MOTOR CONTROL OF RESPONSE TO EXTERNAL PERTURBATIONS IN PEOPLE

POST-STROKE

by

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Abstract

Mechanisms underpinning motor control of standing balance post-stroke remain unclear. Following stroke, ankle plantarflexor muscles demonstrate impairment associated with asymmetrical postural control and decreased balance. Stroke also results in increased attentional demands during challenges to standing balance. This thesis examined motor control impairment post-stroke from medial gastrocnemius motor units to the tri-muscle plantarflexor complex. Investigation of motor, kinematic and kinetic parameters of postural control during external perturbations in standing and associated levels of physiological arousal have furthered understanding of balance impairment post-stroke.

Methods: Medial gastrocnemius motor units were recorded in controls (Chapter 2) and people post-stroke (Chapter 3) in standing as perturbations were sequentially applied at the pelvis under conditions of increased anteriorly-directed challenge. In both studies, motor unit firing rate was calculated during dynamic response to perturbation, and maintenance of steady state between perturbations. Joint kinematics, surface electromyography and movements of the centre of pressure were assessed. In Chapter 4, this methodology was expanded to cross-correlation analysis of electromyography activity of the three plantarflexor muscles with anterior-posterior centre of pressure during steady state. In Chapter 5, attentional demands surrounding timing of external perturbation were manipulated to investigate effects of stroke on physiological arousal and postural reactions.

Results: In healthy subjects, medial gastrocnemius utilized primarily motor unit recruitment to maintain standing with a modest increase in motor unit firing rate only during the dynamic response to external perturbations. The paretic medial gastrocnemius also primarily used motor
unit recruitment; however, lacked firing rate modulation during the dynamic response, albeit firing rate was related to kinematic variables of postural control. In people post-stroke, the three plantarflexors demonstrated asymmetrical motor control of postural sway between-legs but symmetry was improved under conditions of increased challenge to standing balance. Finally, knowledge of timing of perturbations did not decrease the heightened anticipatory postural strategy and level of physiological arousal exhibited post-stroke.

Conclusions: This dissertation provides new understanding of motor control of standing balance post-stroke and reveals anticipatory postural strategies adopted post-stroke under conditions challenging balance. These findings implicate the importance of introducing challenge to standing balance post-stroke in the assessment and rehabilitation of postural control post-stroke.
Preface

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# Table of Contents

Abstract .......................................................................................................................................... ii

Preface ........................................................................................................................................... iv

Table of Contents ......................................................................................................................... vi

List of Tables ................................................................................................................................ xi

List of Figures .............................................................................................................................. xii

Acknowledgements .................................................................................................................... xiv

Dedication ................................................................................................................................... xvi

Chapter 1: Introduction ................................................................................................................1

1.1 General introduction ........................................................................................................... 1

1.2 Background information ..................................................................................................... 2

1.2.1 Gradation of force ........................................................................................................ 2

1.2.2 Postural control ........................................................................................................... 3

1.2.3 Response to external perturbations .......................................................................... 5

1.2.4 Physiological arousal and postural control .............................................................. 6

1.2.5 Motor impairment following stroke ........................................................................ 8

1.2.6 Stroke and motor control of standing balance ....................................................... 9

1.2.7 Physiological arousal and postural control following stroke ............................... 11

1.3 Methodological approach ................................................................................................. 12

1.3.1 External perturbation paradigm ............................................................................. 12

1.3.2 Electromyography .................................................................................................... 13

1.3.3 Kinematics and kinetics ............................................................................................ 14
1.3.4 Electrodermal activation

1.4 Thesis outline

1.4.1 Objectives and hypotheses

Chapter 2: Motor unit recruitment and firing rate in medial gastrocnemius muscles during external perturbations in standing in humans

2.1 Introduction

2.2 Methods

2.2.1 Experimental protocol

2.2.2 Motor unit recordings

2.2.3 Surface EMG recordings

2.2.4 Kinetic and kinematic data

2.2.5 Data analysis

2.2.6 Statistical analysis

2.3 Results

2.3.1 Overview of motor unit recruitment

2.3.2 Dynamic response to the perturbation

2.3.3 Maintenance of steady state between perturbations

2.3.4 Modulation between dynamic response to perturbation and maintenance of steady state

2.3.5 Kinetic, kinematic and surface EMG response to perturbations

2.4 Discussion

2.5 Conclusion
Chapter 3: Behaviour of medial gastrocnemius motor units during postural reactions to external perturbations after stroke

3.1 Introduction

3.2 Methods

3.2.1 Experimental protocol

3.2.2 Motor unit recordings

3.2.3 Surface EMG recordings

3.2.4 Kinetic data

3.2.5 Kinematic data

3.2.6 Statistical analysis

3.3 Results

3.3.1 The postural reaction during the dynamic response to perturbations

3.3.2 Motor unit behaviour of medial gastrocnemius

3.3.3 Kinematic and surface EMG data

3.3.4 Relationship of motor unit ISI to postural reactions

3.4 Discussion

3.5 Conclusion

Chapter 4: Motor control of standing postural sway under anteriorly-directed challenges to standing balance in people post-stroke

4.1 Introduction

4.2 Methods

4.2.1 Experimental protocol

4.2.2 Kinetic and kinematic data
Chapter 5: Influence of knowledge of timing of perturbations on anticipatory postural strategies and postural reactions of people post-stroke

5.1 Introduction........................................................................................................ 88
5.2 Methods.............................................................................................................. 90
  5.2.1 Experimental protocol................................................................................. 91
  5.2.2 Electrodermal activation.............................................................................. 93
  5.2.3 Kinetic and kinematic data.......................................................................... 94
  5.2.4 Surface EMG recordings............................................................................ 95
  5.2.5 Statistical analysis ..................................................................................... 95
5.3 Results ........................................................................................................................... 96
  5.3.1 Participants ........................................................................................................... 96
  5.3.2 Electrodermal activation ....................................................................................... 96
  5.3.3 Kinematics .......................................................................................................... 97
  5.3.4 Kinetics .............................................................................................................. 99
  5.3.5 Electromyography .............................................................................................. 101
  5.4 Discussion ............................................................................................................... 104
  5.5 Conclusion ............................................................................................................ 108

Chapter 6: General discussion ................................................................................................. 109
  6.1 Overview ............................................................................................................... 109
  6.2 Motor unit control of external perturbations ......................................................... 110
  6.3 How does knowledge about the perturbation influence the postural response? ........ 113
  6.4 Motor control of standing balance across the ankle plantarflexors ......................... 116
  6.5 Limitations ........................................................................................................... 117
  6.6 Implications and future directions ......................................................................... 118
  6.7 Conclusion ............................................................................................................ 121

References .......................................................................................................................... 122
List of Tables

Table 2.1 Motor unit recruitment by load level ................................................................. 26
Table 2.2 Anterior-posterior centre of pressure (APCOP) excursion by load level ........... 33
Table 3.1 Participant characteristics .............................................................................. 48
Table 3.2 Anterior-posterior centre of pressure (APCOP) excursion (mm) by load level .... 50
Table 3.3 Anterior-posterior centre of mass (COM) excursion (mm) by load level .......... 50
Table 4.1 Participant characteristics .............................................................................. 71
Table 4.2 Partial correlation controlling for load, between the Z scores for the peak EMG:APCOP correlation coefficients derived from between the paretic and non-paretic medial (MG), lateral gastrocnemius (LG) and soleus (SOL) muscles and APCOP displacement, and external ankle torque, mean RMS EMG amplitude, standard deviation of the APCOP (SD-APCOP)displacement and velocity during postural sway .......................................................... 82
Table 5.1 Participant characteristics .............................................................................. 96
List of Figures

Figure 2.1 Experimental set-up. Schematic of experimental set up for applying perturbations that result in a pull in anterior direction. ................................................................. 22

Figure 2.2 Examples of motor unit dynamic response to load drop. ........................................... 28

Figure 2.3 Duration of the motor unit first 3 ISIs during dynamic response. ......................... 29

Figure 2.4 Motor unit firing behaviour after initial recruitment ............................................ 30

Figure 2.5 Representative example of electromyography recordings from the medial gastrocnemius muscle in standing during incremental increases in external load............. 31

Figure 2.6 Kinetic, surface electromyography (EMG) and kinematic response to increasing levels of perturbation. .................................................................................. 34

Figure 3.1 Experimental set-up. Schematic of experimental set up for applying perturbations that result in a pull in anterior direction. ................................................................. 44

Figure 3.2 Response of the anterior-posterior centre of pressure (APCOP), centre of mass (COM), percentage weight-bearing on the paretic leg, and external ankle torque during the dynamic response to perturbation under increased level of challenge to standing balance........ 51

Figure 3.3 Representative example of electromyography recordings from the paretic and non-paretic medial gastrocnemius muscle in standing.......................................................... 52

Figure 3.4 Interspike interval (ISI) of the paretic medial gastrocnemius muscle with increasing load levels and ISI of the non-paretic medial gastrocnemius muscle collapsed across loads ...... 54

Figure 3.5 Kinematics and surface electromyography of the lower extremity with increased loads. .................................................................................................................. 56

Figure 4.1 Experimental set-up. ........................................................................................... 66
Figure 4.2 Kinematic and kinetic parameters of postural sway....................................................... 73
Figure 4.3 RMS EMG amplitude in response to maintenance of increasing load......................... 75
Figure 4.4 Representative example of cross-correlation calculation function.............................. 78
Figure 5.1 Experimental set-up..................................................................................................... 93
Figure 5.2 Electrodermal activation (EDA), A) prior to perturbation and B) following perturbation during self- and investigator-triggered external perturbations in participants post-stroke and controls. .......................................................................................................................... 97
Figure 5.3 Centre of mass (COM), A) displacement and B) velocity during self- and investigator-triggered external perturbations in participants post-stroke and controls.............................................. 98
Figure 5.4 Centre of pressure (COP) measures during self-triggered (white) and investigator-triggered perturbations.................................................................................................................................................... 100
Figure 5.5 Responses to a single self-triggered perturbation in a participant post-stroke (paretic leg, gray) and a control subject (black).......................................................................................................................................... 101
Figure 5.6 RMS EMG amplitude of the ankle plantarflexor muscles amongst groups............ 103
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Dedication

For my Mother, the strongest influence in my life and my fiercest supporter.
Chapter 1: Introduction

1.1 General introduction

Over 300,000 Canadians currently live with the effects of stroke (HSF, 2011). Regaining the ability to walk is the most commonly cited goal of people post-stroke with hemiparesis, thus, making the rehabilitation of standing balance of paramount importance. Recovery of walking post-stroke has been reported in as many as 88% of all people discharged from hospital (Jorgensen et al. 1995). However, falls occurrence in this population have been also reported to be as high as 73% of all people who recover the ability to walk post-stroke living in the community, with falls commonly occurring within the first few months of returning home from rehabilitation (Mackintosh et al. 2006; Yates et al. 2002). Among ambulatory people living in the community with hemiparesis post-stroke, impaired balance has been associated with low ambulatory activity more so than cardiovascular fitness and has been suggested as an important factor leading to further de-conditioning (Michael et al. 2005). Furthermore, mobility limitations related to impaired balance experienced post-stroke can lead to decreased self-efficacy, loss of independence and restrictions in activities of daily living, community integration and quality of life (de Oliveira et al. 2008; Schmid et al. 2012; Weerdesteyn et al. 2008). Therefore, it is vital to investigate how stroke leads to impaired motor control of balance, specifically under the conditions of challenges to standing balance, with the goal of directing rehabilitation strategies aimed at improving standing balance.

Current literature provides insight into important areas of standing balance post-stroke at the level of performance-based measures of posturography (force platform measurement) during standing balance and in response to perturbations. The distal muscles of the leg, the ankle plantarflexors specifically, are vital to overall postural control of the body during standing
balance (Winter et al. 2003). The ankle plantarflexors are particularly affected by stroke with weakness across the spectrum of stroke severity (Fimland et al. 2011) and such weakness has been suggested to significantly impact standing balance post-stroke (Garland et al. 2009). The overall aim of this thesis is to contribute to the current understanding of standing balance post-stroke by exploring the motor control impairment of the ankle plantarflexors post-stroke. This will be achieved by examining postural control at the level of the fundamental unit of motor control, the motor unit, and extending to measurement of the ability of the activity of each ankle plantarflexor muscle to coordinate the control of standing balance. Finally, as increased attentional demands and decreased confidence have been associated with changes in physiological arousal (Critchley et al. 2000), this thesis will examine how physiological arousal levels post-stroke may affect postural control strategies associated with the ankle plantarflexors.

1.2 Background information

1.2.1 Gradation of force

Force production of a muscle is actively influenced by the activation of motor units. Motor units are comprised of a single motoneurone at the level of the spinal cord and the muscle fibres innervated by the motoneurone axon terminals within a specific muscle. The motor unit (MU) is the final pathway for the central nervous system to generate force and produce movement. Sensory and descending neural inputs result in force production and movement which is modulated by MU recruitment and rate coding (Heckman and Enoka 2012). Muscles differ in their use of motor unit recruitment and rate coding (De Luca et al. 1982; Kukulka and Clamann 1981). Specifically, how the muscle employs motor unit recruitment and rate coding can be influenced by characteristics of the muscle contraction including: joint angle and muscle length (Ballantyne et al. 1993; Kennedy and Cresswell 2001), speed of muscle contraction
(Desmedt and Godaux 1977), nature of the external load (Pascoe et al. 2013), and the type of contraction used (concentric, eccentric or isometric; (Duchateau and Baudry 2013)). Previous studies provided a fundamental understanding of the interaction of motor unit recruitment and rate coding for gradation of force. However, these studies were all performed solely in sitting. Responding to perturbations in standing utilizes task-specific muscle contractions as a result of integration of multiple sensory inputs and synaptic inputs on the motoneurones of postural muscles which differ from that of voluntary isometric contractions (Jacobs and Horak 2007). Therefore, this thesis explores the interaction of motor unit recruitment and rate coding for modulation of force during standing during external perturbations. Determining the behaviour of the healthy motor unit under conditions of standing external perturbations was necessary to facilitate understanding of how sensorimotor impairment following stroke affects motor unit behaviour in response to perturbations.

1.2.2 Postural control

The postural control of upright stance is achieved by maintaining the centre of mass (COM) of the body within the limits of the base of support (BOS) created by the feet. Postural control is commonly quantified using force platforms that measure the movement of the centre of pressure (COP) during standing postural tasks. As described by Winter (2003), the COP signal is a culmination of the vertical projection of the COM and the force generated by the body to maintain the COM within the BOS during standing tasks. The relationship between COM and COP during standing balance is such that COP movement will oscillate to keep COM in a safe position within the BOS. Postural sway and achieving balance is largely achieved with movement about the ankle in quiet stance (Winter et al. 2003). The movement about the ankle in response to modest anteriorly-directed perturbations of the COM is commonly referred to as the
ankle strategy to maintain balance within the BOS and is controlled predominately by the ankle plantarflexor muscles (Horak and Nashner 1986).

The ankle plantarflexor muscles contribute to postural control in standing by way of the interaction between contractile tissue and the series elastic component of the muscles, specifically the Achilles tendon (Loram et al. 2007b). During standing balance the contractile tissue has been shown to be stiffer than the series elastic component and the soleus muscle has been shown to produce higher contractile stiffness than the gastrocnemii muscles (Loram et al. 2007b). During postural sway, the soleus muscle demonstrates tonic activity, whereas the gastrocnemius muscles demonstrates more phasic activation in response to increasing levels of anterior progression of the COP within the BOS (Di Giulio et al. 2009; Vieira et al. 2012). The human gastrocnemius muscle has been described as being composed of approximately 50% slow-twitch fibers, whereas the soleus muscle is composed of 70-100% slow-twitch fibers (Johnson et al. 1973). This may explain the differentiation amongst the ankle plantarflexor muscles during postural control and places particular interest in the role of the gastrocnemius muscles during anteriorly-directed perturbations.

Control of standing balance requires continuous integration of afferent input within the central nervous system (CNS) regarding the position of the COM in the BOS in order to direct motor output to postural muscles controlling postural sway (Bloem et al. 2002; Gatev et al. 1999). Results of cross correlation of the COP of each foot with each respective ankle plantarflexor muscle shows that modulation of plantarflexor activity precedes movement of the COP (Gatev et al. 1999; Masani et al. 2003). This suggests that during quiet stance, the CNS applies a feed-forward control of balance, anticipating the body position based on integration of afferent input pertaining to the maintenance of the COM within the BOS, and providing an
appropriate motor response (Gatev et al. 1999; Masani et al. 2003). Ankle plantarflexor muscles demonstrate a moderate relationship with postural sway and demonstrate an increase in the strength of this relationship during postural challenges such as standing with eyes closed (Gatev et al. 1999). Importantly, the strength of the relationship between modulation of the soleus muscle and postural sway did not differ between young and older adults (Masani et al. 2011). Yet it is not known how neurological impairment, such as stroke, affects the relationship of modulation of the ankle plantarflexor muscle activity and postural sway.

1.2.3 Response to external perturbations

Paradigms applying external perturbations to the body have been employed to explore postural reactions under varying conditions. Postural reactions without movement of the feet are referred to as an 'in-place' strategies whereas stepping reactions aimed at taking steps to reorganize the COM within a re-established BOS, are termed 'change-in- support' strategies (Maki and McIlroy 2005). During in-place reactions, people commonly utilize an ankle strategy in response to modest perturbations and a hip strategy in response to larger perturbations (Horak and Nashner 1986).

Postural reactions are proposed to be long-loop reflexes which integrate afferent input at a supraspinal level and direct the motor response via spinal networks of upper and lower extremity muscles (Bloem et al. 2002; Jacobs and Horak 2007; Misiaszek 2006). External perturbations, which result in forward displacement of the COM with respect to the BOS, generate an ankle strategy controlled by the ankle plantarflexor muscles during in-place reactions (Dunbar et al. 1986; Nashner 1977). The magnitude of the postural reaction is linked to the magnitude of the perturbing stimulus displacing the COM (Diener et al. 1988). The response of the ankle muscles during perturbations is strongly influenced by the proprioceptive inputs from
hip and trunk movement (Bloem et al. 2002). This finding suggests the presence of intra-limb coordination to maintain the COM within the BOS during standing tasks.

External perturbation paradigms can be used to investigate the postural reaction, which follows the perturbation, as well as the anticipatory postural strategies prior to an expected perturbation (Horak et al. 1989; Santos et al. 2010a; Santos et al. 2010b; Sibley et al. 2008). When the magnitude and direction of a perturbation is known, anticipatory postural strategies, typically involving an increase in postural muscle activation, is directed towards resisting the anticipated perturbation prior to perturbation onset (Horak et al. 1989; Santos et al. 2010a).

When these anticipatory postural strategies are used, the magnitude of COP displacement of the associated postural reaction to the perturbation is dampened, particularly when participants know the timing of the external perturbations (Horak et al. 1989; Santos et al. 2010a; Santos et al. 2010b). These centrally-modulated anticipatory postural strategies have been referred to as a change in central set and are influenced by the individuals knowledge of the perturbation stimuli, prior experience, instructions prior to the perturbation, changes in cognitive load or attention, and emotional state (Jacobs and Horak 2007).

1.2.4 **Physiological arousal and postural control**

In addition to the central and somatic pathways which have been shown to interact during postural control tasks, the sympathetic division of the autonomic nervous system (ANS) is associated with perturbation of standing balance (Sibley et al. 2014). During external perturbation paradigms, physiological arousal levels associated with sympathetic drive from the ANS have been shown to be elevated under conditions generating fear of falling (Carpenter et al. 2006) or when external perturbations are expected but the timing is unknown (Sibley et al. 2008). In these studies, modulation of physiological arousal occurs with alterations of postural
reactions to perturbations (Carpenter et al. 2006; Sibley et al. 2008). Carpenter et al (2006) used the height paradigm (standing on an elevated platform) to determine if a postural threat resulted in concomitant change in situational emotional and physiological arousal and force platform-derived measures of standing balance in young and older adults. They found that older adults demonstrated larger changes in arousal than young adults and that arousal levels were positively correlated with levels of reported anxiety during this task (Carpenter et al. 2006). Importantly, although a great deal of literature has focused on association of physiological arousal and fear of falling, it is important to consider that increased levels of arousal in general have been shown to alter postural control in healthy subjects regardless of the nature of the associated emotion (pleasant or unpleasant emotion, (Horslen and Carpenter 2011)).

