Effects of Multi-Directional Surface Perturbations on the Triggered Postural Responses in Hemiplegic Subjects during Standing and walking

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ABSTRACT

Injury to the central nervous system as a result of cerebrovascular accidents (CVA) often leads to impairment in balance and mobility. Very little is known about the strategies that patients with CVA employ to prevent falls during unexpected changes of a support surface while patients are standing and walking. Previous studies showed that light touch increases postural stability in healthy subjects. However, it is not known whether patients with CVA would benefit from light touch in the same way as the healthy subjects do. Three studies in this research were conducted to examine the impact of stroke on postural responses triggered by surface rotations (Triggered Postural Responses; TPRs) during standing and walking and to investigate the effect of light touch on the control of TPRs during both tasks. Pitch and roll surface rotations (amplitude:5^{o}; peak velocity:32^{o}/s) were randomly presented to 11 stroke and 8 healthy age-matched subjects during quiet stance and walking, with similar limb geometry in double limb support. Light touch (<4N) was provided by a load sensor strip mounted on a rail along the walkway. Body kinematics was captured at 120 Hz by a 6-camera Vicon 512 system. Ground reaction forces were acquired at 1080Hz by 2 AMTI force plates. Surface EMGs were recorded at 1080 Hz from 4 bilateral lower limb muscles. Results showed that TPRs in healthy subjects were functionally appropriate to the direction of perturbations and task demands, such that TPRs were tuned down during walking, as compared to standing, suggesting that postural requirements are less during walking. In contrast, CVA disrupts equilibrium control such that TPRs in the stroke subjects were delayed and not modulated as the perturbed directions and task demands changed, possibly due to problems in sensorimotor integration. Asymmetry was characterized by under-activated muscle responses and force generations of the paretic side and hyperactivity of the non-paretic upper and lower

extremities. These impairments led to instability of the trunk and center of pressure, especially in the frontal plane. Light touch increased postural stability in both subjects but its effect was more dominant in the stroke subjects, suggesting that light touch is a potential tool for balance rehabilitation.

ABRÉGÉ

Les lésions du système nerveux central suite à un accident vasculaire cérébral (AVC) affectent fréquemment l'équilibre et la mobilité. Peu d'information est disponible sur les stratégies utilisées par les patients ayant survécu à un AVC pour prévenir les chutes lorsque des changements inattendus de la surface de support se produisent pendant la marche et la position debout. Des études ont établi que des sujets sains voient leur stabilité posturale améliorée avec l'ajout d'information tactile. Cependant, il n'a pas été démontré si les sujets ayant eu un AVC peuvent aussi bénéficier de ces informations tactiles. Cette recherche inclut trois études visant à examiner, en premier lieu, l'impact d'un AVC sur les ajustements posturaux réactifs (APR) déclenchés par des rotations de la surface de support et, en deuxième lieu, l'effet des informations tactiles sur le contrôle de ces APR pendant deux tâches, soit la marche et la position debout. Onze sujets ayant eu un AVC et huit sujets sains, appariés pour le genre et l'âge, ont participé à ces études. Des rotations de la surface de support dans les plans sagittal et frontal (amplitude: 5°; vélocité maximale: 32°/s) étaient présentées aléatoirement aux sujets lors de la marche (phase de double appui) et en position debout (position des membres inférieurs simulant la phase de double appui). Le toucher léger (< 4N) d'une rampe fournissait l'information tactile aux sujets. Un système à six caméras d'analyse tridimensionnelle du mouvement (Vicon 512 system) a été utilisé pour acquérir les données cinématiques du corps à une fréquence de 120 Hz. L'électromyographie de surface de quatre muscles du membre inférieur a été enregistrée bilatéralement à une fréquence de 1080 Hz. Les forces de réaction du sol ont été acquises par deux plate-formes de force (AMTI) à la même fréquence. Les sujets sains ont présenté des APR fonctionnels et modulés par la direction de la perturbation et les exigences de la tâche. Ainsi, les APR

étaient diminués pendant la marche comparativement à la position debout, ce qui suggère que les exigences posturales sont moindres pendant la marche. Au contraire, la perturbation du contrôle de l'équilibre et de l'intégration sensori-motrice chez les sujets ayant eu un AVC entraîne un retard dans les APR de même qu'une absence de modulation selon la tâche et la direction de la rotation de la surface. Les membres inférieur et supérieur du côté parétique ont montré une sous-activation des réponses musculaires et une diminution de force, alors qu'une sur-activation musculaire et une augmentation de force étaient présentes du côté non-parétique. Ces modifications ont entraîné une instabilité du tronc et du centre de pression et ce, surtout dans le plan frontal. L'utilisation d'informations tactiles a amélioré la stabilité posturale chez les sujets ayant survécu à un AVC et, dans une moindre mesure, chez les sujets sains, ce qui suggère une utilité potentielle de cette technique pour la réadaptation de l'équilibre.

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To my family: dad, for forever being my role model, mom: for your support on everything, sisters and brother (Ant, Mod, Art, Oum): for the dedication of your time to help in the clinic so that I can come this far, grandma: for always reminding me to eat and keep healthy and my boyfriend, Vitoon: for always supporting and believing in me.

GLOSSARY

A/P	Anteroposterior
APAs	Anticipatory postural adjustments
BoS	Base of Support
CNS	Central nervous system
CoM	Center of mass
CoP	Center of foot pressure
CVA	Cerebrovascular accidents
H.A.T	Head, arm and trunk
MG	Medial gastrocnemius muscle
M/L	mediolateral
rCoP	resultant center of foot pressure
RFM	Rectus femoris muscle
TA	Tibialis anterior muscle
TFL	Tensor Fascia Latae muscle
TPRs	Triggered postural responses

TABLE OF CONTENTS

CHAPTER 5

CHAPTER 6

LIST OF FIGURES

CHAPTER 3

IMPACT OF STROKE ON POSTURAL RESPONSES TRIGGERED BY UNEXPECTED SURFACE PERTURBATIONS IN THE PITCH AND ROLL PLANES: I. DURING QUIET STANCE.

CHAPTER 4

IMPACT OF STROKE ON POSTURAL RESPONSES TRIGGERED BY UNEXPECTED SURFACE PERTURBATIONS IN THE PITCH AND ROLL PLANES: II. DURING LOCOMOTION.

CHAPTER 5

LIGHT TOUCH IMPROVES CONTROL OF POSTURAL EQUILIBRIUM DURING QUIET STANCE AND LOCOMOTION POST STROKE

LIST OF TABLES

CHAPTER 4

CHAPTER 5

PREFACE

This thesis is organized using a manuscript-based format. This format has been in accordance with the Guidelines for Thesis Preparation of the Faculty of Graduate Studies of McGill University (revised in March 2003).

This thesis consists of 6 chapters:

Chapter 1 is an introduction to the topic of the study, leading to the rationale and the principal objectives of the study.

Chapter 2 is a review of the literature that covers the area relevant to the study. This chapter is organized into 6 sections. Section 1 provides the overview of the control of posture and balance. Sections 2 to 6 review the current knowledge on the postural responses triggered by a sudden movement of the support surface (TPRs). To be specific, the possible control mechanism and control center for TPRs is demonstrated in Section 2. Sections 3 and 4 describe two important characteristics of TPRs regarding tasks and directions of perturbation. Section 5 reviews the impact of cerebrovascular accident (CVA) on TPRs and Section 6 provides the overview regarding the effect of light touch from the fingertip on the control of posture.

Chapters 3 to 5 consist of individual research papers. The results presented in Chapters 3 and 4 aim to determine whether a common mechanism exists for the control of TPRs during static and dynamic tasks. They also investigate the impact of CVA on TPRs during static and dynamic tasks. Chapter 5 examines whether task differences (i.e., static vs. dynamic) influence the effect of light touch on the control of posture. The possibility that stroke subjects may benefit from the use of fingertip cue in the control of posture during static and dynamic tasks is also examined in this chapter.

Chapter 6 presents the final conclusion and summary of the thesis.

STATEMENT OF ORIGINALITY

I attest to the fact that this thesis contains no material previously published or written by another person, except where references are made.

Elements of this doctoral thesis provide original contributions to the fundamental knowledge in the impact of a cerebrovascular accident on the control of balance and mobility functions. The findings of this thesis provide a foundation to identify the relationship of the control mechanisms between static and dynamic equilibrium. This thesis also suggests the use of a novel strategy involving tactile cue provided by the contact of the index fingertip with the fixed surface for balance rehabilitation following stroke. A detailed list of the most important points of original contributions is presented in Chapter 6 (summary and conclusions).

CONTRIBUTIONS OF AUTHORS

The following three manuscripts are included in this thesis:

1. Boonsinsukh R. and Fung J. (2003) Impact of stroke on postural responses triggered by unexpected surface perturbations in the pitch and roll planes: I. during quiet stance. Submitted to: *Brain Research*.
2. Boonsinsukh R. and Fung J. (2003) Impact of stroke on postural responses triggered by unexpected surface perturbations in the pitch and roll planes: II. during locomotion. Submitted to: *Brain Research*
3. Boonsinsukh R., De Serres SJ. and Fung J. (2003) Light touch improves control of postural equilibrium during quiet stance and locomotion post stroke. Submitted to: *Brain.*

I was primarily in charge of all the work included in this thesis, including the design of the studies, the experimentation, the subject recruitment, the data collection and the analysis, and interpretation of the results as well as writing the research papers.

All the work was done under the guidance of Dr. Joyce Fung. She offered the theoretical and practical suggestions on the design of the studies, subject recruitment, research methodology and data analysis and interpretation. All the data collection was carried out in Dr.Fung's laboratory at the Research Center, Jewish Rehabilitation Hospital and supported in part by a grant to her from FRSQ, CIHR, CRIR and the JRH Foundation. She was also a co-author on all papers and participated in the critical review of all manuscripts.

Dr. Sophie De Serres was integrally involved in the writing of the third manuscript by editing the content and offering a critical review of the manuscript.

CHAPTER 1

INTRODUCTION

Changes in the environment disturb posture and balance during static and dynamic conditions. In response to these disturbances, the central nervous system (CNS) generates appropriate postural reactions to restore equilibrium. Two important behavioral goals that need to be accomplished in the control of posture and balance are the control of the position and velocity of the body's center of mass (CoM), as well as the appropriate alignment of the body segments (Horak and Macpherson 1996). Failure in one or both goals leads to a loss of balance or falls. Falls can be a serious issue in health care, as they can lead to severe injuries or even death in the elderly individuals (Binder 2002). The majority of falls in the elderly are due to unexpected external perturbations of the body such as a sudden change of the support surface (Maki et al. 1994). Therefore, the understanding of postural reactions and control mechanisms for balance maintenance during unexpected external perturbations of the body are necessary for fall prevention.

Postural reactions triggered by sudden movement of the support surface (Triggered Postural Responses; TPRs) are functionally appropriate to maintain equilibrium not only during static tasks (i.e., quiet stance) but also during dynamic tasks (i.e., locomotion). However, it is still unclear whether the mechanisms which control static equilibrium could also be applied to the control of dynamic equilibrium. Under static condition, the goal of equilibrium control is to maintain the horizontal projection of the body center of mass (CoM) within the base of support (BoS). This is achieved by activating the muscles in order to adjust the forces exerted through the limbs in contact with the support surface, in a manner that the origin of the exerting forces from the

two feet, the resultant center of pressure (rCoP), coincides with the horizontal projection of the body CoM. Surface movement triggers combined actions of several muscles known as a postural strategy which reflects the control of the CNS in reducing the degree of freedom necessary for coordinating complex postural movements (Nashner and McCollum 1985). It has been shown that postural strategies triggered by movement of the support surface during quiet stance are dependent on the context of the task, such as the direction and velocity of surface movement (Diener et al. 1988; Moore et al. 1988; Runge et al. 1998), the type of base of support (Horak et al. 1997; Horak and Nashner 1986) and the limb geometry (Macpherson et al. 1989).

The goal of equilibrium control during static condition cannot be applied to the dynamic condition. During locomotion, the projection of CoM is not always within the BoS, but its trajectory is precisely controlled to maintain dynamic equilibrium (Jian et al. 1993). It has been suggested that the task of postural control in walking is entirely different from that in standing (Winter 1987). In fact, evidence from clinical practice indicates that the ability to maintain balance in one task does not necessarily ensure the same balance capability in a different task. Such findings imply that postural control strategies differ between static and dynamic tasks. Several previous studies examined postural reactions when surface movement occurred during locomotion. There is a consensus that TPRs during locomotion depend on the phase of gait cycle (Belanger and Patla 1984; Berger et al. 1984; Dietz et al. 1987; Eng et al. 1997; Figura and Felici 1986; Nashner 1980). However, none of the previous studies compares these TPRs with those made under static conditions with the same limb geometry. Therefore, it still remains unknown whether a common mechanism exists to control equilibrium during static condition and dynamic movement. ***The first principal objective of my doctoral thesis is to compare the postural control strategies triggered***

by movement of the support surface during quiet stance and locomotion.

Sensory and motor impairments following cerebrovascular accidents (CVA) can give rise to poor control of balance and mobility (Badke and Duncan 1983). How the cerebral cortex involved in postural responses triggered by unexpected external perturbation is unclear. A recent study revealed that the motor cortex did not play a role in the generation of TPRs, as transcranial magnetic stimulation to motor areas of the cerebral cortex did not modify TPRs (Keck et al. 1998). Nevertheless, abnormal patterns of muscle activities, such as excessive co-contraction and abnormal timing and sequencing of muscle activations, have been found in stroke survivors responding to external perturbations (Badke and Duncan 1983; Di Fabio et al. 1986; Dietz and Berger 1984). These problems in stroke subjects indicate that sensorimotor integration, the transduction of sensory information into appropriate motor outputs, is impaired. Some evidence suggests the misrepresentation of the body in space, for example, a misperception of the trunk orientation in subjects with hemineglect and pusher syndrome (Karnath 1994; Karnath et al. 2000b). Therefore, the central problem may be located at the level of sensorimotor integration which involves an egocentric body coordinate frame of reference leading to a misinterpretation of the body in space and, hence, disordered postural responses seen in the stroke subjects.

Previous studies examining TPRs in stroke patients, however, do not provide the overall picture of the postural responses following CVA as they do not quantify the changes in force and kinematic profiles. Thus it is still unclear as to the extent of postural control mechanisms that are affected by the CVA. So far, the findings regarding TPRs in stroke have been obtained when the perturbation was introduced in the sagittal plane, such as forward/backward translation of the support surface. However, instability in the frontal plane is a common characteristic of subjects with CVA (Dickstein et al. 1984; Shumway-

Cook et al. 1988). Furthermore, most perturbations in daily circumstances do not only take place in the sagittal direction. Therefore, it is crucial to address the postural control strategy used to maintain frontal stability. Finally, none of the studies examines the postural responses during conditions other than the static posture. How the stroke subjects respond to the perturbation in other tasks (i.e., walking) has not yet been answered. Consequently, the information regarding strategies used to improve postural control in the stroke patients is limited. Therefore***, the second principal objective of my thesis research is to determine the impact of CVA on postural responses triggered by support surface movement in both sagittal and frontal planes during quiet stance and locomotion***.

Light touch from the fingertip is likely to be another potential sensory feedback that can be used for improving the control of posture. Fingertip cue attenuates body sway by providing orientation of the body with respect to the touched surface (Jeka and Lackner 1994; Jeka and Lackner 1995). The effectiveness of the fingertip cue in improving postural control has been extensively demonstrated during static equilibrium. What remains unclear is whether light touch has a similar effect on the control of posture during dynamic tasks. Furthermore, light touch may be more useful for controlling equilibrium in subjects with postural control problems, such as stroke patients, much more than healthy subjects. The additional sensory inputs from fingertip cue may provide the potent information used to assist in the sensorimotor integration processes which are impaired in the stroke subjects. Nevertheless, whether stroke patients benefit from the light touch from the fingertip remains unanswered. ***Thus, the third and final principal objective of my thesis research is to determine whether light touch will be able to assist in the restoration of equilibrium and stability in quiet stance and locomotion in stroke subjects.***

All the principal objectives listed have been successfully accomplished in this doctoral thesis. The first two objectives were realized in two companion manuscripts that are currently under review by Brain Research:

Chapter 3 Impact of stroke on postural responses triggered by unexpected surface perturbations in the pitch and roll planes: I. during quiet stance (Boonsinsukh R. and Fung J.)

Chapter 4 Impact of stroke on postural responses triggered by unexpected surface perturbations in the pitch and roll planes: II. during locomotion (Boonsinsukh R. and Fung J.)

The third and final objective was realized in another manuscript that was submitted to Brain:

Chapter 5 Light touch improves control of postural equilibrium during quiet stance and locomotion post stroke (Boonsinsukh R., De Serres SJ. and Fung J.)

CHAPTER 2

REVIEW OF THE LITERATURE

2.1 The control of posture and balance

The ability to control posture and balance is a fundamental prerequisite for the activities of daily living. Two important behavioural goals, postural orientation and postural equilibrium, are implicated in the control of posture (Horak and Macpherson 1996). *Postural orientation* refers to the ability of a body to align body segments in relation to each other and to the environmental variables such as gravity. Postural orientation provides the reference frame for perception and action with respect to the external world (Massion 1994). For example, the relationship between the head, trunk and arm positions is used as a reference frame to calculate the target location in relation to the external environment and to plan the arm movement towards the target. *Postural equilibrium* is the condition where the forces acting on the body are such that the body remains in a targeted posture (static equilibrium) or moves in a controlled manner (dynamic equilibrium). The stability of the body or the equilibrium is mainly achieved through the control of the position and velocity in space of the body center of mass (CoM), the point where the whole body mass is balanced.

Various systems in the body contribute to the control of posture. These include the sensory systems that assess the position and trigger movement of the body in space with respect to the gravity and the musculoskeletal system that adjusts various segments through the internal representation of the body, environmental orientation (such as support conditions) and task expectations (Shumway-Cook and Woollacott 1995). Sensory information comes from the visual, vestibular and somatosensory systems. Visual inputs provide the information about the position and movement of the head in relation to the

surroundings. Inputs from the peripheral and central visual fields have been found to be equally important in providing reference of verticality (Straube et al. 1994). The visual system, however, has problem in differentiating between object (exocentric) and self (egocentric) motions. In other words, it cannot distinguish if it is the body or the surroundings that move. This deficit can be compensated by the vestibular system, as it helps the nervous system to distinguish between exocentric and egocentric motions (Horak and Shupert 1994). The vestibular system reports the head position with respect to gravity and inertial forces so it provides the gravito-inertial frame of reference. This is achieved by two types of receptors in the vestibular system, the semicircular canals that detect the angular acceleration and the otoliths which sense linear position and acceleration of the head. Although the vestibular system is able to report the position of the head, it has difficulty in differentiating the true position and motion of the body in space. For example, the vestibular system cannot distinguish between a simple head nod and a forward bend where both head and trunk move simultaneously (Horak and Shupert 1994). The information about the body segment positions in relation to each other is detected by the somatosensory system that receives inputs from joint and muscle proprioceptors, cutaneous and pressure receptors. The somatosensory information also reports the position and motion of the body in space with respect to the support surface.

Posture and equilibrium are challenged constantly by disturbing forces that arise from either inside or outside the body. Forces due to voluntary movements, such as raising the arm, are referred to as *internal disturbances* while forces which occur from the outside environment such as gravity are called *external disturbances*. External disturbances may be expected (i.e., visible obstacles) or unexpected (i.e., sudden change of the support surface). Such internal and external disturbances act to destabilize the body at its CoM. The postural control system produces appropriate muscular forces acting at

various joints and on the support surface so that the position of the CoM is controlled and equilibrium is maintained. Postural responses associated with voluntary movement (internal perturbation) are called “anticipatory postural adjustments” (APAs) as the responses start before the onset of movement. The APAs act to minimize the effect of the forthcoming disturbances due to voluntary movement in a feedforward manner. For example, the leg muscles were activated 50-100 ms prior the arm movement to maintain balance in the arm raising task during standing (Belenkii et al. 1967). The APAs are also executed to restore the body’s equilibrium during an expected external perturbation. In contrast, unexpected external perturbation triggers quick and unconscious coordinated postural responses called “triggered postural responses (TPRs)”. The TPRs are operated through the feedback circuitry using the external perturbations as triggering stimuli (Massion 1992; 1994). Muscle activations needed to maintain balance on a moving bus are one example of TPRs.

When the postural control system fails to properly respond to disturbances, falls occur. Falls in the elderly are a major concern in health care as they can result in severe injuries, which can affect a person at both personal and social levels. One third of community-dwelling elderly over the age of 65 years experienced at least a fall each year and the number of fallers increases to 50% by the age of 80 years (O'Loughlin et al. 1993; Vellas et al. 1998). It has been found that the majority of falls in the elderly are due to external perturbations of the body. More than half of all falls (54%) are caused by sudden changes in the support surface that result in slips and trips (Maki et al. 1994). Therefore, although both types of postural responses, triggered and anticipatory, are necessary in every day situations, the study of TPRs is the main interest in this review where fall prevention is a major concern.

2.2 TPRs are centrally organized at multiple level of the neuraxis

Triggered postural responses have been extensively studied by using sudden movements of the support surface to simulate slip or trip during standing and walking. Sensory feedback about the nature of the surface movement, such as velocity and amplitude of movement, is necessary for triggering postural responses during unexpected surface perturbation (Dietz 1992; Macpherson et al. 1986). Although integrated feedback information from visual, vestibular and somatosensory inputs are required, it has been shown that most of the TPRs to unexpected surface perturbation rely more on the somatosensory information from the body segments than the vestibular inputs (Dietz et al. 1991; Dietz et al. 1985; Inglis and Macpherson 1995). This may be based on the limitation of the visual and vestibular systems in detecting the orientation and configuration of the body segments as both receptors are located in the head and detect the movements of the head independently from the movement of the trunk and limb whereas somatosensory receptors located throughout the body are more sensitive to changes in body orientation and configuration.

Unlike the simple stretch reflex response where the response is seen only in the muscle that is stretched, a surface perturbation triggers TPRs that can be seen in the whole body. Thus, TPRs cannot be explained in term of simple stretch reflex responses that are mediated through monosynaptic pathways (Ia) (Berger et al. 1984; Dietz and Berger 1984; Dietz et al. 1985; Horak and Nashner 1986). The stretch reflex can even be disadvantageous for postural responses to some types of perturbation (Allum et al. 1989). For example, a toes-up rotational perturbation stretches ankle joint muscles in the same way as does a backward platform translation. The first muscles recruited after platform onset are the plantarflexors due to stretching. However, the continued activity of stretched plantarflexors in the toes-up rotation perturbation is not appropriate since it will bring the body backward

beyond its stability limits. Therefore, the activity of ankle plantarflexors is quickly suppressed and ankle dorsiflexors act to maintain balance in toes-up rotation perturbation.

Supporting the notion that TPRs are not mediated through the stretch reflex pathway, Berger and colleagues (1984) showed that TPRs persisted during ischemic blockage of Ia fibers in the legs. The longer-than-stretch reflex onset latency of leg muscle activities suggests that TPRs may be mediated either through group II afferents over polysynaptic spinal pathways or through supraspinal pathways (Berger et al. 1984). However, recent studies in spinalized cats demonstrate that supraspinal pathways rather than the polysynaptic spinal pathways are possibly the pathways used to control equilibrium following unexpected external disturbances. During standing, the spinalized cats are able to maintain a certain degree of body orientation through spinal reflexes and joint stiffness (Fung and Macpherson 1999) but they cannot maintain balance during displacement of the support surface (Macpherson and Fung 1999). Thus, it appears that different mechanisms are used to control postural orientation and postural equilibrium.

While the control for body orientation relies heavily on spinal circuitry, the control of equilibrium requires more supraspinal control (Fung and Macpherson 1999; Macpherson and Fung 1999). The brainstem and cerebellum are implicated as the supraspinal control center because they are the sites for the integration of vestibular, visual and somatosensory information (Frank and Earl 1990; Horak and Macpherson 1996). Outputs of brainstem and cerebellum are transmitted to the secondary and supplementary motor regions of the cerebral cortex, which may serve to set the direction and gain of the postural responses (Frank and Earl 1990; Horak and Macpherson 1996). Therefore, TPRs are not a combination of segmental stretch reflexes but possibly the result of more complex neural mechanisms that are organized at multiple levels in the central nervous system.

2.3 TPRs are task-dependent

2.3.1 Goal of postural control in quasi-static and dynamic tasks

Postural control is a dynamic process as the body is never completely still, even in a stationary position such as standing. During quiet stance, muscle forces are exerted on the support surface as reflected by the ground reaction force, to control the continuous movement of the body CoM. To be balanced, motion of the point of origin of the ground reaction force, the center of pressure (CoP), should correspond to the movement of the horizontal projection of the CoM. The term “quasi-static postural task” has been introduced to represent this task in which continuous postural control is required.

The task of maintaining balance in a fixed position (quasi-static task) is different from keeping balance when a person is moving from one point to another (dynamic task) as in locomotion. In a quasi-static task, the body moves over the stationary base of support (BoS), the area limited by the contact points between the body segments and the support surface. The goal of postural control in this case is to maintain the horizontal projection of the CoM within the BoS. In contrast, during walking, the CoM moves and the BoS changes its configuration continually. With the forward momentum of the body, the CoM moves anteriorly and the projection of CoM may not pass directly within the BoS. For example, the CoM lies posterior and medial to the stance heel during the start of the single support phase (Winter 1990). Winter and colleagues (1990) proposed that during walking, the body is in a continuous state of imbalance. Fall is avoided by positioning the swing foot ahead of and lateral to the moving CoM. Thus, the goal of postural control in walking must differ from that in standing, suggesting the presence of completely different postural control mechanisms between these two tasks (Winter 1987). On the contrary, the basic joint linkage to be controlled in

either task during the same exact position is very similar so it would seem more advantageous for the nervous system to use similar control strategies with minor adjustments. In fact, Yang and colleagues (1990) found that the same postural control strategies, which counteract perturbations during quiet stance, are also effective during the stance phase of walking (Yang et al. 1990a; 1990b). Their findings, however, are obtained from postural responses during 80 ms after perturbation onset in which they represent the responses from the skeletal and ligamentous systems as well as stretch reflexes. Thus, information about active postural control mechanisms (i.e., muscular system) during standing in relation to during walking is still lacking.

A recent study derived from postural control when adapting to expected changes in surface inclination has revealed different control requirement of the body between standing and walking (Leroux et al. 2002). It has been shown that the trunk and pelvic orientations during standing and walking on the inclined surface are not the same. During walking, the trunk and pelvis tilted forward as the surface inclination changed towards the uphill direction whereas both body segments tilted backward when the surface moved towards downhill slope. It was suggested that these changes in trunk and pelvic orientations during walking were aimed to assist power generation and absorption in accommodating to the changes in surface inclination. In contrast, the trunk and pelvis remained erect aligning with the vertical at any surface inclination during standing. This finding implicated the task-specific characteristic of postural response in which a different control is required to stabilize the body during standing and walking on an expected and fixed surface inclination (Leroux et al. 2002). However, this information may not be valid for postural control during unexpected surface changes. Inputs to the nervous system are different between expected and unexpected perturbations and hence the control mechanism may not be the same. Thus,

it is still unclear as to how postural control, more specifically TPRs, is organized during quasi-static and dynamic tasks.

2.3.2 Postural control of the H.A.T during walking

The challenge for the postural control system during walking is the regulation of the heavy mass of the head, arm and trunk (H.A.T.) over two supporting limbs. In walking, H.A.T travels mostly in the forward-backward direction, thus, a regulation of position and acceleration of H.A.T. in the sagittal plane is necessary. In fact, it has been found that the majority of the work performed during locomotion is in the plane of progression (sagittal) and is done by the hip flexors/extensors (Eng and Winter 1995; Winter et al. 1993). A person normally regulates the H.A.T. in the vertical direction during walking. Human erect locomotion is unique and separates human from other living primates. Grasso et al (2000) demonstrated that the mechanical energy expenditure during erect walking was lesser than when a person developed the bent walking posture (i.e., flexed knee and flexed trunk-flexed knee). Higher level of muscle activities with different muscle activation pattern on the lower limbs were seen during bent walking as compared to erect walking. In the bent posture, the limb was displaced away from the main axis of the limb leading to the reduction of the mechanical advantage of the muscles to support the body weight during the stance and to move the limb during the swing phase. As a result, more muscle activities as well as coupling of new muscle groups were required to generate the appropriate joint torques and to adapt to change in mechanical constraints during bent walking (Grasso et al. 2000). Therefore, the erect regulation of H.A.T. is an efficient strategy to preserve energy expenditure during walking.

2.3.3 TPRs in walking

Another challenge for controlling equilibrium during locomotion is the adaptability of the locomotor system to changes in the environment. Since most falls in the elderly individuals occur when facing unexpected surface perturbation during walking, the control of posture and equilibrium during walking draws a lot of attention from researchers. In 1980, Nashner introduced three types of unexpected surface perturbation: horizontal translation, vertical displacement and rotation were applied during four phases of walking. TPRs were characterized by the EMG activity of tibialis anterior and gastrocnemius muscles, the ground reaction forces and the kinematics of the lower limbs. The results showed that normal walking is maintained by adjusting kinematic, kinetic and EMG profiles of the ongoing step. TPRs are phase-dependent and maximal during heel strike and single support phase of walking (Nashner 1980). Also, it has been found that the strength of TPRs is dependent on the acceleration, amplitude and predictability of the perturbation (Dietz et al. 1987). In addition, TPRs are perturbation specific (Nashner 1980). For example, a toes-down rotation perturbation disrupts the excursion of the supporting ankle joint and changes the force exerted by the limb, which help regulate the rate of forward progression of the body. In contrast, downward platform perturbation alters the vertical height of the supporting leg leading to the extension of the leg to help regulating the height of the body. Therefore, Nashner demonstrated the adaptive ability of the postural control system that can complete the task even in the presence of the perturbation. He also showed that TPRs during walking closely resembled TPRs to the same perturbation during standing. However, this conclusion cannot be applied to a more general situation as only two muscles were investigated in this study.

