p = 0.005$). The interaction between group and measurement point also revealed a significant difference ($Wald \chi^2_{3df} = 24.0, p < 0.001$). Subsequent sub-analyses showed that the hemiparetic group had a significant interaction between duration of walk and measurement point ($Wald \chi^2_{1df} = 9.7, p = 0.022$) whereas the control group had a simple significant effect of measurement point ($Wald \chi^2_{1df} = 12.1, p = 0.007$). For the control group, the significant effect of measurement point was between post-tests 2 and 3 where the largest decrease of ML variability was observed ($0.356 \pm 0.031$ vs. $0.287 \pm 0.025$, $Wald \chi^2_{1df} = 6.5, p = 0.012$). On the other hand, as previously observed for the ML peak-to-peak, the sub-analyses on the hemiparetic group revealed a significant increase of ML variability between baseline and post-test 1 after the 18 minute walk only ($Wald \chi^2_{1df} = 13.8, p = 0.001$).
Figure 4. Average (mean + or - 1 SD) medio-lateral variability center of pressure displacement during double-legged stance for the 6-minute (A) and 18-minute (B) walk in control and hemiparetic participants. The mean center of pressure displacement was measured before the walk (baseline), immediately after the walk (post 1), 15 minutes post-walk (post 2), and 30 minutes post-walk (post 3).
Table 6. Generalized Estimating Equations for the immediate effect of exertion (A) and recuperation pattern (B) with one between-subject factor (hemiparetic participants vs. control participants) and two within-subject or repeated factors (walking duration and measurement points) for medio-lateral variability center of pressure displacement during double-legged stance.
The AP peak-to-peak during sit-to-stand for both groups and for both walking durations is illustrated in Figure 5. Results in Table 7A revealed a significant interaction was found between the group and the measurement point (Wald $\chi^2_{1df} = 9.0$, $p = 0.003$) for the immediate effect of exertion. The analysis of this interaction reveals that at baseline, the control group had a significantly higher AP peak-to-peak than the hemiparetic group. This difference becomes non significant at post-test 1 because the hemiparetic group had an increase of its AP peak-to-peak between baseline and post-test 1 (from $6.762 \pm 0.398$ to $7.115 \pm 0.378$, Wald $\chi^2_{1df} = 4.633$, $p = 0.031$). This increase does not reach the corrected level of significance. Although the decrease between baseline and post-test 1 for the control group is larger in absolute value (from $8.670 \pm 0.272$ to $8.052 \pm 0.325$), the variability is relatively too large to make it significant.

When examining the recuperation pattern after exertion (see Table 7B), the AP peak-to-peak showed a significant interaction between the group and the measurement point (Wald $\chi^2_{3df} = 10.4$, $p = 0.016$). The sub-analyses indicates that there was a significant decrease between baseline and post-test 1 (Wald $\chi^2_{1df} = 10.8$, $p = 0.003$), post-test 2 (Wald $\chi^2_{1df} = 11.3$, $p = 0.002$) and post-test 3 (Wald $\chi^2_{1df} = 14.8$, $p < 0.001$) for the hemiparetic group but not for the control group. Nonetheless, the control group always had a significantly higher AP peak-to-peak than the hemiparetic group except at post-test 1 where it was not significant. The hemiparetic group did not show any significant change in AP peak between the measurement points.
Figure 5. Average (mean ± 1 SD) antero-posterior peak-to-peak center of pressure displacement during sit-to-stand for the 6-minute (A) and 18-minute (B) walk in control and hemiparetic participants. The mean center of pressure displacement was measured before the walk (baseline), immediately after the walk (post 1), 15 minutes post-walk (post 2), and 30 minutes post-walk (post 3).
Table 7. Generalized Estimating Equations for the immediate effect of exertion (A) and recuperation pattern (B) with one between-subject factor (hemiparetic participants vs control participants) and two within-subject or repeated factors (walking duration and measurement points) for antero-posterior peak-to-peak center of pressure displacement during sit-to-stand.
Figure 6 shows ML peak-to-peak during sit-to-stand for both groups and for both walking durations. Table 8A shows that the ML peak-to-peak had a significant triple interaction for the immediate effect of exertion (Wald $\chi^2_{1\text{df}} = 6.1$, $p = 0.013$). The analysis of this interaction reveals that both duration of walk and measurement point had an effect on ML peak-to-peak but only in the hemiparetic group. The hemiparetic group showed a higher ML peak-to-peak after the 18 minute walk compared to after the 6 minute walk ($12.6 \text{ cm} \pm 1.0 \text{ vs. } 10.9 \text{ cm} \pm 0.8$, Wald $\chi^2_{1\text{df}} = 7.9$, $p = 0.005$). The hemiparetic group also showed a higher ML peak-to-peak after exertion at post-test 1 compared to baseline ($12.6 \text{ cm} \pm 1.0 \text{ vs. } 10.9 \text{ cm} \pm 0.8$, Wald $\chi^2_{1\text{df}} = 8.7$, $p = 0.003$). The control group does not show a significant effect of either variable.