Physiological arousal is used as an objective measure of the changes associated with attention, cognitive effort, and emotion (Critchley et al. 2000). The central mechanisms of the interaction between postural control and physiological arousal are not known. However, current evidence suggests that the understanding of modulation of these parameters during postural tasks may be of particular importance to populations with low balance related self-efficacy during standing tasks, such as older adults and people with neurological impairment. Balance related self-efficacy is a term which broadly encompasses constructs such as, fear of falling, anxiety, confidence, etc. (Hauck et al. 2008). Measurement of physiological arousal provides insight into how emotion and/or generalized effort changes when balance is challenged and may represent an objective tool to further refine our knowledge of postural strategies used during response to perturbations (Hauck et al. 2008).
1.2.5 Motor impairment following stroke

Motor impairment following stroke has been characterized primarily during isometric voluntary contractions with the majority of investigations addressing upper extremity muscles. It has been described as the loss of spinal motoneurones (Hara et al. 2004; Li et al. 2011; Lukacs 2005; McComas et al. 1973), followed by collateral sprouting and reinnervation of the muscle fibres which had underwent denervation by remaining motoneurones (Kallenberg and Hermens 2011; Lukacs 2005). Following this process, it has been suggested that chronic paretic muscle is made up of fewer but larger motor units due to the remaining motoneurones innervating a greater number of muscle fibres (Kallenberg and Hermens 2011; Li et al. 2011; Lukacs 2005). It has been reported that there is greater degeneration of type II MUs following stroke (Kallenberg and Hermens 2011). This shift to lower numbers of type II MUs has been demonstrated with more low threshold MUs being active in tasks that would ordinarily have favoured the recruitment of high threshold MUs (Lukacs 2005).

Ultimately, the loss of motoneurones and remodeling of paretic muscle following stroke results in muscle contractions with slower rates of force development and decreased levels of force production (Chou et al. 2013; Garland et al. 2009; Mottram et al. 2014). It is known that the range of firing rates is compressed with lower maximal firing rates after stroke during voluntary isometric contractions of lower extremity muscles (Chou et al. 2013; Frontera et al. 1997). Specifically, Chou et al. (2013) observed that the decreased motor unit firing rate was associated with a decreased rate and magnitude of force production, suggesting that the limitation in firing rate offered a potential mechanism for the decreased speed of voluntary movement in people post-stroke. These characteristics of motor unit behaviour may explain the impairment of muscle activation and associated decreased speed of movement which has been
associated with impaired lower extremity postural control muscles following stroke (Gray et al. 2012a; Gray et al. 2012b).

1.2.6 Stroke and motor control of standing balance

Early research of impaired standing balance following stroke identified the presence of asymmetrical weight-bearing, favoring the non-paretic leg, in patients in the acute phase post-stroke which seemed to become somewhat more symmetrical during recovery (Geurts et al. 2005). This led to intervention studies focused on the restoration of symmetrical weight-bearing; yet the functional results from this approach were limited (Geurts et al. 2005). More recently, research into standing balance post-stroke has shifted away from the static feature of each leg's percentage weight-bearing to the more dynamic feature of the contribution of each leg to control of postural sway. Following stroke, postural sway demonstrates increased displacement along the anterior-posterior axis during quiet stance (Roerdink et al. 2009). When postural sway is compared between the paretic and non-paretic legs of people post-stroke using dual force platforms, during dual task paradigms or oscillations of the standing surface, it can be seen that the COP trajectory of the non-paretic foot demonstrates significantly greater amplitude of displacement and moves at a higher velocity than the paretic leg (Roerdink et al. 2009; van Asseldonk et al. 2006). Importantly, asymmetrical control of postural sway has been shown in people post-stroke despite restoration of symmetrical weight-bearing (van Asseldonk et al. 2006). As the ankle strategy plays an important role in postural control, these findings suggest the importance of understanding the motor control impairment of the ankle plantarflexor muscles during standing balance.

Following stroke, paretic ankle plantarflexors specifically, demonstrated a decreased rate of force development (Fimland et al. 2011), which may be influenced by greater atrophy of the
gastrocnemii muscles than the soleus muscle post-stroke (Ramsay et al. 2011). How the motor impairment of the ankle plantarflexor muscles post-stroke affects motor control of postural sway is not known. Furthermore, the contribution of the paretic and non-paretic ankle plantarflexor muscles to active control of postural sway under conditions of anteriorly-directed challenge to standing balance is not known in people post-stroke, or in healthy older adults. Comparing the modulation of the paretic and non-paretic ankle plantarflexors with postural sway during sustained anteriorly-directed challenges to standing balance will offer insight into the between limb differences in postural control deficits post-stroke.

The impaired response to external perturbations post-stroke has been studied extensively with standing surface translations as an external perturbation. Gastrocnemius muscles specifically have shown delayed postural reflexes (Badke et al. 1987; Marigold et al. 2004a), decreased amplitude of muscle response and variability of intra-limb muscle patterning with other muscles of the ankle and hip in the paretic leg (Badke et al. 1987; Badke and Duncan 1983; Marigold and Eng 2006a) and asymmetrical torque production favoring increased non-paretic ankle plantarflexion torque (van Asseldonk et al. 2006). Although measured in the upper extremity, people post-stroke demonstrate impaired regulation of the long-loop reflex in the biceps brachii during perturbation tasks of the upper extremity (Trumbower et al. 2013). In the lower extremity, impaired sensorimotor integration occurs following stroke with altered hip reflex activity being related to impaired inter-joint coordination of muscle activation during gait (Finley et al. 2008; Hyngstrom et al. 2010). Impaired integration of afferent input related to postural control and associated long-loop reflex responses may account for the impaired motor response to external perturbations post-stroke (Marigold et al. 2004b). This thesis explores how the motor impairment following stroke affects motor unit recruitment and rate coding in the paretic medial
gastrocnemius muscle during external perturbations in order to further understand the impaired motor response to perturbations.

1.2.7 Physiological arousal and postural control following stroke

Impairment of standing balance following stroke has been shown to result in increased attentional demands and conscious control of movement (Brown et al. 2002; Orrell et al. 2009). Additionally, decreased balance self-efficacy influences participation in community level mobility and functional independence following stroke (Botner et al. 2005; Pang and Eng 2008; Salbach et al. 2006; Schmid et al. 2012). As both increased attention to task and emotion influence physiological arousal (Critchley et al. 2000; Sibley et al. 2014), and physiological arousal is known to alter postural control in healthy people (Carpenter et al. 2006; Sibley et al. 2014), it is important to explore how stroke affects physiological arousal concomitantly with postural reactions to external perturbations. Badke et al (1983) demonstrated that during external perturbations delivered by translating force platforms, manipulation of the knowledge of direction and timing of the perturbation, improved the timing of the motor response of the paretic plantarflexor muscles. Similarly, altering conditions of knowledge of timing of the perturbation may facilitate our understanding of modulation of physiological arousal and concurrent postural control during external perturbations in people post-stroke. Specifically, this thesis explores the effect of knowledge of timing of perturbations on physiological arousal and postural control during both the anticipatory postural strategy prior to perturbations and the postural reaction immediately following the perturbation.
1.3 Methodological approach

1.3.1 External perturbation paradigm

The studies of this thesis employ an external perturbation paradigm in which anteriorly-directed external perturbations are applied at the level of the pelvis. Pilot testing of this approach revealed consistent forward progression of the COP in the BOS and increased external ankle torque, with increased ankle plantarflexor muscle activity in healthy people. The set-up is shown in Figure 1.1.

The perturbation paradigm used for Chapters 2 - 4 consisted of external loads applied through the belt by loads dropped into an attached basket from approximately 40 cm. The weights were not removed from the basket so the maintained load increased incrementally until 2.25 kg or 5% body mass was reached. Postural responses were captured during the dynamic response immediately after the perturbations as well as during the maintenance of steady state under progressively increasing loads. Chapter 5 utilized the same cable-pulley system; however, a single load of 2% body mass was dropped into the basket and removed. Repeated application of the load drop was triggered by either the investigator or by the participant.
Figure 1.1 Experimental set-up. A belt was secured around the pelvis of each participant and was attached to a horizontal cable in front of the participant. External loads were applied via a cable-pulley system attached to the front of the belt. A screen was used to ensure participants could not see when the loads were dropped.

1.3.2 Electromyography

To explore how the central nervous system regulates force production at the level of the muscle during postural tasks, this thesis employed various methods of electromyography (EMG). Intramuscular EMG was collected with the insertion of a fine wire bipolar electrode into the muscle belly and allows the recording of motor unit potentials. Bipolar surface (sEMG) uses two electrodes (typically 1 cm in diameter) to record a more global signal from the muscle, representing the activity of multiple motor units within the recording area of the sEMG electrode. High-density surface electromyography (HDsEMG) is a surface electrode comprised of multiple small electrodes which, in this thesis, are configured as multiple bipolar electrodes,
over the surface of the muscle belly and measures EMG activity throughout a broader area of the muscle.

1.3.3 Kinematics and kinetics

The performance of standing balance is measured using kinematic (motion capture) and kinetic (force platform) methodologies depending on the objectives of each study. Eight high-speed digital cameras were employed to capture the movement of passive reflective markers affixed to the body. From marker coordinate data, kinematic parameters were calculated including ankle, knee and hip joint and COM excursion and velocity in the sagittal plane. Joint angular excursion and velocity represents the movement of the limb in reorienting the COM within the BOS during the feet-in-place strategy involving external perturbations.

Dual force platforms allowed measurement of COP movement in the sagittal plane specific to each leg of participants. COP movement is representative of movement of the COM together with the body's actions to maintain COM in a safe position in the BOS (Winter et al. 2003). Therefore, during a postural perturbation with response limited to a feet-in-place response, COP excursion and velocity represents the cumulative effect of the perturbation on the body's movement as controlled by each limb.

1.3.4 Electrodermal activation

Electrodermal activation (EDA) is a measure of electrical conductivity of the surface of the skin or skin conductance. Two surface electrodes on the palm of the hand continually pass a small electrical current between electrodes over the surface of the skin, which provides a continuous measurement of the skins conductivity. Sympathetic discharge controls sweat gland secretion which can be measured on skin surfaces by detection of changes in skin conductivity.
Sympathetic drive can be inferred from EDA measurement during standing perturbation paradigms (Sibley et al. 2014).

1.4 Thesis outline

The overarching objective of this research is to investigate the motor control impairment of postural reactions in people with chronic stroke, with a specific focus on the plantarflexor role in the ankle strategy response to external perturbations. This thesis first explored motor control of postural reactions starting with the motor unit. To accomplish this, the motor unit behaviour of the medial gastrocnemius muscle in response to external standing perturbations in healthy participants was established, as this has not been addressed in existing literature. Next, motor unit behaviour of the paretic medial gastrocnemius of people post-stroke was investigated in response to external perturbations in standing. Motor control of standing balance was explored by measuring the strength of relationship of modulation of each of the three ankle plantarflexor muscles with COP displacement under conditions of maintaining increasing levels of anteriorly-directed loads. Finally, modulation of postural control together with levels of physiological arousal was investigated during investigator- and self-triggered perturbations to explore the influence of knowledge of timing of a perturbation on the anticipatory postural strategy and the postural reactions of people post-stroke.

1.4.1 Objectives and hypotheses

Chapter 2

Objective: The purpose of this study was to examine the motor unit recruitment and firing rate modulation of the gastrocnemius muscle in healthy controls in response to maintaining increased levels of anteriorly-directed load and experiencing external perturbations in standing.
Hypothesis: We hypothesized that the increased force required from the medial gastrocnemius to resist the external loads would increase both motor unit recruitment and firing rate. During the dynamic response to perturbation, we hypothesized that motor unit recruitment would occur with each perturbation and motor unit firing rate would increase with subsequent perturbations experienced with increasing levels of anterior progression of the COP.

Chapter 3

Objective: The purpose of this study was to investigate the behaviour of motor units in the medial gastrocnemius and the postural reaction to external perturbations in standing in people with chronic stroke.

Hypothesis: We hypothesized that limitations in both motor unit firing rate and recruitment in the paretic medial gastrocnemius muscle post-stroke would alter the postural reaction strategy used to resist the external loads. Reflective of an impaired ankle strategy, we anticipated that motor unit firing rate would not modulate with COM, anterior-posterior COP or lower extremity joint angular velocity.

Chapter 4

Objective: The purpose of this study was to examine motor control of each of the three ankle plantarflexor muscles with postural sway during increasing sustained challenges to standing balance. We aimed to compare the between leg differences in participants post-stroke and age-matched controls.

Hypothesis: We hypothesized that the non-paretic limb of people post-stroke would demonstrate stronger relationships between the modulation of plantarflexor activity with postural sway than the paretic limb. We further anticipated that the relationship of modulation of each
plantarflexor muscle activity and postural sway would be lower in the gastrocnemius muscles compared to the soleus muscles in the paretic leg of people post-stroke.

Chapter 5

Objective: The purpose of this study was to explore the influence of knowledge of timing of external perturbations on postural control and the level of physiological arousal in people post-stroke.

Hypothesis: We hypothesized that people post-stroke would demonstrate increased levels of arousal prior to perturbations and in response to perturbations than age-matched controls. We anticipated that the arousal level would be less during self-triggered perturbations than investigator-triggered perturbations for both people post-stroke and controls. We further hypothesized that a heightened pre-perturbation postural preparation during investigator-triggered perturbations in people post-stroke would result in heightened ankle plantarflexor activity prior to perturbation and decreased COM and APCOP displacement and velocity in response to perturbations compared to controls.
Chapter 2: Motor unit recruitment and firing rate in medial gastrocnemius muscles during external perturbations in standing in humans

2.1 Introduction

Gradation of muscle force is controlled by a combination of motor unit recruitment and rate coding (Heckman and Enoka 2012). Muscles differ in their use of motor unit recruitment versus rate coding to modulate force, possibly related to the muscle fiber composition and characteristics of muscle contraction (De Luca et al. 1982; Kukulka and Clamann 1981). For instance, during ramp isometric contractions, muscles composed of a mix of fiber types have been shown to utilize motor unit recruitment throughout a wider range of available force than muscles consisting primarily of slow twitch fibers (Kukulka and Clamann 1981). The characteristics of the muscle contraction that can influence the interaction of motor unit recruitment and rate coding include: joint angle and muscle length (Ballantyne et al. 1993; Kennedy and Cresswell 2001), speed of muscle contraction (Desmedt and Godaux 1977), nature of the external load (Pascoe et al. 2013) and the type of contraction used (concentric, eccentric or isometric; Duchateau and Baudry 2013). All of the aforementioned studies were performed in sitting and the interaction between motor unit recruitment and rate coding for modulating force during standing or in response to standing external perturbations has not been explored. Standing balance integrates multiple sensory inputs and the synaptic inputs on the motoneurones of postural muscles in standing differ from those of isometric ramp contractions (Jacobs and Horak 2007). Therefore, motor unit behaviour in a functional task of standing and withstanding external perturbations must be examined in a task specific paradigm.
Maintaining standing balance requires control of postural muscles, specifically the ankle plantarflexors play a critical role in controlling anterior-posterior movements of the center of mass (COM) within the base of support. It has been suggested that the soleus muscle is the main muscle controlling balance during quiet standing, while the gastrocnemius muscle plays an increasingly active role when the COM travels more anterior to the ankle joint (Di Giulio et al. 2009). Motor unit firing rate of the soleus muscle has been shown to demonstrate rather modest modulation during quiet stance with eyes open or closed (Mochizuki et al. 2007). Conversely, motor unit activity of the medial gastrocnemius muscle has been shown to be intermittent with recruitment of motor units primarily during forward sway of the center of pressure (COP) in quiet standing (Vieira et al. 2012).

Forward sway and the associated length changes of the muscles about the ankle (both plantarflexors and dorsiflexors) have been suggested to provide the sensory information which drives modulation of the gastrocnemius muscles during quiet standing (Di Giulio et al. 2009; Tokuno et al. 2008; Vieira et al. 2012). Plantarflexor activity in standing has been shown to precede anterior displacement of the COP (Masani et al. 2003). However, it has been shown that compared to the COM displacement, the velocity of COM during quiet standing is more associated with activation of the ankle plantarflexors in anticipation of a forward sway (Masani et al. 2003). Responding to perturbations in standing has been shown to integrate both short loop reflexive behaviour, and long loop reflexive behaviour which incorporates supraspinal centers (Jacobs and Horak 2007). The relative weighting of these inputs has been suggested to change with increased challenge to standing balance (Gibbs et al. 1995; Horak 2006). However, it remains unclear as to how the medial gastrocnemius muscle modulates force in response to standing perturbations. The purpose of this study was to determine; 1) if increased firing rate and
recruitment of motor units in medial gastrocnemius muscle occur during the maintenance of standing balance with increasing levels of anteriorly-directed loads, and 2) if the medial gastrocnemius muscle utilizes motor unit recruitment and rate coding in response to abrupt external perturbations of equal magnitude superimposed on the anteriorly-directed loads. Specifically, we hypothesized that the increased force required from the medial gastrocnemius to resist the external loads would increase both motor unit recruitment and firing rate. During the dynamic response to perturbation, we hypothesized that motor unit recruitment would occur with each perturbation and motor unit firing rate would increase with subsequent perturbations experienced with increasing levels of anterior progression of the COP.

2.2 Methods

Six healthy adults (3 women, 42 ± 8.8 years of age, 60.8 ± 9.0 kg, 166.5 ± 10.5 cm) participated on three separate occasions. Individuals were excluded if they had any health conditions that negatively impacted mobility (e.g. severe osteoarthritis, neurological conditions). The study conformed to the standards set by the latest revision of the Declaration of Helsinki and was approved by the University of British Columbia Clinical Research Ethics Board.

2.2.1 Experimental protocol

Participants stood with their feet shoulder-width apart, with each foot on a separate force platform (AMTI OR6-6, Advanced Mechanical Technology, Watertown, MA). Baseline quiet standing data were collected for 30 seconds; then participants were instructed to lean as far forward as possible without needing to take a step to regain their balance. This point represented the perceived anterior limit of stability (LOS). LOS trials were performed twice and the maximum anterior excursion was used as the perceived anterior LOS.
A belt was secured around the pelvis of each participant and was attached to a horizontal cable in front of the participant. External loads were applied via a cable-pulley system (1.6 mm 7x7 galvanized aircraft cable, graded for up to 41 kg with a steel pulley mounted on a tripod) attached to the front of the belt (Fig. 2.1). Participants remained standing in comfortable stance with each foot on a separate force platform as external loads of 0.45 kg were applied five times through the belt by being dropped into an attached basket from approximately 40 cm every 25-40 seconds (random timing); the weights were not removed from the basket so the maintained load increased incrementally until 2.25 kg was reached. The calibrated weights were made from concrete gravel pellets housed in a soft cover bag. A screen in front of the participants prevented them from seeing when the loads were dropped. Application of the load was detected by a force transducer in-line with the cable (Fig. 2.1). Subjects were instructed to maintain their standing balance without taking a step in response to each perturbation. All participants experienced a perturbation in a practice trial prior to the data collection.
Figure 2.1 Experimental set-up. Schematic of experimental set up for applying perturbations that result in a pull in anterior direction. Figure depicts markers (labels underlined) denoting segments used to calculate ankle, knee and hip joint angles (marked with dotted semicircle).

2.2.2 Motor unit recordings

Motor unit potentials were recorded intramuscularly using fine wire bipolar electrodes. Bipolar electrodes were custom made with three 50-µm stainless steel fine wires (California Fine Wire, Grover Beach, CA) fastened together with cyanoacrylate adhesive and inserted into a disposable 2cm, 25 gauge hypodermic needle (Becton Dickinson, Franklin Lakes, NJ). A hook, ~2mm in length, was formed at the recording end of the electrode. The three-wire-electrode allowed selection of a recording from three possible bipolar configurations and choice of the optimal configuration based on signal-to-noise ratio. The electrodes were autoclaved prior to use for 25 min at 120°C. The needle was used to insert the electrode into the medial aspect of the medial gastrocnemius to a depth of approximately 2cm and was extracted, leaving the fine wire
electrode in the muscle. Two electrodes were inserted into the medial gastrocnemius muscle of each leg (4 electrodes in total), with approximately 5-7 cm separating the electrodes.

The electrode position was adjusted to ensure at least one motor unit potential was identifiable from each medial gastrocnemius muscle (in some trials more than one motor unit was identifiable per electrode). Once this was achieved, the electrodes were not moved again. Motor unit recordings were sampled at 20 kHz.

2.2.3 Surface EMG recordings

Bipolar electrodes (1-cm interelectode distance) were used to record surface EMG bilaterally from the medial gastrocnemius (GM), soleus (SOL), rectus femoris (RF), biceps femoris (BF) and lumbar erector spinae (LES) bilaterally. Surface EMG (Delsys Inc., Natick, MA) was sampled at 2000 Hz and saved for off-line analysis.

2.2.4 Kinetic and kinematic data

Kinetic data were collected using two floor-mounted force platforms (detailed above), sampled at 2000 Hz. Anterior-posterior center of pressure (APCOP) displacements and velocity (the derivative of APCOP displacement) with each perturbation were calculated from raw force platform data. Twenty two passive reflective markers were affixed to participants according to a modified Helen Hayes marker set (Kadaba et al. 1989) to allow for motion capture of the arms, trunk and legs bilaterally. Eight high-speed digital cameras (Raptor-E, Motion Analysis Corp, Santa Rosa, CA) sampled the movement of the reflective markers at 100 Hz. Kinematic data were analyzed using a custom-written program (Mathworks Inc., Natick, MA, USA) developed in a previous study to quantify lower extremity kinematics during movement (Pollock et al. 2012). Body segment angles were calculated only in the sagittal plane for the ankle, knee, and hip (Fig. 2.1). External torque applied about the ankles was calculated as the product of the
perpendicular distance of the APCOP from the ankle joint center and the vertical component of the ground reaction force.