Phase-dependent and *perturbation-specific* characteristics of TPRs have been supported in other studies (Belanger and Patla 1984; Berger et al.

1984; Dietz et al. 1987; Eng et al. 1997; Figura and Felici 1986). Deceleration and acceleration of treadmill speed have been used to perturb the walking pattern (Berger et al. 1984; Dietz et al. 1987; Figura and Felici 1986). At the beginning of the stance phase, treadmill deceleration displaced the supporting leg forward in relation to the CoM, causing quick *backward* touch-down of the contralateral foot to counterbalance posterior movement of the CoM. In contrast, treadmill acceleration leads to a backward movement of the weight bearing leg in relation to CoM, resulting in an early *forward* touch-down of the contralateral foot (Berger et al. 1984). Not only the perturbation at the feet elicits perturbation-specific characteristics during walking, a perturbation at the axial segment of the body also triggered similar responses (Misiaszek et al. 2000). Misiaszek et al (2000) used the weight pulling at the waist to trigger postural responses during walking. They found that unexpected backward displacement of the body CoM by pulling the waist backward caused an increase in TA activation on the supported limb at an approximately similar onset latency and amplitude as unexpected backward displacement of the body CoM triggered by deceleration of the treadmill (Berger et al. 1984) or forward translation of the support surface (Tang et al. 1998). In most cases, the EMG pattern of the next step cycle does not change such that the adjustments can be achieved within the perturbed step cycle. The prolongation of the stance phase is occasionally evident and can be viewed as the compensatory mechanism to ensure that the stance leg remains in place to support the body CoM until stability is achieved. On the other hand, the lengthening of the stance duration may reflect the requirement to readjust the position of the CoM after having been displaced and more time is needed to reposition the CoM before allowing the locomotion to continue (Misiaszek et al. 2000).

From the previous findings, it can be seen that the control of posture is effectively integrated into the control of locomotion to ensure that locomotion

continues under different environmental context. It has been suggested that the integrated control of locomotion and posture is possible as both functions share some common organization principles (Lacquaniti et al. 1997; Massion 1992). For example, both postural responses and locomotion seem to use the vertical direction as the frame of reference while the position of the CoM is the controlled variable for both tasks. Although very little is known about the localization of the postural control center in the brain, the fact that TPRs can be easily accomplished during walking suggests a closed connection between postural control and locomotor centers.

The integrated control of posture and locomotion has been shown by stimulation of the specific sites in the brainstem and hypothalamus, causing the cats to change into different locomotion and postural styles (Mori et al. 1989). For example, stimulation of the lateral hypothalamus caused the cat to perform flexed locomotion. Moreover, the basal ganglia has been found to be involved in generating the spatio-temporal framework for the control of trunk geometry and the lower limbs coordination with respect to gravity during walking (Garcia-Rill 1986). Recent study in Parkinson's patients confirmed the role of the basal ganglia in the control of posture during locomotion (Grasso et al. 1999). Parkinson's patients walked with the trunk bent forward and the lower limbs flexed. However, when the internal globi pallidi were stimulated with the implanted electrode, the patients showed a better alignment of the trunk, close to vertical, and a fuller extension of the lower limb prior to heel strike.

Muscles of different body segments contribute unequally to TPRs during locomotion. Figura and colleagues (1986) showed that although TPRs occur on both sides of the body, the responses during the stance phase of walking are larger on the perturbed limb than those on the swing limb. With regards to the supported limb, the more significant changes are accomplished at the ankle (Figura and Felici 1986) and knee joints (Tang et al. 1998) while

muscle activities of the hip and trunk remain relatively unchanged (Tang et al. 1998). This suggests that balance corrections during surface perturbation can be adequately accomplished by distal muscles without requiring a special activation of more proximal muscles (Tang et al. 1998).

Unexpected perturbation during the swing phase of walking has been observed by applying noxious stimuli to the foot (Belanger and Patla 1984). The lift-off and subsequent swing phases are likely facilitated to quickly avoid the stimulus when it is present at the toe-off phase, while the foot is quickly lowered to the ground and the knee is prevented from collapsing when the stimulus is applied at the late swing. Eng and colleagues (1997) proposed two main movement strategies, elevating and lowering strategies, used for postural adjustments during swing phase perturbations. In response to early swing phase perturbation, an elevating strategy, characterized by a flexion of the hip, knee and ankle joints of the swing limb, is operated. This is to enhance toe clearance and CoM elevation so that additional time is allowed for extending the swing limb in preparation for landing. During late swing, the body is decelerated and prepared for landing. Perturbation during this phase of walking leads to the use of a lowering strategy, consisting of a rapid lowering of the swing limb to the ground with a flexed knee and a plantarflexed ankle (Eng et al. 1997).

In brief, it is evident from previous studies that TPRs to unexpected perturbation are functionally appropriate to maintain equilibrium during different tasks. However, the relationship between postural control during quasi-static and dynamic tasks remains unclear. The difference in goal of postural control during each task suggests dissimilar control strategies between the two tasks. In fact, the study of balance training likely supports differences in postural control strategies between standing and walking. It is shown that balance training in standing improves symmetrical standing balance in hemiplegic subjects but does not lead to the reduction in

asymmetrical limb movement patterns associated with hemiplegic locomotion (Winstein et al. 1989). This suggests that postural control is task-specific and the ability to maintain balance in one task (or position) does not necessary ensure the same balance capability in other tasks. Therefore, the study of postural control needs to be done in both quasi-static and dynamic tasks.

2.4. TPRs are context-dependent

2.4.1 Strategies used for responding to perturbed standing

Another characteristic of TPRs is that responses are different according to the context of the task, such as the direction of surface perturbation, the speed of perturbation and the type of base of support (i.e., wide or narrow). Various sets of TPRs can be seen as a result of changes in the nature of the perturbation. TPRs to anteroposterior (A/P) platform translation involve the activation of particular muscle groups with specific amplitude and latency relationships. Two primary patterns of responses have been labeled “ankle” and “hip” strategies (Horak and Nashner 1986). The ankle strategy implicates the exertion of ankle joint torques on the support surface to reverse the direction of CoM displacement. The sequential activation of muscles from ankle, knee and hip (distal to proximal) moves the body around the ankle joint with relatively minimal motion at the hip and knee joints. In the hip strategy, there is marked hip joint rotation and trunk motions with an early recruitment of more proximal trunk and thigh muscles, respectively (Horak and Nashner 1986). Different postural strategies may be due to different goals and contexts of the task. It has been suggested that the ankle strategy is executed during a slow, small perturbation of a firm support surface and it maintains the trunk vertical orientation while moving the CoM (Horak et al. 1997). The hip strategy, on the other hand, is chosen when there is larger amplitude of surface displacement or when standing on a narrow surface (Horak et al. 1997), this strategy being more effective at countering

large disturbances with little concern about trunk vertical orientation (Kuo and Zajac 1993). The rule of strategy selection, however, is not rigid in that a mixed strategy, a combination of hip and ankle strategies, has also been seen (Horak and Nashner 1986). However, this mixed strategy has been viewed as a less stable strategy when compared to the ankle or hip strategies (Ko et al. 2001).

Recent studies suggested that the hip and ankle strategies are not separate responses but rather a continuum of reactions triggered under progressively changing external constraints. Horak and Moore (1993) demonstrated this continuum of reactions when a person leaned forward about the ankles to five different initial stance positions. The continuum involved gradually less ankle strategy and more hip strategy when the amount of leaning forward increased (Horak and Moore 1993). Supporting this concept, Ko et al (2001) reported four distinct strategies when varying the frequency of support surface sinusoidal translation in the anteroposterior direction. For a frequency of surface perturbation varying from low to high, the postural response began with no movement in all joints (rigid strategy) to movement at only the ankle joint (ankle strategy), progressing to movement at the ankle and hip joints (ankle-hip strategy) to finally movements of hip, knee and ankle joints. A person stiffened all joints (rigid strategy) during a slow translation because the inertia forces acting on the body were minimal. However, as the inertial forces increased during the high translation frequencies, stiffening the joints led to postural instability. Thus, reducing joint stiffness and generating movement starting at the ankle joint to dissipate the forces induced by the platform translation become necessary.

The strategy used in reacting to surface perturbation involves the combined action of synergistic muscles. It is believed that in order to reduce the degree of freedom necessary for coordinating complex postural movements, the nervous system produce responses by activating functional

synergies, consisting of a set of muscles regulated as a whole (Nashner and McCollum 1985). These patterns of activation can be modified by intent (Burleigh et al. 1994), anticipation (Burleigh and Horak 1996), surface configuration (Horak and Nashner 1986), initial alignment (Horak and Moore 1993) and practice (Horak et al. 1989).

2.4.2 Central organization of TPRs

Studies of muscle activities to multiple directions of surface translation provide further information about the control of TPRs. Direction-specific characteristics of muscle recruitment have been demonstrated (Henry et al. 1998b; Moore et al. 1988). Distal muscles such as tibialis anterior and medial gastrocnemius are recruited when a perturbation is presented in specific directions with the maximal activation when the perturbation is in one of the diagonal plane. More proximal muscles, hamstring, quadriceps, hip adductors, tensor fascia latae, rectus abdominis, and erector spinae, are activated in the broader direction with two different directions of maximal activities (Henry et al. 1998b). It is proposed that distal muscles may only produce movement at the ankle but more proximal muscles may function as prime movers as well as stabilizers (Moore et al. 1988). In addition, it has been demonstrated that the direction of maximal muscle activation does not always correspond to the anatomical orientation of the muscles.

While it is in agreement that muscle synergies are utilized during postural control to multiple perturbation directions, there is a conflict of opinion on the pattern of postural responses. Moore and colleagues (1988) found that TPRs are discrete in that for any particular perturbation direction there appears to be a single unique response. They argued that the organization of TPRs is likely to be more than just the summation of a few discrete muscle synergies. In contrast, Henry and colleagues (1998) demonstrated that there is a flexible continuum of muscle synergies, not a

unique muscle synergy for each translation direction, and that these synergies can be altered depending on the context of the task. The disagreement may reside in the difference in the experimental procedure. In Moore and colleagues' study, the subjects were changing their foot placement so that they were able to predict the perturbation direction while Henry and colleagues' study used a newly designed platform that moved in any direction in the horizontal plane to reduce anticipatory influences. These contradictory findings show that it is still unclear how TPRs are organized and controlled. To further unravel the TPRs control mechanisms, the study of TPRs, in term of kinematics and kinetics in addition to EMG profiles, to multiple perturbation directions is necessary.

2.4.3 Neural triggering center for TPRs

Modification of the surface perturbation pattern (i.e., translation versus rotation of support surface) provides information about where TPRs are triggered. It was once thought that TPRs are triggered by stretch-related proprioceptive inputs from ankle joint muscles (Horak and Nashner 1986; Nashner 1977). For example, during backward platform translation, the ankle plantarflexors are stretched and are responsible for the balance correction. Results from rotational platform perturbation showed that reflex activities from the stretched muscles are not necessarily the appropriate responses used in balance correction. This can be seen in the inhibition of early reflex activity from the stretched calf muscles during toes-up rotation to prevent the body displacement further backward and activation of the tibialis anterior muscle for correcting the stance posture (Allum et al. 1989). Moreover, TPRs can be triggered independent of ankle stretch inputs (Alexander et al. 1992; Nardone et al. 1995). Allum and colleagues (1995) further support the concept that ankle inputs are not responsible for triggering balance responses during surface movement. They invented the ankle-nulling protocol which was a

combination of backward translation and plantarflexion rotation of the support surface. This protocol minimized the ankle inputs during 200 ms after a surface perturbation, but allowed more inputs from the knee joints because considerable knee flexion was elicited. Their results showed that without ankle inputs, normal onset latencies of TPRs were still present. The findings suggested that the proprioceptive from the knee or trunk and the vestibulo-spinal inputs rather than the ankle sensory inputs are involved in the generation of muscle responses during balance corrections (Allum et al. 1995).

Vestibular inputs have been suggested as the possible triggering signals as the head is accelerated during surface perturbation (Dietz 1992). However, the findings that response latencies were normal in patients with bilateral vestibular loss under no vision condition during surface translation argued that both vestibular and visual inputs are not used to trigger postural responses due to surface translation and surface rotation (Allum et al. 1994; Horak et al. 1990). It has been suggested that the vestibular system may have a role in modulating the magnitude of postural responses as the amplitude of muscle synergy of the lower limbs and the trunk are altered during surface perturbation in patients with vestibular loss (Allum et al. 1994). Allum and colleagues (1998) modulated various sensory inputs (i.e., eyes closed and eyes open, fixed ankle or knee movement conditions) during a combination of surface translation and rotation in subjects with bilateral peripheral vestibular loss. Their results showed that TPRs were preserved when vestibular, ankle and knee inputs were absent. Thus, they suggested that the trunk, hip and upper leg proprioceptive inputs were generating the timing of TPRs whereas the vestibular inputs were influencing the response amplitude modulation (Allum and Honegger 1998).

Subsequent studies were conducted to distinguish the location of triggering center that is more proximal than the ankle. When performing

ankle-nulling technique with diabetic patients with peripheral neuropathy, normal timing of TPRs was preserved even though the patients lost proprioception on the lower leg (Bloem et al. 2000). Therefore, this study confirmed that ankle and lower leg inputs are unlikely to be responsible for triggering the TPRs. The ankle and lower leg inputs are, on the other hand, used to trigger stretch reflex, as can be seen from the diminished or absent early stretch gastrocnemius reflex caused by a toes-up rotation in the patients with peripheral neuropathy (Bloem et al. 2000). Since the ankle-nulling technique elicited larger motion at the knee, Bloem et al. (2000) suggested that ankle and lower leg inputs are mainly used to trigger some automatic postural responses when the knee motion is minimal, such as during an inverted pendulum response. However, when a large knee movement occurs, the triggering center is not at the ankle level but may reside at a more proximal part such as the knee, hip or trunk level (Allum and Honegger 1998; Allum et al. 1995; Bloem et al. 2000). The existence of TPRs in a patient with total proprioceptive loss in the whole legs and using ankle-nulling technique excludes the possibility that the knee inputs are used to elicit TPRs (Bloem et al. 2002). Earlier activity seen in trunk muscles, tensor fascia latae (Henry et al. 1998b) and in paraspinal muscles (Carpenter et al. 1999) than in the lower limb muscles suggests that proprioceptors at the level of the trunk or hip are likely to be triggering centers. In addition, the trunk was suggested to provide information about the direction of perturbation because of its differential sensitivity to perturbation directions, i.e., more sensitive to a roll plane than a pitch plane perturbation (Carpenter et al. 1999).

In brief, TPRs to platform perturbations are dependent on the type and direction of the perturbations. Studies in which the direction of surface perturbation is changed increase our knowledge about the control mechanism of TPRs, including the pattern of responses, the controlled variables and the direction-specific triggering centers. Studies of postural responses to different

perturbation directions not only provide such useful information but they are also important in simulating the perturbation occurring in real life. Studies with perturbations in only one plane will not sufficiently explain how people respond to real life disturbances, which can happen in any plane or any direction.

2.5 TPRs are disrupted by cerebrovascular accidents

2.5.1 The need to study TPRs in patients with stroke

Cerebrovascular accident (CVA) generates a considerable socioeconomic impact worldwide. It is the third most common cause of death after myocardial infarction and cancer (Department of Health 1999). The overall incidence rate of CVA ranges from 200 to 700 per 100,000 populations (Sudlow and Warlow 1997). Approximately 80% of people who suffered from a CVA survive but 65% of the survivors are functionally dependent (Wolfe 2000). This makes CVA the major cause of impairment and disability (Teasell et al. 2002). It was also found that elderly people with CVA are at a higher risk for falling than the healthy elderly individuals (Foster and Young 1995). One third of stroke patients sustained at least one fall in the rehabilitation unit (Teasell et al. 2002) and 73% of them fell at least once in the six months after their discharge from the hospital (Foster and Young 1995). However, the number of serious injuries caused by fall, such as a fracture, is small (Hyndman et al. 2002; Teasell et al. 2002). Nevertheless, falls without serious injury may lead to activity restriction, functional decline and further social isolation together with a loss of independence (Shumway-Cook et al. 1997; Tinetti and Williams 1998). Falls are also associated with extensive increases in annual health care costs (Rizzo et al. 1998).

Major factors causing falls are balance and mobility impairments (Shumway-Cook et al. 1997). For example, stroke patients in the rehabilitation center often fell from beds and wheelchairs and those who fell

tended to have lower motor and mobility functions (Teasell et al. 2002). Likewise, most falls in community-dwelling strokes occurred during walking, primarily as a result of loss of balance (Hyndman et al. 2002). Thus, it is likely that treatment techniques aimed to improve motor and balance functions in CVA survivors may reduce the incidence of falls in this population.

2.5.2 The role of cerebral cortex in TPRs

Following a cerebrovascular accident, areas on the cerebral cortex and some parts of the internal capsule are usually damaged. The resulting sensory and motor impairments often lead to postural control and mobility dysfunctions (Badke and Duncan 1983). While it is well known that motor areas of the cerebral cortex are involved in the execution of voluntary movement, their roles in postural responses to unexpected external perturbation are unclear (Massion 1992). Dietz and colleagues (1984) demonstrated that TPRs during perturbed locomotion are not likely to be mediated through transcortical routes. In fact, a recent study found that the motor areas of the cerebral cortex are not involved in the generation of TPRs during perturbed standing (Keck et al. 1998). In this study, it is shown that transcranial magnetic stimulation to the motor areas of the cerebral cortex during perturbed standing does not modify TPRs. Thus, the motor cortex does not have a role in generating TPRs.

It is found that the cerebral cortex may be involved in providing the body coordinates for the egocentric frame of reference. Information from visual, vestibular and proprioceptive signals as well as tactile and auditory inputs is involved in the organization of these egocentric, body-centered coordinates used for arranging the body configuration and orientation in space (Lackner 1988). The study of hemineglect (i.e., ignoring one side of the body) and pusher syndrome (i.e., constantly pushing the body away from the unaffected side) in stroke patients identified two different neural pathways for

controlling body coordinates; one that senses the orientation perception of the visual world and another that detects the orientation of gravity (Karnath 1994; Karnath et al. 2000a, b). Karnath (1994) demonstrated that unilateral neglect stroke patients perceived the visual target as “straight ahead” when the target was actually deviated approximately 15 degrees toward the nonparetic side. Misperception of visual verticality in the hemineglect stroke patients is due to a disturbance of cortical structures that transform sensory inputs from the periphery into the non-retinal spatial reference frame (Karnath 1994). It is evident that the right inferior-posterior parietal cortex, right premotor frontal cortex and posterior and medial portions of the thalamus play an important role in the non-retinal spatial coordinate transformation in humans (Fogassi et al. 1992; Galletti et al. 1989; MacKay and Riehle 1992). Inaccurate egocentric coordinate perception leads to inaccurate motor performance in space (Bisiach et al. 1990).

While unilateral neglect stroke patients demonstrated misperception of visual verticality, stroke patients with pusher syndrome had normal visual perception in space. However, the “pusher” stroke showed the alteration in perception of body orientation in relation to gravity (Karnath et al. 2000b). They perceived the body as oriented “upright” when the body was tilted 18 degrees to the nonparetic side. The MRI study of “pusher” stroke patients’ brains depicted the infarction of the posterolateral thalamus and its projection into the posterior limb of the internal capsule, suggesting that these areas were likely to be involved in the neural representation of the graviceptive system in human to control upright posture (Karnath et al. 2000a). Thus, two different symptoms in stroke patients lead to the assumption of two separate systems responsible for body orientation with respect to the gravity. Karnath et al. (2000) suggested that both graviceptive systems rely on different sources of inputs. In fact, Mittelstaedt (1992) proposed that the orientation of visual and head verticality was detected through the sensory receptors in the

head and neck, such as visual, vestibular and neck proprioceptors. In contrast, the trunk posture was mainly perceived through the receptor in the trunk, such as information from the kidneys and through the body mass inertia (Mittelstaedt 1992).

2.5.3 The charcteristics of TPRs in patients with stroke

The study of triggered postural responses in stroke population has been used to unravel the role of the supraspinal center in the control of posture. Badke et al. (1983) demonstrated abnormal muscle coactivation of paretic limb in stroke patients during the anterior and posterior surface translations in standing. Healthy subjects contracted their gastrocnemius and hamstring muscles simultaneously during forward sway and paired tibialis anterior and quadriceps muscles during backward sway. In contrast, the stroke patients frequently contracted all four muscles at the same time or contracted both knee muscles simultaneously. The latency of the muscle responses in stroke patients was delayed with the proximal muscles activated earlier than the distal muscles. This proximal-to-distal activation pattern in stroke patients was different from the distal-to-proximal muscle activation pattern found in healthy subjects (Badke and Duncan 1983). Di Fabio et al. (1986) found another pattern of abnormal muscle activation on the paretic limb in stroke patients in response to support surface translation. The patients demonstrated two extreme muscle activation patterns including no coordination between distal to proximal muscles and near simultaneous activation of proximal and distal muscles (Di Fabio et al. 1986). Abnormal patterns of muscle activation indicate that the stroke patients have difficulties in selecting and modulating centrally-programmed adjustments and movement patterns (Badke and Duncan 1983).

Dietz et al. (1984) studied the coordination between the two lower limbs during postural responses by asking the patients to stand on a half

circle surface (the see-saws) and using a tibial nerve electrical stimulation to induce backward sway in standing. The perturbation applied to one lower limb created TPRs in bilateral tibialis anterior muscles (TA) in healthy subjects with a similar onset latency. Hemiplegic subjects showed prolonged and slightly reduced TA responses on the non-paretic side that could be due to the impairment of the ipsilateral uncrossed fibers of the descending pathways (Dietz and Berger 1984). In contrast, the TA responses on the paretic limb were significantly reduced. The amount of EMG responses in the paretic limb has been found to directly correlate with the degree of severity of the cerebrovascular accidents (Dietz and Berger 1984). Dickstein et al. (1989) further reported that low muscle tone on the paretic leg is associated with lower muscle activation, while increased muscle tone corresponds to tonic or co-contraction of antagonistic muscles. To maintain balance when muscle tone on the affected limb is low, the unaffected limb compensates by co-contracting the agonist and antagonist muscles (Dickstein et al. 1989).

In response to the see-saw situation and tibial nerve electrical stimulation, the onset latency was normal on the non-paretic limb but approximately 20-30 ms delayed on the paretic limb, indicating an alteration in the temporal coupling of the interlimb coordination (Dietz and Berger 1984). It is suggested that interlimb coordination is mediated by a spinal interneuronal pathway which is modulated by the supraspinal (i.e., cerebrum and cerebellum) control (Dietz 1992). The fact that delayed onset latency on the paretic limb occurred no matter which limb was perturbed implied that afferent inputs were not responsible for this paretic delay but supraspinal impairments in stroke patients were the cause of delayed and reduced paretic muscle activations (Dietz and Berger 1984). Another possible explanation for the delayed onset latency of TPRs is that the patients likely relied upon redundant visual and vestibular controls (Di Fabio and Badke 1991), hence,

more time was required for the information to be processed through the visual and vestibular pathways (Badke and Duncan 1983).

Despite the impairment in organizing muscle coordination in postural responses, the ability to adjust the TPRs to different context is preserved in the hemiplegic subjects. Stroke patients were able to suppress the activity of the stretched muscles during surface rotation in the toes-up and toes-down tilts as those muscle activities led to enhanced instability and they were able to activate the appropriate muscles to maintain balance. However, the magnitude of the suppression was not uniform with a higher suppression of the proximal (thigh) than the distal (shank) muscles. With the delayed activation of paretic distal muscles, the proximal muscles of the non-paretic limb were activated faster than the healthy subjects' to compensate for the paretic delays (Di Fabio et al. 1986).

From these studies, it can be clearly seen that the impairment of the supraspinal pathways caused by a cerebrovascular accident leads to difficulties in integrating sensory information from the periphery that is crucial in the selection of appropriate muscle activation patterns in response to balance perturbation. As a result, the temporal coupling of the interlimb coordination is disrupted. Also, abnormal TPRs (i.e. muscle activation) on the paretic limb with various compensations on the non-paretic limb were evident. These findings, however, were obtained from studies of postural responses to perturbation in the sagittal plane alone and in standing only. How the hemiplegic subjects maintain balance during perturbation in other directions or during other tasks (i.e. walking) have not yet been answered. A common characteristic of stroke patients during standing is an uneven weight-bearing with more body weight borne on the non-paretic than the paretic limb (Bohannon and Larkin 1985; Dickstein et al. 1984). It has been shown that the asymmetrical limb-loading pattern is associated with excessive body sway in the frontal plane and a decrease in lateral stability (Dickstein et al. 1984;

Shumway-Cook et al. 1988), leading to frequent falls towards the affected side (Diller and Winberg 1970). Therefore, the ability to maintain balance when a perturbation is presented in the medio-lateral direction is also necessary for fall prevention in stroke patients. Moreover, the previous studies focused mainly on EMG profiles, which represent only the information about muscle activities, with little regard to changes in the position of the body (kinematics) and in the forces acting on the body (kinetics). To provide a complete picture of the postural control mechanisms, information from the kinetic and kinematic profiles is required in addition to the pattern of muscle activation provided by the EMG profiles.

2.6 TPRs can be improved by somatosensory cues of the environment

2.6.1 Sensory feedback improves postural control in patients with CVA

During neuromuscular rehabilitation, patients need to develop a new motor program or action plan. Intrinsic (i.e., joint proprioception) and extrinsic (i.e., instruction) feedbacks are necessary for acquiring this new program. Many neurological disorders are partly characterized by a loss or a reduction of sensory feedback, resulting in difficulty in relearning motor tasks. Various forms of sensory feedback given to hemiplegic patients have successfully improved the performance. For example, the combined use of biofeedback and functional electrical stimulation to tibialis anterior and gastrocnemius muscles improves flexion of the knee and ankle during the swing phase of walking. This improvement in gait function is also shown in the increased gait velocity (Cozean et al. 1988). Sensory feedback has also been used to improve postural control in stroke patients. Hemiplegic patients who are provided with auditory (Wannstedt and Herman 1978) or visual feedback (Winstein et al. 1989) about their relative weight distribution (affected vs. unaffected limb) during standing demonstrate a significant improvement in

maintaining balance with more symmetrical weight distribution during standing. It has been found that stroke patients with hemineglect showed a misperception of visual verticality such as perceiving the 15-degree rightward shift of the visual target as “straight ahead”. With the combination of the galvanic stimulation on the left vestibular apparatus and the vibration of the left neck muscles, the hemineglect patients were able to perceive the correct visual verticality by increasing the awareness of the left side of their body (Karnath 1994).

2.6.2 Light touch assists postural control in healthy subjects

Somatosensory information from the fingertip, or haptic cues, is likely to be another potential sensory feedback for helping the control of posture. Obtained from lightly moving the finger across or touching any objects, haptic cues underlie the perception of body image. Cutaneous receptors at the skin surface and proprioceptive information from the muscles, joints and tendons are responsible in the perception and creation of internal representation of the body (Matthews 1988). The use of haptic cues in postural control has been reported earlier by Gurfinkel et al. (1993). In their experiment, the body was slowly rotated while the head was kept stationary. This procedure created the illusory sensation of head rotation with respect to the stationary trunk. Grasping the rigid handle fixed to the ground abolished the illusory head rotation and established the awareness of trunk rotation (Gurfinkel and Levik 1993). Later studies have demonstrated that when touching the rigid immobile object with one finger, somatosensory cues from the fingertip reduce postural sway during quiet tandem standing (Jeka and Lackner 1994; Jeka and Lackner 1995) and single limb standing (Holden et al. 1994) with eyes closed. The fingertip cues are so prominent that they also attenuate body sway even when individuals are allowed sight of the surroundings. During tandem stance position, lateral stability is compromised resulting in a higher fluctuation of the

CoP excursion. With light touch, the excursion of the horizontal CoP and the amount of EMG activities of the peroneal muscles are decreased (Jeka and Lackner 1994; 1995).

Not only does light touch from the fingertip reduces postural sway in the unstable standing posture, such as tandem stance or single-legged stance, it also decreases postural sway in the normal standing posture. Clapp et al. (1999) demonstrated that with light touch given during normal bipedal standing where sagittal plane stability was compromised, CoP fluctuations (calculated from the standard deviation of CoP) in the anteroposterior direction were significantly reduced (Clapp and Wing 1999). A reduction of postural sway, however, was not observed when a subject maintained similar arm configuration as touching the fixed surface but actually not touching it (i.e., keeping the fingertip near and above the touched surface), suggesting that the effect of light touch on postural control was, in fact, due to contacting the fingertip to the fixed surface rather than maintaining the upper extremity at a specific location (Creath et al. 2002).

The effectiveness of fingertip cue in postural control is confirmed by several recent researches. Slijper and Latash (2000) demonstrated a reduction of the anticipatory activity from trunk and leg muscles with light touch during unilateral shoulder flexion when standing on both stable and unstable surfaces (Slijper and Latash 2000). It is also shown that light touch is able to suppress abnormal proprioceptive inputs triggered by tendon vibration that destabilize the body (Lackner et al. 2000). Even passive somatosensory cues are able to stabilize the body during standing (Rogers et al. 2001). Passive somatosensory cues were provided by the flat steel that touched the shoulder or the leg of a person. Both contacting areas of passive touch reduced body sway but the effect was greater when the passive touch was applied on the shoulder, as the sway amplitude in standing is more pronounced at the shoulder than at the leg. It was concluded that the central

nervous system is able to utilize the information from both passive and active touch in modulating postural responses (Rogers et al. 2001).