When examining the recuperation pattern after exertion (see Table 8B), the ML peak-to-peak showed a significant triple interaction (Wald $\chi^2_{3\text{df}} = 14.6$, $p = 0.002$). The analysis of this interaction reveals that, again, duration of walk and measurement point both had an effect on ML peak-to-peak in the hemiparetic group. Hemiparetic participants showed a higher ML peak-to-peak after the 18 minute walk compared to after the 6 minute walk ($12.7 \text{ cm} \pm 1.0 \text{ vs. } 10.8 \text{ cm} \pm 0.7$, Wald $\chi^2_{1\text{df}} = 18.3$, $p < 0.001$). The hemiparetic group not only showed a higher ML peak-to-peak at post-test 1 compared to baseline but also a higher ML peak-to-peak at post-test 3 compared to baseline ($12.1 \text{ cm} \pm 0.7 \text{ vs. } 10.9 \text{ cm} \pm 0.8$, Wald $\chi^2_{1\text{df}} = 19.3$, $p < 0.001$). On the other hand, the control group showed a significant effect on the interaction between the duration of the walk and the measurement point (Wald $\chi^2_{3\text{df}} = 29.3$, $p < 0.001$). The pattern of recuperation after the 6 minute walk consisted of a slow decrease in which the only difference that reached statistical significance was between post-test 1 and post-test 3 (difference of 1.3, $p = 0.023$).
Figure 6. Average (mean + or - 1 SD) medio-lateral peak-to-peak center of pressure displacement during sit-to-stand for the 6-minute (A) and 18-minute (B) walk in control and hemiparetic participants. The mean center of pressure displacement was measured before the walk (baseline), immediately after the walk (post 1), 15 minutes post-walk (post 2), and 30 minutes post-walk (post 3).
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Dependent Variable: ML Peak (STS)
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*Table 8.* Generalized Estimating Equations for the immediate effect of exertion (A) and recuperation pattern (B) with one between-subject factor (hemiparetic participants vs control participants) and two within-subject or repeated factors (walking duration and measurement points) for medio-lateral peak-to-peak center of pressure displacement during sit-to-stand.
The pattern of recuperation after the 18 minute walk consisted of a non-significant initial decrease of ML peak-to-peak, followed by a significant increase between post-test 1 and post-test 2 (increase of 1.4, p = 0.006) and finally a significant decrease between post-test 2 and post-test 3 (decrease of 1.8, p = 0.005).