2.2.5 Data analysis

Motor unit identification was performed in Spike2 (Cambridge Electronic Design, Cambridge, UK) using a template-matching algorithm which classifies motor unit potentials according to their shape and amplitude of the motor unit potentials. Visual inspection of the data allowed the ability to identify instances of misclassification; sections of data which contained misclassifications were eliminated from the analysis.

Motor unit firing rate was calculated during two epochs: (1) the dynamic response to the perturbation – mean of the first three interspike intervals (ISIs) directly following each load drop and fastest ISI of the first three ISIs, and (2) maintenance of steady state between perturbations – mean ISI over a five second epoch taken between five seconds after the load drop and one second prior to the next load drop. The mean, standard deviation and coefficient of variation (CV) of the ISIs during this five second epoch were calculated. Since it has been shown that motor unit behaviour can differ between that observed at initial recruitment versus when a motor unit is already firing (Van Cutsem M. and Duchateau 2005), motor units were analyzed at the recruitment load separately from the subsequent loads. Motor units which were active in the dynamic response to load drop and during maintenance of steady state at the same load level were analyzed to determine the extent of modulation of the motor unit firing rate in response to the load perturbation.

Root Mean Square (RMS) of the surface EMG for each muscle was calculated during the aforementioned time periods of interest, for 500 ms during the dynamic response following load drop and for the same 5s epoch used to measure motor unit ISI during the maintenance of steady
state between perturbations. RMS was normalized to quiet standing taken prior to the application of the first load. The excursion of the APCOP was calculated and converted to a percentage of the LOS. The velocity of the APCOP excursion during the dynamic response to load drop was calculated as the derivative of the APCOP signal. The peak external torque at the ankle during the dynamic response to load drop and mean external torque at the ankle during steady state was calculated and normalized to the external torque calculated during quiet stance. The changes in ankle, knee, and hip angle in the sagittal plane in response to the external loads were calculated with respect to quiet standing.

2.2.6 Statistical analysis

Statistical analysis was performed using SPSS v.20.0 (SPSS, Chicago, IL). ISI data were not normally distributed; therefore, Kruskal–Wallis analysis of variance (ANOVA) tests were performed on the median motor unit ISI duration to test for the effect of load level (1-5) on motor unit firing rate during the dynamic response and during steady state. When significance was noted, Mann-Whitney tests were used to compare between load levels. Motor unit firing rate was compared between initial recruitment load and subsequent loads, and between epochs using Wilcoxon Signed Ranks tests. Motor unit data are reported as median and interquartile range (IQR), all other data are presented as mean ± standard deviation. Kinematic, kinetic and surface EMG data were normally distributed and were compared for the effect of load level (1-5) and side (right, left) using separate mixed model two-way ANOVAs with load as a repeated measure. The RMS amplitude of surface EMG data were compared across muscles and loads (repeated measure) using a two-way ANOVA with Tukey post-hoc analysis. The RMS amplitude of the medial gastrocnemius during the dynamic response to perturbation was compared to steady state using paired t-tests.
2.3 Results

2.3.1 Overview of motor unit recruitment

In total, 57 motor units were identified (yield of 1-4 motor units from each leg) and followed over successive loads once recruited. Motor units were collected successfully from 12 of the 18 experiments performed. Motor units were recruited either during the dynamic response to load drop (total n = 40) or during the steady state periods (total n = 17). Only two of the recorded motor units were active in quiet stance prior to the first load drop. Some motor units were derecruited in the steady state period after the dynamic response to load drop on the recruitment load (total n=10) yet discharged more steadily after the next load drop and for all subsequent loads (Table 2.1).

Table 2.1 Motor unit recruitment by load level

<table>
<thead>
<tr>
<th>Load</th>
<th>Numbers of motor units recruited during Dynamic response Steady state</th>
<th>Dynamic response (derecruited in steady state)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dynamic response</td>
<td>Steady state</td>
</tr>
<tr>
<td>1</td>
<td>18</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>17</td>
</tr>
</tbody>
</table>
2.3.2 Dynamic response to the perturbation

Figure 2.2 shows two representative examples of the dynamic response to load drop. The mean of the first three ISIs immediately after load drop was similar irrespective of the load at which the motor unit was recruited (Fig. 2.3 A, p=0.76). During subsequent load drops, there was also no significant difference in the motor unit firing rate across load levels (Fig. 2.3B, p=0.97). However, for the 40 motor units recruited during the dynamic response to load drop, the median motor unit firing rate was significantly slower at the initial recruitment load (150 ms, IQR 123-170 ms) than during the dynamic response to subsequent load drops (110 ms, IQR 90-125 ms, p < 0.001) or during the steady state discharge at the same load (125 ms, IQR 120-143 ms, p =0.001). The fastest ISI (84 ms, IQR 70-103 ms) was significantly shorter than the average of the first 3 ISI at each load (Fig. 2.4, p <0.01). There was also a significant effect of load on the fastest ISI, with the fastest ISI of load 4 being significantly longer than all other load levels (Fig. 2.4).
Figure 2.2 Examples of motor unit dynamic response to load drop. Dynamic response to load drop at recruitment load (left) and subsequent load (right) in two representative subjects. Tracings from top: Force transducer signal (Force) showing application of perturbation (Load), position of anterior posterior center of pressure (AP COP) as %LOS, AP COP velocity, intramuscular EMG traces of motor units from medial gastrocnemius muscles (GM MU). The first four motor unit potentials (*) that are used to calculate the mean interspike interval in the dynamic response are shown. A) Recruitment load was the first load drop. B) Recruitment load was the fourth load drop. In both examples, the duration of the first 3 ISIs was less with subsequent loads when the motor unit was already active compared to the duration of the first 3 ISIs at recruitment.
Figure 2.3  Duration of the motor unit first 3 ISIs during dynamic response. Boxplot (median) of motor unit interspike intervals (ISI) during dynamic response grouped by recruitment versus subsequent loads.  A) Motor unit behaviour at initial recruitment based on whether recruited at load 1, 2 or 3-5. B) Motor unit behaviour with subsequent loads where motor units were active prior to perturbation. Increased level of perturbation did not influence firing rate (loads 3-5 combined due to lower number of motor units identified at these perturbation levels). At recruitment motor units demonstrated increased duration of the first 3 ISIs during dynamic response compared to motor unit duration of the first 3 ISIs in response to subsequent load drops,* p<0.05.
Figure 2.4  Motor unit firing behaviour after initial recruitment, with subsequent perturbations. Median and interquartile range for motor unit interspike interval (ISI) evoked by the perturbation: fastest of first 3 ISIs (grey circles), mean of first 3 ISIs (black circles) followed by ISI during steady state at the next load increment (open circles). Motor units modulated their firing behaviour by significantly decreasing the duration of ISI during the dynamic response to load drop compared to ISI duration during steady state. The duration of the ISI was significantly longer between steady state and the dynamic response to perturbation and between the mean of the first 3 ISIs and the fastest ISI, *p<0.05. The duration of the fastest ISI during the dynamic response at load 4 was significantly longer than with other perturbations, †p<0.05.
2.3.3 Maintenance of steady state between perturbations

As with the dynamic response to the load drop, there was no significant effect of increasing load on the motor unit firing rate (Fig. 2.4, p = 0.80). The consistency in motor unit firing rate in the steady state phase between perturbations is demonstrated in Figure 2.5 in a representative subject. The CV of motor unit firing rate was significantly larger during the maintenance of steady state following the initial recruitment (18.24%, IQR 14.70-23.00%) than during the steady state after subsequent load drops (15.85%, IQR 11.97-19.30%, p < 0.01).

Figure 2.5 Representative example of electromyography recordings from the medial gastrocnemius muscle in standing during incremental increases in external load. From top row; arrows indicating timing of perturbations (consecutive load drops), raw signal of surface electromyography recorded from medial gastrocnemius (GM sEMG), raw signal of intramuscular EMG recorded from medial gastrocnemius muscle (GM MU), two individual MUs (MU1, 2) identified from the raw signal and displayed as instantaneous firing rate.
2.3.4  Modulation between dynamic response to perturbation and maintenance of steady state

Although there was no effect of load on motor unit firing rate, there was significant modulation of the motor unit firing rate in the dynamic response to the load drop when the motor units were already active prior to the load drop (Fig. 2.2). The motor unit firing rate was significantly faster (110 ms, IQR 90-125 ms) during the dynamic response than steady state (130ms, IQR 110-140 ms) on subsequent load drops excluding the recruitment load (Fig. 2.4, p <0.01).

2.3.5  Kinetic, kinematic and surface EMG response to perturbations

None of the kinetic, kinematic or surface EMG data were different between the left and right legs of participants (p>0.1), reflecting symmetrical responses to the external loads. Therefore, data from both legs were averaged (Fig. 2.6). The average BOS (heel to base of first metatarsal) of all participants was 192.8 ± 14.2 mm. The APCOP excursion at LOS measured 114.8 ± 18.5 mm. The application of the external loads resulted in a significant increase in external torque applied to the ankle both during the dynamic response to load drop (peak torque, p<0.001) and during steady state (mean torque, p<0.01) (Fig. 2.6A). This was associated with a significant forward progression of the APCOP with increasing load, which culminated in an anterior excursion of COP (expressed as change from initial position) of 46.3 ± 22.8 mm by the fifth load level (Table 2.2), an excursion representing 41.7 ± 21.6 % of the LOS (Fig. 2.6A). In contrast to APCOP positions, there was no significant effect of load level on APCOP velocity during the dynamic response to load drop (p = 0.21).
Table 2.2 Anterior-posterior centre of pressure (APCOP) excursion by load level

<table>
<thead>
<tr>
<th>Load</th>
<th>APCOP excursion (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>14.1 ± 7.1</td>
</tr>
<tr>
<td>2</td>
<td>24.2 ± 17.3</td>
</tr>
<tr>
<td>3</td>
<td>33.6 ± 22.0</td>
</tr>
<tr>
<td>4</td>
<td>40.1 ± 22.7</td>
</tr>
<tr>
<td>5</td>
<td>46.3 ± 22.8</td>
</tr>
</tbody>
</table>

The RMS amplitude of each muscle during steady state, normalized to quiet stance, is presented in Figure 2.6B. There was a significant effect of increasing load on RMS amplitude in medial gastrocnemius (p=0.02), soleus (p<0.01) and tibialis anterior (p<0.01), but not for rectus femoris (p=0.19), biceps femoris (p=0.21) or lumbar erector spinae (p=0.15). Compared among muscles at each load, the RMS amplitude of the medial gastrocnemius muscle increased significantly more than soleus, tibialis anterior, biceps femoris, rectus femoris and lumbar erector spinae at each load (Fig. 2.6B, p≤0.05). The RMS amplitude of the medial gastrocnemius muscle during the dynamic response to perturbation was significantly larger than during steady state (p<0.01).

Within each joint, there was a significant effect of increasing load on increasing hip extension (p=0.01) and knee flexion (p=0.02) but only a trend for ankle dorsiflexion (p=0.07). However, the changes in ankle, knee and hip position in response to the external load from quiet stance were small (less than 3 degrees, Fig. 2.6C). The standard error of joint excursions at each load level was less than 1 degree.
Figure 2.6 Kinetic, surface electromyography (EMG) and kinematic response to increasing levels of perturbation. The mean and standard error are presented for all parameters with statistical differences across load being represented with †p<0.05, trend, ‡p=0.07. A) There was a significant increase in peak torque at the ankle during the dynamic response to the perturbation (black squares), mean torque during the maintenance of steady state (open circles) and forward progression of anterior posterior center of pressure (AP COP) expressed as a percentage of anterior limits of stability (% LOS) of the anterior progression of the COP (black asterisks). B) Root mean square (RMS) amplitude of surface EMG normalized to pre-load drop standing for muscles of the lower extremity and low back in response to an anterior pull on the pelvis. There was a significant increase in the RMS amplitude of medial gastrocnemius (GM) and soleus (SOL), and a significant decrease in tibialis anterior (TA). The gradual increase of biceps femoris (BF) and lumbar erector spinae (LES) muscles and the slight decrease in RMS amplitude of the rectus femoris (RF) muscles did not reach significance with increased load levels. The increase in RMS amplitude of medial gastrocnemius (GM) was significantly greater than all other muscles at each load level *p<0.05. C) Joint angle position changes from quiet standing. Ankle dorsiflexion (square), knee flexion (circle) and hip extension (triangle) increased with increasing levels of load, †p<0.05, trend, ‡p=0.07.
2.4 Discussion

The aim of this study was to examine motor unit recruitment and rate coding in the medial gastrocnemius muscle in response to external perturbations experienced under increased levels of challenge to standing balance. Multiple kinematic and kinetic measures supported the potency of the postural manipulations. The loads resulted in a symmetrical response in the two legs including a gradual increase in external ankle torque, forward progression of the COP and small but significant increases in hip extension and knee flexion. There was preferential activation of the medial gastrocnemius muscle, which was significantly higher than all other muscles recorded. Motor units were recruited both in the dynamic response to load drop and during the steady state periods between external loads. Upon initial recruitment, motor units demonstrated a lower firing rate than at subsequent loads. Thereafter, rate coding was only observed as a transient increase in firing rate in response to each perturbation, followed by a return to a lower firing rate that did not change with increases in static anterior loads maintained at the pelvis. Considering the significant increase in the amplitude of medial gastrocnemius muscle EMG with increased static loading, this suggests that motor unit recruitment may have been the prevailing means of force gradation within this postural task.

The range for MU recruitment force during ramp isometric contractions has been shown to differ in muscles with different fiber type composition and function (De Luca et al. 1982; Kukulka and Clamann 1981). In the upper extremity, MU recruitment occurred up to 80% maximal voluntary contraction (MVC) in the biceps brachii (comprised of 34-61% type I muscle fibers), whereas adductor pollicis (72-91% type I muscle fibers) showed no further recruitment beyond 30-40% MVC (Kukulka and Clamann 1981). However, the soleus muscle, also comprised of a large percentage of type I muscle fibers, has shown motor unit recruitment during...
ramp contractions at forces greater than 89% maximum voluntary contraction (Oya et al. 2009). Similar to biceps brachii, medial gastrocnemius muscle is comprised of 47-57% type I fibers (Johnson et al. 1973) and, in the current study, the medial gastrocnemius muscle showed no significant difference in firing rate in standing during maintenance of steady state across all loads. This suggests a larger role for motor unit recruitment to maintain standing in the presence of a progressive forward movement of the COP and the associated increase in external torque applied about the ankle.

Immediately after each load drop, the behaviour of the motor unit showed a transient increase in firing rate compared to steady state after the initial recruitment load. This behaviour mirrored the external peak torque at the ankle that was greater than the mean torque when the same load was maintained during standing. There are a number of factors that may influence the modulation of firing rate which occurred only in response to the dynamic perturbation. It is interesting to consider our motor unit findings in the context of Masani et al. (2003), who noted the importance of the AP COM velocity in modulating gastrocnemius activation. As there is no sensory system that directly measures the velocity of the COM, Masani et al. (2003) suggest an integration of multisensory information at the CNS contributes to the velocity feedback system. In the current study, there was no significant increase in the APCOP velocity or motor unit firing rate during the dynamic response across loads. It is possible that the motor unit firing rate in the medial gastrocnemius muscle following perturbation is related to controlling the COM velocity which is reflected in the COP velocity during the dynamic response. It is also possible that the consistency of the motor unit discharge across loads may reflect the fact that the perturbations were imposed by a load of equal magnitude (0.45 kg), resulting in a similar magnitude of destabilizing effect (relative to the preceding maintained load).
It has been demonstrated that the reflex excitability of the motoneurone pool is altered by the relative direction of the COP sway in quiet standing, with the H-reflex response being heightened during forward sway (Tokuno et al. 2008). Motor unit activity of the medial gastrocnemius muscle in quiet standing has been shown to be intermittent and phase locked with anterior shifts of the COP (Di Giulio et al. 2009; Vieira et al. 2012). In the standing position, with the COP advanced anteriorly within the base of support, modulation of the pre-activated medial gastrocnemius motor units firing rate immediately after load drop may be related to heightened sensitivity of the Ia afferent input in this forward position (Di Giulio et al. 2009). This is supported by the finding that medial gastrocnemius fascicle lengths were responsive to length changes associated with dorsiflexion when the knee is held at 0° but not when the knee was bent (Wakahara et al. 2009), suggesting an increased responsiveness of the gastrocnemius to length changes when the muscle is at a lengthened position as in standing.

Sensory input from stretch of the hip flexors, which would occur as a result of the anterior pull at the pelvis in this paradigm, may have influenced the motor unit firing rate modulation and recruitment of the medial gastrocnemius. The postural reactions of muscles about the ankle and knee in response to perturbations have been shown to be primarily triggered by proprioceptive input from the hip and trunk movement (Bloem et al. 2002). It is interesting to note that on the fourth load (see Fig 2.6C) the slight movement towards hip and knee flexion was accompanied by the fastest ISI being significantly longer than with the each of the other loads. The interaction between changes in joint position for the proximal joints and medial gastrocnemius motor unit firing rate modulation and recruitment warrants further investigation.

The modest increase in firing rate immediately after load drop in motor units active prior to perturbation may be reflective of the properties of the Achilles tendon. During standing
balance the contractile tissue has been shown to be stiffer than the series elastic component, particularly in the Achilles tendon (Loram et al. 2007a; Loram et al. 2007b). This stiffness may have contributed to the limited dorsiflexion at the ankle (less than 1.5° change, Fig 7C) and be playing a buffering role for the plantarflexor force in the dynamic response to perturbation, lessening the need for firing rate modulation.

The muscle activity associated with postural control is modulated by the brainstem with input from Ia afferents and the vestibular system contributing significantly to modulation of this activity level (Creath et al. 2008). Neuromodulation of motoneurones from brainstem inputs has been shown to provide persistent inward currents (PICs) which are known to lower the recruitment threshold of motoneurones, thereby, resulting in amplification of the excitatory input and self-sustained firing (Hultborn et al. 2003; Hyngstrom et al. 2007). While speculative, the motor units in the current study fired more consistently once recruited (lower CV of ISI); behaviour which is consistent with the influence of PICs on the medial gastrocnemius motor units.

Finally, the activation of motor units was in response to perturbations and participants were asked to maintain their standing balance while experiencing these perturbations. For this reason the response to the perturbation would be somewhat controlled. Recruitment of new motor units at a slower firing rate may reflect a strategy to meet the demands of the increased perturbation without further destabilizing standing balance as may occur with a faster firing rate. This suggestion is consistent with Desmedt and Godaux (1977) who observed that the fast initial ISI (< 17 ms) at recruitment was only present during trials which did not attempt to control the force pattern and subjects simply contracted as fast as possible.
Overall, these results support the possibility of task dependency on the interaction of rate coding and recruitment. Within the current standing perturbation paradigm, rate coding and recruitment were used differently for static maintenance of load than when abrupt dynamic perturbations were experienced. It may be the case that increases in the magnitude of perturbation loads or the nature of the response (e.g. able to take a step in response) could affect the initial firing rate response of newly recruited motor units.

2.5 Conclusion

The medial gastrocnemius muscle utilized motor unit recruitment to achieve the increased levels of ankle torque necessary to maintain standing in the presence of external loads. However, there was evidence of modest rate coding during the dynamic response to perturbations which may be attempting to control the velocity of the forward movement associated with perturbations. Multiple sensory inputs are likely integrated to control medial gastrocnemius activation during steady-state maintenance of standing against a load and during abrupt perturbations. Accordingly, these data support the task dependent nature of motor unit recruitment and rate coding and extend these findings to maintaining standing balance.
Chapter 3: Behaviour of medial gastrocnemius motor units during postural reactions to external perturbations after stroke

3.1 Introduction

The ankle plantarflexors are the main muscles controlling standing balance during anteriorly-directed sways (Di Giulio et al. 2009; Tokuno et al. 2007); this is often referred to as the “ankle strategy” (Winter et al. 1998). The medial gastrocnemius muscle has been suggested to be particularly active with anterior sways of increased magnitude (Di Giulio et al. 2009; Vieira et al. 2012) and gastrocnemius muscles have been shown to modulate with centre of mass (COM) velocity and anterior-posterior centre of pressure (APCOP) velocity and excursion during quiet stance (Gatev et al. 1999; Masani et al. 2003; Vieira et al. 2012). For these reasons the gastrocnemius muscles specifically are critical for regaining balance following a standing anterior perturbation.

Decreased postural control and sensorimotor impairments are common after stroke (Garland et al. 2009). Loss of motoneurones and remodeling of paretic muscle following stroke results in muscle contractions with slower rates of force development and decreased levels of force production (Chou et al. 2013; Mottram et al. 2014); these changes may make the motor response to postural perturbations difficult to execute. Paretic ankle plantarflexors, specifically, have demonstrated a decreased rate of force development (Fimland et al. 2011), which may be influenced by greater atrophy of the gastrocnemius muscles than the soleus muscle following stroke (Ramsay et al. 2011).