2.6.3 Mechanisms underlying the use of light touch in postural control

The effect of tactile cue is evident even though the force exerted by the fingertip is insufficient (<1N) to provide mechanical support of the body. Jeka and Lackner (1994) showed that when fingertip force was used to provide a mechanical support or force contact, the body sway, as measured by CoP displacement, was coupled with the force at the fingertip in a phase-log manner. In contrast, light touch force led the CoP excursion by some 300 ms, suggesting that this cue was used to signal sensory information about the body sway (Jeka and Lackner 1994). Muscle activations on the leg used to maintain upright stance were decreased with the force contact, as muscle activations of upper extremities increased to assist in the control of posture. On the contrary, activities of leg muscles were increased, as compared to force or grasp contact, and proceeded the CoP excursion by 150 ms with light touch, indicating that fingertip cue was used in a feedforward mechanism to trigger the activation of postural muscles for controlling body sway (Jeka and Lackner 1995).

It has been proposed that several sensory inputs provide information about body sway during light touch condition. Rapidly adapting cutaneous receptors on the fingertip which are sensitive to local vibration may be responsible for detecting the movement between the skin and the surface, whereas slowly adapting cutaneous receptors responsible for the roughness of the skin surface may convey the information about the direction and size of the forces in relation to the body movement through skin surface deformation or skin stretch (Johansson and Valbo 1983; Johansson and Westling 1984; Johnson and Hsiao 1992; Westling and Johansson 1987). Proprioceptive

receptors in the finger and arm inform about the orientation of the body in relation to the external object or surface (Jeka and Lackner 1994; 1995). These somatosensory receptors function together to confer the combined information of position and velocity of the body sway, as the fingertip cue was shown to be coupled with both position and velocity of the moving touched bar (Jeka et al. 1997; 1998a). This finding was similar to what was observed in the visual system, suggesting that the postural control may utilize the fingertip cue in the same way as the visual information (Jeka et al. 1998a; Jeka et al. 1997). The light touch information is then sent to the cortical areas which control posture, leading to the activation of postural muscles to attenuate sway (Jeka and Lackner 1994). Differences in sensory processing mechanisms between active and passive touch were suggested (Rogers et al. 2001). During the active fingertip touch, a person can control the pressure and movement of the finger, thus output information from the central nervous system about the finger movement as well as cutaneous and proprioceptive information from the finger and arm are processed to control the posture. In contrast, only sensory information from the periphery was available during the passive touch.

Fingertip cues are not always useful in the control of posture. When the somatosensory information from the fingertip is incorrect, postural responses are ineffective (Reginella et al. 1999). Reginella and colleagues investigated the effect of light touch when this information conflicts with the information from other sensory systems. The conflicting information was introduced by moving the touched plate in the same direction as the body swayed (sway-referenced) during eyes closed so that the information from the fingertip conflicts with the inputs from the somatosensory information provided by the lower limbs. They found that light touch on a stable (earth-fixed) surface reduces postural sway, as compared to no touch, while touching a sway-referenced surface increases postural sway (Reginella et al. 1999). The

effectiveness of the light touch is also dependent on the position of the touched surface or arm position with respect to the body sway. Body sway was coupled most strongly to the touched surface that was oriented along the longitudinal axis of the arm or in the same direction of the body sway, such as lateral touched surface and the M/L sway (Jeka et al. 1998b). The findings suggest that inappropriate placement of the touched surface or arm position in relation to body sway could minimize the effectiveness of the tactile cue in the control of posture.

2.6.4 Light touch in balance rehabilitation

Light touch information may not be essential for maintaining balance in a person with no postural control impairment, as there is already a redundancy of sensory cues. However, light touch might provide additional useful information for helping the control of posture in subjects with postural control dysfunctions, such as individuals with vestibular loss, patients with peripheral sensory neuropathy and hemiplegic subjects. The effectiveness of light touch in improving postural control has been shown in patients with bilateral vestibular loss (Lackner et al. 1999). The patients could not maintain the tandem stance posture longer than for a few seconds with their eyes closed and even when vision was provided whereas all the healthy subjects could do the same task with or without vision. However, the patients could maintain tandem stance for as long as 25 seconds in the darkness when provided with the light touch from a fingertip. In fact, the patients with vestibular loss in the darkness with light touch were more stable than healthy subjects in the darkness without touch. Thus, it can be seen that light touch was more effective for the vestibular loss subjects than vestibular cues for the healthy subjects in the tandem stance balance control.

Similarly, patients with sensory neuropathy also demonstrated a greater reduction in postural sway in standing with the use of additional touch,

as compared to healthy subjects (Rogers et al. 2001). Rogers et al. (2001) provided two possible explanations on the larger effect of light touch in patients with sensory neuropathy. One is that as the sensory neuropathy subjects swayed more than the healthy subjects, they were likely to receive enhanced tactile input because greater sway produces greater velocity of the friction stimulus on the skin. The other is that the additional tactile stimulus is not redundant in those who have sensory deficits. A study of the effect of light touch in stroke patients has not been done yet. As the balance problem in hemiplegic subjects may be partly due to sensorimotor impairments and deficits in perception of body orientation, providing supplementary sensory information from fingertip may be sufficient to compensate for these deficits.

Somatosensory cues from the environment, if proven to improve balance control, will be more advantageous for stroke rehabilitation over traditional use of walking devices in terms of posture and gait symmetry. Milczarek and colleagues (1993) found that although a cane promotes a better balance in standing, the use of a cane on the sound side of hemiplegic patients shifts the CoP towards the cane (sound) side. Weight shifting to the unaffected side on locomotion may be beneficial during terminal stance and pre-swing of the affected limb in that walking device assists in shifting the CoM further toward the unaffected limb so that the affected limb is free for lift off (Kuan et al. 1999). However, gait parameters in other phases of walking (Kuan et al. 1999), gait symmetry and muscle activation patterns (Hesse et al. 1998) do not differ when compared between walking with or without aids. Therefore, it is likely that walking aids encourage the use of unaffected side while ignoring the affected side, hence, leading to asymmetrical posture. Light touch, on the other hand, is unlikely to promote postural asymmetry as it does not provide mechanical support, which would encourage leaning toward the touch surface, as walking aids do. Nevertheless, none of the studies available

at the present investigate how hemiplegic patients utilize the somatosensory information from the fingertip during unexpected surface perturbation.

CHAPTER 3

IMPACT OF STROKE ON POSTURAL RESPONSES TRIGGERED BY UNEXPECTED SURFACE PERTURBATIONS IN THE PITCH AND ROLL PLANES: I. DURING QUIET STANCE.

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Previous studies have been shown that stroke lead to impairments of postural responses triggered by unexpected movement of the support surface (Triggered Postural Responses; TPRs) (Badke and Duncan 1983; Di Fabio 1987; Di Fabio et al. 1986; Dickstein et al. 1989; Dietz and Berger 1984). However, the extent to which stroke affects the control of TPRs is still unclear. Previous findings on TPRs during quiet stance in stroke subjects are focused on the postural responses to perturbations that occurred only in the sagittal plane. How the stroke subjects maintain balance when the perturbation is presented in the frontal plane has not yet been answered. In daily activities, perturbations can occur in any planes or directions, thus, the understanding of the control of posture when perturbation occurred in different planes is necessary for fall prevention. The study presented in this chapter was conducted to investigate how stroke affects the modulation of TPRs during quiet stance when the directions of perturbation are varied in the pitch and roll planes.

3.1 Abstract

Stroke resulting from cerebrovascular accidents (CVAs) leads to impairments in balance reactions, but the extent to which stroke affects the organization of postural responses triggered (TPRs) by different axes of surface rotations is still unclear. We hypothesize that TPRs following stroke are underactivated on the paretic side and not modulated by the direction of surface perturbations, as opposed to those observed in healthy subjects. Eleven stroke and 8 age-matched healthy control subjects were exposed randomly to 4 different surface perturbations (peak velocity 32°/s) in the pitch and roll planes during quiet stance while maintaining a step stance posture. TPRs were characterized by body kinematics, forces exerted under the feet and muscle activities recorded from bilateral lower limb muscles. In contrast to healthy controls, TPRs in stroke subjects were not tuned to the direction of perturbations. TPRs were delayed on the paretic side, especially during roll plane perturbations. Asymmetry of TPRs was characterized by under-activation of muscles and force generations in the paretic lower limb, and hyperactivity in the non-paretic side. These impairments resulted in larger sway in the frontal plane and difficulty in stabilizing the trunk and pelvis during all perturbations. Stroke patients also used larger arm movements as a compensatory strategy to assist in the maintenance of equilibrium. These findings suggest that CVAs disrupt the process of sensorimotor integration that is needed to generate the TPRs, thus resulting in balance impairment and overcompensation by the non-paretic side, which could ultimately hinder the recovery of the paretic side.

3.2 Introduction

Unpredictable terrain conditions can disturb upright stance and equilibrium in daily life. The maintenance of equilibrium during stance requires that the postural control system produces appropriate muscular forces at various joints and against the support surface, so that the body is supported against the gravity and the body's center of mass (CoM) is projected within the base of support. Unexpected movement of a support surface elicits rapid, automatic and coordinated postural responses that are triggered primarily by somatosensory afferents depending on the velocity and direction of perturbations (Diener et al. 1988; Moore et al. 1988; Runge et al. 1998). Animal studies have shown that these triggered postural responses (TPRs) are not merely segmental reflexes organized at the level of the spinal cord (Fung and Macpherson 1999; Macpherson and Fung 1999), but rather depend on the integration of proprioceptive, visual and vestibular information at higher levels of the neuraxis, such as the brain stem and cerebellum (Horak and Macpherson 1996).

TPRs can be modulated by the directions of surface perturbations (Carpenter et al. 1999; Henry et al. 1998a, b; Moore et al. 1988). Pitch and roll plane surface perturbations elicit different movements of body segments (Carpenter et al. 1999). Various groups of muscles, i.e., muscle synergies, are recruited and the amplitude is tuned to the different directions of surface translations (Henry et al. 1998b). The control mechanism of directionally specific muscle recruitment, however, is not simple. Muscles recruitment is not necessary predictable based on the line of muscle action or anatomical synergists (Henry et al. 1998b; Macpherson 1988). The timing of muscle recruitment can be complex, in that the onset latencies for the shank and thigh muscles are constant, regardless of surface translation direction, whereas the onset latencies for the trunk muscles and Tensor fascia latae are

either early or late as the direction of perturbation changes (Henry et al. 1998b). The TPRs are likely the product of a complex control mechanism where the central center influences the timing of muscle recruitment and the peripheral processes adjust the amplitude of responses (Henry et al. 1998b). Furthermore, the finding that trunk and TFL muscle activities are modulated to the direction of surface movement suggests that the trunk and pelvis are highly regulated by the central nervous system during various perturbation directions. Selective sensitivity of the trunk motion and paraspinal muscle activations to a particular direction of surface perturbation (Carpenter et al. 1999) also leads to the assumption that proprioceptors located in the trunk and pelvis are used to trigger directionally specific TPRs (Allum and Honegger 1998; Allum et al. 1995).

Injury to the central nervous system, such as cerebrovascular accidents (CVA), results in sensory and motor impairments that often lead to postural control and mobility dysfunctions (Badke and Duncan 1983). TPRs in stroke patients have been studied with respect to sudden surface translations in the antero-posterior directions (Di Fabio 1987; Dickstein et al. 1989; Dietz and Berger 1984), and are characterized by reduction of amplitude and disrupted timing of muscle recruitment on the paretic limb. Delayed onset latencies of the shank muscles (Badke and Duncan 1983; Di Fabio 1987; Di Fabio et al. 1986; Dietz and Berger 1984) and abnormal sequence of distal and proximal muscle activations (Badke and Duncan 1983; Di Fabio et al. 1986) suggest difficulties in selecting and modulating postural adjustments in stroke patients (Badke and Duncan 1983). Dysfunctions of the paretic limb are compensated by the non-paretic limb, such as co-contracting the agonist and antagonist muscles for paretic weaknesses (Dickstein et al. 1989) or earlier activating of thigh muscles for the paretic delays (Di Fabio et al. 1986). However, it is still unclear as to how stroke patients organize TPRs with respect to different directions or planes of perturbations. A common stance

feature of stroke patients is uneven weight-bearing, with increased loading of the non-paretic limb (Bohannon and Larkin 1985; Dickstein et al. 1984). Asymmetrical limb-loading pattern is associated with excessive body sway in the frontal plane and a decrease in lateral stability (Dickstein et al. 1984; Shumway-Cook et al. 1988), leading to frequent falls towards the affected side (Diller and Winberg 1970). Therefore, the ability to maintain balance when a perturbation is presented in the lateral direction is necessary for fall prevention in stroke patients. Thus, the present study was conducted to determine how CVA affects the modulation of TPRs to the direction of perturbation presented in the pitch and roll planes during quiet stance. We hypothesize that, TPRs in stroke patients, due to impairment in sensorimotor integration in the supraspinal neuraxis, are not modulated with respect to directional changes of surface perturbations, and result in a global pattern that show under-response from the paretic limb and exaggerated compensation from the non-paretic limb.

3.3 Methods

3.3.1 Subjects

Eleven subjects with first-time CVA and eight healthy subjects of similar age and gender participated in the study. The age of stroke and healthy subjects ranged from 54-80 years and 53-79 years, respectively. All stroke subjects had motor deficits in the lower extremity on the paretic side (Chedoke-McMaster impairment inventory (Gowland et al. 1995), leg or foot scores of 5/7 – 6/7), were able to stand for 5 seconds or more without external support (Chedoke-McMaster postural control score of 3/7 or higher), and had mild to moderate problem in physical mobility (clinical Timed Up and Go test (Podsiadlo and Richardson 1991), score between 10-29 seconds vs. the normal range of 5-7 seconds).

Stroke subjects were recruited from both the in-patient and out-patient neurological rehabilitation program of the Jewish Rehabilitation Hospital and healthy control subjects were volunteers recruited from the surrounding community. The research protocol was approved by the institutional ethics committee and informed consent was obtained from all subjects. Stroke subjects with expressive and/or comprehensive aphasia, severe hemineglect, cerebral aneurysm, bilateral cerebral impairment, or brainstem and cerebellar lesions were excluded from the study. Stroke and control subjects were excluded if they had: (1) a history of lower extremity musculoskeletal conditions (e.g., pain, contracture and joint replacement) in the past year; (2) hemianopsia; (3) evidence of polyneuropathy in the lower extremity; (4) dizziness or other symptoms indicative of vestibular impairment; or (5) were taking psychotropic medications that may affect balance.

3.3.2 Experimental Procedures

A servo-controlled six degree-freedom-of-movement motion-base embedded with two tri-axial AMTI (OR6-7) force plates (Fung and Johnstone 1998) was used to deliver surface perturbations. Each subject was asked to stand on the force plates and maintain a step stance posture that mimics the double limb support phase of locomotion (for subsequent comparison, see Chapter 4). Each foot was positioned over a force plate, with the right foot (of control subjects) or paretic foot (of stroke subjects) in front (Fig. 3.1A). Each subject's own step length and step width was used to determine the length and width of the foot placement on the force plates. Once the subject was comfortable with the assigned standing posture, positions of the feet were marked on the force plates to ensure the consistency of foot placement throughout the experiment. Each individual wore a body harness that was attached to an overhead rail for safety precautions. None of the subjects used ankle-foot orthosis during the testing procedures.

Ramp perturbations in the pitch and roll planes were randomly delivered at a peak ramp velocity of 32 degree/s and at a peak amplitude of 5 degrees. The chosen velocity and amplitude of surface perturbation were within the ranges that a person was able to maintain balance without changing the base of support (i.e., stepping) (Allum et al. 1993). The axes of surface rotation were centered with respect to the motion platform and the feet position and the perturbation consisted of one of 4 axes: toes-up, toes down, right/paretic side up and left/paretic side down (Figure 3.1A). The perturbation axes were block randomized and four trials of each perturbation direction were collected. Each trial of data collection lasted for 4 seconds. In order to reduce any anticipation by the subject, the onset of surface rotation was varied between trials and four unperturbed standing trials were interspersed and collected with the perturbed trials. Practice sessions were

also given prior to testing to familiarize and habituate subjects to the experimental stimuli.

3.3.3 Data collection and analysis

Thirty-eight retro-reflective markers were placed on anatomical landmarks (Fig. 3.1A) to capture body motions and four additional markers were placed on motion platform to capture surface movements. Three-dimensional motion capture was performed by a high-resolution six-camera VICON motion analysis system (Vicon 512; Oxford Metrics Ltd) at a sampling frequency of 120 Hz. The data were low-pass filtered at 10 Hz using a 2^{nd} order dual-pass Butterworth filter, based on a previous residual analysis of the kinematics (Winter 1990). Anthropometric measurements were obtained from each subject to calculate the position of the body CoM. Segmental and joint angles were calculated based on segments formed by at least three non-coplanar markers.

The triaxial ground reaction forces (GRFs), including the anteroposterior (A/P), mediolateral (M/L) and vertical components (Fx, Fy and Fz respectively) and the moments of force (Mx, My, Mz) were recorded from two force plates (AMTI OR6-7) mounted within the motion base. The force signals were amplified and sampled at 1,080Hz. Any bias in the force signals at the onset of perturbation due to the inertial characteristics of the force plate was subtracted based on inverse dynamics calculation from motions of the platform (Preuss and Fung 2002).

Muscle activities were recorded by an 8-channel telemetric EMG unit (Noraxon USA, Inc.). After appropriate skin preparation, bipolar silver-silver chloride disposable surface electrodes were placed over muscle bellies of four bilateral lower limb muscles: tibialis anterior, medial gastrocnemius, rectus femoris, and tensor fascia latae. EMG recordings were band-pass filtered between 16 and 500 Hz and sampled at 1,080 Hz. The EMG signals

were then full-wave rectified and low-pass filtered at 100 Hz.

The background periods of kinetic, kinematic and EMG activities in the 200 ms before perturbation onset were used as baseline values (Fig. 3.1B). The average baseline activities were subtracted from the subsequent traces. Maximum ranges of displacements (peak-to-peak amplitude) of body CoM, ankle, knee, hip and shoulder joint angles and pelvic and trunk segmental angles in the sagittal and frontal planes from 0 to 1,000 ms after perturbation onset were calculated. The center of pressure (CoP) from each force plate in the A/P (CoPx) and M/L (CoPy) directions was calculated using the following equations.

$$CoPx' = My - (Fx.Wp)/(-Fz), \qquad CoPx = CoPx'.[R_{platform}]$$

$$CoPy' = Mx + (Fy.Wp)/(Fz), \qquad CoPy = CoPy'.[R_{platform}]$$

Where Fx=force in the A/P direction, Fy= force in the M/L direction, Fz=vertical force

Mx=moment of force around x-axis, My=moment of force around y-axis

Wp=width of the forceplate, and

$R_{platform}$ = rotation matrix of platform calculated from the kinematic data of the force plate

The resultant total CoP from the two force plates was then calculated and the peak-to-peak amplitude of the total CoP displacements in the A/P and M/L directions from 0 to 1,000 ms after perturbation were obtained. The root-mean-square (RMS) of CoM and total CoP displacements in A/P and M/L directions were used as a measure of sway from 350 to 1,000 ms after perturbation onset.

Integrals of EMG and GRF were computed based on two time-windows (Fig. 3.1B) (Henry et al. 1998a, b) after the onset of surface movement. For EMG analysis, the integrals were calculated for the *short latency reflex activity period* (E1) and *active response period* (E2), spanning 0-70 ms and 70-320 ms respectively after perturbation onset. Corresponding

periods with 30 ms delay were used for the analysis of the GRF, taking into consideration the electromechanical delay (Henry et al. 1998a, b).

EMG onset latency was selected based on the specific criteria that the first burst of muscle activity must be at least 1 SD greater than the average EMG in the background period and the duration must be at least 25 ms long (Henry et al. 1998b). The EMG activity was normalized to the peak activity of the same muscle for the control subjects and to the peak activity of the non-paretic side for the stroke subjects. A muscle was considered as having a real response only when the firing probability reaches 75% (i.e., activated in 3 out of 4 trials of perturbation) (Henry et al. 1998b). The GRFs were normalized to the subject's body weight. The Fx and Fy vector integrals were summed to produce the resultant horizontal force vector integral under each foot. All analyses were performed with Matlab software (Mathworks Inc)

3.3.4 Statistical analysis

Outcomes in kinetics, kinematics and EMG were averaged for axis of perturbation, as there was no significant difference between trials. All statistical analyses were performed using the Statistica software (StatSoft Inc). Two-way ANOVAs were used to test for any main or interaction effects due to subject group (stroke vs. control) and axis of perturbation (toes-up vs. toes-down vs. right-up vs. left-up). When significant differences were found after multiple comparisons were adjusted with the Bonferroni test ($p<0.05$), pairwise comparisons were performed using the Tukey test.

3.4 Results

3.4.1 Kinematic responses

Figure 3.2 shows how a stroke subject responded differently to surface perturbations as compared to the average kinematic pattern of healthy controls. During toes-up surface rotation, the front (right) limb was elevated while the rear (left) limb was lowered. The feet were displaced into dorsiflexion immediately after perturbation onset while the trunk remained vertical initially due to inertia (Fig. 3.2A). Control subjects executed the kinematic strategy that involved flexion of the trunk and pelvis (opposite to movement of the surface), accompanied by flexion of both hips and the knee on the side of the surface that was tilted up (front limb). The trunk and pelvis reached their maximal displacement around 350 ms and began to return to the initial configuration that was aligned to the vertical axis whereas the lower limbs were held in the new position (flexion). There were minimal movements of the arm opposite to trunk movement. A reverse movement strategy of trunk, pelvis and lower limbs was found in response to toes-down surface tilt as the feet were driven into plantarflexion (Fig. 3.2B). The strategy involved extension of the trunk, pelvis and hips as well as flexion of the knee on the side of the upward tilted surface (rear limb), as well as some flexion of the shoulders.

During left-up surface tilts, the left leg was elevated but the right leg was lowered (Fig. 3.2C). The right foot was driven into inversion whereas the left foot was moved into eversion. A left-up tilting of the pelvis caused abduction of the right hip joint and adduction of the left hip joint. After the pelvis was displaced, the trunk moved in the opposite direction (leftward) and reached its peak around 500 ms when it began to reverse back to its erect position. Knee movements were minimal in the frontal plane due to structural constraint but some knee flexion was evident. Slight movement of both arms opposite to trunk motion was also observed. A reverse pattern of the trunk,

pelvic and lower limbs was seen during right up tilt (Fig. 3.2D). In summary, the movement of the pelvis was in the same direction as the trunk to oppose surface movements during pitch perturbations, whereas pelvis movement was in the same direction as the surface but opposite to the trunk during roll perturbations.

In general, stroke subjects (nine out of eleven) were able to generate similar kinematic responses as control subjects. Only two of eleven stroke subjects (one right and one left hemiparesis with similar clinical postural control score of 5/7) exhibited different kinematic strategy in that the trunk and pelvis moved opposite to healthy subjects. These two subjects also had the lowest walking speed overground (0.4 and 0.5 m/s). Even among stroke subjects who displayed kinematic responses that were similar to control subjects, the movement of each body segments was erratic with over or under-shooting. Large deviations can be seen proximally in the trunk, pelvis and arms with increased response amplitude of the thigh and shank on the non-paretic side, as compared to the paretic side.

The average range of sagittal and frontal trunk and pelvis excursions, as well as hip, knee and ankle excursions in the sagittal plane were contrasted between both groups of subjects in Figure 3.3. In general, changes in the sagittal plane at the trunk and pelvis were smaller than at the hip, knee and ankle, whereas the amount of adjustments in the frontal plane at all segments was approximately the same (<5°). In control subjects, the trunk and pelvis were displaced more in the sagittal plane during toes-up and toes-down tilts and in the frontal plane during right up and left up tilt (Fig. 3.3A, B). The displacement patterns of the trunk and pelvis were also seen in stroke subjects but stroke subjects demonstrated larger trunk and pelvic displacement, as compared to control subjects, in the sagittal plane ($p<0.0001$) and in the frontal plane ($p<0.05$) during all perturbed directions. In control subjects, hip displacements were largest during toes-up tilt where both

hips were moved into flexion (Fig. 3.3C). Maximum knee displacements were seen on the upward tilted limb such as the right knee during toes-up and right up tilts and vice versa for the left knee (Fig. 3.3D). Ankle displacements were generally larger in the front limb (right or paretic) and similar for all directions of perturbations (Fig. 3.3E). Although there were no significant differences in the amount of lower limb angular displacements between the two subject groups, stroke subjects showed smaller displacements in the paretic hip and knee joints as compared to the non-paretic side.

The segmental coordination between the trunk and pelvis in the sagittal and frontal planes are contrasted between a stroke subject and a healthy control in Figure 3.4A. It can be seen that the initial movement of the trunk and pelvis indicated by the arrow was similar in the two subjects during any perturbation direction. However, irregular movement coordination trajectories were evident in the stroke subject for any given perturbation. While the control subjects smoothly returned the trunk and pelvis near the initial neutral position, the stroke subject was far from the initial trunk and pelvis position at the end of the trial. This impaired coordination pattern in stroke subjects suggests instability of the trunk and pelvis during balance corrections. The fact that sagittal and frontal arm movements in stroke subjects were significantly larger ($p<0.005$) than controls during all perturbation directions (Fig. 3.4B) suggests that it might be a compensation strategy used by stroke subjects to assist balance corrections that were deficient in the trunk and pelvis.

3.4.2 The control of body CoM and CoP

Figure 3.5A showed the horizontal displacement of the CoP in relation to the CoM from a control and a stroke subject. The movement patterns of CoM and CoP during any given perturbation were similar in the two groups of subjects. The movement of CoP was always larger in magnitude to

encompass the movement of the CoM. Surface perturbation displaced the CoM towards the direction of downward inclined surface. For example, CoM moved towards the left during right up tilt and towards the right during left up tilt. In contrast, CoP initially moved opposite to the CoM movement as there was increased loading of the upward tilted limb during the first 225 ms after perturbation onset. This increased force was partly due to passive stiffness and partly voluntary control to resist the upward motion of the surface. CoP then followed and encompassed the CoM movement after 225 ms. Despite similar movement patterns of CoM and CoP observed in both groups of subjects, stroke subjects demonstrated larger CoP trajectories during roll perturbations but not during pitch perturbations.

Figure 3.5B shows the amount of average A/P and M/L peak-to-peak displacements of CoM and CoP in the two groups of subjects. CoM and CoP were displaced maximally in the A/P direction during toes-up/down tilts ($p<0.0001$) with the maximum displacement at toes-down perturbation, whereas CoM and CoP were displaced similarly in the M/L direction for all perturbation directions. There were no significant differences in CoM displacements between the two groups of subjects. However, CoP displacements were larger in stroke subjects, especially in the M/L direction during both pitch and roll plane perturbations. Problems in the control of CoP in the M/L direction by stroke subjects were most evident in the period 350-1,000 ms after perturbation onset, as seen in the significant increase in variability (RMS) (Fig. 3.5C).

3.4.3 Ground reaction forces

The pattern of vertical force changes was similar for all perturbation directions in the stroke and control subjects. This pattern involved alternate increase and decrease in loading force between the front and rear limbs (Fig. 3.6A). During the passive period (50-100 ms), the vertical force was

increased on the upward lifted limb and decreased on the lowered limb. Changes in vertical forces during this period were due to passive viscoelastic properties of the musculoskeletal system because no muscle activation was found in the passive period. Early in the active period (100-350 ms), increased loading on the elevated limb was continued for a short period as a person resisted the upward movement of the surface prior to yielding to the force of gravity by unloading the raised limb and loading the other limb.

The example of loading-unloading pattern was seen from an individual trace shown in Figure 3.6A-left column. The loading pattern during toes-up perturbation in a control and a stroke subject was characterized by initial loading of the right or paretic limb and unloading of the left or non-paretic limb followed later by the reverse action of each limb. Although the pattern of vertical force changes was similar between the two groups, the amount of change was significantly different. Average vertical force integrals during the active period in two groups were shown in Figure 3.6A-right column. The paretic limb significantly accepted less weight during any perturbation direction, as can be seen from less load acceptance during left-up and toes-down perturbations and more unloading during toes-up and right-up perturbations. In contrast, the non-paretic limb appeared to accept more weight, even though significant differences were found only when the non-paretic limb was loaded during toes-up and right-up tilts. The findings indicate an inability for stroke subjects to adjust the loading force by bearing more weight on the paretic limb.

Figure 3.6B shows the characteristics of shear force during the passive and active periods in a control and a stroke subject and the group average during the active period. In general, the amount of shear force changes was less than the vertical force changes and the pattern of shear force was the same during the passive and active period, thus only the pattern of shear force during the active period was presented in Figure3.6B. Similar pattern

and amplitude of shear forces were observed in both groups of subjects and the vector directions were generally similar to the direction of body CoM movements, i.e., backward during toes-up perturbation and rightward during left-up tilt, and vice-versa for toes-down and right-up tilts.

3.4.4 Muscle responses

Figure 3.7A demonstrates bilateral medial gastrocnemius (MG) muscle activities during all perturbation directions in a control and a stroke subject. MG responses began only in the active response period (70-320 ms) as no activity could be detected in the short latency stretch reflex period (0-70 ms). Bilateral MG activities were modulated by the direction of surface rotation, whereas the amount of MG activities in the stroke patient was rather constant during all perturbation directions. Furthermore, while the control subject showed equal amount of MG activations on the right and left leg, the stroke patient showed lower MG activities on the paretic limb and larger MG activities on the non-paretic limb.

Figure 3.7B contrasts the average EMG integrals during the active period (70-320 ms) of bilateral lower limb muscles in the two groups of subjects. It is evident that muscle activations in control subjects were tuned to the perturbation directions. Bilateral tibialis anterior (TA) were activated maximally during toes-up tilt to bring the body forward while bilateral MG were recruited during toes-down tilt to bring the body backward. Rectus femoris (RFM) and Tensor Fascia Latae (TFL) on the right and left limb functioned alternately to load the limb that was dropped by the surface tilt. For example, the right RFM and right TFL were activated during toes-down and left-up perturbations whereas the left RFM and left TFL were activated during toes-up and right-up perturbations. Thus, RFM and TFL showed two maximum activation directions.