The step reaction time on the hemiparetic side of stroke participants and right side of control participants for both walking durations is illustrated in Figure 7. The step reaction time on the hemiparetic side did not reveal any significant interaction but was significantly associated with the group (Table 9). The hemiparetic group had a longer reaction time compared to the control group. When the hemiparetic group stepped with the unaffected side (Figure 8), there was again no significant interaction, but was significantly associated with the group (Table 10). As previously observed, the hemiparetic group had a longer reaction time compared to the control group.
Figure 7. Average (mean ± 1 SD) step reaction time for the 6-minute (A) and 18-minute (B) walk in control (right side) and hemiparetic (hemiparetic side) participants. The step reaction time was measured before the walk (baseline), immediately after the walk (post 1), 15 minutes post-walk (post 2), and 30 minutes post-walk (post 3).
Table 9. Generalized Estimating Equations for the immediate effect of exertion (A) and recuperation pattern (B) with one between-subject factor (hemiparetic participants vs control participants) and two within-subject or repeated factors (walking duration and measurement points) for step reaction time on hemiparetic side of stroke participants and right side of control participants.
Figure 8. Average (mean + or - 1 SD) step reaction time for the 6-minute (A) and 18-minute (B) walk in control (right side) and hemiparetic (unaffected side) participants. The step reaction time was measured before the walk (baseline), immediately after the walk (post 1), 15 minutes post-walk (post 2), and 30 minutes post-walk (post 3).
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Dependent Variable: Reaction time healthy side
Model: (Intercept), Duration, Group, test, Duration * Group * test, Duration * Group, Duration * test, Group * test

B  Tests of Model Effects

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Dependent Variable: Reaction time healthy side
Model: (Intercept), Duration, Group, test, Duration * Group * test, Duration * Group, Duration * test, Group * test

Table 10. Generalized Estimating Equations for the immediate effect of exertion (A) and recuperation pattern (B) with one between-subject factor (hemiparetic participants vs control participants) and two within-subject or repeated factors (walking duration and measurement points) for step reaction time on the unaffected side of stroke participants and right side of the control participants.
Discussion

The results from this study indicated that increasing the duration of walking can significantly affect postural stability in persons with chronic stroke. With respect to the primary outcome measures, significant triple interactions of increased center of pressure in the mediolateral excursion in static (double-legged stance) and in dynamic (sit-to-stand) tasks were found. This was observed in the hemiparetic group when walking a greater distance over 18 minutes in comparison with a distance covered in 6 minutes at similar comfortable speeds. Furthermore, this destabilizing effect was not found in the control group.

*Effects of Walking Duration on Physical Exertion Parameters*

The notion of comparativeness of both groups is of interest in this study. Both groups were matched in that every sex and aged matched control participant maintained the same average heart rate as their hemiparetic counterpart. Also, the average comfortable speeds of each group were similar for both of their walking durations. It is important to note that the walking speed of the control group was adjusted throughout the walk so that their heart rate equalled that of their matched hemiparetic participant. For this reason, the control group was not necessarily walking at their self selected comfortable speed. A further demonstration of the comparativeness of both groups was that the control group walked an average "comfortable" speed that was 66% of their maximal speed, and the hemiparetic group walked an average comfortable speed that was 63% of their maximal speed. Furthermore, both the hemiparetic and control groups covered a distance in the 18 minute walk that was 3.2 times greater than the distance they walked in the 6 minute walk.
This study demonstrated that physical exertion measures increased when the duration of activity was increased from 6 minutes to 18 minutes at a comfortable speed. This was the case for both the hemiparetic and the control group. This was supported by both the objective and subjective measures. With regard to the objective measures, the matched heart rate was significantly higher in both groups during the 18-minute walk. The subjective measures of physical exertion followed similar trends for both groups in that they had higher scores for the 18-minute walk in comparison with the 6-minute walk.

On the other hand, the oxygen uptake did not reveal a significant change when the duration of walk increased from 6 minutes to 18 minutes. This lack of significant change can be explained by the fact that the walking speed that both groups maintained was similar for both of their walking durations. The hemiparetic participants walked at lower speeds and had lower oxygen uptake. In fact, it has been previously shown that the oxygen uptake was closely related to the walking speed (Waters & Mulroy, 1999). Furthermore, the rate of oxygen consumption during walking for persons with hemiplegia is less than the rate for normal participants walking at their comfortable speed despite the inefficiency of the gait pattern and high energy cost (Zamparo, 1995). Although the primary gait disability is the slow speed, the rate of energy expenditure during walking still represents a higher percentage of the patient's maximal capability than for able bodied adults due to the deconditioning and aging (Macko, Smith, Dobrovolny, Sorkin, Goldberg, & Silver, 2001; Bard, 1963).