The impaired response to external perturbations in standing following stroke has been studied extensively with surface translations. Gastrocnemius muscles have shown delayed
postural reflexes (Badke et al. 1987; Di Fabio et al. 1986; Marigold et al. 2004a), decreased amplitude of muscle response and variability of the muscle patterning compared with non-paretic limb and controls (Badke et al. 1987), and asymmetrical torque production favouring increased non-paretic ankle plantarflexion torque (van Asseldonk et al. 2006). It is known that firing rate modulation is reduced after stroke during voluntary tasks (Chou et al. 2013; Frontera et al. 1997; Gemperline et al. 1995). However, the role of motor unit recruitment and firing rate in the plantarflexors when utilizing the ankle strategy during anteriorly-directed external perturbations after stroke is not known. Understanding medial gastrocnemius motor unit response to a standing perturbation will add to our fundamental understanding of the muscle impairment which is functionally known to limit the effectiveness of the ankle strategy during postural control tasks post-stroke.

In previous work, we used a novel anteriorly-directed external perturbation paradigm in standing to investigate motor unit recruitment and rate coding in the medial gastrocnemius muscle of healthy people. Dynamic perturbations were applied as participants maintained increasing levels of external load which resulted in a forward progression of the APCOP (Pollock et al. 2014). Although there was an increase in instantaneous firing rate in medial gastrocnemius motor units in response to abrupt external perturbations, motor unit recruitment was used primarily for the maintenance of standing with increased external torque about the ankle (Pollock et al. 2014).

The aim of this study was to investigate the behaviour of motor units in the medial gastrocnemius, in the context of the postural response as described by kinematic and kinetic variables, during external perturbations in standing in people with chronic stroke. We aimed to 1) determine if the paretic medial gastrocnemius muscle uses motor unit recruitment and rate
coding in response to abrupt external perturbations experienced under conditions of increasing anteriorly-directed loads, and 2) to explore any relationships of the motor unit firing rate with postural control strategies of the lower extremities. Measurement of joint kinematics of the lower extremities, movement of the COM, anterio-posterior centre of pressure and external ankle torque allowed motor unit behavior to be put in the context of the postural strategy specific to each leg during external perturbations. As post-stroke, the ankle strategy has demonstrated impairment in response to perturbations (Badke et al. 1987; Di Fabio et al. 1986; Marigold et al. 2004a; van Asseldonk et al. 2006), we hypothesized that limitations in both motor unit firing rate and recruitment in the paretic medial gastrocnemius muscle post-stroke would alter the postural reaction strategy used to resist the external loads. Reflective of an impaired ankle strategy, we anticipated that motor unit firing rate would not modulate with COM, APCOP or lower extremity joint angular velocity.

3.2 Methods

Nine people with chronic stroke (>3months) participated. Individuals post-stroke were included if they were ambulatory with or without a walking aid, and could stand independently for a minimum of five minutes. Individuals were excluded if, in addition to stroke, they had any health conditions that negatively impacted mobility (e.g. severe osteoarthritis). The severity of motor impairment following stroke was measured at the foot and ankle using the Chedoke-McMaster Stroke Assessment (CMSA, (Gowland et al. 1993). Community-level walking balance was measured using the Community Balance and Mobility scale (CB&M, (Howe et al. 2006; Knorr et al. 2010). The study conformed to the standards set by the latest revision of the Declaration of Helsinki and was approved by the University of British Columbia Clinical Research Ethics Board.
3.2.1 Experimental protocol

Participants stood with their feet shoulder-width apart and on separate floor-mounted force platforms (AMTI OR6-6, Advanced Mechanical Technology, Watertown, MA). Baseline data in quiet standing were collected for 30 seconds, then participants were instructed to lean as far forward as possible without needing to take a step to regain their balance. This point represented their perceived anterior limit of stability (LOS). These trials were performed twice and the maximum anterior excursion was used as the perceived anterior LOS.

External loads were applied via a cable-pulley system attached to the front of a belt secured around the pelvis of each participant (Fig. 3.1). Participants stood in comfortable stance and external perturbations were introduced every 25-40 s (random timing) by loads of one percent body mass (1% BM) being dropped five times into a basket 40 cm below; the weights were not removed from the basket so the maintained load increased incrementally until 5% BM was reached. Participants were asked to focus on maintaining equal weight-bearing on the paretic and non-paretic legs and were instructed to maintain their standing balance without taking a step in response to each perturbation. Participants could not see when the loads were dropped. The timing of the application of the load was detected by a force transducer in-line with the cable (Fig. 3.1). The force signal was sampled at 2000 Hz and recorded for off-line analysis.
3.2.2 Motor unit recordings

Motor unit potentials were recorded intramuscularly using fine wire (California Fine Wire, Grover Beach, CA) bipolar electrodes. Briefly, bipolar electrodes were custom made with three 50-µm stainless steel fine wires and two electrodes were inserted into the medial gastrocnemius of both legs to a depth of 2cm (4 electrodes in total), with 5-7 cm separating the electrodes. Prior to beginning each trial, the electrode position was adjusted to ensure that at least one motor unit potential was identifiable from each medial gastrocnemius muscle (in some
trials more than one motor unit was identifiable per electrode). Once this was achieved, the electrodes were not moved again. Motor unit recordings were sampled at 20 kHz. Motor unit identification was performed in Spike2 (Cambridge Electronics, Cambridge, UK) with visual inspection to identify any instances of misclassification. Sections of data which contained misclassifications were eliminated from the analysis.

Motor unit firing rate was calculated during two epochs: (1) the dynamic response to the perturbation – mean of the first three interspike intervals (ISIs) directly following each load drop, and (2) maintenance of steady state between perturbations – mean ISI over a five second epoch taken at least five seconds after the load drop and one second prior to the next load drop. The mean and standard deviation of the ISIs during this five second epoch were calculated. Motor units that were active in the dynamic response to load drop and during maintenance of steady state at the same load level were analyzed to determine if there was any modulation of the motor unit firing rate in response to the perturbation.

3.2.3 Surface EMG recordings

Whereas intramuscular electrodes were used to record motor unit potentials, surface EMG electrodes (Delsys Trigno, Delsys Inc., Natick, MA, USA) were placed over the medial gastrocnemius (GM), soleus (SOL), and tibialis anterior (TA) muscles bilaterally. Surface EMG was sampled at 2000 Hz and saved for off-line analysis. Root Mean Square (RMS) amplitude of the surface EMG for each muscle was calculated for one second during the dynamic response to perturbation and for the same five second epoch that was used to measure motor unit ISI during steady state between perturbations. RMS amplitude was normalized to the baseline quiet standing measurement taken prior to the application of the first load.
3.2.4 Kinetic data

Kinetic data were collected using two floor-mounted force platforms (detailed above), sampled at 2000 Hz. APCOP displacement was calculated and converted to a percentage of the base of support for each foot as calculated from the kinematic markers of the heel (0%) and the base of the 1st metatarsal (100%). The velocity of the APCOP displacement was the derivative, calculated from the APCOP signal data points averaged over a time constant of 0.03s. The peak APCOP velocity and displacement values were calculated during the dynamic response to load drop. Percentage weight bearing through the paretic leg was calculated from the vertical component of the ground reaction force of the paretic limb divided by the total vertical ground reaction force from both platforms. External torque applied about each ankle was calculated as the product of the vertical component of the ground reaction force and its perpendicular distance of the APCOP from the ankle joint centre. The peak external torque about each ankle was identified during the dynamic response to load drop and normalized to the external torque calculated during quiet stance.

3.2.5 Kinematic data

Passive reflective markers were affixed to participants according to a modified Helen Hayes marker set to allow for motion capture of the arms, trunk and legs bilaterally (Kadaba et al. 1989). Eight high-speed digital cameras (Raptor-E, Motion Analysis Corp, Santa Rosa, CA, USA) sampled the movement of the reflective markers at 100 Hz. Kinematic data were analyzed using a custom-written program in MATLAB (The Mathworks Inc., Natick, MA, USA) that was used in a previous study to quantify kinematics during movement (Pollock et al. 2012). Body segment angles were calculated in the sagittal plane for the ankle, knee, and hip (Fig. 1). Joint angular velocities calculated from the joint angle data points averaged over a time constant of
0.03s were measured for each perturbation. Trunk rotation was calculated as rotation in the transverse plane of the segment created by bilateral shoulder markers (upper trunk) and the segment created by the anterior-superior iliac spine (ASIS) markers (lower trunk) with respect to the base of support created by lateral ankle markers of each ankle. Peak angular excursion and peak angular velocity of each joint and kinematic segment were measured during the dynamic response to perturbation. COM was estimated as the central point of the pelvic marker coordinates (ASIS markers anteriorly and L5/S1 junction posteriorly). COM velocity was calculated from the COM signal data points averaged over a time constant of 0.03s.

### 3.2.6 Statistical analysis

Statistical analysis was performed using SPSS (IBM SPSS Statistics for Windows, Armonk, NY, USA). ISI data were not normally distributed and some units were not active across all loads; therefore, Kruskal–Wallis ANOVA tests were performed on the median motor unit ISI duration to test for the effect of load level (1-5) on motor unit firing rate during the dynamic response and during the maintenance of steady state. Motor unit ISI during the dynamic response to load drop and maintenance of steady state were compared using Wilcoxon Signed Ranks tests. Motor unit ISI from paretic and non-paretic medial gastrocnemius muscles were compared for all loads combined using Mann-Whitney U tests.

Kinematic, kinetic and surface EMG data were also not normally distributed; however, data were complete across loads. Therefore, Friedman ANOVA tests were performed for the effect of load level (1% BM – 5% BM). Wilcoxon Signed Ranks tests were used with these data to compare paretic and non-paretic leg performance during postural reactions. All data are reported as median, interquartile range (IQR).
Relationships between motor unit ISI during the dynamic response to perturbation and kinematic performance variables were explored using Spearman correlations with all perturbations pooled regardless of load.

### 3.3 Results

Participant characteristics are outlined in Table 3.1. Participants were in the chronic stage post-stroke and had mild to moderate impairments in walking balance and motor control of the foot and ankle. The median BOS (heel to base of first metatarsal) across participants was 203.0 mm, IQR 197.0-211.5 mm for the paretic foot, and 198.0 mm, IQR 191.5-208.0 mm for the non-paretic foot. The APCOP excursion at LOS measured 185.2 mm, IQR 179.7-197.5 mm for the paretic foot and 199.2 mm, IQR 192.7-205.5 mm for the non-paretic foot. The APCOP excursion during the LOS testing was significantly further forward on the non-paretic foot (100.6%BOS, IQR 97.3-103.8%), compared to the paretic foot (91.4%BOS, IQR 88.5-97.3%, p<0.01). APCOP excursion of 100%BOS indicates that the COP was located at the marker placed at the base of the 1st metatarsal.

### Table 3.1 Participant characteristics

<table>
<thead>
<tr>
<th>Age</th>
<th>Gender</th>
<th>Time since stroke, mo</th>
<th>Paretic side</th>
<th>CB&amp;M</th>
<th>CMSA Foot</th>
</tr>
</thead>
<tbody>
<tr>
<td>68.1 ± 7.7</td>
<td>7M/2F</td>
<td>63.0 ± 44.1</td>
<td>4R/5L</td>
<td>30.0 ± 16.7</td>
<td>4.3 ± 1.3</td>
</tr>
</tbody>
</table>

Mean ±SD of participant characteristics and functional tests, including the Community Balance and Mobility Scale (CB&M) out of a maximum score of 96; Chedoke-McMaster Stroke Assessment (CMSA) out of a maximal score of 7.
3.3.1 The postural reaction during the dynamic response to perturbations

Postural reactions were not the same on the paretic and non-paretic sides (Fig. 3.2). That is, the peak APCOP displacement (expressed as change from initial position) progressed significantly more forward on the non-paretic side compared to the paretic side (Table 3.2, Fig 3.2A, p<0.01). The peak APCOP velocity was also significantly smaller on the paretic than the non-paretic leg (p<0.01), with only the non-paretic leg demonstrating a trend towards a significant effect of increased external load (Fig. 3.2B, p=0.06). The peak anterior-posterior COM excursion (expressed as change from initial position) showed no significant effect of load level (Table 3.3, p=0.2). The peak COM velocity was significantly different across loads (Fig. 3.2C, p=0.03). Specifically, the trajectories of both the APCOP velocity and the COM velocity were non-linear across loads (Fig. 3.2B, 3.2C), revealing that participants exhibited an initial increase followed by a decrease in velocity with larger maintained loads. This behaviour can be seen also in the representative figure with a decreased forward progression of the APCOP in both legs with the application of the last 2 loads (Fig. 3.3). The non-paretic ankle had to resist a sequential increase in peak external torque with increased external load levels (Fig 3.2D, p<0.01); although a similar trend was observed on the paretic side; the increase in peak ankle external torque was only a trend (p=0.09). It is important to note that any differences between legs were not simply the result of increased weight bearing through the non-paretic leg as the percentage weight-bearing was not affected by load (p>0.05) and was close to 50% across all perturbations (50%, IQR 45-58%).
Table 3.2 Anterior-posterior centre of pressure (APCOP) excursion (mm) by load level

<table>
<thead>
<tr>
<th>Load</th>
<th>Paretic</th>
<th>Non-Paretic</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2.2 (27.2- -14.8)</td>
<td>9.8 (19.1- -7.7)</td>
</tr>
<tr>
<td>2</td>
<td>7.7 (33.2- -11.1)</td>
<td>31.0 (36.4- 1.8)</td>
</tr>
<tr>
<td>3</td>
<td>14.9 (37.8- -9.5)</td>
<td>26.1 (35.1- 19.3)</td>
</tr>
<tr>
<td>4</td>
<td>18.5 (42.0- -1.1)</td>
<td>34.2 (52.4- 17.2)</td>
</tr>
<tr>
<td>5</td>
<td>28.9 (43.6- -10.8)</td>
<td>48.1 (63.9- 33.4)</td>
</tr>
</tbody>
</table>

Reported as median (interquartile range)

Table 3.3 Anterior-posterior centre of mass (COM) excursion (mm) by load level

<table>
<thead>
<tr>
<th>Load</th>
<th>COM excursion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>13.3 (19.8- 4.0)</td>
</tr>
<tr>
<td>2</td>
<td>15.3 (19.0- 4.4)</td>
</tr>
<tr>
<td>3</td>
<td>11.5 (24.5- 6.0)</td>
</tr>
<tr>
<td>4</td>
<td>13.9 (25.5- -0.9)</td>
</tr>
<tr>
<td>5</td>
<td>11.4 (29.3- -5.5)</td>
</tr>
</tbody>
</table>

Reported as median (interquartile range)
Figure 3.2 Response of the anterior-posterior centre of pressure (APCOP), centre of mass (COM), percentage weight-bearing on the paretic leg, and external ankle torque during the dynamic response to perturbation under increased level of challenge to standing balance.  

A) The peak APCOP displacement progressed significantly more forward on the non-paretic than the paretic side (*p<0.01) and reached 85%BOS on the non-paretic leg.  

B) The peak APCOP velocity was non-linear across loads and significantly smaller on the paretic than the non-paretic leg (*p<0.01), with a trend towards a significant effect of load on the non-paretic leg (‡p=0.06).  

C) Peak COM velocity demonstrated a significant effect of load (†p<0.05), with an initial increase followed by a decrease by the highest level of load. There was no effect of load on the percentage of weight-bearing on the paretic leg.  

D) The non-paretic ankle had to resist a roughly linear increase in peak external torque with increased load level (†p<0.05), whereas there was only a trend towards an increase on the paretic side (‡p=0.09).
Figure 3.3 Representative example of electromyography recordings from the paretic and non-paretic medial gastrocnemius muscle in standing during incremental increases in external load. From top row; force depicting application of perturbations (consecutive load drops), raw signal recorded from the anterior-posterior centre of pressure (APCOP, %BOS) of the paretic leg, raw signal of intramuscular electromyography recorded from the paretic medial gastrocnemius (GM MU), two individual MUs (MU1, 2) identified from the raw signal and displayed as instantaneous firing rate. Order is repeated for the non-paretic medial gastrocnemius muscle. There was no modulation of firing rate in MU1, whereas MU2 on the paretic side was a higher threshold motor unit that showed evidence of recruitment and derecruitment during the task.
3.3.2 Motor unit behaviour of medial gastrocnemius

In total, 36 motor units (26 - paretic muscle, 10 - non-paretic muscle) were identified (1-3 motor units from each leg). Because collection of motor units during a standing task proved challenging, priority was given to obtaining motor unit data from the paretic muscle. The motor unit firing rate during the dynamic response to load drop (mean of first three ISIs) did not change with increasing loads in the paretic (Fig. 3.4, p=0.73) or the non-paretic medial gastrocnemius muscle (p=0.37). There was no significant difference in the motor unit firing rate between the paretic and non-paretic medial gastrocnemius muscles during the dynamic response to load drop with all loads combined (paretic ISI 168 ms, IQR 141-207 ms; non-paretic 158 ms, IQR 115-225 ms, p=0.25). Despite a wide range of chronicity (26 months to 156 months, the time since stroke did not affect the motor unit results; as an example the participants who were 26 months and 156 months post-stroke both demonstrated the main finding of a lack of modulation of the firing rate in response to perturbations.

Despite the above noted increase in external torque at the ankle with increased loads, there was no effect of maintaining an increased load on the motor unit firing rate in the paretic (Fig. 3.4) or non-paretic medial gastrocnemius (p =0.91 and 0.96, respectively) during steady state between perturbations. Figure 3.3 depicts a representative example of the lack of modulation of firing rate in MU1 during steady state with increasing load levels. However, when combined across all load levels, the motor unit ISI of the paretic medial gastrocnemius muscle (167 ms, IQR 145-193 ms) was significantly longer than the non-paretic muscle (129 ms, IQR 116-173 ms, p<0.01) during steady state (Fig 3.4B).
Figure 3.4 Interspike interval (ISI) of the paretic medial gastrocnemius muscle with increasing load levels and ISI of the non-paretic medial gastrocnemius muscle collapsed across loads during; (A) dynamic response and (B) steady state maintenance of standing balance with increasing load levels. During steady state, the ISI of the non-paretic medial gastrocnemius was significantly shorter than the ISI of the paretic medial gastrocnemius collapsed across loads (p<0.01). There was no effect of load on the ISI of the paretic or the non-paretic medial gastrocnemius muscles during the dynamic response or steady state maintenance of standing balance.

3.3.3 Kinematic and surface EMG data

To investigate whether the lack of firing rate modulation in response to perturbation was because people after stroke did not use the ankle strategy but responded to the perturbation with a different postural control strategy, we examined the kinematics of the lower extremity and trunk movements. In the sagittal plane, only the position of the paretic hip demonstrated a significant increase in peak angular excursion with load (Fig. 3.5, p<0.01), whereas the knee and
ankle position remained relatively unchanged with load. There was also rotation of the upper and lower trunk during quiet stance prior to any perturbations, such that the both the upper and lower trunk were rotated more anteriorly on the non-paretic side with respect to the paretic side (upper trunk 4.07°, IQR 0.32-7.13°, lower trunk 2.99°, IQR 2.09-7.01°). This anterior trunk rotation on the non-paretic side was maintained throughout the perturbations with only the lower trunk rotation demonstrating a significant, albeit small, decrease with increasing levels of load (1.59°, IQR -1.28-8.86°). Angular velocity of the hip (paretic (P) 0.91°/s, IQR 0.35-1.75°/s, non-paretic (NP), 1.04°/s, IQR 0.37-2.04°/s), knee (P 0.61°/s, IQR 0.08-1.15°/s, NP 0.54°/s, IQR 0.19-1.31°/s) and ankle joints (P 0.33°/s, IQR 0.02-0.78°/s, NP 0.33°/s, IQR 0.03-0.84°/s) during the dynamic response was not different between legs (p>0.05), nor was there an effect of load level (p>0.05).

Surface EMG, measured during the maintenance of steady state between loads, showed a significant increase in the RMS amplitude in both the non-paretic and paretic medial gastrocnemius (Fig. 3.5, p<0.01) and tibialis anterior (p<0.05) muscles with increasing load levels. The paretic soleus muscle also showed a significant increase in the RMS amplitude (Fig. 3.5, p<0.01). RMS amplitude of paretic medial gastrocnemius was significantly higher during the dynamic response to perturbations than during steady state (p<0.01).
Figure 3.5 Kinematics and surface electromyography of the lower extremity with increased loads. The median and inter-quartile range are presented. A) Joint angle position changes from quiet standing. Ankle dorsiflexion (triangle), knee flexion (circle) and hip extension (diamond). The paretic hip demonstrated a significant effect of load on peak angular excursion in response to perturbations with initial flexion followed by extension in response to increased loads †p<0.05. B) Root mean square (RMS) amplitude of surface EMG normalized to pre-load drop standing for muscles of the ankle in response to an anterior pull on the pelvis. There was a significant increase in the RMS amplitude of the paretic and non-paretic medial gastrocnemius (GM) and tibialis anterior (TA) muscles. The paretic soleus muscle also showed a significant increase in the RMS amplitude †p<0.05.
3.3.4 Relationship of motor unit ISI to postural reactions

Although group data revealed no modulation of firing rate during the dynamic response to perturbation, we sought to examine whether the absolute firing rate during the dynamic response to perturbations was associated with the postural reaction. In this analysis, the data were pooled across load levels. The peak angular velocity of the ankle demonstrated a significant, yet small, negative correlation with the mean ISI of paretic medial gastrocnemius during the dynamic response to perturbations (rho=-0.26, p=0.03). The peak COM velocity (rho=-0.41, p<0.01) and the peak hip angular velocity (rho=-0.34, p<0.01) had moderate negative correlations with the mean ISI of paretic medial gastrocnemius, meaning that faster movements of the COM, hip and ankle were associated with shorter ISIs.