In contrast, there was no distinct pattern of direction-specific muscle

modulation in stroke subjects. Moreover, while the amount of muscle activations in control subjects was relatively similar for both limbs, stroke subjects demonstrated asymmetry of muscle recruitment on the paretic and non-paretic limb. The amount of muscle activations were generally less on the paretic limb but larger on the non-paretic limb. Muscle activations on the paretic limb, even when recruited for balance correction, were also significantly lower than control subjects, suggesting that muscle responses to surface perturbations in stroke subjects were under-activated on the paretic side. In contrast, muscle activations on the non-paretic limb, when recruited for balance correction, were not significantly different from the control subjects, even though most of them appeared to be larger than in control subjects. However, significantly large muscle activations were observed for the non-paretic limb, as compared to control subjects, during perturbed directions that normally did not require actions of those muscles. Therefore, there was an overall increase of muscle activities in the non-paretic limb, probably used to compensate for insufficient muscle activities in the paretic side.

Figure 3.8 shows the average muscle onset latencies in the two groups of subjects. In general, stroke subjects showed similar sequence of muscle recruitment as control subjects, but the activation was significantly delayed on the paretic limb, except for MG that showed short latency activation in response to toes-up perturbation. Similar early onset of TA activation was observed in stroke subjects during toes-down surface tilts, suggesting the recruitment of ankle muscles by stretch reflex activations.

In response to right-up perturbation, right TFL, right RFM and left TA were recruited early in control subjects. This activation pattern corresponded to the initial loading of the right limb and unloading of the left limb. Then left TFL, left RFM and right TA were activated as the left limb began loading and right limb started unloading. This pattern was reversed during left-up

perturbations. A similar pattern of muscle recruitment was observed in stroke subjects, but with significant delays in the paretic limb. In addition, muscle recruitment in stroke subjects was more delayed for right-up and left-up perturbations as compared to during toes-up and toes-down perturbations.

3.5 Discussion

Results from this study showed that following CVA, postural responses to surface displacements were disrupted, in that responses were delayed and not directionally tuned. Reactions were asymmetric, with hypoactivity in the paretic and hyperactivity in the non-paretic lower extremity. The loading forces were concomitantly under-activated in the paretic limb with overcompensation from the non-paretic limb. Deficits in the control of lower extremities gave rise to erratic movements of body segments with significant impairment at the trunk and pelvic levels, as demonstrated by irregular and larger trunk and pelvic displacements in both planes during all perturbed directions. Larger CoP displacements in the M/L direction suggest difficulties in the control of CoM in this plane, which required the assistance from arm and upper body movement. Deficits in directional modulation of TPRs in stroke subjects suggested that the integration of somatosensory and motor commands was affected as a result of CVA. The impairments seen in the paretic limbs could be attributed directly to the cortical lesions whereas abnormality seen in non-paretic limb may be a compensatory reaction of the nervous system, long-term use of which could be detrimental to the competency of the paretic limb during motor recovery.

3.5.1 Active control of the trunk and pelvis in balance corrections

Axial control of the trunk and pelvis is important in the maintenance of equilibrium during quiet stance. The trunk and pelvis together contain most of the body mass and hence the central nervous system could control the body CoM through the regulation of the trunk and pelvis (Massion 1992; 1994). In our present study, movements of the trunk and pelvis in the control subjects, which were mostly opposite to the surface tilting direction, suggest that the active neural control of the trunk and pelvis were employed early in the

balance correction during surface tilt; otherwise the trunk and pelvis would be displaced passively in the same direction as the tilting surface due to gravity and inertia. Active trunk and pelvis movements opposite to the perturbed direction were used to restore the position of the CoM back over the base of support (Henry et al. 1998a). Rapid early movements of the trunk and pelvis can generate active hip torques to maintain equilibrium when standing on a beam (Horak and Nashner 1986), as this strategy is more effective to encounter large and rapid disturbances (Kuo and Zajac 1993). Such a postural strategy can be achieved through early activations of rectus abdominis, erector spinae and TFL muscles (Henry et al. 2001).

The present study shows that coupling of trunk and pelvis movements is different for perturbations occurring in the pitch or the roll plane. Opposite movements of the trunk and pelvis during the roll perturbation may act to minimize the displacement of the body CoM in the frontal plane. In fact, minimal frontal displacement of the CoM is necessary when the BoS is limited in the frontal plane due to the step stance posture. In contrast, we found that the trunk and knee responses were similar, regardless of the tilting direction. Knee motion could be used to assist regulation of the CoM, especially in the vertical direction (Ko et al. 2001). Similar trunk and knee responses to pitch and roll perturbations were in agreement with the general principle that the control of postural equilibrium is similar for sagittal and lateral perturbations (Henry et al. 1998a). Different responses during pitch and roll perturbations likely result from different mechanical constraints of foot position as well as biomechanical constraints of the trunk and lower limbs in the step stance posture, permitting larger movements in the sagittal plane.

Most stroke subjects were able to initiate similar trunk, pelvic and lower limb responses to surface tilts, suggesting that the ability to select the motor programs is not likely to be affected by the CVA. However, they had difficulty in restoring equilibrium of the trunk and pelvis, as seen in overshooting and

increased stabilizing time. Such difficulty could be related to impairments at the trunk, pelvis and lower limb levels, such as reduced sensory inputs and muscle weaknesses, or at the level of sensorimotor process regarding perception of the body in space. Larger pelvic movements with increased time for stabilization could be resulted from impairment of bilateral hip abductors and gluteus medius muscles (Wing et al. 1993). After CVA, bilateral deterioration of the trunk muscles during active movements were also reported (Bohannon 1995; Bohannon et al. 1995; Dickstein et al. 2000; Tanaka et al. 1997, 1998). It has been shown that the erector spinae was more affected than rectus abdominis and external oblique muscles, as demonstrated by the decreased torque and the synchronicity between paretic and non-paretic side (Dickstein et al. 2000; Tanaka et al. 1998). Impairments of the trunk function in stroke subjects could be caused by disuse atrophy or impaired descending pathways (Tanaka et al. 1998). Each side of the trunk muscles is innervated by both sides of the brain, which both are necessary for normal trunk function (Carr et al. 1994); therefore lesions on one side of the brain could disrupt function of bilateral trunk muscles (Tanaka et al. 1998).

However, in upright stance where the heavy trunk and pelvis are balanced on two supporting limbs, stability of the trunk and pelvis also requires the intact control of the lower limbs. It has been shown that inadequate control of the lower limbs in stroke subjects leads to larger pelvic sway during quiet standing with eye closed (Dickstein and Abulaffio 2000). Other studies reported the disruption of standing balance in the stroke subjects due to impairment in the integration of the somatosensory information from the lower limbs (Di Fabio and Badke 1991), causing the stroke subjects to rely more on the visual and vestibular systems (Badke and Duncan 1983), as these systems are not commonly affected by the CVA. In our study, movements of the knee and hip joints in the stroke subjects were found to be smaller on the paretic side but larger on the non-paretic side.

Asymmetrical control of the lower limbs could, therefore, contribute partly to poor stabilization of the trunk and pelvis seen in this study.

An internal representation of the body is a product of multiple sensorimotor processes that generate a reference system so as to perceive the position of body segments with each other and position of the body with respect to external space (Gurfinkel et al. 1986). Various sensory inputs from vestibular, visual, somatosensory and proprioceptive systems contribute to the estimation of body orientation with respect to the external environment (see review by Massion, 1994). Neurons in the posterolateral thalamus which also pass through the posterior limb of the internal capsule are believed to inform the position of the body in relation to the gravity (Karnath et al. 2000a). Lesions of these neurons correspond to the lateral tilt of the body as seen in the stroke subjects with pusher syndrome (Karnath et al. 2000b). In contrast, stroke subjects with hemineglect showed impairment in different aspects of body perception, known as visual verticality, which can be assessed by misaligning the visual target to the vertical (Karnath 1994). Right inferior-posterior parietal cortex, right premotor frontal cortex and posterior and medial portions of the thalamus are involved in the perception of visual verticality (Fogassi et al. 1992; Galletti et al. 1989; MacKay and Riehle 1992). Although the stroke subjects in our study were not presented with hemineglect and pusher syndrome, we could not entirely exclude impairments in the internal representation of the body, as the CVA may disrupt other sensory pathways involved in processing the internal representation of the body, the impairment of which could lead to malalignment and increased body sway.

3.5.2 Control of CoM projection and CoP in the horizontal plane

The central nervous system (CNS) controls many muscles acting across multiple body segments, reflected in the displacement of the foot CoP,

in order to maintain the CoM within the base of support (Winter et al. 1990). In response to surface tilts, larger excursions of the CoM and CoP were found in the A/P direction during toes-up and toes-down perturbations. This behavior may again be related to the step stance posture with increased stability in the A/P axis, as compared to the M/L axis. Therefore, by limiting the excursion of the CoM and CoP in the M/L direction, the CNS could easily maintain the CoM within the base of support. In addition, larger forward excursion of CoP during toes-down tilt as compared to backward CoP excursion during toes-up tilt could be explained by the longer ankle joint lever arm in the forward direction. In contrast, when stance is perturbed by M/L translations when standing with feet together, the peak M/L displacement of the CoP was larger (Henry et al. 1998a).

Winter et al. (1996) revealed that the functions from both ankle and hip muscles contribute to the maintenance of equilibrium during the step stance posture. However, the contribution from these two groups of muscles is more complicated than those recruited in side-to-side or tandem stance foot position (Winter et al. 1996). During side-to-side or tandem foot position, the ankle and hip mechanisms function independent of each other to maintain the A/P and M/L equilibrium. In contrast, these two mechanisms need to be work in concert to control the equilibrium during step stance foot position and require more control from the CNS implicated in the integration and selection of the tasks. Therefore, it has been suggested that this step stance posture would be the more appropriate position for the stroke subjects to challenge the control of equilibrium.

The control of M/L CoP excursion during external perturbation in standing is the result of major actions from the paraspinal (lateral bending of the trunk) and hip (loading the limb) muscles with minimal but important contribution from the ankle (loading the M/L border of the foot) muscles (Rietdyk et al. 1999). Therefore, larger variability of M/L CoP excursion in

stroke subjects could be due to impaired functions of the muscles that control lateral movements of the trunk, pelvis, hips and ankles. For instance, larger displacement of the trunk and pelvis in the lateral direction, resulted from deficits in the control of trunk muscles and delayed recruitment of pelvic muscles (TFL and RFM), could be one factor contributing to M/L instability. Other studies examining weight bearing on each foot during quiet stance in stroke subjects also reported a major problem of control in the M/L balance (Dickstein et al. 1984; Shumway-Cook et al. 1988). Lateral instability has been considered as a primary cause of falls towards the paretic side in stroke subjects (Diller and Winberg 1970).

3.5.3 Principles of muscle recruitment and force generation in equilibrium control

The CNS produces appropriate muscular forces at various joints and on the support surface to regulate the CoM. Two components of the force exerted under the feet, the vertical and the shear force are controlled during surface tilts. The vertical force change reflects active weight bearing (loading/unloading) of each limb. An initial resistance of upward displacement of the support surface (0-150 ms) leads to an increase in loading of the upward tilted lower limb, while the downward tilted limb was concurrently unloaded. A reverse pattern of limb loading occurred later such that the downward tilted limb was loaded and the upward tilted limb was unloaded, a pattern seen during standing on an inclined plane that is affected by gravitational force. Different patterns of vertical forces observed in the passive and active integral periods indicate that complex neural mechanisms, rather than mere passive joint stiffness, contribute to the control of the vertical force. In fact, the general pattern of flexor and extensor activities in the lower limb corresponds well to the unloading/loading force changes. For example, right TFL and right RFM were recruited early in response to initial right limb loading

during right-up or toes-up perturbation, followed by the activation of left TFL and left RFM as the left limb began to be loaded later in the active period.

Early activation of the TFL and RFM to load the limb has also been suggested in previous studies in which TFL could play a role in stabilizing the pelvis whereas RFM may control the knee during limb loading (Burleigh et al. 1994; Henry et al. 1998a, b). As previously shown (Carpenter et al. 1999), the ankle muscles, TA and MG, were maximally recruited when the perturbations were in the pitch plane. However, the present study found short latency reflex activation of TA and MG in stroke subjects but not in healthy controls. This is probably due to the fact that the ability to decrease the gain of short latency reflex responses is affected as a result of CVA, similar to what was found in the study examining the anticipatory responses to bimanual loading task in the healthy and stroke subjects (Bennis et al. 1996). We also found less weight acceptance on the paretic limb during all directions of perturbations. The decreased loading could be contributed by the significant delay of RFM and TFL muscle recruitments on the paretic limb. Lower weight acceptance in the paretic limb has been reported in stroke subjects performing a simple leg flexion (Rogers et al. 1993) or gait initiation (Brunt et al. 1995).

The shear forces on both feet were exerted in the same direction as the horizontal displacement of the body CoM to generate the recovering forces on the ground, which were equal in magnitude but opposite in direction. For example, the shear forces during toes-up tilt were displaced backward whereas the recovering forces were generated forward, which helps to counteract the movement of the CoM. Furthermore, similar patterns of shear force during the passive and active integral period suggest that passive mechanics of the body may play a relatively important role in regulating the shear force. This pattern of shear force was similar to the force pattern observed during wide base of support (Henry et al. 2001) and might be a result of increased passive stiffness of the pelvis and lower extremities

(Winter et al. 1998). This may explain why stroke subjects who have deficits in neural control show similar pattern and magnitude of shear forces as the control subjects.

Direction-specific muscle recruitment is likely a result of sensorimotor integration, the process involves in the transduction of sensory stimuli into biochemical energy and muscle contractions (Cohen and Anderson 2002). This sensorimotor integration is disrupted after stroke, as shown in the lack of direction tuning. Stroke could disrupt the sensorimotor integration at several levels of the processing such as sensory perception, internal representation of the body, neural integration circuit and motor control outputs. It has been suggested that the sensorimotor integration circuit is located in the brainstem and cerebellum, as they are the sites for integrating vestibular, visual and somatosensory information (Frank and Earl 1990; Horak and Macpherson 1996). However, brainstem and cerebellum lesions are excluded from the present study. Recent studies, however, have demonstrated that neurons in specific areas on the cerebral cortex, such as the dorsal premotor area (Shen and Alexander 1997) and posterior parietal cortex (Cohen and Anderson 2002), can play a role in sensorimotor integration.

In contrast to the symmetrical activation of the right and left lower limb muscles in healthy subjects, stroke subjects show larger muscle activation in the non-paretic limb and vice versa in the paretic limb. Decreased muscle responses in the paretic limb have also been reported during standing on moveable see-saws (Dietz and Berger 1984) or on a sinusoidal moving surface (Dickstein et al. 1989). Reduced postural responses on the paretic limb were partly due to decreased supraspinal activation to the motor neuron pool of the leg muscles and partly due to changes in mechanical properties of the muscles following the lesion (Dietz and Berger 1984). On the other hand deficits in the non-paretic limb may be due to an adaptive or compensatory mechanism of the nervous system. Although the compensatory mechanism

from the non-paretic limb could assist in maintaining balance, the long-term reliance on the non-paretic limb could delay the recovery of the paretic side.

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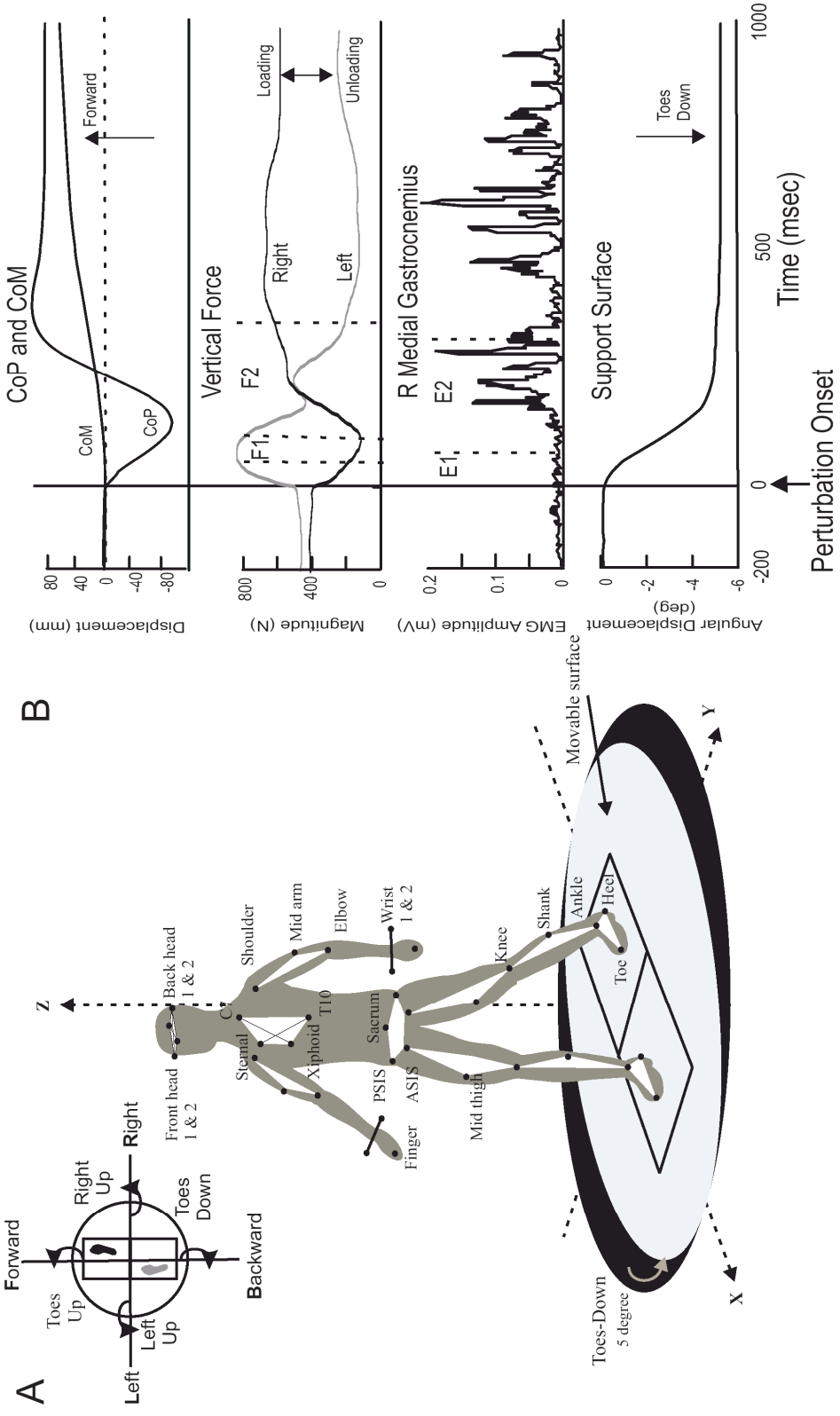
Figure 3.1. A: A subject was exposed to the surface tilt in four different directions; toes-up, right-up, toes-down and left-up, during semi-tandem quiet stance with the right or the paretic foot in front. Each foot was placed on a separate force plate embedded on top of the movable surface. Reflective markers shown by filled circles were attached on the specified body landmarks. *B*: Examples traces from a representative control subject during a toes-down perturbation. The perturbation onset was denoted as time 0, preceded by a background period of 200 ms. Postural responses were characterized by changes from the background period after perturbation onset. The force data were analyzed in the two integral windows, passive (F1:50-100 ms after perturbation onset) and active response periods (F2:100-350 ms after perturbation onset). Two integral periods of the EMG data were 0-70 ms after perturbation onset (E1:short latency reflex activity period) and 70-320 ms after perturbation onset (E2:active response period).


A
Forward
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z
Front head 1 & 2
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C7
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T10
Shoulder
Mid arm
Elbow
Wrist 1 & 2
PSIS
ASIS
Sacrum
Finger
Mid thigh
Knee
Shank
Ankle
Heel
Toe
Movable surface
Toes-Down 5 degree
X
Y
B
CoP and CoM
CoM
CoP
Forward
Vertical Force
F1
F2
Loading
Unloading
Right
Left
R Medial Gastrocnemius
E1
E2
Support Surface
Toes Down
Displacement (mm)
Magnitude (N)
EMG Amplitude (mV)
Angular Displacement (deg)
Time (msec)
Perturbation Onset

Figure 3.2. Stick figures and traces illustrating the kinematic responses from all control subjects and one stroke subject. Only the relevant plane was shown, i.e., sagittal plane for toes-up (A)/ toes-down (B) perturbations and frontal plane for left-up (C)/ right-up (D) perturbations. The control traces shown are the average of 8 control subjects (thin black) with 95% confident intervals (gray shade), while the stroke traces were selected from a representative stroke subject. The arrow indicates the perturbation onset.

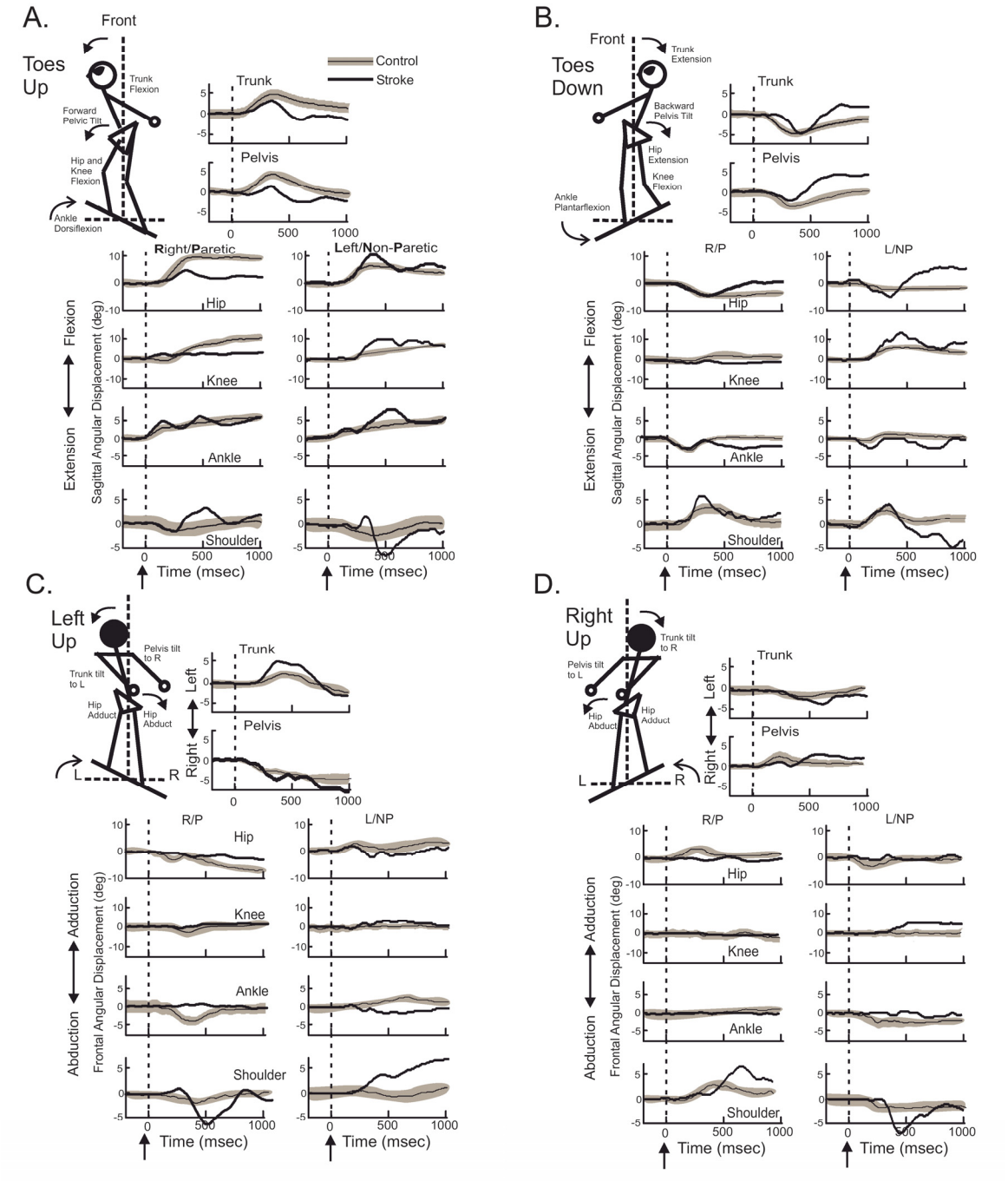

A.
Front
Toes Up
Trunk Flexion
Forward Pelvic Tilt
Hip and Knee Flexion
Ankle Dorsiflexion
Control
Stroke
Trunk
Pelvis
Right/Paretic
Left/Non-Paretic
Hip
Knee
Ankle
Shoulder
Flexion
Extension
Sagittal Angular Displacement (deg)
Time (msec)
B.
Front
Toes Down
Trunk Extension
Backward Pelvis Tilt
Hip Extension
Knee Flexion
Ankle Plantarflexion
R/P
L/NP
C.
Left Up
Pelvis tilt to R
Trunk tilt to L
Hip Adduct
Hip Abduct
Left
Right
Adduction
Abduction
Frontal Angular Displacement (deg)
D.
Right Up
Trunk tilt to R
Pelvis tilt to L

Figure 3.3. Average (±1 SE) peak excursions of the kinematic responses from 0-1,000 ms after perturbation onset in 8 control and 11 stroke subjects plotted against the four different axes of perturbations. Kinematic responses included sagittal and frontal segmental excursion of the trunk (A) and pelvis (B) and sagittal joint excursion of the right or paretic (R/P) and left or non-paretic (L/NP) hip (C), knee (D) and ankle (E).

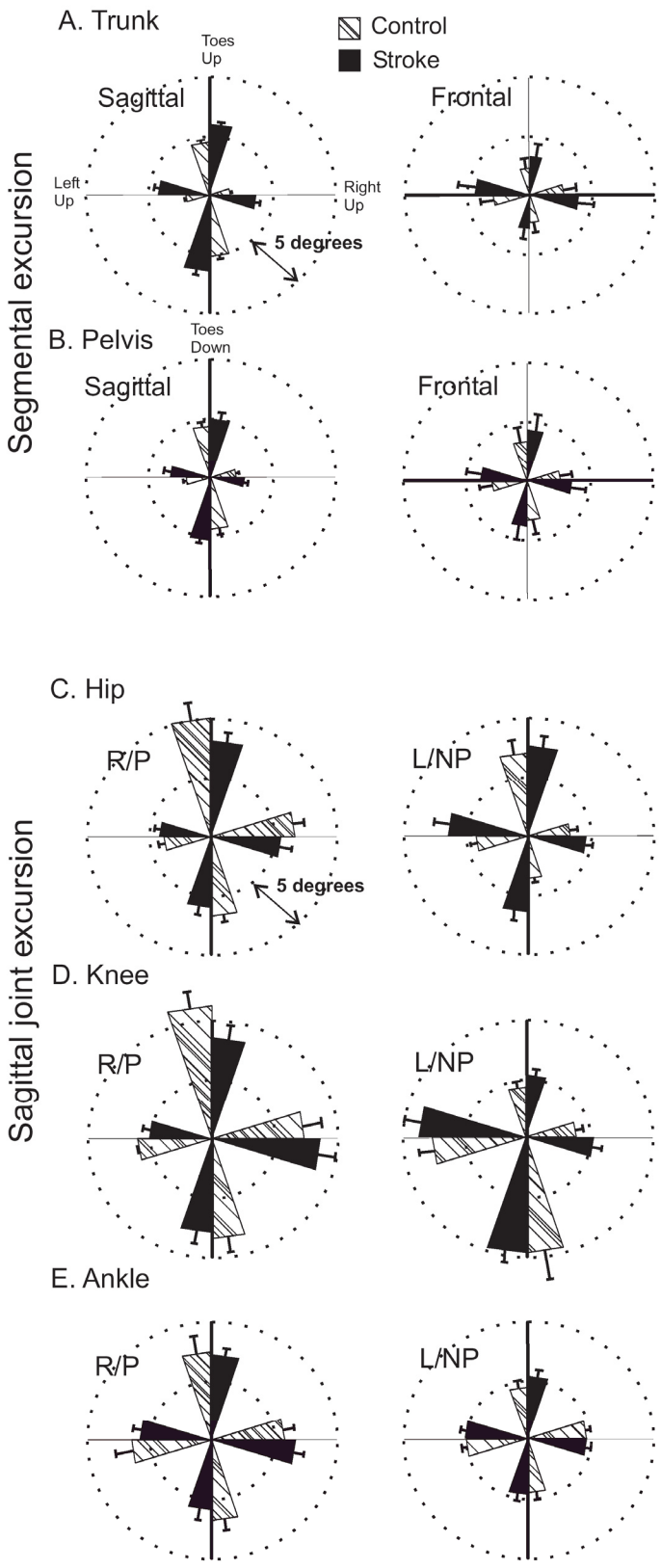
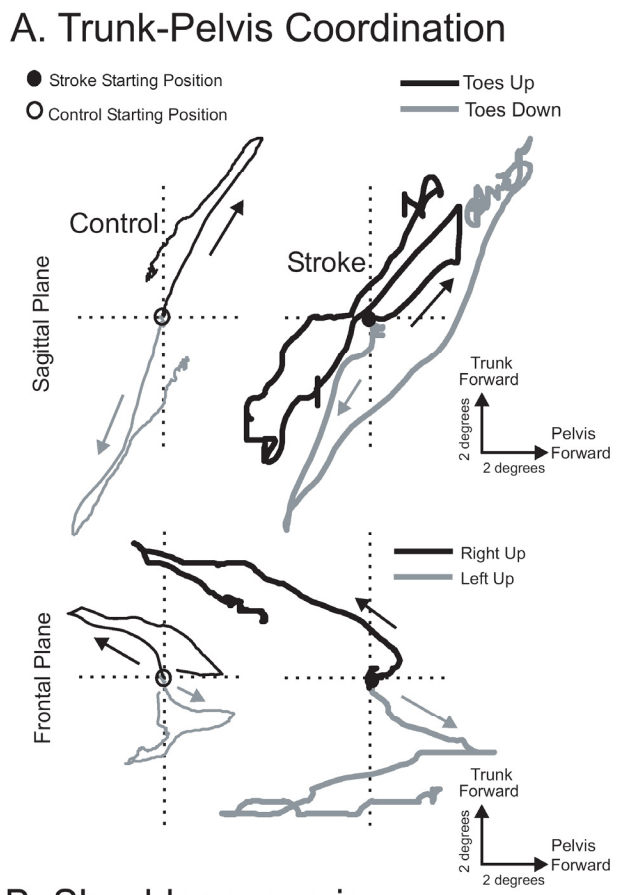

A. Trunk
Control
Stroke
Toes Up
Sagittal
Frontal
Left Up
Right Up
5 degrees
Segmental excursion
B. Pelvis
Toes Down
Sagittal
Frontal
C. Hip
R/P
L/NP
5 degrees
Sagittal joint excursion
D. Knee
R/P
L/NP
E. Ankle
R/P
L/NP

Figure 3.4. A: Contrast of trunk-pelvis coordination between an individual healthy control and a stroke subject from 0-1,000 ms after perturbation onset. Only the relevant plane was shown; i.e., sagittal plane (forward/backward) during toes-up/ toes-down perturbations and frontal plane (rightward/leftward) during right up/left up perturbations. The arrow indicates the initial direction of trunk-pelvic movement. *B*: Average (±1 SE) peak-to-peak amplitude of right or paretic (R/P) and left or non-paretic (L/NP) shoulder excursions from 0 to 1,000ms after perturbation onset in the sagittal and frontal plane of 8 control and 11 stroke subjects plotted against the four different axes of perturbation.