In fact, in our study, when performing the 18 minute walk at the comfortable speed, the average VO\textsubscript{2} level of the hemiparetic participants was 10.1ml/kg/min. Cress and colleagues (2003) found that a peak VO\textsubscript{2} of 20 ml/kg/min was needed for independent living for adults aged 65-97 years. Unfortunately, individuals who have
sustained a stroke, have an averaged VO\(_2\) level of 14ml/kg/min. (Ivey, Hafer-Macko, & Macko, 2006; Eng, Dawson, & Chu, 2004; MacKay-Lyons & Makrides, 2004; Fujitani, Ishikawa, Akai, & Kakurai, 1999). Based on this reference value, our hemiparetic group achieved 71.8% of their peak fitness level during the 18 minute walk. This suggests that walking 18 minutes was physically demanding and probably tiring.

When examining walking speed and oxygen uptake, it is important to also examine the energy cost of walking. The energy cost of walking is the amount of oxygen consumed per kilogram body weight per unit distance travelled (i.e. oxygen uptake divided by the speed of walking) (Waters & Mulroy, 1999). The value for healthy adults between the ages of 29-59 is 0.15 ml/kg/m and for healthy adults between the ages of 60 and 80 is 0.16 ml/kg/m (Waters & Mulroy, 1999). In our present study, the control group fell close to this range at 0.14 ml/kg/m. while the hemiparetic group had a higher energy cost of walking (0.19 ml/kg/m). Interestingly, there was no significant difference in energy cost for both groups when increasing the duration of the walk from 6 minutes to 18 minutes. The cost remained exactly the same. One would expect to see a difference due to the higher energy expenditure that the hemiparetic participants would have to make when increasing the duration. In fact, studies have shown that the energy cost of walking of hemiparetic participants was 1.5 to 2 times greater than healthy controls and that the energy expenditure was elevated by 55-100% in hemiparetic participants compared with healthy controls (Zamparo, Francescato, De Luca, Lovati, & di Prampero, 1995).

Zamparo, Francescato, De Luca, Lovati, & di Prampero (1995) also found that the difference in energy cost of level walking between hemiplegic participants and healthy controls was even increased at lower speeds. Individuals with impaired walking function are reported to choose lower speeds of progression, probably because of fear of falling, difficulties of coordination or balance problems (Mattsson, Brostrom, Borg, & Karlsson,
Among other possible factors contributing to this increase of energy expenditure, the level of spasticity following stroke has been correlated with the energy cost of walking (Bard, 1963). In addition, physical deconditioning along with age-associated declines in fitness and muscle mass can further contribute to activity intolerance, therefore compromising patients' capacity to meet the high-energy demands of hemiparetic gait (Macko, Smith, Dobrovolny, Sorkin, Goldberg, & Silver, 2001). Eng and colleagues (2002) postulated that the increased RPE can be attributed to the presence of stroke-specific impairments (e.g. muscle weakness or spasticity), which may increase peripheral muscular discomfort and fatigue and be perceived as requiring more exertion.

Endurance has been defined as the time limit of a person's ability to sustain a particular level of physical effort (McArdle & Katch, 1996). A reduction in speed over the test would have indicated that endurance was challenged during this test. In our present study it was expected that the walking speed for the hemiparetic group would be lower when walking 18 minutes vs. 6 minutes. This could have demonstrated an increase in physical exertion levels and fatigue (Ivey, Hafer-Macko, & Macko, 2006). However, our findings showed that hemiparetic participants walked at similar speeds for both durations. When looking at the subjective measures of exertion and fatigue, the change from 6 to 18 minutes represented a shift from a light to a hard physical exertion. This implies that the physical effort was tiring for the hemiparetic participants but that they were able to sustain their effort throughout the walk since they kept their walking speed constant until the end of the walk. This phenomenon has been previously reported when hemiparetic participants were asked to maintain their comfortable walking speed for 6 and 12 minute durations and they were able to maintain a constant speed throughout (Eng, Chu, Dawson, Kim, & Hepburn, 2002; Dean, Richards, & Malouin, 2001). Our results suggest that the strategy of maintaining a constant speed throughout the walk
exists for both durations and does not necessary mean that they were not fatigued or exerting themselves enough.