3.4 Discussion

In this study, people after stroke used primarily motor unit recruitment in the medial gastrocnemius muscle rather than increased firing rate to meet the challenge of anteriorly-directed perturbations. Furthermore, firing rate during the dynamic response to perturbation compared to maintenance of steady state was not significantly different, contrary to that found in healthy individuals (Pollock et al. 2014). This lack of modulation may be influenced by alterations in postural reaction strategies.

While participants demonstrated a forward progression of APCOP with repeated load application, and an initial increase in COM velocity and APCOP velocity with load application, by the fifth load, the velocity parameters had returned to that exhibited with the first load (see Fig 3.2), suggesting an attempt to control the velocity of movement created by the later perturbations. Limiting movement of the COM during external perturbation tasks has been demonstrated in healthy adults and was suggested to be due to anticipatory postural muscle
activation when external perturbations were expected but the exact timing of the perturbation was unknown (Brown and Frank 1997). We also found in the current study that there was an increase in ankle plantarflexor and dorsiflexor activation in both paretic and non-paretic legs during the maintenance of steady state at increasing levels of load, consistent with co-contraction about the ankle. Co-contraction was not typically seen in healthy adults in a similar task who demonstrated an increased activation of ankle plantarflexors and decreased activation of dorsiflexors with increased load level (Pollock et al. 2014). Finally, the larger APCOP velocity and greater increase in external ankle torque applied about the non-paretic ankle than the paretic ankle, suggests a greater capacity of the non-paretic leg to meet the demands of the task compared to the paretic leg, even when there is equal weight-bearing on the paretic leg.

Postural strategy employed during external perturbations represents central nervous system adaptations, which govern the motor response utilized to withstand a destabilizing stimulus and maintain standing balance (Carpenter et al. 2001; Horak et al. 1989). A postural aim to limit the COM movement velocity in response to perturbations may have decreased the postural reaction required of the medial gastrocnemius muscle; therefore, the modulation of motor units would not be necessary. Such a strategy is consistent with the relationship found between the motor unit firing rate of the paretic medial gastrocnemius muscle in response to each perturbation (regardless of load level) and the COM velocity. This suggests that although modulation in response to a perturbation was not identified with each load drop in the current study as has been demonstrated in healthy controls (Pollock et al. 2014), motor unit firing rate is, to some extent, associated with postural reaction strategies employed by people post-stroke. Motor unit ISI during the dynamic response to perturbation also showed a significant negative correlation with angular hip velocity, such that motor unit ISI was shorter when hip angular
Postural reactions of lower extremity muscles in response to perturbations have been shown to be triggered primarily by proprioceptive input from the hip and trunk movement (Bloem et al. 2002). These postural reactions are proposed to be long-loop reflexes which integrate afferent input regarding trunk movement at a supraspinal level and then direct the motor response via spinal networks of upper and lower extremity muscles (Bloem et al. 2002; Misiaszek 2006). People post-stroke have been shown to demonstrate impaired regulation of the long-loop reflex in the biceps brachii during perturbation tasks of the upper extremity (Trumbower et al. 2013). Impaired sensorimotor integration was also shown following stroke with altered hip reflex activity being related to impaired inter-joint coordination of muscle activation during gait (Finley et al. 2008; Hyngstrom et al. 2010). Altered integration of afferent input related to postural control and associated long-loop reflex responses have also been suggested to account for increased instability and falls during standing perturbation tasks experienced under additional sensory conflict in people post-stroke (Marigold et al. 2004b). However, the presence of a relationship between the velocity of movement of the hip and the COM with motor unit firing rate of the medial gastrocnemius muscle (albeit moderate) suggests that sensorimotor integration of long-loop postural reflexes associated with postural control may be somewhat intact.

All subjects in the current study had balance impairment, as demonstrated on the CB&M, so it is possible that the participants in this study had decreased confidence in their ability to maintain balance (Pang and Eng 2008). The reduced balance ability of the participants may have motivated them to use an asymmetrical strategy including the postural bias of rotation of the trunk anteriorly on the non-paretic leg side and the larger external ankle torque, APCOP displacement and velocity on the non-paretic leg. This asymmetrical reliance on the non-paretic
leg to accommodate for increasing challenge of postural reactions is in agreement with previous studies utilizing surface translation perturbations (Marigold et al. 2004a; van Asseldonk et al. 2006) and is different from simply shifting more weight onto the non-paretic side and may be reflective of a change in central set or an anticipatory postural strategy when a perturbation is expected (Brown and Frank 1997). Central set with respect to postural control represents the preparation aspect of a postural strategy to an upcoming balance disturbance which is scaled to an individual's perception of their ability to withstand a perturbation (Brown and Frank 1997). Although the non-paretic limb may be physiologically affected at the level of the muscle by stroke as well (Frontera et al. 1997), a change in central set to limit the body's movement in response to the external perturbations, would explain the lack of motor unit firing rate modulation also noted in the non-paretic medial gastrocnemius muscle during the dynamic response to perturbation. Also consistent with a change in central set, both the paretic and non-paretic legs showed increased tibialis anterior activation associated with increased levels of load, a pattern not noted in healthy participants in a comparable task (Pollock et al. 2014), but increased activation of the tibialis anterior was associated with a postural strategy which aimed to limit movement during challenges to balance (Carpenter et al. 2001).

Our results support the current understanding of motoneurone firing behaviour following stroke but extend these findings to that of a postural task. That is, the significantly longer ISI of the paretic medial gastrocnemius muscle compared to non-paretic muscle during steady state is in agreement with previous research which has demonstrated decreased firing rate of paretic muscles following stroke (Chou et al. 2013; Frontera et al. 1997; Gemperline et al. 1995; Mottram et al. 2014). The mean ISI of the non-paretic medial gastrocnemius muscle during steady state was similar to that reported for the medial gastrocnemius muscles of healthy
individuals (130 ms, IQR 110-140 ms) during a similar postural task which also demonstrated no effect of maintaining an increasing anterior progression of the APCOP and load level on motor unit firing rate (Pollock et al. 2014). These findings, combined with the increased surface EMG recordings, suggest that motor unit recruitment was used to meet the increased torque demands of the standing task in both paretic and non-paretic medial gastrocnemius muscles.

3.5 Conclusion

The current study adds to the understanding of the motor control of postural reactions following stroke. Both the paretic and non-paretic medial gastrocnemius muscles appear to demonstrate a lack of modulation of motor unit firing rate in response to perturbations, therefore relying on recruitment. Further analysis in the paretic medial gastrocnemius revealed that there may be an important relationship between motor unit firing rate and the velocity of the COM and hip movement during performance of the postural reaction. This suggests that underlying long-loop postural reflexes in people post-stroke may be somewhat intact. The change in postural central set adopted by people post-stroke during this task may limit the need for modulation of motor unit firing rate in response to the abrupt external perturbations.
Chapter 4: Motor control of standing postural sway under anteriorly-directed challenges to standing balance in people post-stroke

4.1 Introduction

Decreased balance following stroke is associated with decreased independent mobility and an increased risk of falls, which could lead to injury and further disability (Garland et al. 2009; Geurts et al. 2005). Early research aimed at improvement in balance following stroke focused on the presence of asymmetrical weight-bearing, and restoring symmetrical weight-bearing to improve standing balance. However, outcomes using this approach were weakly linked to functional improvement (Geurts et al. 2005). This is likely due to the static nature of weight-bearing as opposed to the active nature of postural control. More recently, greater emphasis has been placed on understanding impaired active postural control following stroke that has been shown to be asymmetrical favoring the non-paretic leg to maintain balance (de Haart et al. 2004; Marigold and Eng 2006b; Roerdink et al. 2009; van Asseldonk et al. 2006). Importantly, standing balance following stroke is suggested to be more strongly related to decreased control about the ankle than the hip (Dickstein and Abulaffio 2000; Hocherman et al. 1988). Following stroke, ankle plantarflexor muscle weakness is common across the spectrum of severity (Fimland et al. 2011). However, the paretic gastrocnemius muscles of people post-stroke demonstrate greater proportions of muscle atrophy than the soleus muscle (Ramsay et al. 2011). Therefore, understanding of active postural control about the ankle post-stroke requires the investigation of each of the plantarflexor muscles in the context of postural sway.

During quiet stance, maintaining the center of mass (COM) of the body within the base of support (BOS) provided by the feet is largely achieved by modulation of muscle activity about
the ankle (Di Giulio et al. 2009; Horak and Nashner 1986; Winter et al. 2003). Using cross-correlation analysis of the electromyography (EMG) signals of the ankle plantarflexor muscles and force platform recordings of postural sway, it has been shown that modulation of the ankle plantarflexor muscle activity is moderately correlated with, and largely precedes, postural sway during quiet standing in healthy controls (Gatev et al. 1999; Masani et al. 2003; Masani et al. 2011). Sensorimotor impairment and asymmetrical between leg strategies of postural control post-stroke may influence this relationship between plantarflexor muscle activity and postural sway.

Interestingly, despite the asymmetry of postural control following stroke, during maintenance of standing balance under external loading, there remains a moderate level of common drive to the medial gastrocnemius motor units bilaterally in people post-stroke, suggesting at least partial preservation of a common command between paretic and non-paretic plantarflexor muscles during standing (Garland et al. 2014, in press). However, how the plantarflexor muscles of the paretic and non-paretic legs of people post-stroke each modulate with postural sway to control standing balance is not known. Examining this aspect of motor control between the legs of people post-stroke will provide further insight into the asymmetrical postural control strategy of people post-stroke. Use of high-density electromyography (EMG) will provide sampling from a broad area of each muscle reflecting the global activity of each muscle. The purpose of this study was to examine the modulation of the three ankle plantarflexor muscles (medial and lateral gastrocnemius and soleus) with postural sway in people post-stroke and age-matched controls, in response to an increasing anteriorly-directed challenge to standing balance.
As the non-paretic leg of people post-stroke has been shown to more actively control postural sway during standing than the paretic leg (Roerdink et al. 2009; van Asseldonk et al. 2006), we hypothesized that the ankle plantarflexors on the non-paretic side of people post-stroke would demonstrate a stronger relationship of modulation of plantarflexor activity with postural sway than the paretic plantarflexors muscles. We further anticipated that the relationship of modulation of each plantarflexor muscle activity and postural sway would be lower in the gastrocnemius muscles compared to the soleus muscles in the paretic leg of people post-stroke. Finally, we hypothesized that the relationship of co-modulation of paretic plantarflexor muscle activity with postural sway would be stronger with increased anteriorly-directed challenge to standing balance.

4.2 Methods

Ten people with chronic stroke (> 3 months post-stroke) and ten age-matched controls participated. Individuals post-stroke were included if they were ambulatory with or without a walking aid and could stand independently for a minimum of five minutes. Individuals were excluded if they had any health conditions that negatively impacted mobility (e.g. severe osteoarthritis). Controls were included if they were free from neurological or musculoskeletal impairment which resulted in mobility restrictions and/or balance deficits. The study conformed to the standards set by the latest revision of the Declaration of Helsinki and was approved by the University of British Columbia Clinical Research Ethics Board.

Participants post-stroke were assessed for motor recovery of the foot and ankle using the Chedoke McMaster Stroke Assessment Scale (CMSA, 0-flaccid to 7-normal, (Gowland et al. 1993) and both participants post-stroke and controls were assessed for ambulatory balance with the Community Balance and Mobility Scale (CB&M, /96, (Howe et al. 2006; Knorr et al. 2010).
4.2.1 Experimental protocol

Participants stood with their feet shoulder-width apart, with each foot on a separate force platform (AMTI OR6-6, Advanced Mechanical Technology, Watertown, MA).

A postural control challenge paradigm was employed which has been shown to increase external torque applied to the ankle joint (Pollock et al. 2014). A belt was secured around the pelvis of each participant and was attached to a horizontal cable in front of the participant. External loads were applied via a cable-pulley system attached to the front of the belt (Fig. 4.1). Participants remained standing in comfortable stance and external loads were applied through the belt by incremental loads of one percent body mass (1%BM) being dropped into a basket from 40 cm above the basket every 25-40 seconds (random timing) until five percent body mass (5%BM) was maintained. A screen in front of the participants prevented them from seeing when the loads were dropped. Application of the load was detected (deflection from baseline) by a force transducer in-line with the cable (Fig. 4.1).
Figure 4.1 Experimental set-up. Participants stood with each foot on a separate force platform. Anteriorly-directed external loads were dropped by a cable and pulley system attached to a belt around the participants' pelvis. Incremental loads of 1% body mass were applied every 25-30 s until a total of 5% body mass was reached.

4.2.2 Kinetic and kinematic data

Kinetic data were collected using two floor-mounted force platforms (detailed above). Anterior-posterior center of pressure (APCOP) displacement and the vertical ground reaction force were measured for each foot. Percentage weight-bearing through the paretic leg of participants post-stroke and the right leg of controls was calculated from the vertical ground component of the ground reaction force of the limb divided by the total vertical ground reaction force of both limbs of the participant. APCOP velocity was calculated as the derivative of the APCOP displacement signal.
Passive reflective markers were affixed to participants to capture the anterior and posterior limits of the foot and the ankle joint centre. Markers were affixed to the heel and at the head of the first metatarsal, and on the lateral malleoli bilaterally. Eight high-speed digital cameras (Raptor-E, Motion Analysis Corp, Santa Rosa, CA) sampled the movement of the reflective markers at 100 Hz. Kinematic data were analyzed using a custom-written program (Mathworks Inc., Natick, MA, USA). To compare the excursion of COP among participants, APCOP displacement was converted to a percentage of the length of the foot or base of support (BOS) within each foot; BOS was calculated from the kinematic markers of the heel (0%) and the head of the first metatarsal (100%). External torque about the ankles was calculated bilaterally as the product of the perpendicular distance of the APCOP position from the ankle joint center (estimated as in line with the lateral ankle marker) and the vertical component of the ground reaction force.

4.2.3 Electromyography

High-density surface electromyography (HDsEMG) was collected from the soleus (24 electrode grid, 2 cm interelectrode distance), medial and lateral gastrocnemius (20 electrode grids each, 1.5 cm interelectrode distance) bilaterally (OT Bioelectronica, Turin, Italy), sampled at 2048Hz. HDsEMG signals were analyzed in bipolar configurations resulting in 18 EMG signals from the soleus (SOL) muscle and 16 from each of the medial (MG) and lateral gastrocnemius (LG) muscles. EMG signals were bandpass filtered (20-400Hz) and a notch filter at 60Hz was applied. RMS EMG amplitude was calculated for each EMG signal from each muscle and normalized to the RMS EMG amplitude measured during quiet standing measured at baseline.
4.2.4 Outcome measures

Parameters were measured for 15 second epochs while participants maintained each load level to capture the EMG specific to controlling postural sway. This epoch was chosen to ensure the centre of pressure behaviour was more reflective of postural control under sustained anteriorly-directed challenge to standing balance rather than postural control in response to abrupt external forces acting on the body. Each epoch was measured beginning approximately 5 seconds post-application of each load to ensure the initial response to load application was not included in the measures. Four parameters were the main dependent measures calculated for each leg: APCOP displacement and velocity, external ankle torque and RMS EMG amplitude. The APCOP displacement signals were visually inspected and epochs were selected that did not include any intentional shift of position beyond sway (sometimes participants post-stroke would adjust their body position posteriorly as the postural challenge increased). The mean APCOP displacement as a percentage of the BOS and the percentage weight-bearing on the paretic leg of participants post-stroke and the right leg of controls were calculated during each epoch to determine the absolute displacement and symmetry of stance at each load level. The standard deviation of the APCOP (APCOP SD) displacement (cm) and the APCOP SD-velocity (cm/s) were calculated to explore the variability of postural sway of each leg at each load level. External ankle torque and RMS EMG amplitude of the medial and lateral gastrocnemius and soleus muscles were also calculated during each epoch to quantify the change in external ankle torque and change in muscle activation levels from quiet stance at each load level.

4.2.5 Cross-correlation

The EMG signals were full-wave rectified. Both EMGs and APCOP data were low-pass filtered using a fourth-order, zero-phase-lag Butterworth filter with 4Hz cutoff (Masani et al.
2003). Using a custom-written program (Mathworks Inc., Natick, MA, USA), cross-correlation was applied to each detrended EMG signal from each muscle (16 for the medial and lateral gastrocnemius muscles, 18 for the soleus muscle) with the APCOP displacement in a moving 1 second window across the 15 second epoch at each load level. Peak correlation coefficients within the epoch at each load level, and the corresponding timing of the peak cross correlation function were calculated for each signal within each grid.

Correlation functions with peaks that fell outside of a range of -50ms to -500 ms were excluded from calculation of the median correlation coefficient of each grid as peaks outside of this range would be approximately twice that previously established in healthy participants (Gatev et al. 1999; Masani et al. 2003; Masani et al. 2011).

4.2.6 Statistical analysis

Mixed methods two way ANOVAs with post-hoc paired comparisons with Bonferroni adjustment were employed in each group separately (participants post-stroke and controls) to explore the effect of load as a repeated measure (1-5%BM) and leg (paretic and non-paretic of participants post-stroke; right and left legs of controls) for the following parameters: APCOP displacement (% of BOS), APCOP SD-displacement, APCOP SD-velocity, external ankle torque, and percentage weight-bearing (paretic leg and control right leg for this parameter). This analysis explored the kinetic and kinematic response to the perturbation paradigm in each group. To examine the main outcome of interest, the peak value of the EMG:APCOP correlation coefficients were transformed using a Z-transformation. Separate mixed methods two way ANOVAs with post-hoc paired comparisons with Bonferroni adjustment were performed in each group (stroke and controls) and each muscle (medial, lateral gastrocnemius and soleus muscles) to explore effect of load as a repeated measure (1-5%BM) and between leg (paretic and non-
paretic of participants post-stroke; right and left legs of controls) differences in the correlation Z-scores, timing of the peak correlation and RMS EMG amplitude. One-way ANOVAs with Tukey post-hoc tests were performed in each leg (right, left, paretic, non-paretic) to explore between muscle (medial, lateral gastrocnemius and soleus muscles) differences in the correlation Z-scores.

A complementary partial correlation analysis, controlling for load, was performed separately in the paretic and non-paretic muscles to explore the relationships between the correlation Z-scores and the RMS EMG amplitude, external ankle torque and APCOP SD-displacement and velocity. This analysis serves to explore if changes in the level of muscle RMS EMG amplitude, external ankle torque and the variability of APCOP displacement and velocity during postural sway is related to change in the level of EMG:COP correlation.

Level of significance was set at \( p = 0.05 \). Data are presented as mean and standard deviation unless otherwise noted. Peak correlation coefficients, rather than Z-scores, are presented in the results and discussion to assist the reader’s interpretation of results.

4.3 Results

4.3.1 Participants

Table 1 shows the characteristics of participants post-stroke and controls. Age was not significantly different between groups \( (p = 0.65) \). Participants post-stroke scored significantly lower than controls in ambulatory balance as measured by the CB&M \( (p < 0.01) \).
Table 4.1 Participant characteristics

<table>
<thead>
<tr>
<th></th>
<th>Age (yrs)</th>
<th>Sex (m/f)</th>
<th>Post onset (yrs)</th>
<th>Paretic side (R/L)</th>
<th>CMSA * (0-7)</th>
<th>CB&amp;M (0-96)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke</td>
<td>66.2 ± 9.2</td>
<td>8m /2f</td>
<td>6.6 ± 3.6</td>
<td>5 R / 5L</td>
<td>3 (IQR 3-6)</td>
<td>31.9 ± 23.8</td>
</tr>
<tr>
<td>Control</td>
<td>68.0 ± 8.2</td>
<td>7m / 3f</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>80.9 ± 7.8</td>
</tr>
</tbody>
</table>

*CMSA reported as median and interquartile range.

4.3.2 Kinematic and kinetic parameters of postural sway

The mean APCOP displacement as a percentage of BOS and external ankle torque are shown in Figure 4.2A and 4.2B, respectively. The APCOP displacement, expressed as a percentage of the BOS, showed a significant anterior progression with the addition of loads (p < 0.01) with no significant difference between legs of participants post-stroke (p = 0.87) or controls (p = 0.92). There was a significant effect of load on the change in external ankle torque from quiet stance for each leg of participants post-stroke and controls (p < 0.01). The external ankle torque on the non-paretic side tended to be higher than on the paretic side (p = 0.10), whereas, there was no significant difference between the legs of controls (p = 0.58). In summary, APCOP displacement progressed significantly forward in each foot and external ankle torque increased in each ankle with a tendency towards higher external ankle torque in the non-paretic legs of participants post-stroke.