A. Trunk-Pelvis Coordination
Stroke Starting Position
Control Starting Position
Toes Up
Toes Down
Control
Stroke
Sagittal Plane
Trunk Forward
2 degrees
Pelvis Forward
2 degrees
Right Up
Left Up
Frontal Plane
Trunk Forward
2 degrees
Pelvis Forward
2 degrees

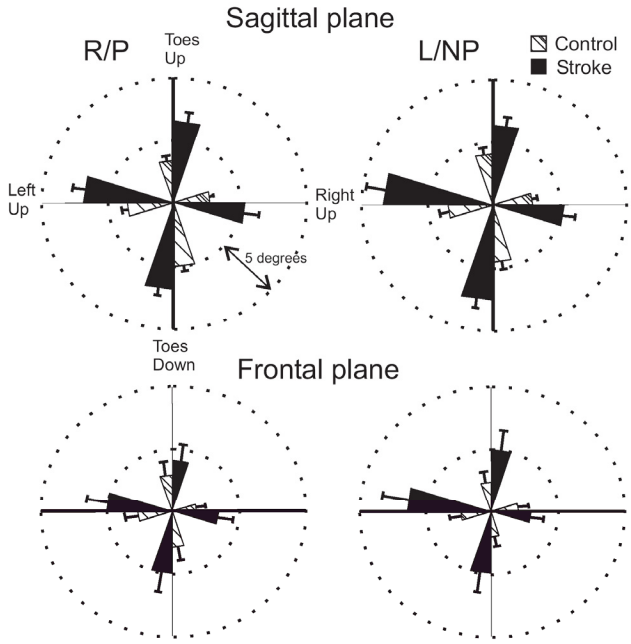
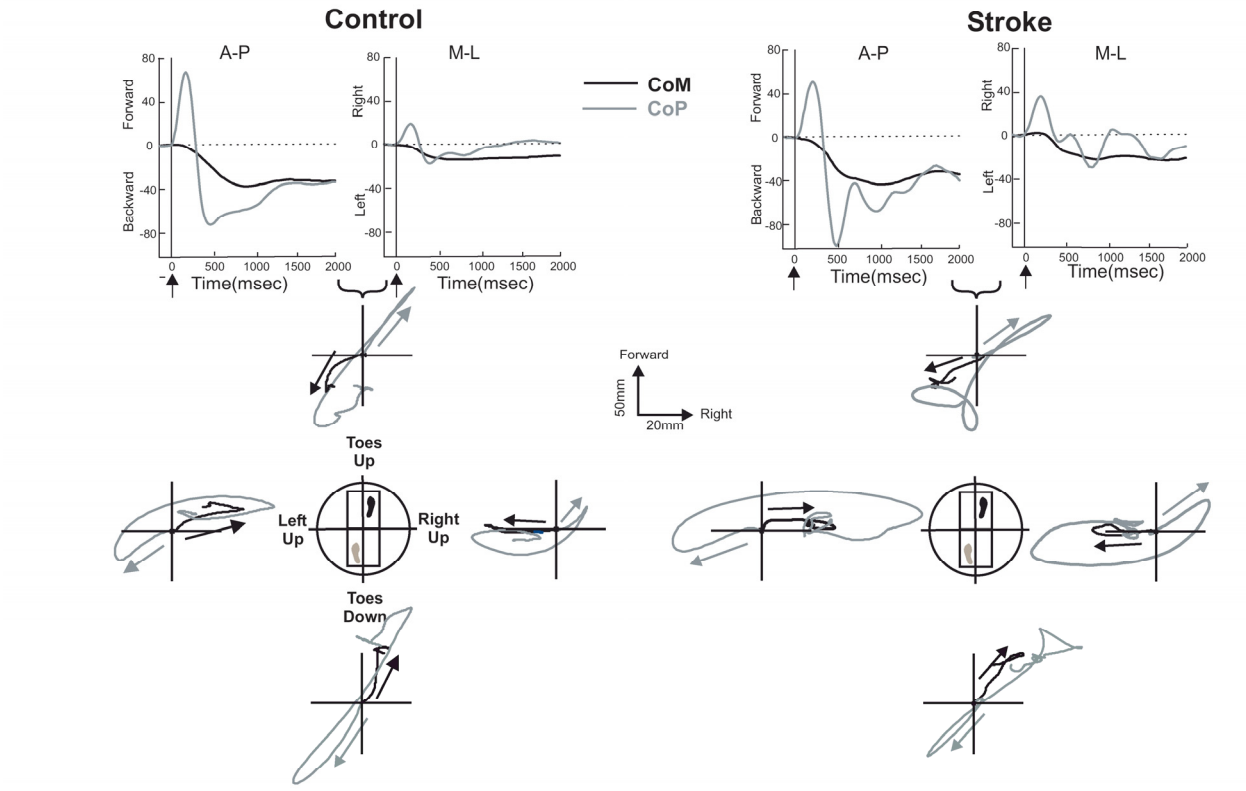

B. Shoulder excursion
Sagittal plane
R/P
Toes Up
L/NP
Control
Stroke
Left Up
Right Up
5 degrees
Toes Down
Frontal plane

Figure 3.5. A: CoP and CoM A/P and M/L displacements from a healthy control and a stroke subject in response to a toes-up surface perturbation. Examples of resultant horizontal displacements of the CoM and CoP for the four axes of perturbations are also shown. The black and grey arrows indicate the initial direction of CoM and CoP displacements, respectively. *B*: Average (±1 SE) peak excursions and CoP displacements from 0-1,000 ms after perturbation onset in the A/P and M/L directions of 8 control and 11 stroke subjects plotted against the four different axes of perturbations C: Average sway variability expressed as RMS of CoP from 350-1,000 ms after perturbation onset in the A/P and M/L directions of 8 control and 11 stroke subjects plotted against the four different axes of perturbation. The asterisk indicates a significant difference between groups.

A. Individual CoP and CoM displacements



B. Group average peak excursions

C. Variability of CoP (RMS)

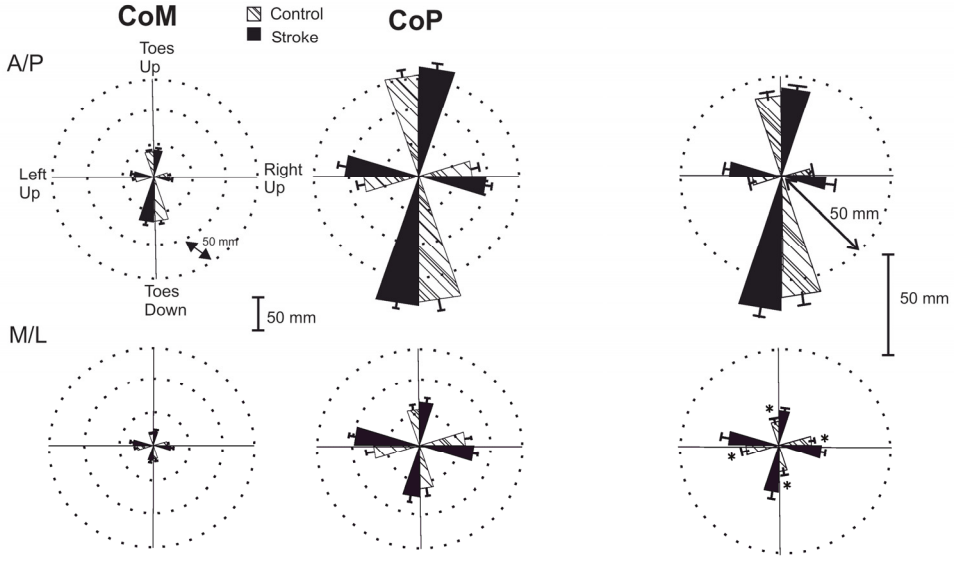
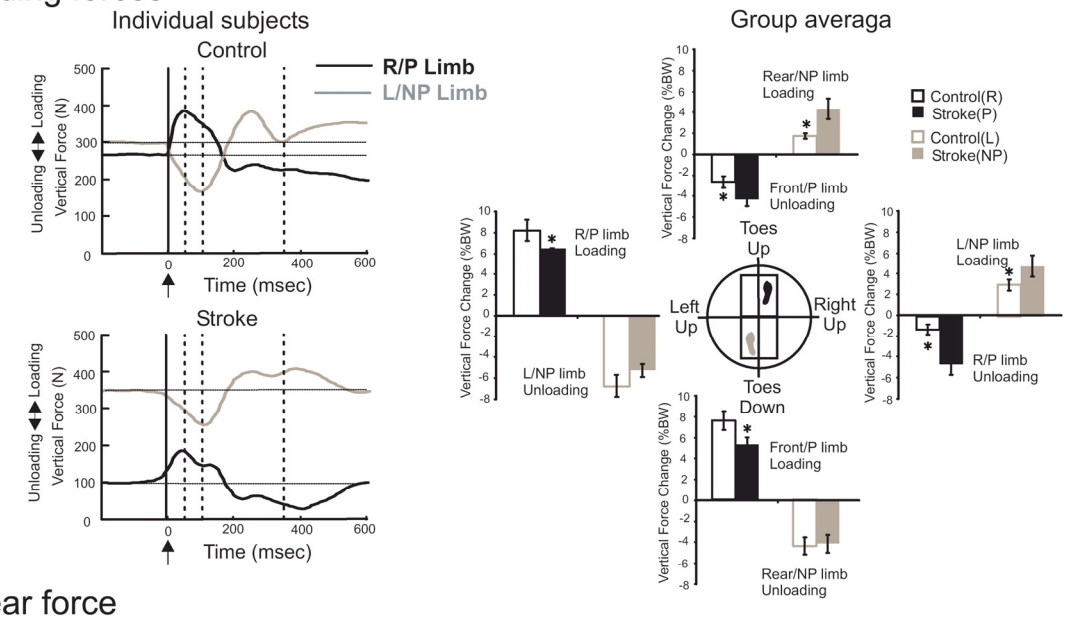


Figure 3.6. A: Left-handed panel showing loading forces on the right/paretic (black) and left/non-paretic (grey) limb in response to toes-up perturbation in a representative healthy control and a stroke subject. The arrow and the vertical solid line indicate the perturbation onset whereas the vertical dash lines delimit the integral windows. The horizontal solid line indicates the background force prior to perturbation onset. Right-handed panel shows average (±1 SE) integrals of loading force (background force removed) on each limb during the active response period (100-350 ms) in response to the different axes of perturbation in 8 control and 11 stroke subjects. Loading force changes were calculated as percentage of body weight where the positive value represents increased loading and the negative value indicates unloading. The asterisk indicates statistical significance between groups. *B*: Integrals of shear force on each foot in response to the different axes of perturbation in control and stroke subjects during the active response period. Thin lines represent the average shear force integral from each individual subject while the arrow represents the grand mean. The shear force integral is a resultant obtained by summing the A/P and M/L force integrals. Loading force changes were calculated as percentage of body weight.

A. Loading forces



B. Shear force

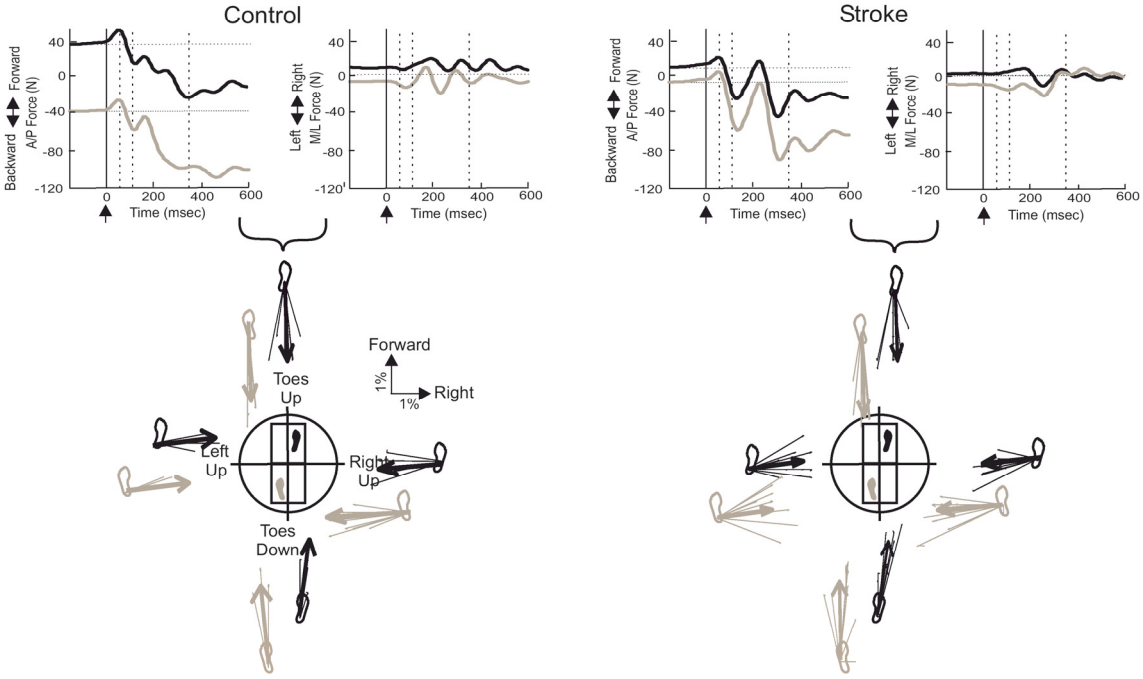
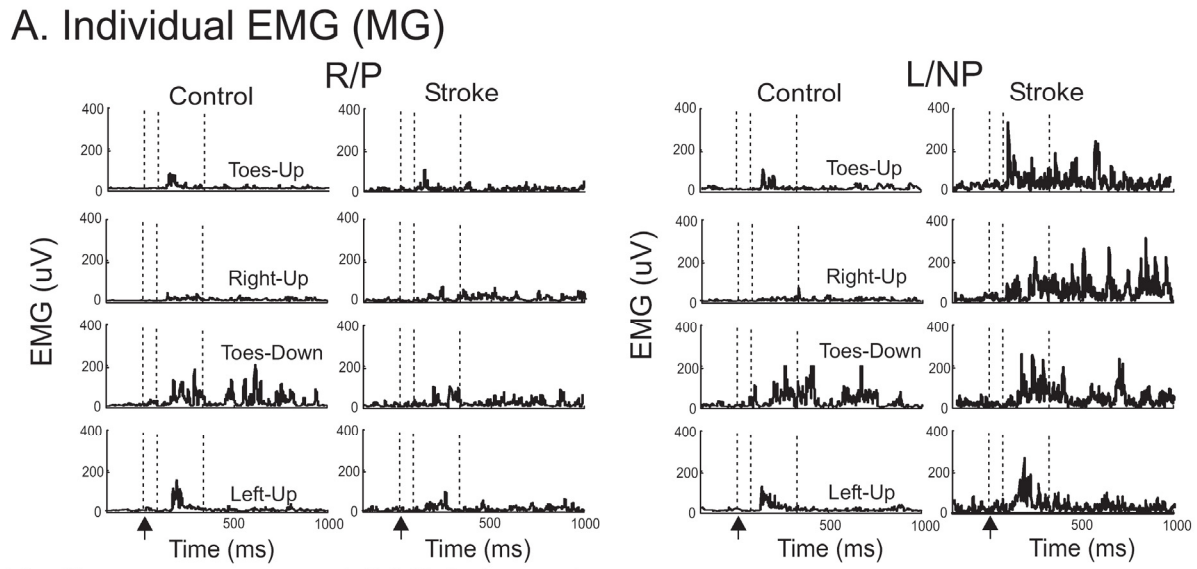


Figure 3.7. A: The full-wave rectified and filtered EMG traces from bilateral medial gastrocnemius muscles taken from a representative healthy control and a stroke subject during the four different axes of perturbation. The arrow and vertical solid line indicate the perturbation onset, whereas the vertical dash lines delimit the integral windows. *B*: Average (±1 SE) EMG integrals normalized to maximum responses during perturbed standing of the same muscle of healthy subject or the same non-paretic muscle of the stroke subjects in the active response period (70-320 ms) of four bilateral lower limb muscles in 8 control and 11 stroke subjects against different axes of perturbation. The asterisk indicates statistical significance between groups.


A. Individual EMG (MG)
R/P
L/NP
Control
Stroke
Toes-Up
Right-Up
Toes-Down
Left-Up
EMG (uV)
Time (ms)
400
200
0
500
1000

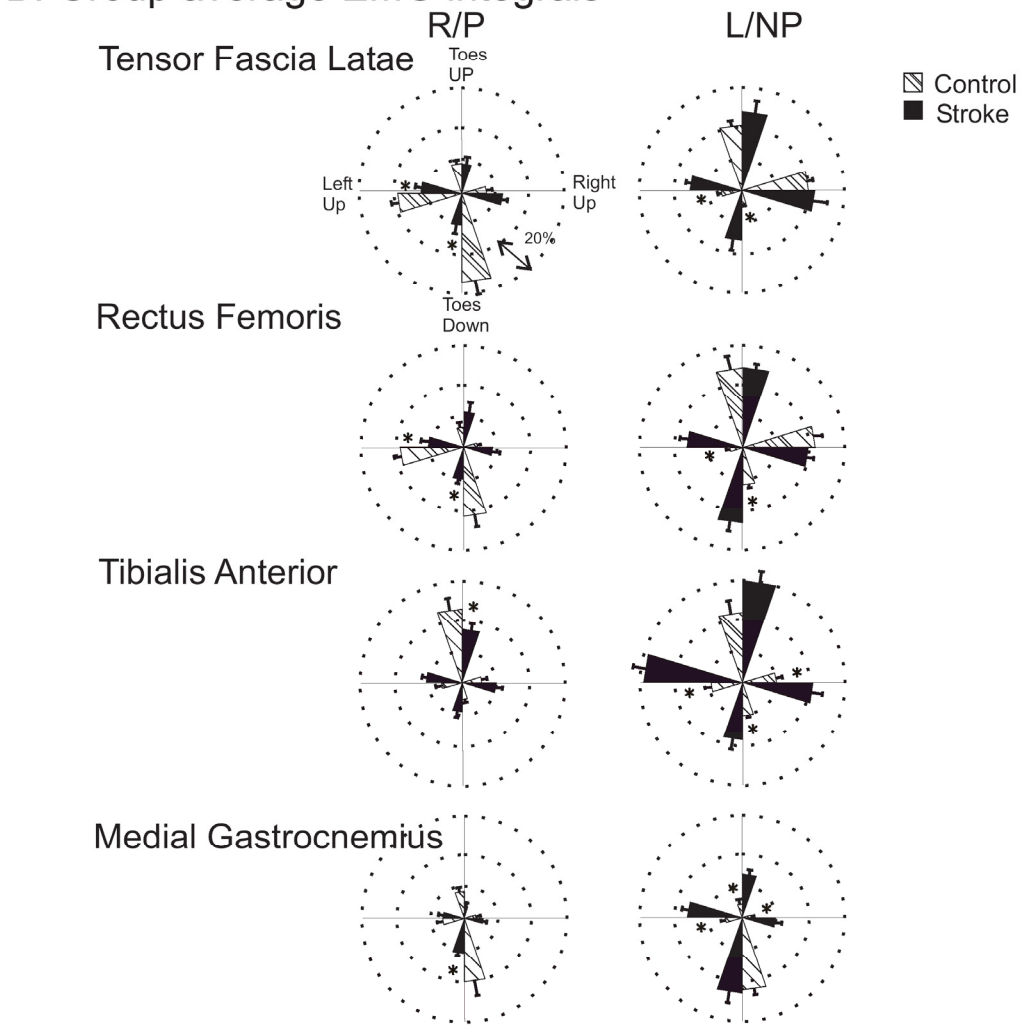
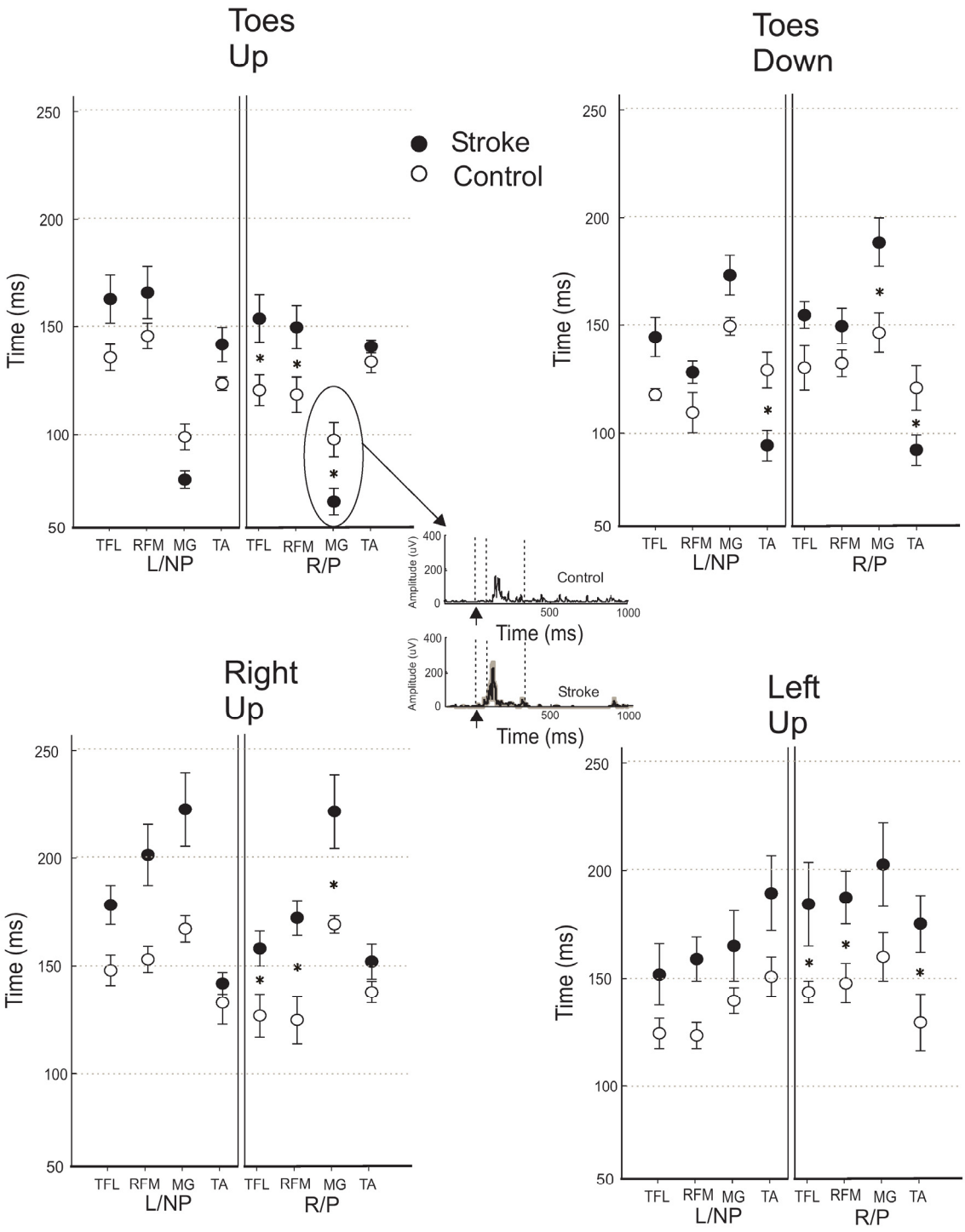

B. Group average EMG integrals
R/P
L/NP
Tensor Fascia Latae
Rectus Femoris
Tibialis Anterior
Medial Gastrocnemius
Toes UP
Toes Down
Left Up
Right Up
20%
Control
Stroke

Figure 3.8. Average (±1 SE) EMG onset latency of four bilateral lower limb muscles; tibialis anterior (TA), medial gastrocnemius (MG), rectus femoris (RFM) and tensor fascia latae (TFL) in 8 control and 11 stroke subjects during 4 directions of perturbation. The oval highlights that the onset latency of MG during toes-up perturbation in the stroke subjects is markedly shorter, in the range of a short latency stretch reflex. Insets show the average traces of MG activation from a representative control and a stroke subject. The arrow and the vertical solid line indicate the perturbation onset, while the vertical dash lines represent the integral windows. The asterisk indicates statistical significance between groups.


Toes
Up
Toes
Down
Right
Up
Left
Up
Stroke
Control
Time (ms)
250
200
150
100
50
TFL RFM MG TA
L/NP
R/P
Amplitude (uV)
400
200
0
500
1000
Control
Stroke

CHAPTER 4

IMPACT OF STROKE ON POSTURAL RESPONSES TRIGGERED BY UNEXPECTED SURFACE PERTURBATIONS IN THE PITCH AND ROLL PLANES: II. DURING LOCOMOTION.

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Submitted to: Brain Research

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The postural control system needs to be able to generate triggered postural responses (TPRs) that are functionally appropriate to maintain equilibrium not only during static condition (i.e., quiet stance) but also during dynamic movement (i.e., locomotion). Results from the study in Chapter 3 showed that stroke disrupted TPRs during quiet stance such that the modulation of TPRs to directions of perturbation was absent. Subjects with stroke demonstrated asymmetrical of TPRs where the muscle activation and force generations of the paretic lower limb were under-activated but these profiles of the non-paretic limb were exaggerated. Nevertheless, it remains unclear as to how the stroke subjects maintain equilibrium when the support surface unexpectedly changes during walking. The study in this chapter was conducted to examine the impact of stroke on TPRs when the perturbation occurred in the pitch and roll planes during walking and to compare the TPRs during two tasks; standing vs. walking.

4.1 Abstract

Postural responses triggered (TPRs) by surface perturbations are modulated as the requirement of the tasks changes, but how this modulation is affected by stroke is unclear. This study aimed to examine the impact of stroke on TPRs during walking as compared to standing. Eleven stroke and 8 age-matched healthy controls were exposed to 4 different surface perturbations (peak velocity 32°/s) in the pitch and roll planes during the double support phase of walking and similar posture during quiet stance. TPRs were characterized by body kinematics, forces exerted under the feet and muscle activities recorded from four bilateral lower limb muscles. TPRs in healthy controls were markedly reduced during walking, as compared to standing. TPRs during walking were apparent only when perturbations occurred in the plane of forward progression, as in toes-up/down direction. Stroke disrupted TPRs such that muscle activations were underactivated on the paretic leg and not modulated according to the directions or tasks. Difficulties in stabilizing the body's center of mass could arise from problems in the control of the trunk, pelvis and the feet center of pressure, more during standing than walking. Forward progression was impeded in stroke subjects, especially during a toes-up perturbation. The findings suggest that balance requirements depend on task goals and the postural adjustments are smaller during walking when the main goal is to maintain forward progression of the body. The task goals of balance and locomotion can be achieved simultaneously in healthy controls but not in stroke subjects who have difficulty in sensorimotor integration.

4.2 Introduction

Locomotion requires not only that the body be propelled in the direction of progression but also that posture and equilibrium be maintained during the course of progression. The control of posture and equilibrium during locomotion involves the regulation of the heavy mass of the trunk over two supporting limbs and the control of position and velocity of the body's center of mass (CoM) in space. Humans keep the trunk erect during locomotion; partly due to lesser mechanical energy expenditure during walking with an erect posture as compared to other postures (Grasso et al. 2000). Control of the trunk motion in the sagittal plane is achieved through the active function of hip flexors and extensors, while the regulation of the trunk in the frontal plane is performed through the foot placement and actions of hip abductors and trunk muscles (MacKinnon and Winter 1993; Winter et al. 1993). During forward locomotion, the CoM advances forward and medial to the supporting foot, creating a continual state of imbalance. This temporary imbalance is corrected by changing the foot center of pressure (CoP) as the swinging foot is placed ahead of and lateral to the moving CoM (Winter et al. 1990).