We have seen that the physical exertion measures were similar for both groups but the significant difference was in the subjective values of RPE and VAS. This variable demonstrated that the hemiparetic participants found it more difficult to walk the longer duration of 18 minutes compared to 6 minutes. The results are in accordance with other studies with regard to heart rate, distance, and RPE. During 6 minute and 12 minute walks in a study performed by Eng and colleagues (2002), the average heart rate represented 63% of the age-predicted maximum heart rate which was exactly the case in the current study. The intensity of the exercise can, therefore, be classified as moderate according to the heart rate (American College of Sports Medicine, 2000).

**Effect of Physical Exertion on Balance**

The results of this study support the hypothesis that walking over a longer duration (18 min) produced a greater destabilizing effect than walking over a shorter duration (6 min) in hemiparetic participants but not in healthy control participants. This is evident by the significant increase in COP sway in both static (double-legged stance) and dynamic (sit-to-stand) situations. This destabilizing effect was observed immediately after the end of the walk while COP excursions returned to baseline values 15-minute post walk. It should also be noted that a combined effect of walking duration and group was found in COP ML and AP variability in double-legged stance indicating that both walking durations had a destabilizing effect in hemiparetic participants. These results are in accordance with findings by Nardone, Tarantola, Galante, & Schieppati (1998) who examined the effect of physical exertion on postural stability of young healthy adults. While investigating the effects of fatiguing (above the anaerobic threshold) and non-
fatiguing (below the anaerobic threshold) exercise, Nardone and colleagues (1998) found that body sway area during double-legged stance increased significantly after fatiguing exercises only. To induce fatigue, young healthy participants performed 25 minutes of running on a treadmill at 85% of their age predicted maximal heart rate. When COP was assessed before and after the exercise, the results showed a significant increase in body sway measures similar to our present study. Furthermore, the postural sway significantly increased immediately after the end of the exercise and returned to initial value at 15 minutes post-exercise. In the present study, the destabilizing effect of exercise observed in hemiparetic participants was induced at a much lower intensity of work (i.e. 65% of the participants’ age predicted maximal heart rate). This is likely due to the fact that a majority of hemiparetic individuals experience the phenomenon of body deconditioning after stroke (Ivey, Macko, Ryan, & Hafer-Macko, 2005) This is characterized by a dramatic decrease of exercise tolerance and physical fitness due mainly to the loss of mobility and the reduction of cardiovascular capacities (Micheal, Allen, & Macko, 2005; Macko, DeSouza, Tretter, Silver, Smith, Anderson, Tomoyasu, Gorman, & Dengel, 1997; Potempa, Braun, Tinknell, Popovich, & Benefits, 1996; Macko, Ivey, Forrester, Hanley, Sorkin, Katzel, Silver, & Goldberg, 2005). This lowering of cardiovascular capacities is expressed by a decrease of peak fitness levels, the peak oxygen consumption rate of hemiparetic participants representing about half that of sedentary age-matched controls (Ivey, Macko, Ryan, & Hafer-Macko, 2005; Micheal, Allen, & Macko, 2005).