In participants post-stroke, there were trends towards an effect of load on postural sway parameters of APCOP SD-displacement (Fig. 4.2C, p = 0.13) and APCOP SD-velocity (Fig. 4.2D, p = 0.14) on the paretic leg. The non-paretic leg also tended to demonstrate greater APCOP SD-displacement (p = 0.07) and significantly greater APCOP SD-velocity (p = 0.05) than the paretic leg, suggesting greater variability of postural sway parameters in the non-paretic
leg than the paretic leg. In controls, there was no significant effect of load on APCOP SD-displacement (Fig. 4.2C, \( p = 0.22 \)) or APCOP SD-velocity (Fig. 4.2C, \( p = 0.80 \)). There was also no difference between the right and left legs of controls in APCOP SD-displacement (\( p = 0.55 \)) or APCOP SD-velocity (\( p = 0.51 \)).

There was no significant effect of load on percentage weight-bearing on the right leg of controls or the paretic leg of participants post-stroke (\( p = 0.20 \)). There was also no significant difference between groups (stroke and controls, \( p=0.32 \)) for the percentage of weight-bearing when expressed as the grand mean across loads; paretic leg was 47.56 ± 6.95% and control right leg was 50.33 ± 5.17%.
Figure 4.2 Kinematic and kinetic parameters of postural sway. Mean and SE of parameters for participants post-stroke; paretic leg (white bars) and non-paretic leg (black bars), and controls; left leg (light grey) and right leg (dark grey).  

A) APCOP progression in BOS (%BOS - 0% represents the heel and 100% represents the marker placed at the base of the metatarsal head of the great toe). Both the legs of participants post-stroke and controls demonstrate significant increase forward progression of the APCOP in the BOS with increase loads. There was no significant difference between the legs of participants post-stroke or controls.  

B) Change in external ankle torque from quiet stance (Nm). Both the legs of participants post-stroke and controls demonstrate significant increase in external ankle torque with increase loads. The non-paretic leg tended to demonstrate higher levels of change in external ankle torque than the paretic leg in participants post-stroke.  

C) Mean APCOP SD-displacement (cm).  

D) Mean APCOP SD-velocity (cm/s). There was a trend towards effect of load in variance of APCOP displacement and velocity of postural sway in participants post-stroke, this effect was somewhat non-linear, however, suggests an increase in variance with load. There also tended to be higher variance of postural sway displacement and velocity in the non-paretic leg than paretic leg. There was no effect of load or between leg differences in controls. Taken together, kinematic and kinetic parameters of postural sway suggest an asymmetrical control of standing balance.
4.3.3 RMS amplitude of plantar flexor muscles

The increase of RMS EMG amplitude of each muscle is shown in Figure 4.3. Each plantarflexor muscle of participants post-stroke and controls showed a significant effect of load on RMS EMG amplitude (p < 0.01). There was no significant between leg differences in the ankle plantarflexor muscles (p>0.05), with the exception of a trend in the lateral gastrocnemius muscles in participants post-stroke towards greater RMS EMG amplitude in the non-paretic leg than the paretic leg (p=0.09).
Figure 4.3 RMS EMG amplitude in response to maintenance of increasing load, normalized to quiet stance, depicted as mean and SE. In participants post-stroke (open symbols) and controls (black symbols), the medial (MG) and lateral (LG) gastrocnemius and soleus (SOL) muscles demonstrated a significant increase with load ($p < 0.01$). Only the lateral gastrocnemius (LG) muscle of participants post-stroke demonstrated a between-group difference, with a tendency for the non-paretic (open triangles) to increase activation more than the paretic (open circles) ($\dagger p = 0.09$).
4.3.4 Peak correlation coefficients between modulation of muscle activity and APCOP displacement

For the HDsEMG grids on control and non-paretic muscles, an average of only 1 ± 1 signal displayed timing of the peak correlation coefficient which fell outside the range of -50 to -500ms and was removed prior to calculation of the median correlation coefficient for each muscle for each participant. For the HDsEMG grids on the paretic muscles, 3 ± 4 signals were outside of this range and were removed.

A representative figure of the cross-correlations is shown in Figure 4.4. Moderate positive correlations were found between EMG activity of the medial and lateral gastrocnemius muscles and APCOP displacement and weak to moderate positive correlations were found in soleus muscles across load levels in both participants post-stroke and controls. There were no significant differences between legs and no effect of load in the ankle plantarflexor muscles of controls (Figure 4.5, p > 0.05).

In participants post-stroke, medial gastrocnemius muscle EMG:APCOP correlation coefficients showed a significant main effect of leg (p = 0.03), but not of load (p = 0.71) with a trend towards an interaction between leg x load (p=0.06); that is, there was a stronger relationship on the non-paretic than the paretic side at load levels 1-3%BM (Fig. 4.5, p < 0.05). Lateral gastrocnemius muscle EMG:APCOP correlation coefficients showed a significant main effect of leg (p = 0.05) and of load (p = 0.05) and a significant interaction between leg group x load (p=0.05), such that the correlation was stronger on the non-paretic side than the paretic side at load levels 1-2%BM (Fig. 4.5, p < 0.05). The soleus muscle showed a trend towards an effect of leg (p = 0.14) and no effect of load (p = 0.26) or interaction between leg group and load.
(p=0.27), with the non-paretic correlation tending to be stronger than the paretic at load levels 1-2%BM (Fig. 4.5, p = 0.06).

EMG:APCOP correlation coefficients collapsed across loads revealed a significant effect of muscle in controls (p < 0.05), with stronger correlations in the medial gastrocnemius than the lateral gastrocnemius and soleus muscles in the left (p < 0.01) and right leg (p < 0.10). In participants post-stroke, EMG:APCOP correlation coefficients collapsed across loads also showed a significant effect of muscle in the non-paretic leg (p < 0.01). In the non-paretic leg, correlations were stronger in both the medial and lateral gastrocnemius muscles than the soleus muscle (p < 0.01). In the paretic leg of participants post-stroke, there was only a trend towards an effect of muscle on EMG:APCOP correlation coefficients (p = 0.13), such that the correlation was stronger in the paretic lateral gastrocnemius than the soleus muscle (p = 0.13).
Figure 4.4  Representative example of cross-correlation calculation function between modulation of paretic medial gastrocnemius muscle activity (MG EMG) and APCOP displacement (anterior-posterior postural sway) in a participant post-stroke. Tracings from top: Force transducer signal showing application of perturbation (Load), position of anterior posterior center of pressure (AP COP), raw EMG trace from paretic medial gastrocnemius muscles. Inset boxes (bottom) show signal variance for one channel of paretic MG EMG and APCOP displacement while maintaining 1%BM (left) and 5%BM (right). Cross-correlation functions between EMG and APCOP signals (top of the inset) show higher peak values at 5%BM than 1%BM.
Figure 4.5  Relationship between modulation of each ankle plantarflexor muscle activity (EMG) and APCOP displacement (anterior-posterior postural sway). Mean and SE of the coefficients of the peak EMG:APCOP displacement correlation function taken during the maintenance of increasing levels of anteriorly-directed loads in standing in participants post-stroke (open symbols) and controls (black symbols). While there was little difference across loads or between legs in control participants, the non-paretic ankle plantarflexor muscles (open triangles) demonstrated stronger EMG:APCOP displacement relationships than paretic ankle plantarflexors (open circles) at lower load levels († p < 0.05, ‡ p = 0.06).
4.3.5  Timing of the peak EMG:APCOP displacement correlation

There was no significant difference between the legs or a significant effect of load on the timing of the peak EMG:APCOP displacement correlations in participants post-stroke or controls (Fig. 4.6, p > 0.05).

Figure 4.6  Timing of the peak correlation between muscle activation and postural sway. Grand mean and SE (collapsed across loads) for participants post-stroke; paretic leg (white bars) and non-paretic leg (black bars), and controls; left leg (light grey) and right leg (dark grey). There was also no significant difference in the timing of the peak correlation in participants post-stroke or controls in any ankle plantarflexor muscle.

4.3.6  Association of performance parameters with EMG:APCOP Correlation

In the plantarflexor muscles of participants post-stroke, the narrowing of the difference between paretic and non-paretic peak EMG:APCOP displacement correlation coefficients cannot be entirely explained by an effect of load. Parameters of postural sway, external ankle torque, RMS EMG amplitude and the correlation Z-scores were explored with partial correlations,
controlling for load (Table 4.2). The significant moderate positive correlations between the RMS EMG amplitude and the Z-scores in both paretic gastrocnemii muscles suggest that as muscle activation levels increase in these muscles, so does the modulation of muscle activity with APCOP displacement. Conversely, there was a weak negative correlation between RMS EMG amplitude and Z-scores in the paretic soleus muscle. There was a significant moderate relationship between Z-scores and mean RMS EMG amplitude in the non-paretic soleus muscle, and contrary to the paretic side, no relationship between Z-scores and RMS EMG amplitude in the non-paretic gastrocnemii muscles. The Z-scores of all non-paretic plantarflexor muscles, but not paretic muscles, demonstrated a significant weak to moderate negative relationship with external ankle torque, suggesting that the reduction in the EMG:APCOP displacement correlation on the non-paretic side is influenced by the increased external ankle torque but the increase in external ankle torque on the paretic side was not influencing the increase in the EMG:APCOP displacement correlation. Parameters of postural sway (APCOP SD-displacement and APCOP SD-velocity) both demonstrated significant positive relationships with paretic and non-paretic plantarflexor muscles, suggesting that the increase in the variance of sway was associated with increased strength of EMG modulation with postural sway.
Table 4.2  Partial correlation controlling for load, between the Z scores for the peak EMG:APCOP correlation coefficients derived from between the paretic and non-paretic medial (MG), lateral gastrocnemius (LG) and soleus (SOL) muscles and APCOP displacement, and external ankle torque, mean RMS EMG amplitude, standard deviation of the APCOP (SD-APCOP) displacement and velocity during postural sway.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>External ankle torque</th>
<th>RMS EMG amplitude</th>
<th>APCOP SD-displacement</th>
<th>SD-APCOP velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paretic MG</td>
<td>-0.08</td>
<td>0.38 *</td>
<td>0.69 *</td>
<td>0.68 *</td>
</tr>
<tr>
<td>Paretic LG</td>
<td>-0.09</td>
<td>0.51 *</td>
<td>0.63 *</td>
<td>0.42 *</td>
</tr>
<tr>
<td>Paretic SOL</td>
<td>-0.17</td>
<td>-0.28 ***</td>
<td>0.71 *</td>
<td>0.64 *</td>
</tr>
<tr>
<td>Non-paretic MG</td>
<td>-0.38 *</td>
<td>-0.17</td>
<td>0.42 *</td>
<td>0.50 *</td>
</tr>
<tr>
<td>Non-paretic LG</td>
<td>-0.27 ***</td>
<td>-0.15</td>
<td>0.53 *</td>
<td>0.53 *</td>
</tr>
<tr>
<td>Non-paretic SOL</td>
<td>-0.31 **</td>
<td>-0.48 *</td>
<td>0.45 *</td>
<td>0.53 *</td>
</tr>
</tbody>
</table>

* P ≤ 0.01, ** p ≤ 0.05, *** p ≤ 0.1

4.4 Discussion

During anteriorly-directed progressive challenges to maintaining standing balance, the plantarflexor muscle activity of participants post-stroke and age-matched controls was positively correlated with postural sway. There was no significant difference in the timing in which the modulation of each muscle preceded postural sway in participants post-stroke and controls. In participants post-stroke, the relationships of the ankle plantarflexor muscles with postural sway were significantly stronger in the non-paretic leg than the paretic leg at the lower two load levels which was lost at the higher (3-5%BM) load levels. The difference between EMG:APCOP displacement correlation on the non-paretic and paretic sides suggests that, initially, there was an asymmetrical motor control strategy favoring the non-paretic leg but with increased levels of postural challenge this asymmetry appears to be reduced.

The peak correlation coefficients as a measure of the relationship between the modulations of ankle plantarflexor muscle activity with postural sway in the current study are in
agreement with correlation coefficients during quiet stance in healthy subjects (Gatev et al. 1999; Masani et al. 2003). The active control of the ankle plantarflexors, as first described by Gatev et al. (1999), suggests that the CNS maintains standing balance in a feed-forward manner, using the constant input of afferent information to anticipate the position of the COM in the next instance and actively controls this position with activation of the ankle plantarflexor muscles to maintain balance (Gatev et al. 1999; Masani et al. 2003).

The timing between the EMG and postural sway signals is composed of the time associated with afferent feedback and motor command, and therefore, is suggested to be representative of the central integrative command (Gatev et al. 1999; Masani et al. 2011). It is important to note that 3±4 EMG grid signals were removed from calculation of the median correlation coefficient of each paretic muscle, as the timing of correlation between these EMG signals and postural sway fell outside of the feed-forward range previously established in healthy controls (Gatev et al. 1999; Masani et al. 2003). Therefore, this suggests a decreased within-muscle homogeneity of this aspect of motor control in paretic plantarflexor muscles. However, the lack of difference in the timing of the EMG:APCOP displacement peak correlation in participants post-stroke and age-matched controls suggests that this feed-forward mechanism of postural control is somewhat maintained in participants post-stroke. Masani et al. (2011) investigated the timing of the peak correlation between soleus EMG activity and postural sway in young and older adults and found no effect of age. Our findings suggest that the timing between the EMG activity and the resultant postural sway is also similar in people post-stroke. That is, a time delay associated with central integrative command does not appear to explain the destabilization of postural control noted in these participants with chronic stroke.
The “lateralization” of postural control favoring the non-paretic leg following stroke has been established by comparison of measures of postural sway measured separately under each foot (Roerdink et al. 2009; van Asseldonk et al. 2006). Roerdink et al. (2009) proposed that this asymmetrical postural control strategy was a result of motor impairment of the distal muscles of the leg following stroke. The tendency for larger variance of postural sway displacement and velocity in the current study on the non-paretic side suggests compensatory control provided by the non-paretic leg. Our examination of the variance of postural sway together with the nature of the relationship between modulation of muscle activity and postural sway showed that greater variability of the APCOP SD-displacement and velocity was associated with a stronger EMG:APCOP displacement relationship (Table 4.2).

Interestingly, the increased variance of postural sway of the non-paretic leg is suggestive of the exploratory nature of postural sway (Carpenter et al. 2010; Gatev et al. 1999). As postural sway has been found to include feed-forward control mechanisms, it has been proposed that postural sway may serve to continually gather afferent input regarding position of the body relative to the BOS, allowing for anticipation of the body's position in the next movement instance and motor commands to maintain balance (Carpenter et al. 2010; Gatev et al. 1999). Our results suggest that challenge to standing balance may somewhat lead to a more active role of the paretic leg in the exploratory aspect of postural sway as the EMG:APCOP displacement correlations increase on loads 4-5%BM.

Larger RMS EMG amplitude of the paretic medial and lateral gastrocnemius muscles was associated with increased modulation of EMG activity of the muscles with postural sway. Conversely, the non-paretic ankle plantarflexor muscles showed negative relationships with external ankle torque. This relationship between the non-paretic plantarflexors and external
ankle torque is counterintuitive yet fit the nature of the change reflected in the plots of strength of modulation of the non-paretic plantarflexors with postural sway (Fig. 4.5). Although speculative, taken together, these relationships suggests that the challenge of this task may involve an inter-limb motor control strategy such that the paretic plantarflexor muscles must increasingly participate in the active control of standing balance, thereby requiring less from the non-paretic.

Comparison of strength of EMG:APCOP correlation coefficients between muscles within each leg is suggestive of stronger relationship of postural sway with gastrocnemius muscles than soleus muscles in non-paretic and control legs. This was less evident in the paretic leg of participants post-stroke. Furthermore, the paretic gastrocnemius muscles of participants post-stroke demonstrated stronger relationships with postural sway as RMS EMG amplitude increased, whereas, the strength of the relationship of the paretic soleus muscle with postural sway was not positively related to increase in activation. The roles of the plantarflexor muscles are differentiated during postural sway in quiet stance (Loram et al. 2011; Vieira et al. 2012). The soleus muscle demonstrates tonic activation, whereas the gastrocnemius muscle tends to demonstrate more phasic activity in response to anterior displacement of the COP with respect to the ankle joint. The human gastrocnemius muscle has been described as being composed of approximately 50% fast-twitch fibers, whereas the soleus muscle is composed of 70-100% slow-twitch fibers (Johnson et al. 1973). Paresis following stroke has been shown to lead to greater atrophy in the gastrocnemius muscles than the soleus muscle (Ramsay et al. 2011). These patterns of atrophy may be related to the greater proportion of type II motor unit loss reported to occur in paretic muscle following stroke (Lukacs et al. 2008). The current findings may suggest
that the remodeling of paretic muscle that occurs post-stroke impacts the gastrocnemius muscle more than the soleus during challenging postural control tasks.

Rehabilitation incorporating postures which challenge balance in patients post-stroke by maintaining postures with increased anteriorly-directed challenges to balance have reported improved bilateral control of postural sway (Lin et al. 2007; McCombe and Prettyman 2012) and therefore, may be useful to incorporate into rehabilitation of standing balance. It is important to note that the asymmetry of postural control favoring the non-paretic leg post stroke has been shown to be less evident amongst people with mild motor impairment (Marigold and Eng 2006b; Roerdink et al. 2009). This study did not include sufficient number of participants across the range of sensorimotor impairment following stroke to explore this aspect of motor control of standing balance. Nevertheless, this study suggests that future research into rehabilitation strategies for standing balance post-stroke to determine how the paretic leg may be best challenged is warranted.

4.5 Conclusion

The results of this study suggest that, similar to age-matched controls, moderate relationships exist between the paretic and non-paretic ankle plantarflexor muscles and postural sway in participants post-stroke. The timing of the EMG activity in the ankle plantarflexor muscles preceding postural sway appears to be reasonably maintained post-stroke. At the lower levels of challenge to standing balance in this paradigm, the non-paretic ankle plantarflexor muscles demonstrate stronger relationships with postural sway than paretic ankle plantarflexor muscles, indicative of asymmetrical motor control strategies. However, an increased level of challenge to standing balance results in a reduction in this asymmetry in participants post-stroke,
suggesting a benefit of the inclusion of sustained challenges to standing balance in rehabilitation of postural control post-stroke.
Chapter 5: Influence of knowledge of timing of perturbations on anticipatory postural strategies and postural reactions of people post-stroke

5.1 Introduction

Neuromuscular control of standing balance is known to be impaired after stroke (Garland et al. 2009). Response to surface translations have characterized the postural reactions of people post-stroke as being asymmetrical favoring the non-paretic leg (Marigold et al. 2004a; van Asseldonk et al. 2006), accompanied by muscle activation which is delayed and of decreased amplitude in the paretic limb, with increased variability of intralimb muscle patterning (Badke and Duncan 1983; Marigold and Eng 2006a). Furthermore, impairment in standing balance following stroke has been shown to result in increased attentional demands and conscious control of movement (Brown et al. 2002; Orrell et al. 2009). Taken together, this suggests that functional balance following stroke may be affected by neuromuscular impairment as shown in Chapters 3 and 4, as well as changes in attention and/or emotion related to maintenance of standing balance.

Mounting evidence suggests that physiological arousal modulated by the autonomic nervous system (ANS) may influence postural control (Sibley et al. 2014). Physiological arousal can be measured indirectly by electrodermal activation (EDA); a measurement of skin conductance. Measurements of EDA have been used to examine changes in physiological arousal associated with changes in level of attention, cognitive effort, and emotion during tasks (Critchley et al. 2000). The change in levels of attention and/or emotion surrounding maintenance of standing balance following stroke may result in increased levels of physiological arousal during tasks that threaten balance, and would be reflected in changes in EDA measures. This is important to consider as increased levels of arousal in general have been shown to alter
postural control in healthy subjects regardless of the nature of the associated emotion (Horslen and Carpenter 2011). Perturbation tasks which manipulate the perception of threat and the knowledge of timing of a perturbation have demonstrated modulation of levels of physiological arousal both in anticipation of a perturbation, and in response to perturbations (Sibley et al. 2008; Sibley et al. 2010; Sibley et al. 2014). Larger increases in physiological arousal have been found in older adults than in young adults in response to postural threat (Carpenter et al. 2006). The autonomic nervous system can be affected by stroke (Korpelainen et al. 1999). It is possible that attentional demands and emotional state may interact with balance-related neuromuscular impairments after stroke. The attentional demands can be explored by manipulating knowledge of the impending postural disturbances (Badke et al. 1987; Sibley et al. 2008).