Locomotion is often challenged under unpredictable situations in daily activities. The central nervous system (CNS) must adapt the locomotor pattern to the environmental changes so that locomotion continues and equilibrium is maintained. Such adaptation requires supraspinal control of goal-directed behaviour (Armstrong 1988; Drew 1988). Quick and unconscious muscle activations with specific spatio-temporal patterns are part of the postural responses triggered (Triggered Postural Responses; TPRs) by an unexpected movement of the support surface. Sudden surface movements have been introduced during various points in the stance (Belanger and Patla 1984; Berger et al. 1984; Dietz et al. 1987; Eng et al. 1997; Figura and Felici 1986; Nashner 1980) or swing phase of walking (Belanger and Patla 1984;

Eng et al. 1997). In those studies, normal locomotion was observed to be maintained with the presence of TPRs that are phase-dependent, being maximal during heel strike and double phase of walking (Nashner 1980). Such phase-modulation is related to the mechanical effectiveness of the responses at different times in the walking cycle (Belanger and Patla 1984; Berger et al. 1984; Nashner 1980). The perturbation-specific characteristic of TPRs is also evident. For instance, toes-down surface rotation changes the excursion and force on the supporting limb resulting in the increased rate of forward progression, whereas downward surface movement alters the vertical height of the supporting leg leading to the extension of the leg to regulate the height of the body (Nashner 1980). In most cases, the adjustments could be achieved within the perturbed step cycle as muscle response pattern did not change in the subsequent step cycle (Tang et al. 1998).

Although very little is known about the location of the postural control center in the brain, the fact that TPRs can easily be accomplished during walking suggests a closed connection between postural control and locomotor centers. The integrated control of locomotion and posture is possible as both tasks share some common principles, such as the control variable is the position of the CoM and the vertical is used as a reference frame (Lacquaniti et al. 1997; Massion 1992). The integrated control of posture and locomotion has been shown by stimulation of the specific sites in the brainstem and hypothalamus, causing the cats to change into different locomotion and postural styles (Mori et al. 1989). Furthermore, the basal ganglia have been found to be involved in the generation of the spatio-temporal framework for the regulation of trunk geometry and the lower limbs coordination with respect to gravity during walking (Garcia-Rill 1986; Grasso et al. 1999).

The goal of postural control during quiet stance and locomotion is different (Winter 1987). The task of maintaining the horizontal projection of

CoM within the base of support (BoS) in quiet stance cannot be applied in locomotion, as the CoM is not always remained within the BoS during walking. Different goals of the task may suggest different postural control strategies between quiet stance and locomotion. However, it would seem more advantageous for the nervous system to use similar control strategies with small adjustments. In fact, it has been shown that passive response strategies, which counteract perturbations during quiet stance, are also effective during walking (Yang et al. 1990a, b). On the contrary, our preliminary results showed that kinematic response strategies are different between standing and walking, and that the responses are much larger in standing as compared to walking. These findings suggest that kinematic responses are organized differently depending on the task and the maintenance of CoM with respect to the base of support (Fung et al. 2002).

Pathologies causing motor or sensory impairments, as seen in stroke subjects who survived a cerebrovascular accident (CVA), often lead to deficits in postural control (Badke and Duncan 1983). TPRs in stroke subjects are characterized by dysfunctions of the paretic limb with over compensation from the non-paretic limb (Badke and Duncan 1983; Di Fabio 1987; Di Fabio et al. 1986; Dickstein et al. 1989; Dietz and Berger 1984). However, all of what is known about TPRs in stroke subjects is derived from postural responses observed during quiet stance. The purpose of this study was to determine how CVA affects the organization of TPRs with respect to different task goals and directions of perturbations. We hypothesized that, due to a disruption in the sensorimotor integration process, TPRs in stroke subjects are more affected by perturbations in quiet stance than in locomotion, and that stroke subjects show no modulation of TPRs with respect to the direction of perturbations during standing or walking.

4.3 Methods

4.3.1 Subjects

Eleven subjects with CVA and eight healthy subjects of similar age and gender participated in the study (Table 4.1). The age of stroke and healthy subjects ranged from 54-80 years and 53-79 years, respectively. All stroke subjects had motor deficits in the lower extremity on the paretic side (Chedoke-McMaster impairment inventory (Gowland et al. 1995), leg or foot scores of 5/7 – 6/7), were able to stand for 5 seconds or more without external support (Chedoke-McMaster postural control score of 3 or more), and had mild to moderate problem in physical mobility (clinical Timed Up and Go test (Podsiadlo and Richardson 1991), score between 10-29 seconds vs. the normal range of 5-7 seconds).

Stroke subjects were recruited from both the in-patient and out-patient neurological rehabilitation program of the Jewish Rehabilitation Hospital and healthy control subjects were volunteers recruited from the surrounding community. The research protocol was approved by the institutional ethics committee and informed consent was obtained from all subjects. Stroke subjects with expressive and/or comprehensive aphasia, severe hemineglect, cerebral aneurysm, bilateral cerebral impairment, or brainstem and cerebellar lesions were excluded from the study. Stroke and control subjects were excluded if they had: (1) a history of lower extremity musculoskeletal conditions (e.g. pain, contracture) in the past year; (2) hemianopsia; (3) evidence of polyneuropathy in the lower extremity; (4) dizziness or other symptoms indicative of vestibular impairment; or (5) were taking psychotropic medications that may affect balance.

4.3.2 Experimental Procedures

A servo-controlled six degree-freedom-of-movement motion-base embedded with two tri-axial AMTI (OR6-7) force plates (Fung and Johnstone 1998) was used to deliver surface perturbations. Each subject was instructed to walk along a 7-meter walkway, where the motion-base device was positioned in the middle, at their own comfortable speed. Subjects were instructed to continue walking when the perturbation occurred as if they were walking on an uneven surface. The starting distance was adjusted for each individual so that the right (in control subjects) or paretic (in stroke subjects) foot always led the other foot and landed with each foot on a different force plate (Fig. 4.1). The length and width of the foot placement on the force plates of each subject during walking were not different from those observed during standing (see Chapter3). None of the subjects used walking aids or ankle-foot orthosis during testing procedures. Each individual wore a body harness that was attached to an overhead rail for safety precaution.

Moderate surface perturbation was given in the pitch and roll planes. Ramp perturbation was delivered at a peak ramp velocity of 32 degree/s and at the maximum amplitude of 5 degree for 150 ms. The chosen velocity and amplitude of surface perturbation was within the range that a person was able to maintain balance without changing the base of support (i.e., stepping) (Allum et al. 1993). The axes of surface rotation were centered with respect to the motion platform and the feet position and the perturbation consisted of one of 4 axes: toes-up, toes-down, right/paretic-up and left/paretic-down (Fig. 4.2). Each trial of data collection lasted for 6 seconds. The platform rotation was triggered during the double support phase of walking when the loading force on each plate was relatively symmetrical and corresponded to approximately 50% of the body weight. In order to reduce any anticipation by the subject, the onset of surface rotation was varied between trials and four unperturbed walking trials were interspersed and collected with the perturbed

trials. Practice sessions were also given prior to testing to familiarize and habituate subjects to the experimental stimuli. A rest period between trials was given to prevent fatigue and all experimental procedures were completed within a single day.

4.3.3 Data collection and analysis

Thirty-eight retro-reflective markers were placed on anatomical landmarks (Fig. 4.1A) to capture body motions and four additional markers were placed on motion platform to capture surface movements. Three-dimensional motion capture was performed by a high-resolution six-camera VICON motion analysis system (Vicon 512; Oxford Metrics Ltd) at a sampling frequency of 120 Hz. The data were low-pass filtered at 10 Hz using a 2^{nd} order dual-pass Butterworth filter, based on a previous residual analysis of the kinematics (Winter 1990). Anthropometric measurements were obtained from each subject to calculate the position of the body CoM. Segmental and joint angles were calculated based on segments formed by at least three non-coplanar markers.

The triaxial ground reaction forces (GRFs), including the anteroposterior (A/P), mediolateral (M/L), and vertical components (Fx, Fy and Fz respectively) and the moments of force (Mx, My, Mz) were recorded from two force plates (AMTI OR6-7) mounted within the motion base. The force signals were amplified and sampled at 1,080 Hz. Any bias in force signals at the onset of perturbation due to the inertial characteristics of the force plate was subtracted based on inverse dynamics calculation from motions of the platform (Preuss and Fung 2002).

Muscle activities were recorded by an 8-channel telemetric EMG unit (Noraxon USA, Inc.). After appropriate skin preparation, bipolar silver-silver chloride disposable surface electrodes were placed over muscle bellies of four bilateral lower limb muscles: tibialis anterior, medial gastrocnemius,

rectus femoris, and tensor fascia latae. EMG recordings were band-pass filtered between 16 and 500 Hz and sampled at 1,080 Hz. The EMG signals were then full-wave rectified and low-pass filtered at 100 Hz.

Data were further analyzed using BodyBuilder (Oxford Metrics Ltd.) and Matlab (MathWorks Inc.) software. Data were normalized to the gait cycle starting from one initial contact of the foot to the next. An average of four unperturbed walking trials was used as the reference. This reference was subtracted from the data in the perturbed walking trial to obtain the relevant postural responses due to surface perturbation. Maximum ranges of displacements (peak-to-peak amplitude) of body CoM, ankle, knee, hip and shoulder joint angles and pelvic and trunk segmental angles in the sagittal and frontal planes from 0-350 ms after perturbation onset were calculated (Fig. 1). This period of interest was shorter than the period during the standing task (0-1,000 ms), as changes during the walking task occurred within 350 ms after perturbation onset before rapidly returning to the reference level. The variation in postural restoration during locomotion was determined by computing the root-mean-square (RMS) of body CoM, trunk and pelvis excursion in the sagittal and frontal planes between 350 to 1,000 ms after perturbation onset with respect to the unperturbed walking trials. The average gait speed was calculated by dividing the average distance of CoM progression by the cycle duration for 2 consecutive gait cycles in the four unperturbed walking trials. The change of instantaneous gait velocity due to the perturbation was calculated by subtracting the CoM velocity at 350 ms after perturbation onset from the CoM velocity at 350 ms before perturbation onset. The center of foot pressure (CoP) from each force plate in the anteroposterior (A/P, CoPx) and mediolateral (M/L, CoPy) directions was obtained and the resultant center of pressure (rCoP) from the 2 force plates was then calculated (Henry et al. 1998a). Peak-to-peak amplitude of rCoP displacements in the A/P and M/L directions from 0-350 ms after perturbation

and RMS of rCoP displacements from 350-1,000 ms in the A/P and M/L directions were obtained.

Integrals of EMG and GRF were computed based on two time-windows (Fig. 4.1B) (Henry et al. 1998a; Henry et al. 1998b) after the onset of surface movement. For EMG analysis, the integrals were calculated for the *short latency reflex activity period* (E1) and *active response period* (E2), spanning 0-70 ms and 70-320 ms respectively after perturbation onset. Corresponding periods with 30 ms delay were used for GRF analysis, taking into consideration the electromechanical delay. The EMG activity was normalized to the peak activity of the same muscle for control subjects and to the peak activity of the non-paretic side for stroke subjects. A muscle was considered as having a real response only when the firing probability reaches 75% (i.e., activated in 3 out of 4 trials of perturbation) (Henry et al. 1998b). The GRFs were normalized to the subject's body weight. The Fx and Fy vector integrals were summed to produce the resultant horizontal force vector integral under each foot.

4.3.4 Statistical analysis

Outcomes in kinetics, kinematics and EMG were averaged for each axis of perturbation, as there was no significant difference between trials. All statistical analyses were performed using the Statistica software (StatSoft Inc.). Two-way ANOVAs were used to test for any main or interaction effects due to subject group (stroke vs. control) and axis of perturbation (toes-up vs. toes-down vs. right-up vs. left-up). The main or interaction effect due to tasks (stand vs. walk), subject group and axis of perturbation was evaluated by using a three-way ANOVA. When significant differences were found after multiple comparisons had been adjusted with the Bonferroni test ($p<0.05$), pairwise comparisons were performed using the Tukey test.

4.4 Results

4.4.1 Kinematic responses

Pitch and roll surface perturbations introduced during the double support phase of walking displaced both lower limbs. However, most of postural adjustments occurred on the leading limb (right or paretic limb), as the trailing limb was soon lifted off the ground during swing and all the weight was transferred onto the leading limb in front to support the body. Figure 4.2 shows the kinematic responses in the sagittal plane of the trunk, pelvis and the leading limb's hip, knee and ankle joints during 4 directions of perturbation in a representative healthy control subject, as compared to the average kinematic profiles (with the 95% confidence intervals) obtained from all healthy subjects during the unperturbed cycle. In response to surface rotations, little kinematic adjustments were made at all segments and joints, except at the ankle joint which moved into dorsiflexion (during toes-up and right-up rotations) or plantarflexion (during toes-down and left-up rotations). This finding indicates that the maintenance of equilibrium during walking in control subjects can be adequately accomplished by adjusting only at the ankle joint of the stance limb. In contrast, kinematic responses in stroke subjects (Fig. 4.3) involved adjustments at multiple segments, including the trunk, pelvis and the leading lower limb. Stroke subjects responded by flexinging the trunk, regardless of perturbed directions, except during toes-up rotation where the trunk was displaced into extension. The pelvis was tilted in the forward direction with the flexion of the hip and knee joints. Ankle dorsiflexion and plantarflexion were also seen in the stroke subjects, depending on the direction of perturbations, to accommodate for the change in surface inclination.

Figure 4.4 compares the average peak-to-peak changes in segmental angles of the trunk and pelvis and joint angles of the hip, knee, ankle and shoulder between control and stroke subjects. In general, the amount of

changes at the axial segments, the trunk and pelvis, was approximately the same in the sagittal and frontal planes, whereas the adjustment at the distal segments such as the lower limb occurred mainly in the sagittal plane with minimal adjustments in the frontal plane (<3°). In the control subjects, maximum changes were seen at the ankle joint (Fig. 4.4C), whereas changes at the trunk and pelvis (Fig. 4.4A) were minimal (~1°) with the highest changes occurring in the sagittal plane during toes-up or toes-down rotations. It is evident that, in response to surface rotation during walking, the control subjects made only the adjustment at the ankle joint to adapt to the surface changes and maintained axial stability and postural changes that normally occurred with unperturbed walking. In the stroke subjects, in addition to the adaptation seen at the ankle joint, some changes were also evident at the hip and knee joints (Fig. 4.4C) but the average changes were not significantly different from the control subjects, due to the variability in kinematic profiles of stroke subjects. Surface rotation in the toes-up and toes-down directions triggered significantly large changes of the trunk and pelvis in the sagittal plane, as compared to the frontal plane in stroke subjects as well as for the control subjects ($p<0.001$). Relevant differences between the two groups of subjects, however, were found in the amount of trunk and pelvic excursions. The amount of excursion changes at the trunk ($p<0.01$) and pelvic ($p<0.05$) segments in the stroke subjects was significantly larger in both sagittal and frontal planes during all perturbed directions, as compared to the control subjects (Fig. 4.4A). The ability to maintain the trunk and pelvic positions was affected in the stroke subjects, as can be seen from significantly larger RMS of changes in the excursions of the trunk ($p<0.01$) and pelvis ($p<0.05$) in the sagittal and frontal planes between 350-1,000 ms after perturbation onset (Fig. 4.4B). RMS of trunk and pelvic excursions reflected the stability of the trunk and pelvis during the maintenance of equilibrium after disturbances, where larger RMS indicated less stability of the observed segments.

Significantly larger bilateral arm movements ($p<0.05$) in the sagittal and frontal planes of the stroke subjects (Fig. 4.4D) suggested that the patients used larger movements of the paretic and non-paretic arms to compensate for poor trunk and pelvis stabilization.

Kinematic responses when perturbation occurred during walking were different from the responses during stance perturbations. Control subjects can correct the disturbances due to surface rotation during walking by adjusting the distal segment without any involvement from the axial segments. In contrast, the adjustments of both distal and axial segments were required during stance perturbations. Another important feature that distinguished kinematic responses during standing and walking was the magnitude of responses. Regardless of the directions of perturbation, the amount of responses at the trunk, pelvis and stance lower limb in the control subjects was much smaller ($p<0.001$) when surface perturbations occurred during walking, as compared to standing (Fig. 4.5). Surprisingly, stroke subjects also demonstrated significant lower magnitude of kinematic responses at the axial and distal segments during walking than during standing ($p<0.001$), even though kinematic responses during both tasks were impaired such that both involved the adjustments at the distal and axial segments. However, the amount of decrease in peak excursions, as the task changed from standing to walking, was smaller in stroke subjects (~42%) as compared to control subjects (~58%). Nevertheless, these findings indicate that the equilibrium demand was likely to be much higher during quiet stance as compared to forward locomotion. The fact that stroke subjects also showed smaller kinematic adjustments during walking may suggest that postural control in stroke subjects, specifically the stabilization of the trunk and pelvis, were less affected when responding to perturbations during locomotion, as compared to quiet stance.

4.4.2 The control of body CoM and CoP

In forward walking without any surface perturbation, when the right foot was placed in front of the left foot, the CoM moved forward and rightward from the left (non-paretic) to the right (paretic) foot (Fig. 4.6A). The CoP was displaced in the same direction but with a larger magnitude than the CoM. The position of CoP was shifted in a larger distance during the transition from left to right foot. Then the CoP moved in a smaller distance within the area limited by one foot as the left foot changed into the swing phase, leaving only the right foot to support the body. Rotation of the support surface occurring during walking changed the trajectory of the CoM and CoP (Fig. 4.6A). Although the CoM still moved in the forward direction, its forward displacement was reduced for all perturbed directions, except during toes-down rotation where the forward displacement was increased in the control subjects. The CoM excursion in the lateral direction was also altered with no specific pattern. The CoP moved backward and leftward during toes-up and right-up rotation but forward and rightward during toes-down and left-up rotation.

Figure 4.6B shows the amount of average changes in A/P and M/L displacements of CoM and CoP when perturbation occurred during walking in control and stroke subjects. In general, the changes of CoM and CoP displacements were also smaller during walking, as compared to standing. During walking, changes of CoM and CoP displacements were higher in the A/P than M/L direction with the maximum changes occurred during toes-up/down rotations ($p<0.0001$). Stroke subjects demonstrated significantly larger CoM changes in the A/P direction during all perturbed directions ($p<0.05$), as compared to control subjects. Both A/P and M/L CoP changes were also significantly increased in the stroke subjects during all perturbed directions ($p<0.05$), indicating that stroke subjects had difficulty in controlling the body CoM during any direction of perturbation. Instability of the body CoM

in stroke subjects was confirmed by larger RMS of changes in CoM displacements in the A/P and M/L directions during 350-1,000 ms after perturbation onset which was the duration when only the right or paretic foot was left on the ground (Fig. 4.6C). Stroke subjects demonstrated significant higher RMS of changes in CoP displacement in the M/L ($p<0.01$) but not in the A/P direction, suggesting that the maintenance of M/L rather than A/P stability may be more impaired in balance corrections during walking.

The speed of forward progression as demonstrated by the changes in instantaneous CoM velocity in the A/P direction during 4 directions of perturbation was shown in Figure 4.7. The average gait velocity during normal comfortable walking (with no perturbation) in control and stroke subjects was 1.4 ms^{-1} and 0.7 ms^{-1}, respectively. The healthy subject maintained steady CoM velocity during normal forward locomotion but the instantaneous velocity was affected by the unexpected movement of the support surface (Fig. 4.7A). In control subjects, the speed of forward progression was not altered when perturbation occurred in the right-up or left-up direction, whereas it changed when the surface moved in the toes-up or toes-down direction. Toes-up rotation reduced the CoM velocity in control subjects by 10% ($p<0.005$), while toes-down rotation increased the speed of forward progression by 9% ($p<0.01$). The speed of forward progression in the stroke subjects, however, was substantially and significantly reduced when perturbation occurred in all perturbed directions, except during toes-down rotation where a minimal reduction was evident. Toes-up rotation was found to be the most disturbing direction, as the speed of forward progression was maximally decreased ($p<0.001$). Two distinct patterns of changes were observed. In the 3 stroke subjects who had the lowest unperturbed gait velocity overground (0.4 - 0.5 ms^{-1}, see Table 4.1), sudden surface perturbations during walking induced a decrease in CoM velocity by more than 90%, indicating almost a total arrest of locomotion. The other 8 stroke subjects (unperturbed gait velocity ranged

from 0.6 - 1.0 ms^{-1}) could continue locomotion while maintaining equilibrium, although the speed of progression was much slower (decreased by 50%) than the control subjects. Figure 4.7C demonstrates the correlation of postural instability with gait velocity change during toes-up perturbation in two groups of subjects. The correlation coefficient (r) in the control subjects ranged from 0.20-0.35, whereas it ranged from 0.70-0.79 in the stroke subjects. Low correlation in the control subjects was due to the fact that all subjects had smaller changes in the gait velocity and RMS of CoM. High correlation of changes in CoM velocity and RMS of CoM in the A/P and M/L direction suggest that a stroke subject who made larger decrease in speed of forward progression also have larger instability in the control of the CoM. Therefore, it is likely that the problem in stabilizing the CoM during walking in stroke subjects corresponds to the ability to maintain constant speed of forward progression and the severity of this problem may determine how the stroke subjects would respond to the toes-up perturbation occurred during walking.

4.4.3 Contact forces under the feet

Changes in surface inclination during double support phase of walking triggered adjustments of the contact force under the feet, mainly in the leading (right or paretic) limb. Most of the adjustments occurred in the vertical force while adjustments of the shear force were minimal. The pattern of changes in shear forces of the leading limb during walking was similar to those during standing. Such pattern involved the displacement of the shear forces in the opposite direction to the direction of the CoM displacements, to generate the recovering force for counteracting the motion of the CoM. Both groups of subjects showed similar pattern and magnitude of shear force adjustments during walking.

Figure 4.8A demonstrates the trajectories of vertical force when

perturbation occurred in the toes-up direction during walking in control and stroke subjects. During the passive period (50-100 ms), the increase in the vertical force was likely related to passive viscoelastic properties of the musculoskeletal system, as no increased muscle activities was seen. However, increase in loading still persisted shortly during early active period due to active resistance to upward surface movement, followed by unloading during the entire active period (100-350 ms) to accommodate the change in surface inclination. However, loading of the leading stance limb occurred after the active period to generate the push-off power used in forward propulsion. It can also be seen that changes in the vertical force were rapidly adjusted back to normal walking at the push off peak of the same stance cycle, suggesting that force adjustment during walking was brief and a person was able to resume normal pattern of walking within the same stance cycle. The opposite pattern of vertical force adjustments was found when perturbation occurred in the toes-down direction during walking. Vertical force adjustments during walking, however, were negligible in response to right-up or left-up perturbations. Such adjustments during walking were different from those during standing where the adjustments of vertical force occurred during all perturbed directions.

Both groups of subjects showed a similar pattern of vertical force adjustments during walking. However, the difference was seen at the magnitude of average vertical force changes on the stance limb during the active period, in response to all perturbed directions ($p<0.001$) (Fig. 4.8B). Stroke subjects showed more unloading on the paretic limb during all perturbed directions, except during toes-down rotation where less loading occurred. Less loading and more unloading may imply that force generation in stroke subjects was not sufficient to propel the body forward. This finding may underlie the larger reduction in the speed of forward progression when encountering the perturbation during walking in stroke subjects.

4.4.4 Muscle activities

Figure 4.9A showed muscle responses on the leading stance (right or paretic) limb during right-up perturbation in a representative control and stroke subject. In general, muscle responses began in the active period (70-320 ms) and there were no changes in muscle activities occurring in the short latency reflex activity period (0-70 ms). The responses of the four muscles, tibialis anterior (TA), medial gastrocnemius (MG), rectus femoris (RFM) and tensor fascia latae (TFL), were minimal in the control subjects. In contrast, large activation and co-contraction were common features in the stroke subjects. The pattern of directional modulation in the four muscles during the active period was shown in Figure 4.9B. In control subjects, TFL, RFM and MG were activated maximally during toes-down perturbation, whereas TA was activated highest during toes-up rotation. The amount of muscle activations was minimal when perturbation occurred in the right-up and left-up direction. This pattern of muscle modulation in the control subjects corresponds to the pattern of loading or unloading in that TA was activated to unload the limb. Stroke subjects, on the other hand, showed no muscle directional modulation, as the amount of muscle activations was approximately the same during any perturbed direction. Responses from all recorded paretic muscles were always larger than those observed in the control subjects when perturbations occurred in the right-up or left-up directions. In contrast, during toes-up or toes-down perturbations, the muscles that were normally recruited for balance corrections were markedly smaller in activation amplitude in the paretic limb of stroke subjects ($p<0.05$).

Figure 4.9C contrasted the average changes in EMG integrals in the active period across all perturbed directions in standing and walking. In control subjects, EMG integrals were much smaller when recruited for balance corrections during walking, as compared to standing ($p<0.005$). This finding indicates that muscle adjustments were less demanding when

perturbation occurred during walking, and that control subjects could modulate the responses depending on the demand of the task. In contrast, the amount of EMG activation in the paretic limb of stroke subjects was not significantly different between standing and walking, suggesting that the ability to modulate the muscular responses to the task demand was impaired following stroke.

4.5 Discussion

This study shows that TPRs were task-specific such that they were much larger during quiet stance, as compared to locomotion. TPRs during quiet stance were seen when perturbations occurred in all observed pitch and roll perturbations, whereas TPRs during walking were elicited only when perturbations occurred in the plane of progression as in the toes-up and toes-down direction. Our original hypothesis was supported, in that following CVA, TPRs were impaired such that they were not modulated with respect to the directions and demands of the task. An under-activation of the paretic lower limb muscles was seen with difficulty in stabilizing the trunk and pelvis, more during quiet stance than walking. Stroke subjects demonstrated impairment in continuing locomotion when encountering the perturbations, especially in the toes-up direction that directly impeded forward progression.

4.5.1 The integration of balance and locomotion

Successful locomotion requires the ability to generate the locomotor pattern as well as the ability to maintain dynamic equilibrium over the changing BoS and during various internal and external disturbances (Shik and Orlovsky 1976). In our present study, when surface perturbation occurred during walking, healthy subjects could easily maintain body equilibrium without interrupting the forward progression, as demonstrated by minimal changes in the CoM velocity after the perturbations, suggesting that the integration of postural control and locomotion can be simultaneously achieved by the intact CNS. Such integration is necessary to ensure safe transport across different environmental contexts (Winter 1987). It is likely that this dual task integration to maintain body equilibrium and progression is organized at the supraspinal levels (Teasdale et al. 1992; Teasdale et al. 1993), in which the amount of supraspinal control is dependent on the level of instability of the

body (Lajoie et al. 1993). Several studies suggested that the control of posture and equilibrium is mutually dependent at multiple levels of the CNS, including the motor cortex, basal ganglia, brainstem and the spinal cord (Garcia-Rill 1986; Grasso et al. 1999; Jankowska and Edgley 1993; Mori 1987; Mori et al. 1989).

The ability to perform the dual task of postural control and locomotion in the control subjects, however, was varied depending on the direction of perturbation. Postural adjustments were integrated into the locomotion pattern without changing the rate of forward progression when the perturbation occurred in the right-up or left-up direction. In contrast, when the perturbation opposed the plane of progression, as in toes-up and toes-down perturbation, the rate of forward progression was slower (during toes-up perturbation) or faster (during toes-down perturbation). It has been suggested that changes in the force exerted by the stance limb during surface tilts may underlie the regulation of rate of body forward progression (Nashner 1980). In fact, we found that minimal adjustment in loading force during right-up and left-up perturbation corresponded to slight change in the speed of forward progression. Increased loading force may facilitate the speed up of forward progression during toes-down perturbation, whereas unloading of the stance leg during toes-up tilt was related to decreased rate of forward progression.

In contrast to the control subjects, the integration of postural control and locomotion was vastly disrupted following stroke. Stroke subjects showed larger decreased in the speed of forward progression during all perturbed directions. Loading force adjustments in the stroke subjects were also impaired such that the paretic stance limb always bore less loading force, as compared to the control subjects. Less load bearing may give rise to inadequate propelling force to bring the body forward, and hence lead to significant decrease in the forward progression speed. Furthermore, the magnitude of decrease in weight bearing on the paretic limb may be related to

the amount of reduction in the speed of forward progression. For instance, we found that minimal reduction in the forward progression speed occurred when the perturbation was in the toes-down direction, where the magnitude of load bearing on the paretic limb was the highest. It is also shown that in the stroke subjects, the speed of forward progression and the stability of CoM in the A/P and M/L directions were highly correlated. The high correlation between the speed of forward progression and the A/P stability of CoM is expected, as forward walking primarily involves the displacement of the body in the anterior direction. However, we had not expected a high correlation between the speed of forward progression and M/L stability of CoM. This finding may be due to the fact that the period of response falls into the single support phase, where the stability in the frontal plane is compromised, more in the stroke than the control subjects. Therefore, problems in the frontal stability of stroke subjects could also contribute to decreased speed of forward progression, leading to high correlation between the decrease in speed of forward progression and M/L CoP during perturbed locomotion.

In the performance of dual tasks, one or both tasks could be disturbed if both tasks exceed the shared central processing capacity (Kerr et al. 1985). Several previous studies have shown that stroke subjects had difficulties in performing dual tasks. For instance, stroke subjects reduced the speed of walking or even stopped walking while performing a cognitive task such as talking (Bowen et al. 2001; Haggard et al. 2000) or they reduced the speed of arm raising while adjusting the anticipatory postural responses (Garland et al. 1997). We also found that both right and left stroke subjects had similar difficulty in the integration of balance and locomotion. This finding is in agreement with the previous study examining the interference between locomotion and cognitive tasks (Haggard et al. 2000), in which it is concluded that brain laterality did not influence the disruption of dual tasks differently. Haggard et al. (2000) suggested that the impairment of one task in the dual

task performance was due to competition for limited central capacity which was impaired following stroke, such that the additional capacity for another concurrent task was diminished. Therefore, in our study, the marked reduction in the speed of forward progression (i.e., concurrent task) in the stroke subjects could also reflect the limitation of the central processing circuit involving in the control of posture and equilibrium.