The question that must be addressed is why hemiparetic participants mainly increase their postural sway in the medio-lateral direction after walking. This increase of postural sway may be due to the greater asymmetry observed in hemiparetic participants. In fact, weight-bearing asymmetry during standing has shown to be
significantly correlated to the increased ML COP sway in individuals with stroke (Marigold & Eng, 2006). Furthermore, it has been shown that physical exertion induced by whole-body endurance exercises produces a destabilizing effect on the human body (Nardone, Tarantola, Giordano, & Schieppati, 1997; Lepers, Bigard, Diard, Gouteyron, Guezenne, 1997; Corbelli, Blouin, Begin, Nougier, & Teasdale, 2003; Wilson, Madigan, Davidson, & Nussbaum, 2006; Vuillerme, Nougier, & Teasdale, 2002; Thomas, Cotten, Spieth, & Abraham, 1975; Caron, 2004; Caron, 2003; Nardone, Tarantola, Galante, & Schieppati, 1998; Derave, De Clercq, Bouckaert, & Pannier, 1998; Seliga, Bhattacharya, Succop, Wickstrom, Smith, & Willeke, 1991; Ochsendorf, Mattacola, & Arnold, 2000; Wilkins, Valovich McLeod, Perrin, & Gansneder, 2004). Among the possible factors contributing to the transient destabilizing effect induced by fatiguing exercises, the decrease in muscle force has been proposed as one possible mechanism (Nardone, Tarantola, Galante, & Schieppati, 1998). This phenomenon is known to be produced by physiological mechanisms occurring at the central and peripheral levels (Balestra, Duchateau, & Hainaut, 1992; Sharpe & Miles, 1993; Enoka & Stuart, 1992; Gandevia, Enoka, McComas, Stuart, & Thomas, 1995; Nordlund, Thorstensson, & Cresswell, 2004). Two physiological factors have been identified as key elements in the decrease of muscle force observed after strenuous exercise. The first factor is of short duration and involves the resynthesis of phosphocreatine (Baker, Kostov, Miller, & Weiner, 1993). This factor plays an important role in the lowering of muscle strength in the first few minutes after the end of the exercise. The second factor is of longer duration and is related to disorders of the excitation-contraction coupling (Skof & Strojnik, 2006). This decreased somatosensory information from the lower limbs and, especially the hemiparetic limb, could be causing the increased postural sway (Di Fabio, Badke, & Duncan, 1986). Furthermore, the paretic muscles have been shown to primarily recruit glycolytic Type 11 fibers to initiate contraction which can result in diminished capacity for
oxidative metabolism and eventually decreased exercise endurance (DePeyne, Hafer-Macko, Ivey, Ryan, & Macko, 2004). Therefore, the paretic limb is further affected and the already greater asymmetry when standing creates more postural sway in the medio-lateral direction after having performed physical exertion. It can then be postulated that increasing the duration of physical exertion will increase the amount of ML COP sway due to a higher reliance on the unaffected limb.

A similar relationship between peripheral physical exertion and significant increases in ML COP excursion was found in young healthy adults. Corbeil, Blouin, Begin, Nougier, & Teasdale (2003) performed a study that examined the effects of peripheral muscular fatigue on balance in healthy males. The participants were asked to perform repeated plantar flexion of both legs at 50-75% of their maximal workload while sitting. The participants exhibited an increased postural sway characterized by faster center of pressure velocity and greater mean and median frequency of COP displacements while standing on a force platform. The authors suggested that fatigue places higher demands on the postural control system by increasing the frequency of actions needed to regulate the upright stance. These actions would be associated with discrete control of the postural oscillations required to compensate the motor and/or sensory deficiencies induced by peripheral muscular fatigue. The altered force production by the fatigued muscles would require an increase in the frequency of the corrections in order to avoid greater displacements of the COP. Due to the increased physical exertion of walking a longer duration of 18 minutes, the results of this study showed a greater postural sway in the ML direction compared to when walking a duration of 6 minutes.
When examining the results for the antero-posterior (AP) COP excursion, our results showed that there was increased AP COP sway for the hemiparetic group when standing. Furthermore, when examining variability, both groups had higher AP COP values after having performed the 18-minute walk (vs. 6-minute walk). When examining dynamic balance using the sit-to-stand task, the results were very different. In fact, control participants showed greater AP COP sway. Would this suggest that control participants were more destabilized? Studies have shown that AP COP sway was larger than the ML COP sway for healthy control participants (Cheng, Liaw, Wong, Tang, Lee, & Lin, 1998). This might be due to excessive momentum during the momentum-transfer phase while rising from a chair (Schenkman, Berger, Riley, Mann, & Hodge, 1990). Therefore, the unexpected non-significant interactions in our present study are in accordance with other studies.