The relationship between the perception of threat to standing balance and the postural strategy, associated with increased postural muscle activity, adopted during a standing balance task has been established in healthy individuals (Adkin et al. 2002; Brown and Frank 1997; Carpenter et al. 2001; Carpenter et al. 2006; Sibley et al. 2008). During external perturbations, healthy individuals demonstrate a lower centre of mass (COM) and anterior-posterior centre of pressure (APCOP) velocity and displacement when expected perturbations are introduced under conditions of perceived risk to standing balance (Brown and Frank 1997; Carpenter et al. 2001). These changes have been suggested to be secondary to change in the postural control strategy related to anticipation of a challenge to standing balance. Anticipatory postural strategies are associated with a tighter control of the COM and APCOP, which is suggested to ensure a faster and more effective postural reaction to a perturbation, such that there is less resulting displacement of the COM within the BOS and therefore less risk of a fall (Brown and Frank 1997; Horak et al. 1989; Horslen et al. 2013; Santos et al. 2010a).
The purpose of this study was to examine the influence of attentional demands, by using investigator-triggered versus self-triggered external perturbations, on the level of physiological arousal, anticipatory postural strategies prior to perturbations, and postural reactions following perturbations in people post-stroke compared to age-matched controls. We hypothesized that people post-stroke would demonstrate increased levels of physiological arousal, as measured by electrodermal activation, and heightened ankle plantarflexor muscle activity than age-matched controls in anticipation of external perturbations. We further hypothesized that anticipatory levels of physiological arousal and plantarflexor muscle activation would be less during self-triggered perturbations than investigator-triggered perturbations for both people post-stroke and controls. Lastly we hypothesized that the anticipatory postural strategies used in participants post-stroke would result in less COM and APCOP displacement and velocity in response to perturbations compared to controls.

5.2 Methods

Ten people with chronic stroke (>1 year post-stroke) and ten age-matched controls participated. Individuals post-stroke were included if they were ambulatory, with or without a walking aid, and could stand independently for a minimum of five minutes. Individuals were excluded if in addition to stroke, they had any health conditions that negatively impacted mobility (e.g. severe osteoarthritis). Controls were included if they were free from neurological or musculoskeletal impairment which resulted in decreased mobility and/or balance. The study conformed to the standards set by the latest revision of the Declaration of Helsinki and was approved by the University of British Columbia Clinical Research Ethics Board.

Participants post-stroke were assessed for motor recovery of the foot and ankle using the Chedoke McMaster Stroke Assessment Scale (CMSA, 0-flaccid to 7-normal, (Gowland et al.
1993)). Both participants post-stroke and controls were assessed for ambulatory balance with the Community Balance and Mobility Scale (CB&M, /96, (Howe et al. 2006; Knorr et al. 2010)), and for balance related self-efficacy using the Activities-specific Balance Confidence Scale (ABC, /100, (Botner et al. 2005; Myers et al. 1996)).

5.2.1 Experimental protocol

Participants stood with their feet shoulder-width apart, with each foot on a separate force platform (AMTI OR6-6, Advanced Mechanical Technology, Watertown, MA). Baseline quiet standing data were collected for 30 seconds in order to investigate a change in anticipation of a perturbation.

A belt was secured around the pelvis of each participant and was attached to a horizontal cable in front of the participant. External loads were applied via a cable-pulley system attached to the front of the belt (Fig. 5.1). All participants were exposed to 5 unexpected load drops prior to data collection for familiarization. The paradigm used in this experiment utilized repetitions of the same stimulus, rather than incremental increases in load drops used in the previous studies, in order to examine the EDA and postural response to the same stimuli with only knowledge of timing altered. During data collection, participants remained standing in comfortable stance and external loads were applied through the belt by dropping loads of 2% body mass (BM) into a basket from a height of 40 cm. The load was maintained in the basket for 10 seconds and then removed. This was repeated ten times, with 15-30 seconds (random timing) of quiet standing between perturbations. Load drops were either self-triggered by participants or investigator-triggered using a button which initiated the release of the load suspended by an electromagnet. Half of the participants experienced the self-triggered prior to investigator-triggered perturbations and the other half had the opposite order. In the self-triggered mode, participants
were instructed to regain comfortable stance once the load was removed and to press the button when they felt ready for the next perturbation. A screen in front of the participants prevented them from seeing when the loads were dropped which was of importance particularly during the investigator-triggered condition in which the exact timing of the load drop was not known. The exact timing of application of the load was detected by a force transducer in-line with the cable (Fig. 5.1). During the investigator-triggered perturbations, loads were dropped once the APCOP position returned to that during quiet stance. In some instances, participants did not return to this position, therefore the load drop was triggered once their COP position appeared stable on visual inspection.
Figure 5.1  Experimental set-up. Participants stood with each foot on a separate force platform. Anteriorly-directed external loads of 2% body weight were dropped by a cable and pulley system attached to a belt around the participants’ pelvis. Ten loads were released with investigator-triggered behind the screen or self-triggered with an electromagnet suspending the load.

5.2.2 Electrodermal activation

EDA was measured using electrodes affixed to the palmer surface of the right hand in controls and the non-paretic hand of participants post-stroke (Sibley et al. 2014); the EDA response has been shown to be suppressed on the paretic side (Muslumanoglu et al. 2002). Electrodes were placed on the hypothenar and thenar eminences. Resistance of a 50 mV signal was measured between the two electrodes representing the skin conductance, an estimate of physiological arousal of each participant. EDA was collected at a sampling frequency of 2000 Hz. The EDA was measured for 30 seconds during quiet stance, prior to participants donning the
hip belt described above, and this represented a baseline level of physiological arousal during standing. During the perturbation trials, the mean EDA was measured 1 second prior to the perturbation and for peak activation directly following the perturbation, and the change from the baseline quiet stance trial was calculated.

5.2.3 Kinetic and kinematic data

Kinetic data were collected using two floor-mounted force platforms (detailed above), sampled at 2048 Hz. Mean APCOP displacement and velocity (the derivative of the APCOP signal) were calculated one second prior to the perturbation and peak values were measured one second immediately following load drop. Percentage weight-bearing through the paretic leg for participants post-stroke and the right leg for controls was calculated from the vertical component of the ground reaction force of the each limb divided by the total vertical ground reaction force from both platforms, multiplied by 100.

Twenty two passive reflective markers were affixed to participants according to a modified Helen Hayes marker set (Kadaba et al. 1989). Eight high-speed digital cameras (Raptor-E, Motion Analysis Corp, Santa Rosa, CA, USA) sampled the movement of the reflective markers at 120 Hz. Kinematic data were analyzed using a custom-written program in MATLAB (The Mathworks Inc., Natick, MA, USA) that was used in a previous study to quantify kinematics during movement (Pollock et al. 2012). COM was calculated from marker coordinate data and using published anthropometric values (Dempster and Gaughran 1967). COM velocity was calculated as the derivative of COM displacement. Anterior-posterior mean COM displacement and COM velocity were calculated one second prior to the perturbation and peak values were measured one second following load drop. Position of APCOP within the base of support prior to load drop was calculated using APCOP displacement converted to a
percentage of the base of support for each foot as calculated from the kinematic markers of the heel (0%) and the base of the 1st metatarsal (100%).

5.2.4 Surface EMG recordings

High-density surface electromyography (HDsEMG) data from the soleus (SOL) (24 electrode grid, 2 cm interelectrode distance), medial (MG) and lateral gastrocnemius (LG) (20 electrode grids each, 1.5 cm interelectrode distance) were collected bilaterally (OT Bioelectronica, Turin, Italy) at 2048 Hz. HDsEMG signals were analyzed in bipolar configurations resulting in 18 EMG signals from the SOL muscle and 16 from each of the MG and LG muscles. RMS amplitude of EMG of each bipolar configuration was measured for one second prior to the perturbation and one second immediately following the load drop. The median RMS amplitude was calculated across the bipolar signals for each plantarflexor muscle before and after the perturbation.

5.2.5 Statistical analysis

The performance of right and left legs of controls were first compared using paired t-tests. As there were no significant differences between the right and left legs of controls for all outcomes, data from the two legs were averaged in the control group. Pre-perturbation RMS EMG amplitude from each ankle plantarflexor was compared to that during the baseline quiet stance trial (when there was no possibility of a perturbation) using one sample t-tests. This addressed, within each group specifically, the change in pre-perturbation activation of plantarflexors comparatively to quiet standing under conditions of no anticipated perturbation. RMS amplitude of EMG of each plantarflexor muscle in response to the perturbation were compared using a mixed model two way ANOVA for within subject factor of condition, self-triggered and investigator-triggered, and between group factor of control (C), non-paretic (NP)
and paretic (P) leg. EDA and COM parameters were compared using mixed model two way ANOVAs for the within subject factor of condition (self-triggered and investigator-triggered), and between group factor (stroke and control). Centre of pressure parameters in response to perturbations were compared using similar mixed model two way ANOVAs, but with the between-group factor comprising control (C), non-paretic (NP) and paretic (P) legs. When condition or group main effects were evident, post-hoc pairwise comparison revealed the nature of the condition or group differences. Significance was set at p = 0.05.

5.3 Results

5.3.1 Participants

Table 5.1 shows the characteristics of participants post-stroke and controls. Age was not significantly different between groups (p = 0.65). Participants post-stroke scored significantly lower than controls in ambulatory balance as measured by the CB&M (p < 0.01). Balance self-efficacy as measured by the ABC tended to be lower in participants post-stroke than controls (p = 0.14).

<table>
<thead>
<tr>
<th></th>
<th>Age (yrs)</th>
<th>Gender (m/f)</th>
<th>Post onset (yrs)</th>
<th>Paretic side (R/L)</th>
<th>CMSA * (0-7)</th>
<th>CB&amp;M (0-96)</th>
<th>ABC (100%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke</td>
<td>66.2 ± 9.2</td>
<td>8m /2f</td>
<td>6.6 ± 3.6</td>
<td>5 R / 5L</td>
<td>31.9 ± 23.8</td>
<td>83.0 ± 17.6</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>68.0 ± 8.2</td>
<td>7m / 3f</td>
<td>n/a</td>
<td>n/a</td>
<td>80.9 ± 7.8</td>
<td>93.0 ± 6.7</td>
<td></td>
</tr>
</tbody>
</table>

*CMSA reported as median and interquartile range.

5.3.2 Electrodermal activation

Participants post-stroke demonstrated significantly higher levels of pre-perturbation EDA than controls for both perturbation conditions (Fig. 5.2A, p=0.04). The EDA response to
perturbation tended to be higher in participants post-stroke compared to controls (Fig. 5.2B, p=0.08). Whereas the EDA response did not modulate with perturbation condition in participants post-stroke (p=0.81), it did in controls (p=0.01).

![Graph A) Pre-perturbation EDA change from QS (µv) and B) Peak EDA response (µv) for Stroke and Control groups](image)

Figure 5.2 Electrodermal activation (EDA), A) prior to perturbation and B) following perturbation during self- and investigator-triggered external perturbations in participants post-stroke and controls. Participants post-stroke demonstrated significantly higher levels of pre-perturbation EDA than controls in both perturbation conditions (*p=0.04). The EDA response to perturbation (change from pre-perturbation EDA level) tended to be higher in participants post-stroke compared to controls (**p=0.08). Whereas the EDA response did not modulate with perturbation condition in participants post-stroke, it did in controls (†p=0.01).

5.3.3 Kinematics

The COM anterior-posterior position pre-perturbation did not show significant difference between conditions of self- and investigator-triggered perturbations in both groups (p=0.60); therefore, even though we could not control the APCOP position upon load release in the self-
triggered mode, there was no difference in the body position between the two conditions. Peak COM excursion in response to perturbation was not significantly different groups (Fig. 5.3A, p=0.65). However, in controls there was a significant effect of condition such that there was less COM excursion in response to investigator-triggered perturbations compared to self-triggered perturbations (p=0.05). COM velocity in response to perturbations was not significantly different between groups (Fig. 5.3B, p=0.89) or conditions (p=0.87).

Figure 5.3 Centre of mass (COM), A) displacement and B) velocity during self- and investigator-triggered external perturbations in participants post-stroke and controls. The peak COM displacement in response to perturbation was significantly higher during self-triggered perturbations than investigator-triggered perturbations in controls (†p=0.03).
5.3.4 Kinetics

Mean APCOP position immediately prior to perturbations, expressed as a percentage of the BOS, was significantly more anterior on the paretic leg than the control leg (Fig. 5.4A, \( p = 0.05 \)). The mean APCOP position pre-perturbations was significantly more anterior during the self-triggered perturbation than investigator-triggered perturbations on the paretic side of participants post-stroke (\( p = 0.03 \)). There was no effect of condition on percentage weight-bearing during the response to perturbation on the paretic side in participants post-stroke (self-, 54.0 ± 5.3%, investigator-triggered, 51.6 ± 5.0%) or the right side in controls (self-, 46.0 ± 2.7%, investigator-triggered, 49.8 ± 2.2%, \( p = 0.82 \)). The peak APCOP displacement post-perturbation was significantly less in the paretic leg of participants post-stroke in both conditions compared to the non-paretic leg and control leg (Fig. 5.4B, \( p = 0.01 \)). A representative example is depicted in Figure 5. Between conditions, the peak APCOP displacement was significantly greater in response to investigator- than self-triggered perturbations in the paretic leg (\( p = 0.02 \)). The peak APCOP velocity was significantly slower post-perturbation in both conditions in the paretic leg of participants post-stroke compared to the non-paretic leg and control legs (Fig. 5.4C, \( p = 0.01 \); Fig. 5.5). In addition, the peak APCOP velocity was higher in response to investigator- than self-triggered perturbations in the control (\( p = 0.01 \)) and paretic legs (\( p = 0.08 \)).
Figure 5.4 Centre of pressure (COP) measures during self-triggered (white) and investigator-triggered perturbations. The mean and standard error are presented for all parameters with statistical differences indicated by between leg differences *p ≤ 0.05, and between condition, †p ≤ 0.05, ‡p=0.08. A) APCOP position immediately prior to perturbations amongst groups: control, non-paretic, paretic. Mean APCOP position was significantly more forward immediately prior to the perturbation in the paretic compared to the control leg. The mean APCOP position of the paretic leg was significantly more forward prior to self-triggered perturbations than investigator-triggered perturbation. B) Anteriorly-directed APCOP displacement post-perturbation. Peak APCOP displacement was significantly less in the paretic compared to the non-paretic and control legs. Peak APCOP displacement was significantly greater in response to investigator- than self-triggered perturbations in paretic legs. C) Peak APCOP velocity post-perturbation. Peak APCOP velocity was significantly slower in paretic leg compared to control and non-paretic legs. Peak APCOP velocity was higher in response to investigator- than self-triggered perturbations in control and paretic legs.
Figure 5.5 Responses to a single self-triggered perturbation in a participant post-stroke (paretic leg, gray) and a control subject (black). From top: force, representing application of load drop, anterior-posterior centre of pressure (APCOP) anterior displacement, muscle activation (amplitude normalized to quiet stance (QS)) for medial and lateral gastrocnemius (MG, LG) and soleus (SOL). Pre-perturbation muscle activation is larger in paretic than control muscles. APCOP of the paretic leg demonstrates a smaller peak anterior displacement with lower velocity compared to the control leg.

5.3.5 Electromyography

Pre-perturbation, RMS EMG amplitude was significantly increased from quiet stance in the paretic medial gastrocnemius and soleus muscles of participants post-stroke during both self- and investigator-triggered perturbations (Fig. 5.6A, p<0.05). Amongst the ankle plantarflexors of the control leg, the lateral gastrocnemius muscle demonstrated a significant increase from quiet stance levels during investigator-triggered perturbations only (Fig. 5.6A, p<0.05).
Representative ankle plantarflexor EMG in response to perturbation are shown in Figure 5 and are reflective of an increase in RMS EMG amplitude from directly pre-perturbation. In response to perturbations, participants post-stroke demonstrated larger muscle activation in both gastrocnemii muscles during investigator-triggered perturbations than during self-triggered perturbations (Fig. 5.6B, p<0.05), which was not seen in controls.
Figure 5.6 RMS EMG amplitude of the ankle plantarflexor muscles amongst groups C-Control, P-paretic, NP- non-paretic, during self-triggered (white) and investigator-triggered (black) perturbations. A) RMS EMG amplitude prior to perturbations. The dotted horizontal line represents RMS EMG amplitude of each muscle during quiet stance prior to any perturbations. Participants post-stroke had higher RMS amplitude than quiet stance in the paretic medial gastrocnemius and soleus muscles in both conditions, and in the lateral gastrocnemius during self-triggered perturbations (†p<0.05), while controls demonstrated a significant increase in RMS amplitude in the lateral gastrocnemius muscle during investigator-triggered perturbations only. B) Change of RMS EMG amplitude from pre- to post-perturbation. Participants post-stroke demonstrated larger muscle activation in the paretic.
5.4 Discussion

The purpose of this study was to compare the level of physiological arousal and postural control strategy adopted by participants post-stroke and age-matched controls during conditions of self-triggered and investigator-triggered external perturbations. Regardless of condition, participants post-stroke demonstrated higher anticipatory levels of physiological arousal than controls and these levels did not modulate between conditions. During both conditions, participants post-stroke demonstrated increased anticipatory activation of the paretic medial gastrocnemii and soleus muscles compared to quiet stance. Self-triggered timing of perturbations resulted in a response of lower peak physiological arousal in controls whereas there was no effect of trigger condition on the peak physiological arousal response of participants post-stroke. However, between conditions, the paretic leg demonstrated less plantarflexor activity and APCOP displacement and velocity during self-triggered than investigator-triggered perturbations. These findings suggest that the effect of stroke resulted in increased levels of anticipatory physiological arousal and use of anticipatory postural control strategies during perturbation trials, which were not decreased under self-triggered perturbation conditions.

Heightened anticipatory postural control strategies in participants post-stroke, including increased ankle plantarflexor muscle activity, would limit the anterior displacement of the APCOP in response to a perturbation rather than relying on reacting to a perturbation (Brown and Frank 1997; Horak et al. 1989; Santos et al. 2010a). This postural strategy appears to be particularly evident in the paretic leg of participants post-stroke, indicating an asymmetrical adaptation of the anticipatory postural strategy under conditions of external perturbations.

This study is the first to show that physiological arousal during external perturbations is heightened following stroke. In this paradigm, perturbation characteristics of direction and
magnitude are known and therefore are somewhat predictable (only the exact timing of the investigator-triggered perturbations was not known) and this knowledge has been shown to inform the postural strategy adopted during perturbations (Badke et al. 1987; Cordo and Nashner 1982; Santos et al. 2010a). We anticipated that the arousal of participants post-stroke would be higher than controls. However, we also anticipated the arousal associated with certainty of the timing of the perturbation would be less during self-triggered perturbations in both groups, as has previously been shown in healthy subjects (Sibley et al. 2008). Our results show that despite knowledge of the perturbation characteristics and the additional control of timing, participants post-stroke maintained higher levels of arousal than controls, perhaps reflecting a higher perception of postural threat.

Control subjects demonstrated increased physiological arousal and decreased COM displacement in response to investigator-triggered perturbations, suggestive of a change in postural control strategy. These results are in agreement with previous studies in healthy participants that show a heightened physiological arousal response and dampened COM displacement in response to perturbations under conditions of increased postural threat and unknown timing of a perturbation (Brown and Frank 1997; Sibley et al. 2008). However, if there was an associated change of anticipatory postural strategy or postural reaction in controls, it does not appear to be captured by the parameters collected in the current paradigm and may include components of postural control such as the hip strategy.

Conversely, participants post-stroke demonstrated heightened anticipatory activation of plantarflexor muscles in both conditions and modulation of paretic plantarflexor activity and APCOP during postural reactions between conditions. Participants post-stroke demonstrated heightened plantarflexor muscle activity, particularly in the paretic leg, regardless of the trigger
condition, suggesting that the anticipatory postural strategy at the ankle did not change with knowledge of timing. Amongst participants post-stroke the postural reactions in response to perturbations in the paretic leg did demonstrate an effect of knowledge of timing. The change of postural reaction was seen specifically in the paretic leg with decreased activation of both gastrocnemii muscles in response to the perturbation and decreased APCOP excursion and velocity when perturbations were self-triggered. Prior knowledge of direction and timing of a perturbation have been shown to improve the timing of gastrocnemius muscle burst onset and temporal coordination with other muscles of the paretic leg during anteriorly directed perturbations (Badke et al. 1987). Badke et al. (1987) suggested that attentional variables may be a critical source of influence to preprogrammed postural reactions. In the current study, it is possible that improved timing and coordination of lower extremity muscle activation associated with increased knowledge of timing may have resulted in decreased overall amplitude of muscle activation required to respond to the perturbation.