4.5.2 Task-specific postural requirements

Posture and equilibrium are disturbed constantly under static and dynamic conditions during daily activities. We showed that postural adjustments in response to surface tilts in healthy controls were modulated according to the task, such that the amplitude decreased from standing to walking when the main goal was to keep the CoM advancing over the changing BoS. Different kinematic responses were seen when perturbation occurred during static or dynamic tasks. Similar to the previous findings (Figura and Felici 1986), we found that most of the responses during walking were seen in the stance limb during all perturbed directions. In contrast, both lower limbs contributed significantly to balance corrections during standing. We also showed that postural responses during standing occurred at the axial and distal segments. In contrast, postural responses during walking involved the adjustments only at the ankle joint with negligible adjustments from the axial segment (i.e., trunk and pelvis). This finding is in agreement with the previous reports examining postural responses to surface translation during walking (Tang et al. 1998) and showing that activities from muscles of the legs and thighs were the primary contributor to restore balance during walking. Another study suggested that muscles activities around the hip generated an extensor moment during early stance to control the upper body and prevent collapse of the lower limbs at the initial onset of perturbation, whereas a knee extensor moment was generated during late stance to

prevent the collapse of the lower extremities (Ferber et al. 2002). The patterns of force and muscle responses during static and dynamic equilibrium, however, could be either similar or different, depending on the direction of perturbation. Force and muscle adjustments were different when perturbation occurred in the lateral direction, whereas they were similar when the perturbation was presented in the A/P direction. Thus, the previous findings (Nashner 1980) supporting that postural responses were similar during standing and walking did not contradict our results, as the conclusion was based only on muscle activities (i.e., TA and MG) during antero-posterior surface tilts.

The distinction of postural responses between static and dynamic task could be due to difference in the goal of the task. The maintenance of equilibrium during static condition as in quiet stance involves the maintenance of the projection on the ground of the body CoM within the BoS. In contrast, while the CoM rarely passes in the BoS during walking (Winter 1990), equilibrium maintenance is achieved through the control of the body CoM to new positions across a given trajectory (Massion 1984). Minimal postural adjustments during walking suggested that equilibrium requirements were less during walking, as compared to standing. It is also likely that the postural control system takes the advantage of a more excitable motoneuronal pool that controls the ongoing forward progression. Differences in responding strategy and postural requirements between the two tasks, however, do not imply different neural mechanisms in controlling equilibrium during static and dynamic tasks. It is suggested that static and dynamic equilibrium could possibly share the same neural mechanisms, as both tasks are involved in the control of the position of body CoM in space (Pozzo et al. 1990) and both require the integration of inputs from visual, vestibular and proprioceptive systems to generate the appropriate egocentric and exocentric frame of reference (Paillard 1988).

Impairment in the task-specific modulation of the paretic muscle can be related directly to lesions of the descending pathways as a result of stroke. It should be noted that task-specific modulation of the non-paretic muscles was not observed in this study. The fact that stroke subjects could adjust postural responses, in terms of kinematic and force profiles, to the task, may suggest that the CNS retains partially the ability to modulate postural responses, at least for the non-paretic muscles, according to different task goals.

4.5.3 Functional significance

In the rehabilitation of posture and locomotion for patients with stroke, several studies showed that training of the performance in one task did not improve the performance of another untrained task (Dean and Shepherd 1997; Engardt et al. 1995; Winstein et al. 1989). For instance, a more symmetrical standing posture was resulted from balance retraining program using augmented visual feedback to promote symmetrical weight bearing in standing but this training did not improve the locomotor performance (Winstein et al. 1989). Thus, these previous studies suggested that the ability to maintain posture in standing could not be transferred to balance control during walking. Our finding that postural adjustments were different between standing and walking may help explain why the ability to maintain equilibrium does not transfer between static and dynamic tasks. We agree that the stroke patients need to be able to maintain equilibrium during both static and dynamic conditions, as balance could be disturbed during static and dynamic activities. To achieve this goal, we suggest that the stroke patients should be allowed to practice equilibrium control in both standing and walking, in which balance training in standing would aim to improve the static equilibrium control, whereas balance training during walking would encourage the practice of dynamic equilibrium control. This suggestion is, in fact, in agreement with the task-specific training, in which the patients learn what

they practice. Furthermore, the finding that balance requirements (and adjustments) were smaller during walking than standing even after stroke may encourage the therapist to begin the balance training program in walking earlier than waiting for normal standing balance to be accomplished.

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Figure 4.1. *Top*: An example of the experimental setup showing that a subject was exposed to surface rotation in the toes-down direction during the double support phase of walking when the right or paretic foot was stepping onto the force plate in front while the trailing limb was pushing off the rear force plate. *Bottom*: Example of recording from a representative control subject during walking with (black traces) and without (grey traces) a toes-down perturbation. The perturbation occurred when the loading force in each foot was relatively symmetrical. Profiles from four unperturbed cycles were averaged to establish the reference (grey traces). Postural responses are characterized by changes from the reference during 0-350 ms after perturbation onset. The force data were analyzed in two epochs, passive (F1, 50-100 ms after perturbation onset) and active response periods (F2, 100-350 ms after perturbation onset). Integrals of EMG data were computed for two periods, 0-70 ms after perturbation onset (E1, short latency reflex activity period), and 70-320 ms after perturbation onset (E2, active response period).

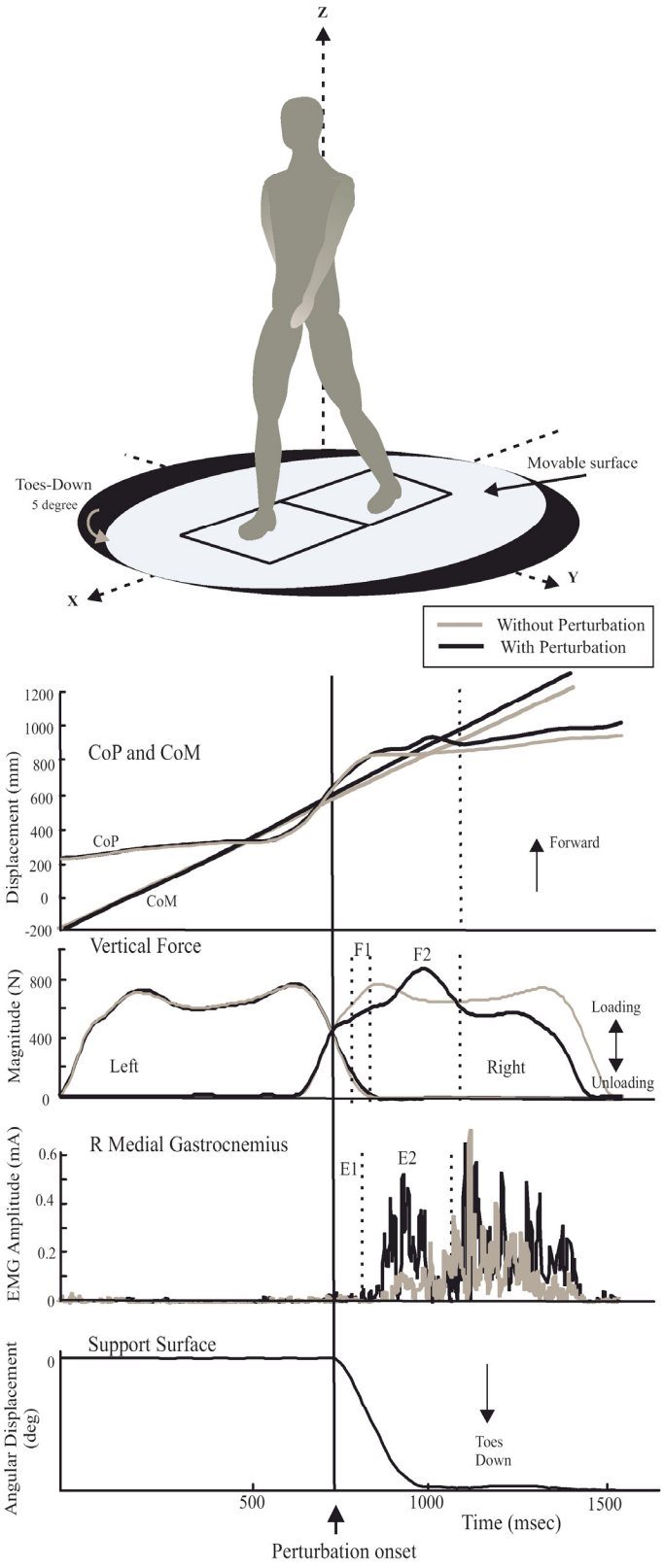

Z
Movable surface
Toes-Down
5 degree
X
Y
Without Perturbation
With Perturbation
CoP and CoM
Displacement (mm)
1200
1000
800
600
400
200
0
-200
CoP
CoM
Forward
Vertical Force
Magnitude (N)
800
400
0
F1
F2
Left
Right
Loading
Unloading
R Medial Gastrocnemius
EMG Amplitude (mA)
0.6
0.4
0.2
0
E1
E2
Support Surface
Angular Displacement (deg)
0
Toes
Down
500
1000
1500
Time (msec)
Perturbation onset

Figure 4.2. Stick figures and traces representing the kinematic strategies in response to pitch and roll perturbations in the control subjects. Figures and traces are shown only for the sagittal plane, and normalized to 100% of the gait cycle from one initial foot contact to the next. The control traces are the average of 10 unperturbed walking trials (thin black) with the 95% confident intervals (grey shade), while the bold traces are the average of 4 perturbed walking trials from one representative control subject. The oval circle highlights the response and the arrow indicates the perturbation onset.

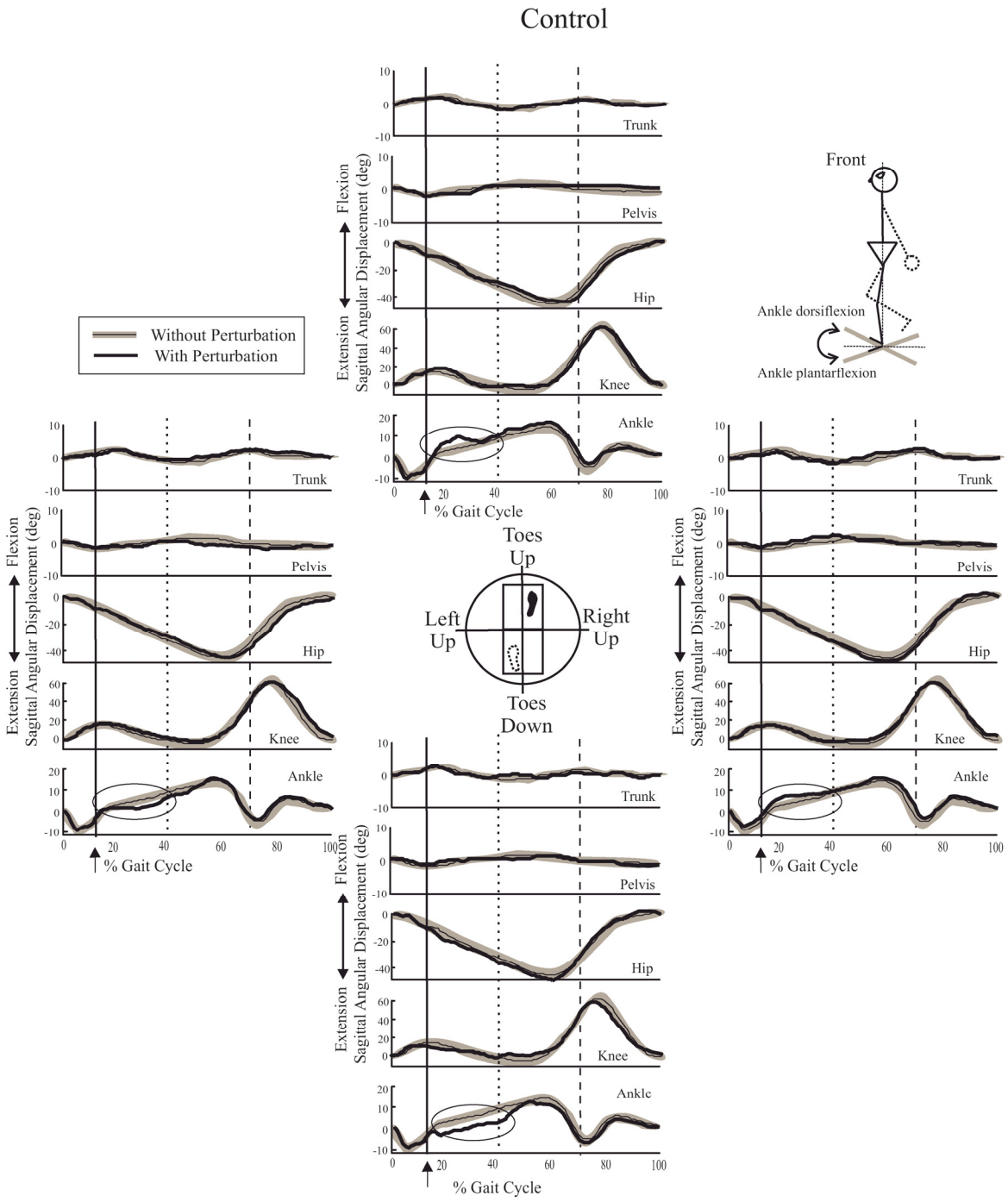

Control
Without Perturbation
With Perturbation
Trunk
Pelvis
Hip
Knee
Ankle
Flexion
Extension
Sagittal Angular Displacement (deg)
% Gait Cycle
Front
Ankle dorsiflexion
Ankle plantarflexion
Toes Up
Left Up
Right Up
Toes Down

Figure 4.3. Stick figures and traces representing the kinematic strategies in response to pitch and roll perturbations from one stroke subject. Figures and traces are shown only for the sagittal plane, and normalized to 100% of the gait cycle from one initial foot contact to the next. The control traces are the average of 10 unperturbed walking trials (thin black) with the 95% confident intervals (grey shade), while the bold traces are the average of 4 perturbed walking trials. The oval circle highlights the response and the arrow indicates the perturbation onset.

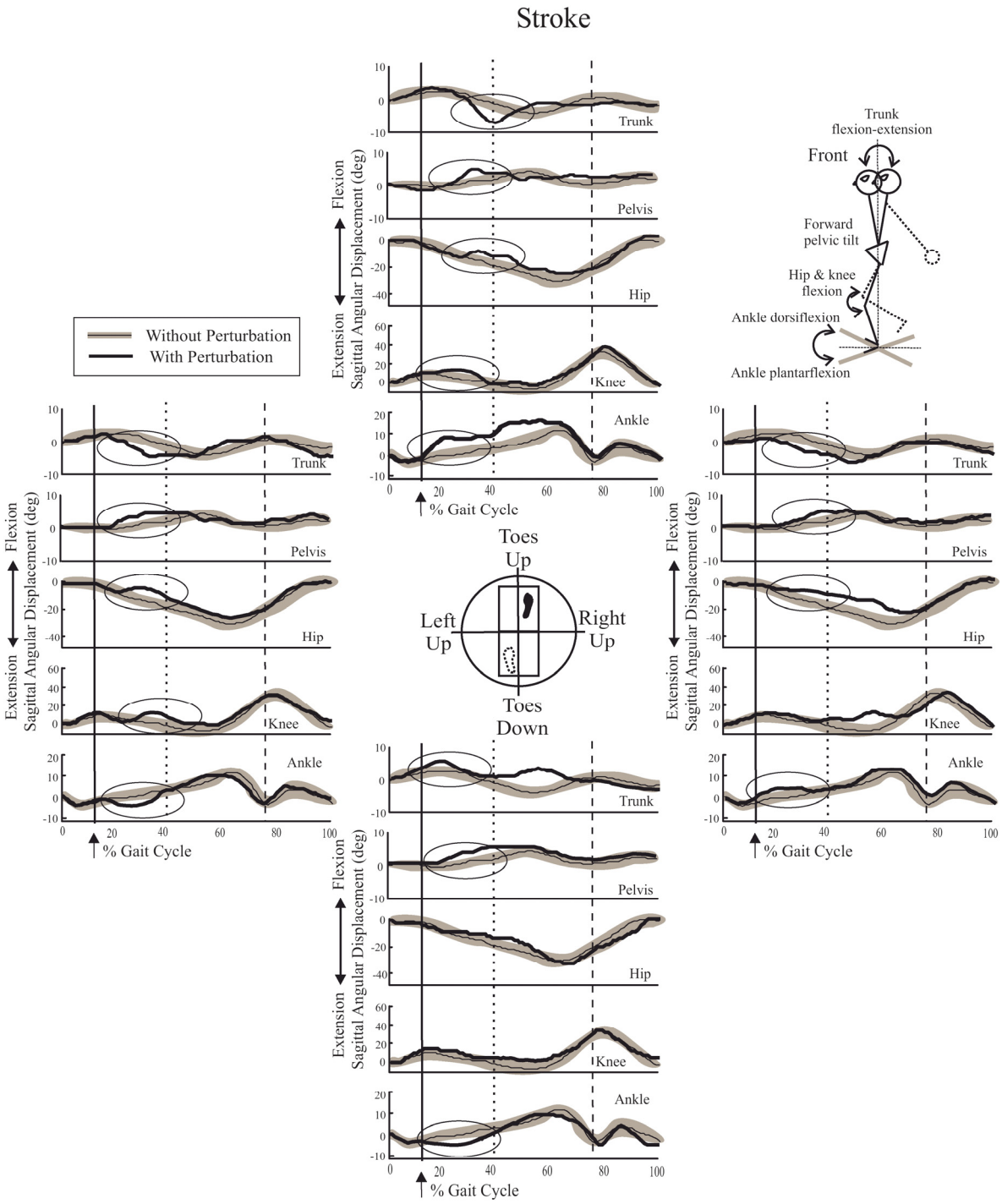

Stroke
Without Perturbation
With Perturbation
Trunk
Pelvis
Hip
Knee
Ankle
Flexion
Extension
Sagittal Angular Displacement (deg)
% Gait Cycle
Toes Up
Left Up
Right Up
Toes Down
Trunk flexion-extension
Front
Forward pelvic tilt
Hip & knee flexion
Ankle dorsiflexion
Ankle plantarflexion

Figure 4.4. Average (±1 SE) maximum kinematic responses (changes from the reference obtained during unperturbed walking) in 8 control and 11 stroke subjects. Kinematic responses include sagittal and frontal segmental excursion of the trunk and pelvis in the first 350 ms (A) and RMS of the trunk and pelvis excursions from 350 to 1000 ms after perturbation (B); as well as sagittal angular displacements of right or paretic (R/P) hip, knee and ankle (C) and of the bilateral shoulders (D). The asterisk indicates a significant difference between groups.

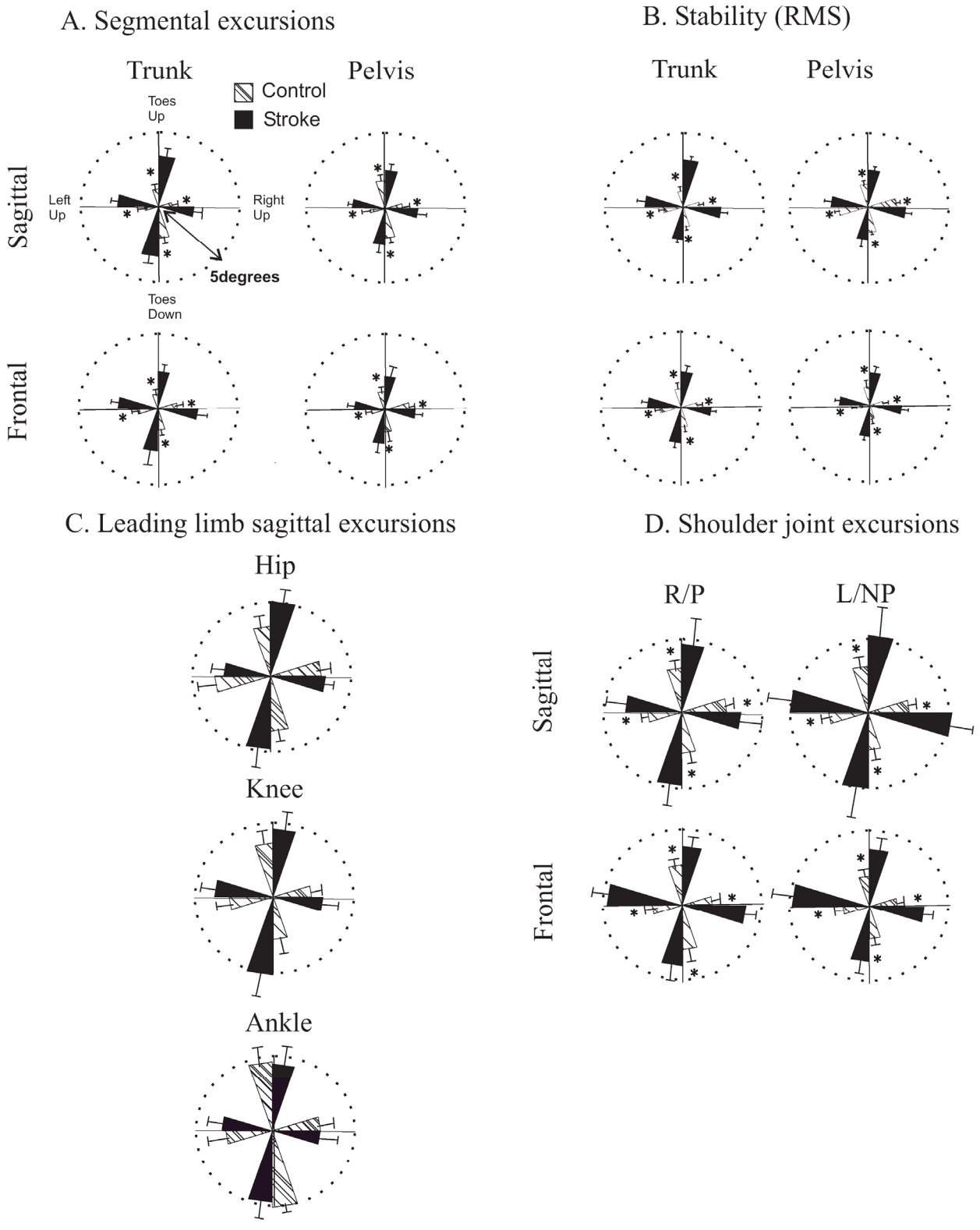
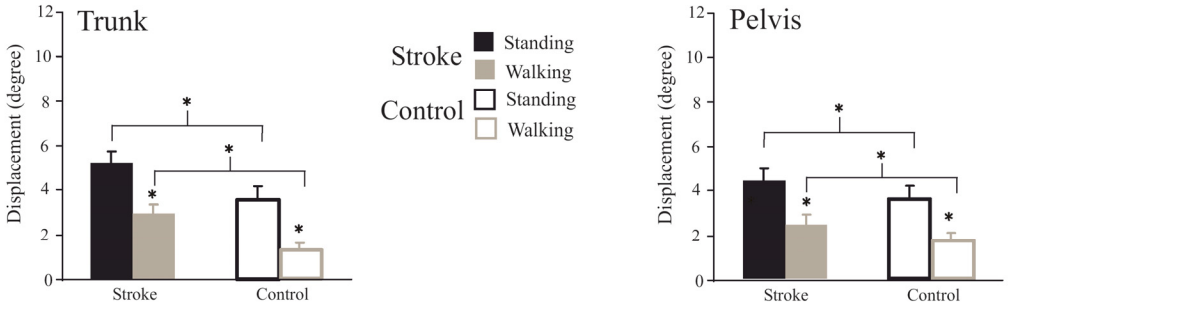

A. Segmental excursions
Trunk
Pelvis
Control
Stroke
Toes Up
Left Up
Right Up
5degrees
Toes Down
Sagittal
Frontal
B. Stability (RMS)
Trunk
Pelvis
C. Leading limb sagittal excursions
Hip
Knee
Ankle
D. Shoulder joint excursions
R/P
L/NP
Sagittal
Frontal

Figure 4.5. Average maximum sagittal angular excursions of the trunk and pelvis (A) and the right or paretic hip, knee and ankle (B) contrasted between the task of standing and walking in 8 control and 11 stroke subjects. The asterisk indicates significant difference between tasks (standing vs. walking) and the asterisk-over-solid-line indicates significant difference between groups of subjects (healthy vs. stroke).

A. Segmental sagittal excursions



B. Right/Paretic limb sagittal excursions

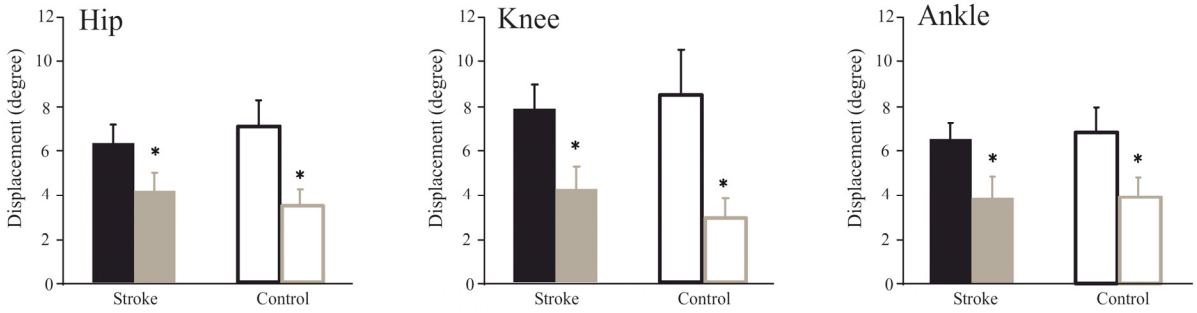


Figure 4.6. *A*: Traces of CoP and CoM displacements in the A/P and M/L direction during walking with and without toes-up perturbation in a healthy control and a stroke subject. *B*: Average (±1 SE) maximum CoM and CoP displacements (changes from the reference obtained during unperturbed walking during the first 350 ms) in the A/P and M/L directions of 8 control and 11 stroke subjects. *C*: Average (±1 SE) RMS of CoM and CoP displacements (changes from the reference obtained during unperturbed walking from 350 to 1000 ms) in the A/P and M/L directions of 8 healthy controls and 11 stroke subjects. The asterisk indicates significant difference between groups.

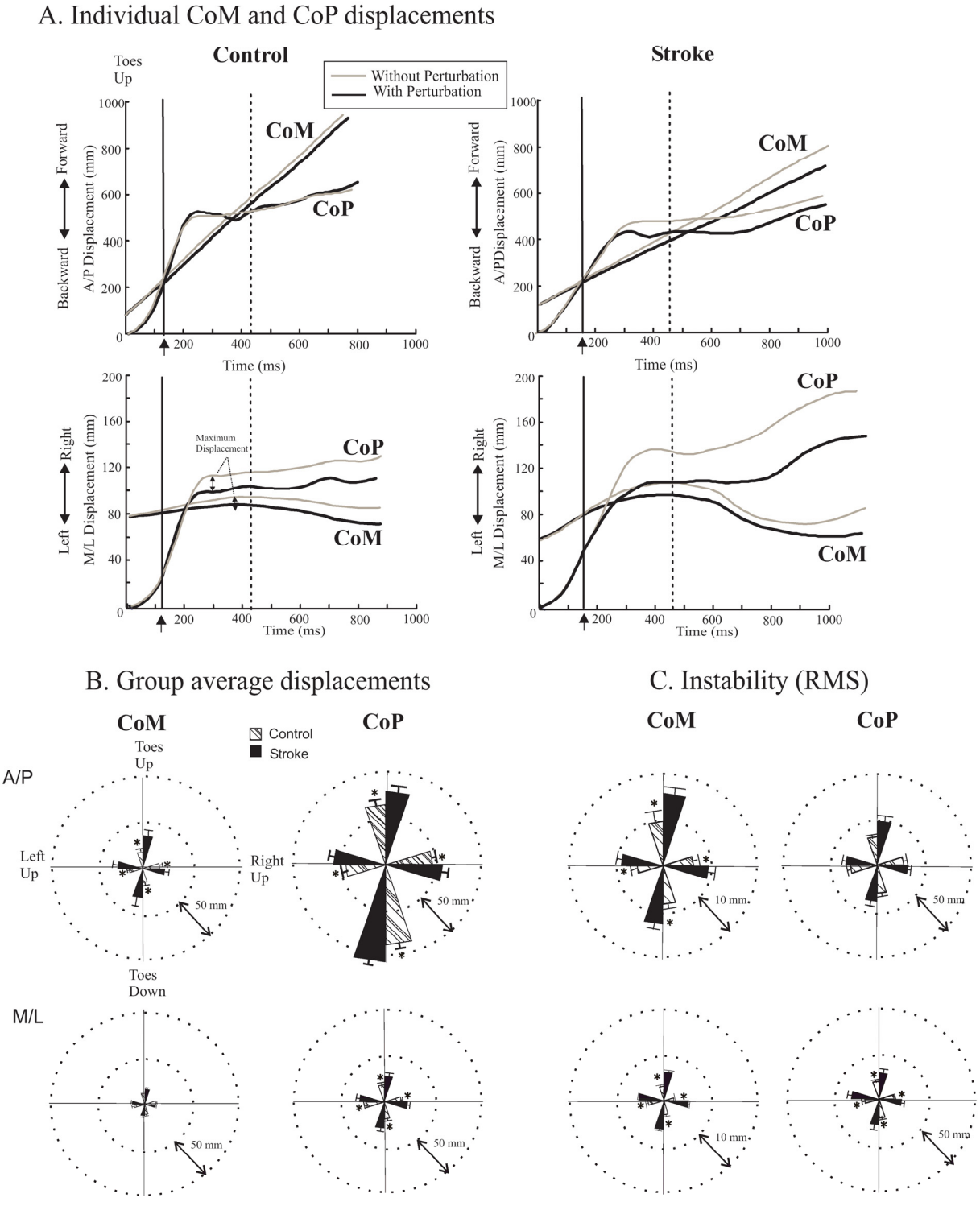

A. Individual CoM and CoP displacements
Control
Stroke
Without Perturbation
With Perturbation
Toes Up
Forward
Backward
A/P Displacement (mm)
A/PDisplacement (mm)
CoM
CoP
Time (ms)
Right
Left
M/L Displacement (mm)
Maximum Displacement
B. Group average displacements
C. Instability (RMS)
Control
Stroke
A/P
M/L
Toes Up
Toes Down
Left Up
Right Up
50 mm
10 mm

Figure 4.7. *A*: Traces of instantaneous CoM velocity displacement in the sagittal plane during walking with and without pitch and roll plane perturbations in a healthy control and a stroke subject. The thin black trace represents the average of 10 unperturbed walking trials with the 95% confident intervals (grey shade) while the other traces correspond to different axes of perturbation. The arrow indicates the perturbation onset and the thin vertical line indicates the 350 ms after perturbation onset. *B*: Average ($\pm$1 SE) change in CoM velocity (expressed as a percentage of the average CoM velocity of the unperturbed cycle) in the first 350 ms after pitch or roll plane perturbations during walking in 8 control and 11 stroke subjects. The asterisk indicates a significant difference from unperturbed walking (0%). *C*: Percent decrease in CoM velocity due to toes-up perturbation plotted against the RMS of CoM displacements in the A/P and M/L directions, fitted with a linear regression and the R^2 (% variance explained by the linear regression).