It was hypothesized that walking over a longer period of time would create a destabilizing effect in hemiparetic participants but not in control participants. Indeed, increasing the duration of the walk produced greater ML COP excursion for the hemiparetic group. Is this increase in ML COP excursion associated with an increased risk of falling? Cheng and colleagues (1998) reported that stroke fallers had significantly increasing ML COP sway during sit-to-stand task, as compared to stroke non fallers and healthy controls. The greater ML COP sway for the stroke fallers indicated poor dynamic postural stability which would place those individuals at greater risk of fall. More recently, Cheng and colleagues (2001) followed 54 hemiparetic participants and found that a postural stability rehabilitation program resulted in smaller ML COP excursion when performing the sit-to-stand task as compared to an ‘untrained’ control group of hemiparetic participants. Furthermore, after 6 months follow-up, participants who completed the program fell less often than the control group. This tends to show that a
lower COP excursion in the medio-lateral direction lowers the risk of falling and, conversely, that an increase in ML COP sway could increase the risk of falling. In our present study, the ML COP excursion during double-legged stance and sit-to-stand tasks showed a significant increase following the 18 minute walk vs. the 6 minute walk. This increase represented a change of 19.4 and 39% after performing the 18 minute walk for double-legged stance and sit-to-stand respectively. This increase could imply that the hemiparetic participants in our study were at a higher risk of falling when walking the longer duration.

An unexpected result was the lack of any significant interaction for the step reaction time on both the hemiparetic side and the unaffected side for the hemiparetic group. A generalized slowing in step reaction time is part of human aging (Stelmach & Worringham, 1985) and is a strong sensorimotor predictor of the risk of falls (Lord & Fitzpatrick, 2001). Studies have shown that older adults with stroke have delayed paretic limb postural reflexes in the form of coordinated muscle activity. (Di Fabio, Badke, & Duncan, 1986). Rogers, Johnson, Martinez, Mille, & Hedman (2003) suggested that older healthy adults have slower reaction time and that it is further exacerbated by stroke impairments. The purpose of this task was to examine the effect of physical exertion on postural reactions. The step reaction times of the current study were similar to other studies performing the same task with hemiparetic participants (Marigold, Eng, Dawson, Inglis, Harris, & Gylfadottir, 2005). However, the lack of significant changes in our study after performing 6 or 18 minutes of walking may be due to a learning effect of the participants. The fact that this task was performed repetitively may have contributed to a learning effect in which participants would have improved their performance after repeating the task several times. In future research, the implementation of the step reaction time trials should be improved. Marigold, Eng, Dawson, Inglis, Harris, &
Gylfadottir (2005) designed the repetitive step reaction time trials in such a way that the first two and last two trials were with the unaffected limb, and the middle trial was with the paretic limb to reduce any standing postural bias.

**CONCLUSION**

This study demonstrated that physical exertion induced by walking produces a destabilizing effect in postural stability of hemiparetic participants. The results showed a significant triple interaction for increased ML COP for the hemiparetic participants in static (double-legged stance) and dynamic (sit-to-stand) when walking over a longer duration. In contrast, no significant effects were found for the step reaction time. The changes observed in postural stability of hemiparetic participants after performing the 18-minute walk could be due to body deconditioning and muscle weakness of their hemiparetic leg, which could increase their risk of falling.

The results of this study could help to identify future directions in stroke research. The effect of exercise duration and intensity, and the time course of balance recovery for stroke patients should be further investigated. The goal of future research would be to provide information to health professionals on the safest ways for individuals with stroke to perform their activities of daily living, thereby helping them to achieve a level of independence and to break the vicious cycle of deconditioning so often associated with this population.
References


Conference of the New England American College of Sports Medicine. Rhode Island, USA.


Appendix A

Power calculation
**Power calculation**

The main outcome variable of this study is postural sway after a double-leg stance task and the objective is to compare the change between pre-intervention and post-intervention postural sway for stroke subjects compared to healthy controls. We computed the power using preliminary data on three stroke subjects and two healthy controls. With the proposed sample size of 12 stroke subjects and 12 healthy controls, the study will have a power of 80% to yield a statistically significant result. This computation assumes that the mean difference is 0.6 (corresponding to a change between pre- and post-measures in postural sway of 0.6 for the stroke subjects versus minimal change for the healthy controls) and the common within-group standard deviation is 0.5\(^A\).