Anticipatory postural strategies inclusive of increased activation of muscles able to dampen the displacement effect of an expected perturbation are scaled to an individual's perception of their ability to withstand the perturbation (Brown and Frank 1997; Horak et al. 1989; Jacobs and Horak 2007; Santos et al. 2010a). Therefore, the heightened anticipatory postural strategy across conditions in participants post-stroke may reflect an increased perception of threat to standing balance during external perturbations in participants post-stroke more so than controls. This is further supported by the increased levels of physiological arousal in participants post-stroke. Interestingly, it appears that greater emphasis of the postural control strategy during external perturbations is placed on limiting the effect of the perturbation on the paretic leg specifically.
As decreased balance confidence has been suggested to influence the risk of falls more so than many physical measures of balance post-stroke (Pang and Eng 2008), it is tempting to suggest that the heightened level of physiological arousal noted in participants post-stroke in the current study which lacks modulation, together with heightened muscle activation in the anticipatory postural strategy which is not reduced by knowledge of timing of the perturbation is a negative sequel of stroke. However, it is important to note that the mean ABC score in the current study is higher (denoting increased balance confidence) than that previously reported by Botner et al. (2005, ABC mean 68.3 (SD 17.5)) for participants with chronic stroke. In fact, the participants post-stroke in the current study demonstrate only a 10% lower self-reported balance confidence compared to healthy controls. Conversely, the CB&M scores of participants within the current study are lower (denoting more impaired walking balance) than that of people at 2 and 8 months post-stroke (Knorr et al. 2010). Despite low functional walking balance, participants in the current study report rather high balance confidence. Therefore, perhaps these anticipatory adaptations of postural control are an appropriate adaptation. The sensorimotor consequence of stroke on standing balance is significant (Garland et al. 2009). Maintenance of standing balance following stroke has been shown to utilize increased attentional demands (Brown et al. 2002; Orrell et al. 2009). Therefore, it is possible that the individuals post-stroke participating in the current study have established a physiological response, including autonomic arousal related to increased attentional focus and/or emotion, and change in anticipatory postural strategy when faced with a postural challenge, which is successful for them maintaining independent mobility at the community level, inclusive of the ability to withstand perturbations under conditions which they may perceive as a challenge to standing balance.
5.5 Conclusion

Manipulation of the control of timing of an anticipated perturbation showed that participants post-stroke demonstrated similar levels of increased physiological arousal and paretic plantarflexor muscle activity in anticipation of a perturbation regardless of whether it was a self- or investigator-triggered perturbation. This is the first report of ANS and postural control responses in people post-stroke. The between-leg asymmetrical anticipatory postural strategy may explain the decreased APCOP displacement and velocity of the paretic leg compared to the non-paretic and control legs. However, the postural reaction of the paretic leg of participants post-stroke demonstrated between-condition modulation of paretic gastrocnemii muscles and APCOP parameters, which suggests that the paretic leg is influenced by the addition of knowledge of timing of the perturbation.
Chapter 6: General discussion

6.1 Overview

The results of this thesis provide a greater understanding of the balance impairment following stroke by adding to the understanding of the underlying changes in motor control of key postural muscles following stroke. Specifically, how ankle plantarflexor muscles respond to conditions of anteriorly-directed perturbations to standing balance were examined. This thesis advances the understanding of fundamental motor unit behaviour of the healthy medial gastrocnemius muscle during standing perturbations in Chapter 2 and reveals the task-specific nature of motor unit recruitment and rate coding during challenges to standing balance. These results formed the foundation from which to examine the motor unit behaviour of paretic medial gastrocnemius muscles of people post-stroke during standing perturbations. In Chapter 3, paretic medial gastrocnemius muscles demonstrated impaired rate coding during postural reactions and decreased firing rate during maintenance of standing balance under increased challenge. Additionally, the relationship between motor unit firing rate and centre of mass (COM) and hip movement velocity was suggestive of an interaction of motor unit firing rate and a postural strategy which increasingly aimed to limit movement velocity of the COM as perturbations increased. Chapter 4 provides an examination of the modulation of muscle activity of all three ankle plantarflexors with anterior-posterior postural sway, revealing that the EMG preceded the postural sway with similar timing noted in paretic muscle as in non-paretic and control muscles. The non-paretic ankle plantarflexor muscles of people post-stroke demonstrated a stronger level of active control of postural sway than the paretic ankle plantarflexor muscles at low-levels of anteriorly-directed challenge to standing balance, which decreased at higher-levels of postural
challenge as the paretic muscle activation increased. In comparison to the non-paretic plantrarflexors, there was some suggestion that the relationship of modulation of EMG activity and postural sway appeared to be effected in the paretic gastrocnemii muscles more so than the soleus muscles. Finally, by manipulating the knowledge of the timing of an anticipated perturbation in Chapter 5, we showed that people post-stroke demonstrated both increased levels of physiological arousal and paretic ankle plantarflexor muscle activity in anticipation of a perturbation regardless of whether it was a self- or investigator-triggered perturbation. This was not seen in age-matched controls and offers initial insight into the possible relationship between physiological arousal and postural control following stroke.

6.2 Motor unit control of external perturbations

Motor unit behaviour in response to standing external perturbations has not previously been explored, therefore, Chapter 2 first established medial gastrocnemius muscle motor unit behaviour in healthy participants in response to external perturbations experienced under increased levels of challenge to standing balance. Healthy participants demonstrated a symmetrical response to perturbations with a gradual increase in external ankle torque and amplitude of medial gastrocnemius muscle activity while maintaining increased anteriorly-directed loads. The medial gastrocnemius muscle of healthy people utilized motor unit recruitment to achieve the increased levels of muscle activity required to maintain steady state standing balance under conditions of increasing levels of external ankle torque. During the dynamic response to abrupt perturbations, the medial gastrocnemius muscles of healthy individuals used firing rate modulation of motor units which were active at the time of perturbation together with recruitment of new motor units to resist the perturbation. These data suggest that the gradation of force by motor unit recruitment and rate coding is task-specific.
That is, in standing, modest increases in force required of the medial gastrocnemius muscle to meet the increase in external ankle torque is modulated by motor unit recruitment, whereas rate coding is used to a greater extent when a faster reaction is required of the muscle to resist an abrupt perturbation.

Exploring this motor unit behaviour in Chapter 3 in the paretic medial gastrocnemius muscles of people post-stroke revealed that they primarily exhibit motor unit recruitment with lower motor unit firing rates than previously-measured in control participants during both the static and dynamic aspects of this perturbation paradigm. The firing rate of the paretic medial gastrocnemius muscle was slower, as revealed by a longer interspike interval (ISI 167 ms, IQR 145-193 ms), than that of healthy controls (ISI 130ms, IQR 110-140 ms). Considering this finding in the context of what is known about muscle re-modeling following stroke provides insight into the impairment at the level of the muscle post-stroke. Specifically, paretic muscle is known to be comprised of a fewer number of motor units with a particular loss of larger type II motor units (Lukacs et al. 2008). Of the ankle plantarflexor muscles, the paretic gastrocnemius muscle experiences significant atrophy following stroke (Ramsay et al. 2011). These findings, together with that of Chapter 3, suggest that force production of the paretic medial gastrocnemius muscle directed towards maintaining standing balance under postural challenges is impaired not only by a smaller number of motor units and subsequent atrophy, but also by a decreased firing rate of available motor units.

There are two possible explanations for the lack of firing rate modulation in paretic muscle during postural reactions. One explanation is that the blunted motor unit firing rate is a reflection of impairment of long loop reflexes, which have been suggested to be a component of impaired postural reflexes of lower extremity muscles post-stroke (Marigold and Eng 2006a).
Postural long-loop reflexes are proposed to integrate afferent input at a supraspinal level and
direct the motor response via spinal networks of upper and lower extremity muscles (Bloem et al.
2002; Jacobs and Horak 2007; Misiaszek 2006). If the long-loop reflexes are impaired, this
might explain the relative lack of motor unit response during external perturbations post-stroke.
That being said, the analysis of the motor unit firing rate with the kinematic parameters in
Chapter 3 revealed relationships of motor unit firing rate with COM and hip movement velocity.
The relationship between the afferent input and motor response during perturbations has been
described by Bloem et al., (2002); they found that the response of the ankle muscles during
perturbations was strongly influenced by the proprioceptive inputs from hip and trunk movement
(Bloem et al. 2002). It is possible then, that despite the finding of a lack of an increase in motor
unit firing rate dynamically in response to a perturbation in the paretic medial gastrocnemius
muscle, sensorimotor integration of longloop postural reflexes associated with postural control
may be partially preserved.

A second explanation for the lack of modulation of the paretic motor units in response to
perturbations is that the compensatory postural strategy used by participants post-stroke did not
necessitate a change in firing rate. Postural reactions at increasing load levels in Chapter 3
showed decreased COM and COP velocity in the paretic leg in response to perturbations in
participants post-stroke, in keeping with an anticipatory postural control strategy aimed at
limiting movement following perturbations. A stiffening strategy has been suggested by others
after stroke (Hocherman et al. 1988). In contrast, healthy controls in Chapter 2, motor unit
modulation occurred concurrently with a consistent COP velocity in response to perturbations
despite maintaining increased levels of loads. Therefore, the relationship between motor unit
firing rate and velocity of movement in participants post-stroke may also suggest that
anticipatory postural strategies adopted by people post-stroke further impacts paretic medial gastrocnemius motor unit response to perturbation by decreasing the requirements of the muscle to respond to a perturbation.

6.3 How does knowledge about the perturbation influence the postural response?

During external perturbations, postural reactions of the paretic ankle plantarflexor muscles of people post-stroke have been shown to be delayed and of decreased amplitude/magnitude (Garland et al. 2009). Therefore, the use of an anticipatory postural control strategy in people post-stroke as suggested in Chapter 3 may reflect a component of compensatory postural control. This hypothesis was supported by the findings of Chapter 5, which revealed increased muscle activation of all three paretic plantarflexors in anticipation of a perturbation. This preactivation of plantarflexor muscles was somewhat unique to the paretic leg and therefore, further reflects the between-leg assymetrical postural control strategies of people post-stroke.

This finding of preactivation of the plantarflexors is suggestive of a change in central postural set that incorporates compensation for the sensorimotor impairment of the paretic limb post-stroke. When anticipatory postural strategies are used, the magnitude of COP displacement of the associated postural reaction to the perturbation is dampened, particularly when the timing of the external perturbations is known by participants (Horak et al. 1989; Santos et al. 2010a; Santos et al. 2010b). Importantly this anticipatory strategy was not changed in participants post-stroke when perturbations were self-triggered, and was not observed in either triggering condition in the control group. The pre-activation in anticipation of a perturbation was paralleled by the level of physiological arousal, such that both parameters, physiological arousal and
plantarflexor muscle activation, were elevated more so in people post-stroke than in controls, with no effect of control of timing of the perturbation.

Mounting evidence suggests the autonomic nervous system may influence postural control (Carpenter et al. 2006; Sibley et al. 2014). Although a great deal of research has focused on constructs such as generalized decreased balance related self-efficacy, fear of falling, increased attentional focus on balance following stroke, all of which have potential to provoke an autonomic response, this is the first study to reveal a heightened level of physiological arousal and change in postural strategy in anticipation of and in response to perturbations in people post-stroke. Although the interaction of physiological arousal and postural control is not understood fully, these data suggest that physiological arousal may play a role in the anticipatory postural strategies adopted by people post-stroke when there is a challenge to balance that might be perceived as a threat.

Importantly, there was no modulation of the peak EDA amplitude in response to perturbations between investigator- and self-triggered perturbations. The physiological mechanism underlying the interaction of the ANS with the motor response of the postural reaction is not known (Sibley et al. 2010; Sibley et al. 2014). Modulation of the postural reaction in people post-stroke between triggering conditions, in the absence of modulation of anticipatory postural strategies and electrodermal activation, suggests additional influence on modulation of the motor control of the postural reaction specifically in people post-stroke. After stroke, there may be cortical influence (attention) on postural reactions, which may be more evident in the presence of self-controlled timing (Badke et al. 1987; Jacobs and Horak 2007). It has been suggested that attentional variables may be a critical source of influence to preprogrammed postural reactions in people post-stroke (Badke et al. 1987).
Balance function following stroke has been shown to be related to increased attentional demands (Brown et al. 2002; Orrell et al. 2009) and decreased balance self-efficacy (perception of abilities to successfully overcome challenges to standing balance; (Pang and Eng 2008; Schmid et al. 2012; Schmid and Rittman 2007). Mounting evidence suggests that balance impairment, both at the level of motor control impairment and decreased self-efficacy, impacts successful reintegration into the community, independence and quality of life (Pang and Eng 2008; Schmid et al. 2012). Recent evidence in healthy subjects suggests a relationship between the CNS and autonomic nervous system (ANS) during postural control tasks (Carpenter et al. 2006; Sibley et al. 2014). Therefore, as both heightened attention and emotion may result in increases in physiological arousal response of the ANS (Critchley et al. 2000), the interaction of these constructs with physiological arousal and postural control may be an important new area to explore further in people post-stroke.

Badke et al. (1987) also suggested that prior knowledge of direction and timing appeared to improve the timing of gastrocnemius muscle burst onset and temporal coordination with other muscles of the paretic leg during anteriorly-directed perturbations in people post-stroke. In Chapter 5, participants post-stroke demonstrated a decreased activation of the paretic plantarflexors together with decreased APCOP displacement and velocity during self-triggered perturbations. This may reflect the influence of improved timing and coordination of muscle activation resulting in decreased overall amplitude of muscle activation required to respond to the perturbation. This modulation of the postural reaction in response to the perturbation, without modulation of the anticipatory postural strategy, suggests that the postural reaction was not simply reflective of a change in anticipatory postural strategy in people post-stroke and may be shaped by other influences related to prior knowledge of a perturbation.
6.4 **Motor control of standing balance across the ankle plantarflexors**

In quiet stance, the movement of the COP is influenced by modulation of the ankle plantarflexor muscles which precedes postural sway (Gatev et al. 1999; Masani et al. 2003). The motor impairment of the paretic muscles of the ankle has been suggested to influence the decrease in postural sway parameters of displacement and velocity noted in the paretic leg compared the non-paretic leg during quiet standing (Roerdink et al. 2009). Chapter 4 further supports this understanding of impaired postural control and between-leg differences of people post-stroke by demonstrating that the plantarflexor muscles demonstrate larger active control of postural sway in the non-paretic leg compared to the paretic leg, suggestive of the compensatory role of the non-paretic leg in control of postural sway. However, the difference between paretic and non-paretic plantarflexor muscles is reduced as increased anteriorly-directed challenge is applied to the body. These data suggest that paretic plantarflexor muscles respond to postural challenges, involving increased muscle activation demands, with increased active control of postural sway which in more similar to that of the non-paretic leg. This may suggest that increased postural challenge further engages the paretic leg as a more active contributor to postural control.

The roles of the plantarflexor muscles are differentiated during postural sway in quiet stance (Loram et al. 2011; Vieira et al. 2012). The gastrocnemius muscle tends to demonstrate more phasic activity in response to anterior displacement of the COP with respect to the ankle joint whereas, the soleus muscle demonstrates more tonic activity during postural sway. The change in strength of relationship of EMG:APCOP modulation with increased load that was noted between paretic and non-paretic plantarflexors was more evident in the gastrocnemii
muscles than the soleus muscle. Importantly, in control and non-paretic legs there was greater differentiation between gastrocnemii and soleus muscles with respect to strength of the relationship of EMG:APCOP modulation than paretic legs and the strength of this relationship was positively influenced by increased activation of the muscle in both paretic gastrocnemii muscle but not in paretic soleus muscle. These findings suggest that the gastrocnemii muscles, in particular, require increased challenges to standing balance to play a more active role in postural control.

6.5 Limitations

There are several important limitations in the interpretation of the results of these studies:

1) The participants in these studies were all ambulatory and living independently in the community. Although the participants in the chronic phase post-stroke encompassed a range of mild to moderate stroke severity as measured with the CMSA and dynamic balance abilities as measured with the CB&M, the results of this study are limited to those who have achieved sufficient community-level ambulation. It is not known if the current findings of motor control of standing balance would be found in those who are not ambulatory, or have achieved limited ambulation (e.g. with support of a walker indoors only).

2) The stage of chronicity (> 1 year post-stroke) of the participants post-stroke in these studies further limits interpretation of these aspects of motor control. How these findings of motor control have emerged from the acute phase post-stroke is not known.

3) The number of participants in these studies precludes the ability to investigate how the severity of motor impairment after stroke influences the aspects of the motor control parameters measured in Chapters 3-5. A larger number of participants with a broad range of stroke severity would be required.
4) Data collection in Chapter 2 was performed with younger participants than the participants post-stroke in Chapter 3. The lack of age-matched controls in Chapter 3 somewhat limits the interpretation of changes secondary to stroke as the effect of aging is not known. Importantly, both studies in Chapters 4 and 5 include age-matched controls.

5) This thesis utilizes normalization of the surface RMS EMG amplitude during perturbations to the surface RMS EMG amplitude during quiet standing. This limits the interpretation of influence of perturbations on the amplitude of muscle activation to that of a change of amplitude from quiet standing specifically. No interpretation of the absolute RMS EMG amplitude can be made.

6) Kinematic measurements utilized in this thesis represent primarily sagittal joint excursion and therefore, restrict the understanding of postural strategy in response to perturbation to movement in the sagittal plane. Movement in the frontal and transverse planes of movement may reflect important components of the postural strategy used by people post-stroke.

7) It was not possible to employ a power calculation based on previous research to inform the number of participants required in each of the studies of this thesis. Therefore, it is possible that type II errors may be present.

6.6 Implications and future directions

These chapters suggest the importance of investigation of motor unit behaviour under conditions of functional tasks in both healthy and stroke populations in order to further understand fundamental aspect of motor control at the level of the motor unit. The use of standing postural challenges which employ increasing levels of load to challenge the ability to maintain postural control, together with abrupt perturbations, have revealed important and novel features of motor control impairment specific to postural control in people post-stroke.
Gradation of force in healthy and paretic medial gastrocnemius muscle primarily employs motor unit recruitment to meet increased challenges to standing balance. Modulation of firing rate is more specifically employed when a faster reaction is required. However, absolute firing rate and modulation of firing rate is impaired in paretic medial gastrocnemius muscles. The ability to produce fast responses to postural challenges may be influenced by an interaction of motor unit firing rate and postural strategies adopted by people post-stroke. Specifically, the anticipatory postural strategy employed by people post-stroke suggests an influence of limiting the displacement and velocity of the paretic leg during postural reactions.

The current research captures postural control of participants with chronic stroke. Future research should characterize motor control during postural reactions to external perturbations in the sub-acute phase of stroke recovery, which is more reflective of the timing of rehabilitation post-stroke. Research in the sub-acute phase post-stroke may provide further insight into the motor unit impairment post-stroke; specifically addressing if a lack of motor unit modulation during abrupt perturbations is a reflection of the impact of compensation in the form of movement limiting anticipatory postural control strategies related to chronicity versus a reflection of sensorimotor impairment specific to the nature of the paresis post-stroke. This is important to the development of rehabilitation strategies aimed at improving postural control during response to external perturbations and to understand the aspect of motor control at which these interventions are expected to influence postural control post-stroke. Rehabilitation incorporating activities promoting fast contraction of the muscle may address the sensorimotor impairment at the level of the muscle response; whereas, rehabilitation focused on the training of motor control strategies at the level of the whole body response, may develop effective compensation for the motor impairment.
Future research should explore physiological arousal and postural control strategies in people post-stroke, together with participant reports of measures of attention and self-efficacy specific to the task used in the study paradigm, to more comprehensively understand how these constructs influence postural control in people post-stroke. Furthermore, determining interactions of physiological arousal and postural control which may represent positive and negative functional outcomes for standing balance post-stroke is important to begin to understand compensation versus maladaptation. Once understood in more detail, the interaction between attention, self-efficacy and postural control in people post-stroke may lead to rehabilitation strategies which more comprehensively address impaired balance post-stroke, resulting in improved community re-integration and quality of life.

This thesis highlights the between-leg and between-muscle differences in the strength of the relationship of the paretic and non-paretic plantarflexor muscle activation with postural sway. A common finding of postural control following stroke is the shift of control of postural sway to the non-paretic leg (Roerdink et al. 2009; van Asseldonk et al. 2006). This shift to the non-paretic leg is particularly evident in the gastrocnemii muscles more so than in the soleus muscles of people following stroke, although it was diminished with increased levels of activation. These findings may imply that rehabilitation of the plantarflexor roles of postural control should include sufficient challenge to balance to address the role of the gastrocnemii muscle specifically as the gastrocnemii muscles and their role in postural control appear to be effected more so than the soleus muscles by stroke. Future research should explore the relationships of each plantarflexor with postural sway under conditions of challenges to standing balance following interventions that specifically target postural control under maintained and sufficient challenges to standing balance. This line of research could also explore if
improvements in the modulation of muscle activity with postural sway is associated with functional changes in standing balance following stroke.

6.7 Conclusion

This thesis provides new insight into the motor control of standing balance following stroke. The motor control impairments identified at the level of the motor unit and at the level of integrated muscle activation show important changes under conditions of increased postural challenge which further the understanding of control of standing balance following stroke. Importantly, aspects of motor control have been presented in association with kinematic and kinetic description of movement which allows connection between motor control and resultant movement impairment following stroke. This thesis also provides initial insight into the possible influence of the ANS on motor control underlying postural control strategies associated with anticipation of a loss of balance following stroke. Further research is needed to more comprehensively understand modulation of the motor control parameters examined in this thesis under conditions of different challenges to standing balance, at acute and sub-acute stages of stroke recovery, and following treatment of standing balance post-stroke.
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