A second aspect of this study is to estimate the mean difference between the two populations. On average, a study of this design would enable us to report the mean difference between the two populations with a precision (95.0% confidence level) of plus/minus 0.5 points. For example, an observed difference of 0.6 would be reported with a 95.0% confidence interval of 1.1 to 0.1.

\(^A\) We used preliminary data to compute the common standard deviation as follows. For each group we computed the standard deviation of the pre-post differences using:

\[ s = \text{SQRT} \left[ s_{\text{pre}}^2 + s_{\text{post}}^2 - 2 \times (t_{\text{pre/post}} \times s_{\text{pre}} \times s_{\text{post}}) \right] \]

then computed the common standard deviation equation using the square root of the following equation:

\[
\sqrt{\frac{\left( \frac{s_1^2}{n_1} + \frac{s_2^2}{n_2} \right)^2}{\frac{(s_1^2/n_1)^2}{n_1-1} + \frac{(s_2^2/n_2)^2}{n_2-1}}}
\]
Appendix B

Cummings Jewish Centre for Seniors Physical Activity

Questionnaire (modified Par-Q form)
Cummings Jewish Centre for Seniors Physical Activity

Questionnaire (modified Par-Q form)

DATE: ________________________________

NAME: __________________________________________________________

ADDRESS: _______________________________________________________

Number Street Apt. pc

TELEPHONE NUMBER: ________________________________

PHYSICIAN'S NAME: ________________________ TEL #: ________________

DATE OF LAST CHECK-UP: _________________________________________

PRESENT MEDICATIONS: ___________________________________________

________________________________________________________________

In case of emergency, please list two contact persons whose home addresses are different from your own:

Name: _______________________ Relationship: _______________________

Phone#: ______________________

________________________________________________________________

Name: _______________________ Relationship: _______________________

Phone#: ______________________

________________________________________________________________

DATE OF BIRTH: ________________________________
AGE: _____

GENDER: M / F

MEDICAL HISTORY: (Check (x) if YES and give details)

**Cardiovascular**
- High Blood Pressure: ( )
- Angina: ( )
- Heart Attack: ( )
- Valve Disease: ( )
- Irregular Heartbeat: ( )
- Pain in legs while walking: ( )

**Cerebral Vascular**
- Stroke: ( )
- Side of body affected: ( )
- TYPE: Hemorrhagic ( ) Ischemic ( )

**Walking aids:** ( )
**Rehab facility:** ( )

**Musculo-skeletal**
- Arthritis: ( )
- Swelling ( )
- Stiffness ( )
- Pain ( )
- Back Problems: ( )
- Foot Problems: ( )
- Joint replacements: ( )
- Movement Limitations: ( )

**Osteoporosis**
- Date of diagnosis: ( )
- Movement Limitations: ( )
- Activity Limitations: ( )
- Related Fractures: ________________ Cause: __________________

**Respiratory Problems:** ( )

**Diabetes:** ( )
Epilepsy: ( )

Hernia: ( )

Others: ( )

Recent Hospitalizations (in past 6 months): ________________________________

**REVIEW OF PRESENT MEDICAL SYMPTOMS** (within the past 2 months)

Have you recently had:

Spells of dizziness: ( )

Chest pain: ( )

Heart Palpitations: ( )

Feeling Faint: ( )

Do you smoke? ( )

**PHYSICAL HEALTH STATUS**

Do you engage in physical activity? Yes ( ) No ( )

Which Activities? __________________________________________________________

How many times per week? _______________ Duration: _______________

What is the degree of difficulty? (Compared to walking up stairs)

Very Easy: ( )

Easy: ( )

Moderate: ( )

Difficult: ( )

Very Difficult: ( )

Can you get down and up off the floor? Yes ( ) No ( )

Can you go up and down stairs? Yes ( ) No ( )
Can you ride a stationary bicycle? Yes ( ) No ( )

Can you walk safely in the community? Yes ( ) No ( )

**Balance**

How do you think your balance is compared to other people your age in general?

a. Much more than others my age
b. More than others my age
c. About the same as others my age
d. Less than others my age
e. Much less than others my age

How is your balance after having performed exercise such as walking compared to before exercising?

a. The same
b. A little worse
c. A lot worse

*Thank you for your time!*