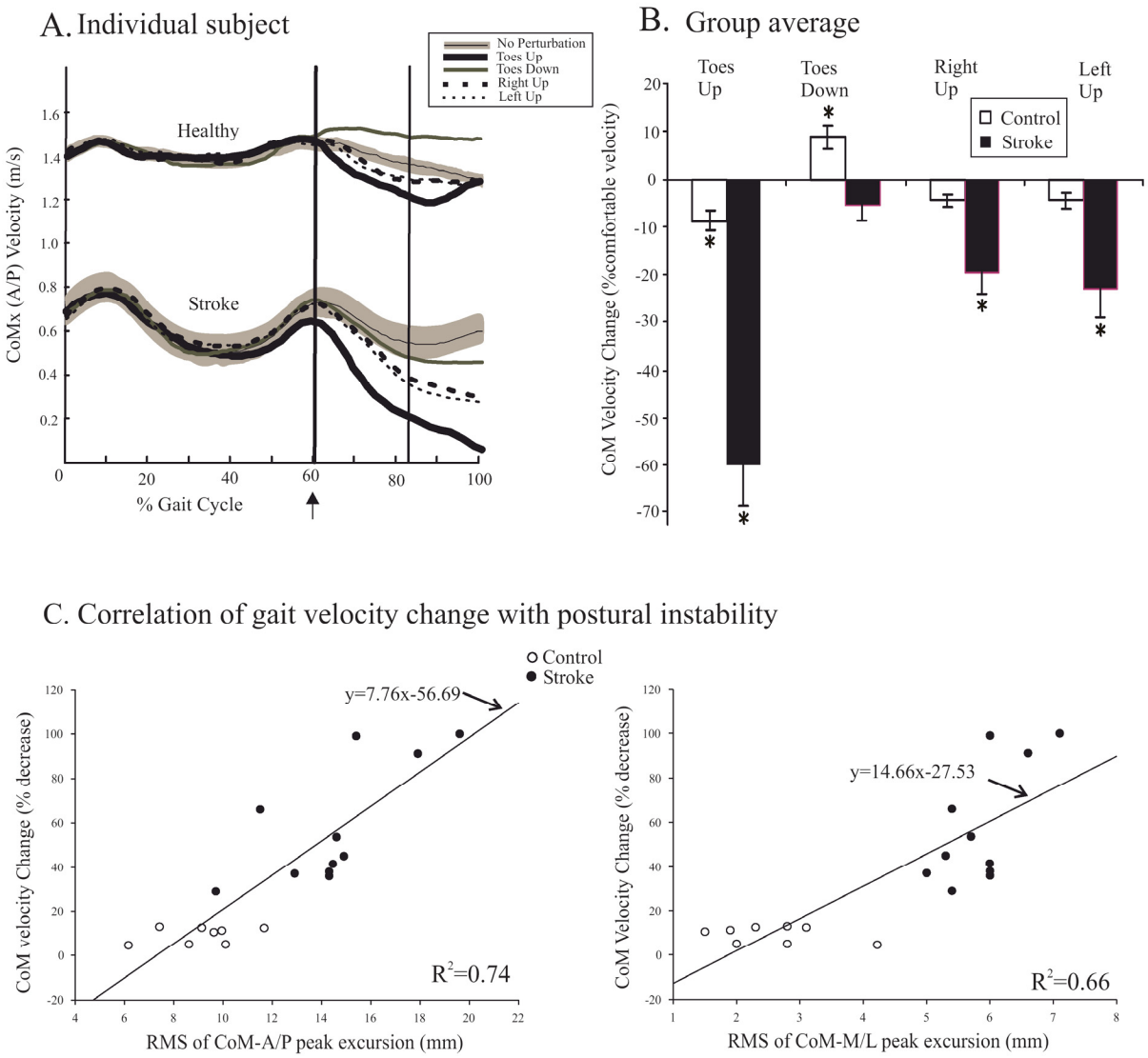
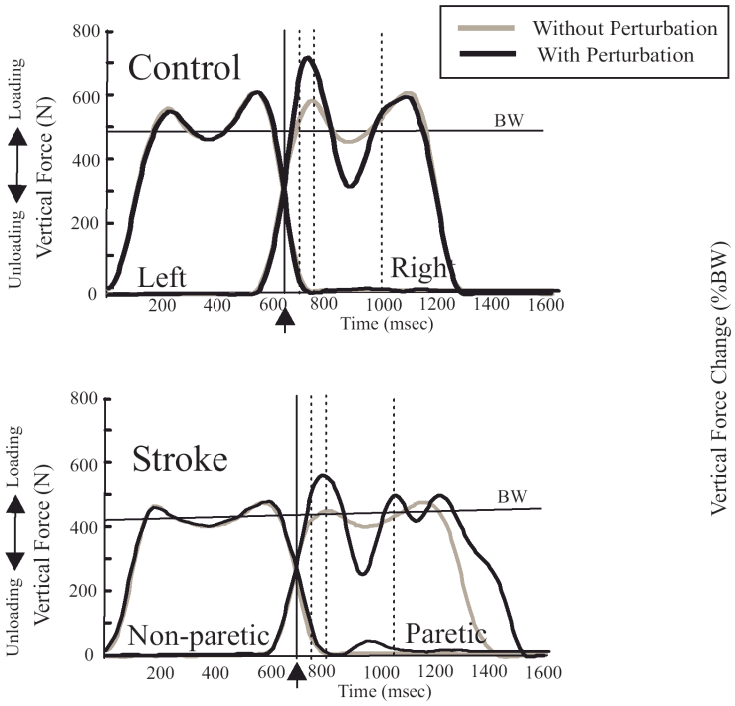

A. Individual subject
No Perturbation
Toes Up
Toes Down
Right Up
Left Up
Healthy
Stroke
CoMx (A/P) Velocity (m/s)
% Gait Cycle
B. Group average
Toes Up
Toes Down
Right Up
Left Up
Control
Stroke
CoM Velocity Change (%comfortable velocity)
C. Correlation of gait velocity change with postural instability
Control
Stroke
y=7.76x-56.69
R²=0.74
CoM velocity Change (% decrease)
RMS of CoM-A/P peak excursion (mm)
y=14.66x-27.53
R²=0.66
CoM Velocity Change (% decrease)
RMS of CoM-M/L peak excursion (mm)

Figure 4.8. Left-sided panel shows loading forces on the right/paretic (black) and left/non-paretic (grey) limb during walking with and without perturbation in the toes-up direction from a healthy control and a stroke subject. The arrow and vertical solid line indicate the perturbation onset while the vertical dash lines represent the integral periods. The horizontal solid line indicates the subject's body weight. Right-sided panel shows average (±1 SE) integrals of loading force responses from the right/paretic limb during the active response period (100-350 ms) in response to pitch and roll plane perturbations in 8 healthy control and 11 stroke subjects. Loading force changes were calculated as the percentage of body weight where the positive value represents increased loading and the negative value indicates unloading. The asterisk indicates significant difference between groups.

Loading Forces

Individual Subject



Group average

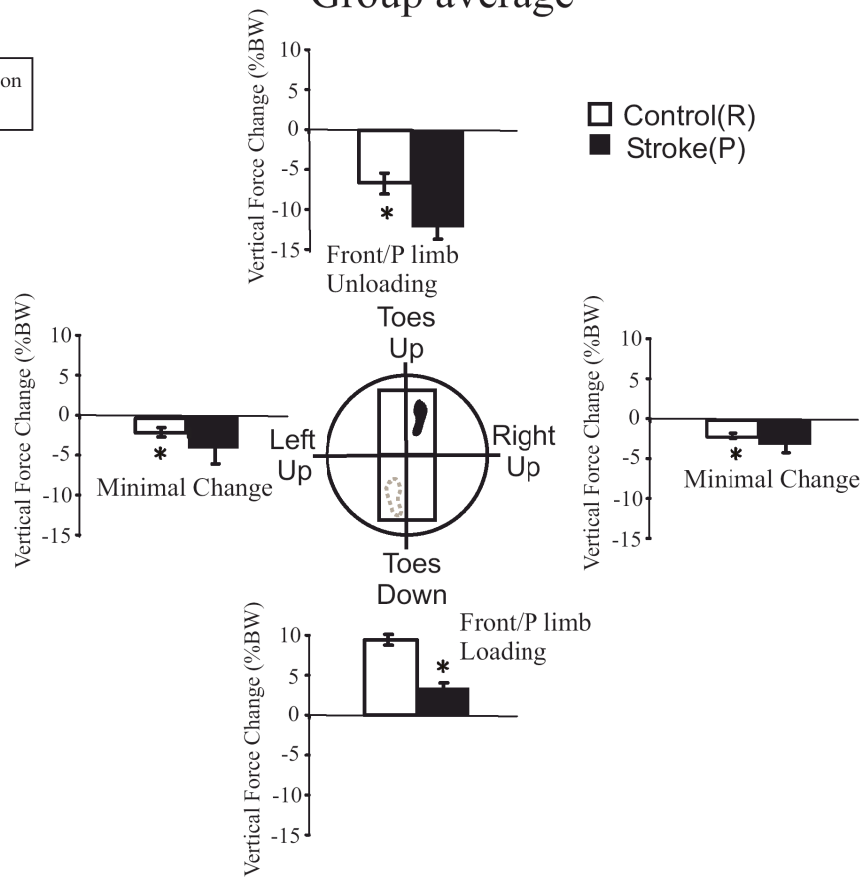


Figure 4.9. *A*: Full-wave rectified and filtered EMG traces from the right/paretic tibialis anterior (TA), medial gastrocnemius (MG), rectus femoris (RFM) and tensor fascia latae (TFL) muscles of a healthy control and a stroke subject during walking with and without roll (right-up) perturbation. “IC” indicates the initial contact of the right or paretic limb. The arrow and vertical solid line indicate the perturbation onset, whereas the vertical dash lines represent the integral periods. *B*: Average (±1 SE) EMG integrals in the active response period (70-320 ms) of the right/paretic limb muscles in 8 healthy control and 11 stroke subjects against perturbed directions. The asterisk indicates significant difference between groups. *C*: Average EMG responses from the right or paretic leg of 8 healthy controls and 11 stroke subjects during the active period (70-320 ms) contrasted between the tasks of standing and walking. The asterisk indicates significant difference between tasks.

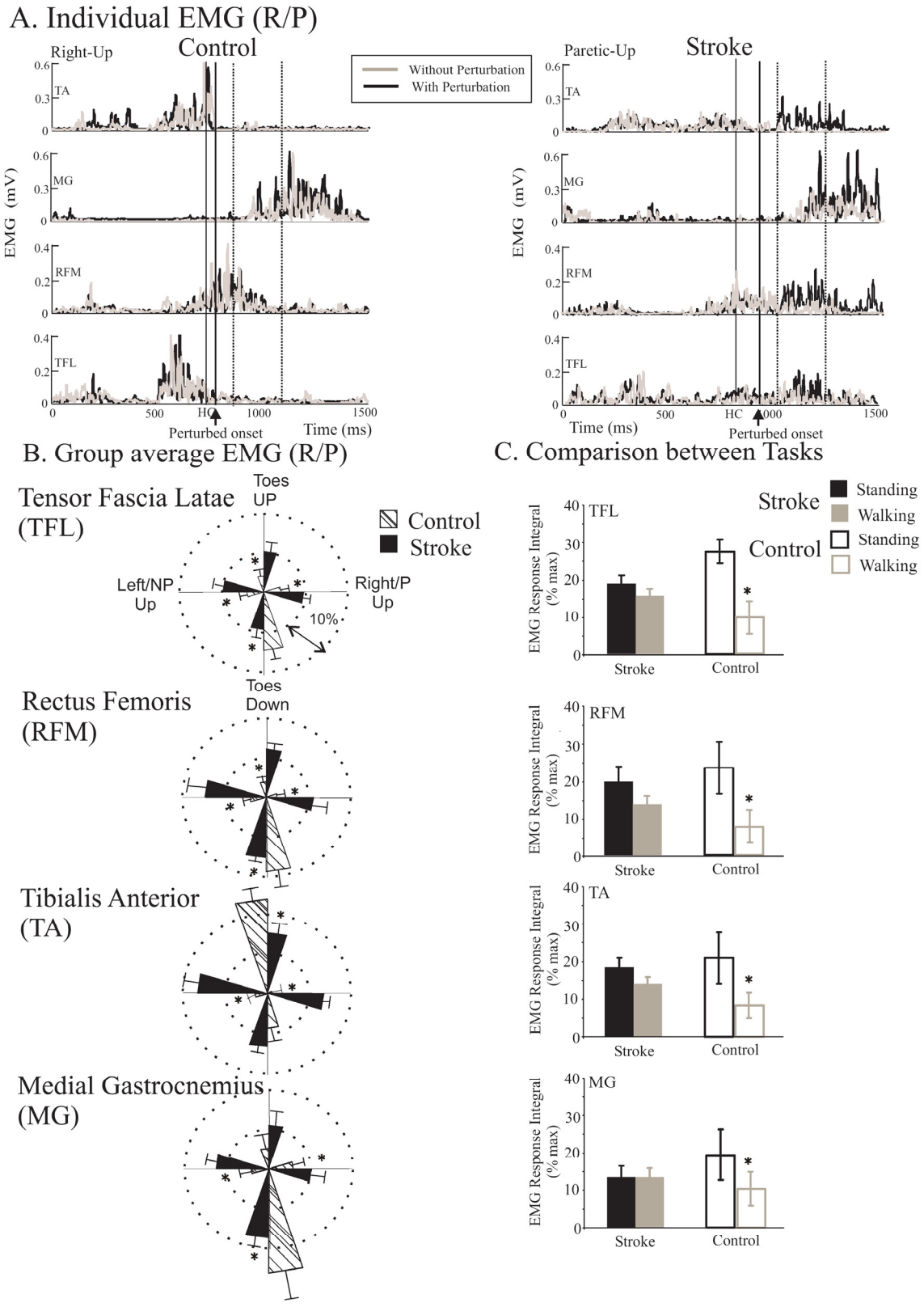

A. Individual EMG (R/P)
Control
Stroke
Right-Up
Paretic-Up
Without Perturbation
With Perturbation
EMG (mV)
TA
MG
RFM
TFL
Perturbed onset
Time (ms)
B. Group average EMG (R/P)
C. Comparison between Tasks
Tensor Fascia Latae (TFL)
Rectus Femoris (RFM)
Tibialis Anterior (TA)
Medial Gastrocnemius (MG)
Toes UP
Toes Down
Left/NP Up
Right/P Up
10%
Control
Stroke
Standing
Walking
EMG Response Integral (% max)

Table 4.1: Subject Characteristics

Stroke Subjects	Age	Gender	Side of Paralysis	Chedoke-McMaster Posture/leg/foot clinical score*	Average overground walking speed (ms^{-1})
1	54	F	L	6/5/5	0.5
2	60	M	R	6/6/6	0.7
3	63	M	R	6/6/6	0.9
4	64	F	R	6/5/5	0.6
5	64	M	R	6/3/5	0.7
6	65	F	R	6/6/6	1.0
7	66	M	R	5/3/5	0.6
8	66	M	L	5/5/5	0.8
9	70	M	R	5/4/5	0.6
10	79	F	R	5/5/5	0.4
11	80	M	R	5/4/5	0.5

R=Right; L=Left
*** Chedoke-McMaster Stroke Impairment : maximum score =7**

CHAPTER 5

LIGHT TOUCH IMPROVES CONTROL OF POSTURAL EQUILIBRIUM DURING QUIET STANCE AND LOCOMOTION POST STROKE

Rumpa Boonsinsukh, Sophie J. De Serres and Joyce Fung

Submitted to: Brain

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Results from the study in Chapter 4 suggested that stroke disrupted the process of sensorimotor integration which leads to the impairment in the modulation of triggered postural responses (TPRs) to the task requirements. Light touch from the fingertip has been shown to improve balance maintenance during quiet stance in healthy subjects by providing precise somatosensory and proprioceptive information regarding body orientation in space (Jeka and Lackner 1994; Jeka and Lackner 1995). Thus, fingertip cue may be able to assist in the control of posture in stroke subjects who were presented with sensorimotor integration problems. To date, none of the studies investigates the effect of light touch on TPRs in stroke subjects. Therefore, the study in this chapter was carried out to examine whether stroke subjects could benefit from the fingertip cue on equilibrium control in the same way as the healthy subjects and whether the effectiveness of light touch on postural control is similar for the static and dynamic tasks. The

perturbation used in this study was a surface rotation in the toes-up direction as it was the most disturbing perturbed direction which caused the maximal reduction in gait speed in the stroke subjects (as shown in Chapter 4).

5.1 Abstract

A tactile cue in the form of light touch applied through the fingertip has been shown to reduce body sway during standing in healthy subjects, but its effects are not known in stroke subjects. This study examined the effects of tactile cue on postural control during standing and walking in stroke subjects. Eleven stroke and 8 healthy age-matched subjects were exposed to toes-up surface tilt (peak velocity 32°/s) during the double limb support phase of walking and similar posture in quiet stance, with and without light touch (<4N) exerted through the index finger on a firm surface. Postural responses were characterized by body kinematics, center of pressure (CoP) and muscle activities recorded from 4 bilateral lower limb muscles. Results show that healthy subjects utilized tactile cue differently depending on the task demands, such that their balance improved more during quiet stance than walking. The gains in balance control were much more prominent in stroke subjects who benefited from the tactile cue during both standing and walking. With increased trunk and CoP stability, stroke subjects also reduced the use of compensatory arm movements to restore balance. The speed of forward progression that was markedly reduced by a sudden surface perturbation in stroke subjects was significantly improved with tactile cue. The different responses in stroke and healthy subjects may be due to the different degree of redundancy in available sensory information. The use of tactile cue for balance rehabilitation is discussed.

5.2 Introduction

Tactile information provided through lightly touching a rigid surface has been shown to decrease postural sway during quiet stance (Jeka and Lackner, 1994; 1995) and reduce the anticipatory postural adjustments from trunk and leg muscles during a unilateral shoulder flexion task (Slijper and Latash, 2000). Even passive light touch delivered to the shoulder or leg by an object fixed to the environment can stabilize the body during standing (Rogers et al., 2001). Light touch provided information on the position and velocity of the body in relation to the external objects or surface (Jeka et al., 1998; Jeka et al., 1997). Several sensory inputs, including rapidly and slowly adapting cutaneous receptors on the fingertip and proprioceptive receptors in the finger and arm, can provide information about body sway for balance control (Jeka and Lackner, 1994; 1995). It has been shown that the frequency of body sway was found to follow that of an oscillating surface that was in contact with either the feet or the fingertip (Jeka et al., 1998; Jeka et al., 1997). Thus, the central nervous system (CNS) may utilize tactile information to control posture in the same way that visual cues are employed as anchors of the environment (Jeka et al., 2000).

Injury to the CNS such as cerebrovascular accidents (CVA) often leads to balance impairment and mobility dysfunctions (Badke and Duncan, 1983). We have previously shown that the postural responses triggered by sudden surface tilts were disrupted by CVA such that they were delayed and not modulated according to the demands of the task (Boonsinsukh et al., 2002). Additional sensory information such as auditory (Wannstedt and Herman, 1978) or visual cues (Winstein et al., 1989) were found to improve balance control in standing following stroke. However, the effects of tactile cue on the control of posture in stroke subjects are unclear. A recent study demonstrated that light touch provided through the paretic hand showed no favorable effect

on the anticipatory postural adjustments during unilateral shoulder flexion in quiet stance. However, the study did not examine the effect of tactile cue from the non-paretic hand which can provide more effective sensory cues than the paretic hand that is likely to be impaired in sensation. We have previously reported that postural responses triggered by surface tilts are task-specific, being larger in quiet stance than during walking (Fung et al., 2003). The question arises as to whether the effect of tactile cue would also be modulated by the demand of the task or a CVA. Therefore, the present study was conducted to contrast the effect of tactile cue on postural responses triggered by unexpected surface tilts during quiet stance and locomotion between stroke and healthy subjects. We hypothesize that light touch is more effective in assisting the maintenance of equilibrium in stroke than in the control subjects and that the effects are more pronounced in standing as compared to walking.

5.3 Methods

5.3.1 Subjects

Eleven subjects who suffered a cerebrovascular accident (CVA) that ranged in onset from 2 weeks to one year participated in this study (Table 5.1). They were recruited from both the in-patient and out-patient neurology program at the Jewish Rehabilitation Hospital (Laval, Quebec, Canada). Their functional mobility as measured by the Timed Up and Go test (Podsiadlo and Richardson, 1991) ranged from mildly to moderate impaired (10-29 s vs. the normal range of 5-7 s). All stroke subjects showed evidence of unilateral lower limb motor deficits as indicated by the Chedoke-McMaster impairment scale (Gowland et al., 1995, see also Table 1), but they were able to stand longer than 5 s without external support (postural control scores of 3/7 or higher on the impairment scale). All stroke subjects were ambulatory and able to walk for at least 5 meter without rest. Stroke subjects were excluded from the study if they had 1) cognitive or language impairment, 2) severe hemineglect, 3) cerebral aneurysm, 4) bilateral cerebral impairment, 5) brainstem and cerebellar lesions or 6) impaired touch and pressure sensation on the non-paretic hand.

Eight healthy subjects who matched the stroke subjects by age and gender participated in the study. They were recruited from the surrounding community. The healthy subjects were all right-hand dominant, as determined by the Edinburgh Handedness Inventory (Oldfield, 1971). Informed consent was obtained from all subjects and the study was approved by the institutional ethics board.

5.3.2 Experimental Procedure

A servo-controlled six degree-freedom-of-movement motion-base (Fung and Johnstone, 1998) was used to deliver surface perturbations. Embedded within the top of the motion-base at the ground level were two tri-axial AMTI (OR6-7) force plates measuring forces exerted by the subject. Unexpected surface perturbation was delivered at a peak ramp velocity of 32 degree/s and at the maximum amplitude of 5 degree for 150 ms in the direction of a *toes-up tilt* (Fig. 5.1A), the direction that is most disturbing for balance control during locomotion as it impedes the forward progression of the body's center of mass (CoM) (Boonsinsukh et al., 2002). The magnitude of surface perturbation was within the range that a person was able to maintain balance without stepping (Allum et al., 1993).

A 5-meter wide wood plank was mounted firmly beside the walkway to provide somatosensory information from the environment through the fingertip (Fig. 5.1A). It was mounted on the non-paretic for stroke subjects and on the right side for healthy controls. The top of the rail was adjusted at the level of each individual's hip level. A thin strip of load sensors (0.15 m x 2.45 m dimension) was secured on the surface of the plank to measure the amount of force exerted by the fingertip. A force that exceeded 4N would trigger a beep. Subjects were habituated to stand or walk while touching or sliding, respectively, their fingertip along the sensor strip without triggering the sound. This amount of force was chosen because it provided sensory information rather than the mechanical support (Slijper et al., 2002). A trial was rejected when a beep was triggered.

In each trial, subjects were asked to maintain balance during quiet stance or locomotion with or without touching the plank. During quiet stance, subjects were asked to maintain a step stance posture with the right (healthy control) or paretic foot (stroke) in front (Fig. 5.1A) to mimic the same posture during the double limb support phase of walking. The foot position was

determined by the individual subject's step length and step width, with markings on the force plates to ensure the consistency of foot placement throughout the experiment. .

During locomotion, surface tilt was triggered during the double limb support phase of walking when the loading force on each plate was relatively equal. Individuals were asked to walk along the 5-meter walkway across the movable surface at their own comfortable speed. Subjects were instructed to continue walking even when the perturbation occurred and to respond to the perturbation as if they were walking on an uneven surface. None of the subjects used walking aids or ankle-foot orthosis during testing procedures and a suspended body harness (without providing any weight support) was worn by all subjects for safety precautions. Each individual participated in four blocks of standing and four blocks of walking trials. One block of data collection consisted of four perturbed (two with touch and two without touch) trials and four unperturbed (two with touch and two without touch) trials, randomly assigned. Each standing and walking trial lasted for 4 and 6 seconds, respectively, with at least a 1-minute rest period between trials.

5.3.3 Data collection and analysis

Three-dimensional body segment positions were acquired by a six-camera VICON motion analysis system (Vicon 512; Oxford Metrics Ltd) at the sampling frequency of 120 Hz. Thirty-eight retro-reflective markers were placed on anatomical landmarks to capture body motions (Fig. 5.1). Four additional markers were placed on the four corners of the platform to capture surface movements. The data were low-pass filtered at 10 Hz with a 2^{nd} order dual-pass Butterworth filter, based on a previous residual analysis of the kinematics (Winter, 1990). Anthropometric measurements were obtained from each subject to calculate the position of the body CoM. Segmental and joint

angles were calculated based on segments formed by at least three non-coplanar markers.

The triaxial ground reaction forces (GRFs), including the anteroposterior (A/P), mediolateral (M/L) and vertical components (Fx, Fy and Fz respectively), were acquired using two force plates (AMTI OR6-7) mounted within the motion base at the sampling rate of 1,080Hz. Any bias in force signals at the onset of perturbation due to the inertial characteristics of the force plates was subtracted based on inverse dynamics calculation from motions of the platform (Preuss and Fung, 2003). The adjusted force was then filtered at 10Hz and the center of pressure (CoP) from each foot in the A/P (CoPx) and M/L (CoPy) directions were computed. The resultant center of pressure (rCoP) from the two feet was then calculated (Henry et al., 1998a).

The electromyographic (EMG) activities from four bilateral lower limb muscles, right and left tibialis anterior (TA), medial gastrocnemius (MG), rectus femoris (RFM), and tensor fascia latae (TFL) were recorded with an 8-channel TELEMYO (Noraxon USA, Inc.) system at 1,080 Hz. The EMG signals were band-pass filtered (16-500 Hz), full-wave rectified, and then further low-pass filtered at 100 Hz (Gottlieb and Myklebust, 1993) for off-line analysis.

BodyBuilder (Oxford Metrics Ltd) and Matlab (MathWorks Inc.) software programs were used to perform subsequent data analyses. The perturbation onset was calculated from the initial change in the position of reflective markers placed on four corners of the force plates. In quiet stance, the period of 200 ms before the perturbation onset was used as the baseline. In walking, data were initially normalized to the gait cycle, starting from one initial contact of the foot to the next and the baseline was the average of four unperturbed walking trials. This baseline was subtracted from the perturbed data to determine postural responses due to surface perturbation. Peak-to-peak

excursions of the body CoM, rCoP, ankle, knee, hip and shoulder joints and trunk and pelvic segments in the sagittal and frontal planes between 0-1,000 ms during quiet stance and between 0-350 ms during walking were calculated. A shorter period was chosen to analyse the walking trials because any postural responses that occurred during walking were elicited within 350 ms after the perturbation onset before rapidly returning to the baseline (Boonsinsukh et al., 2002). Stability of the body was measured as the root-mean-square (RMS) of the body CoM, rCoP, trunk and pelvic excursion in the sagittal and frontal planes between 350 and 1,000 ms after perturbation onset. The average gait speed was calculated by dividing the average distance of CoM progression by the cycle duration for 2 consecutive gait cycles in the four unperturbed walking trials. The change of instantaneous gait velocity due to the perturbation was calculated by subtracting the CoM velocity at 350 ms after perturbation onset from the CoM velocity at 350 ms before perturbation onset.

Integrals of EMG data were calculated for two intervals (Fig. 5.1B): 1) *short latency reflex activity period* (E1), 0-70 ms after perturbation onset and 2) *active response period* (E2), 70-320 ms after perturbation onset. EMG onset latency was selected based on the criteria that the first burst of muscle activity must be at least 2 sd greater than the average EMG in the background period and the duration of activation must be at least 25 ms long (Henry et al., 1998b). The EMG activity was normalized to the peak activity of the same muscle for control subjects and to the peak activity of the non-paretic side for stroke subjects. A muscle was considered as to be recruited as a postural response only when the firing probability reached 75% (i.e., activated in 3 out of 4 trials of perturbation).

5.3.4 Statistical analysis

Statistica (StatSoft Inc) software was used to perform statistical analyses. Two-way analyses of variance (ANOVAs) were used to test for any main or interaction effects due to subject group (stroke vs. control) and conditions of somatosensory information (touch vs. no-touch) during each standing and walking task. When significant differences were found after multiple comparisons were adjusted with the Bonferroni test ($p<0.05$), pairwise comparisons were performed using the Tukey test.

5.4 Results

5.4.1 Effects of light touch on TPRs during quiet stance

5.4.1.1 Kinematic responses

Kinematic responses to toes-up perturbation occurred mainly in the sagittal plane and representative examples of the responses during quiet stance, in the presence or absence of light touch, are shown in Figure 5.2A. The main kinematic strategy in either group of subjects without light touch consists of forward flexion of the trunk and pelvis, bilateral flexion of the hip and knee joints and bilateral ankle dorsiflexion. In stroke subjects, the movement trajectory of each body segment was irregular with some overshooting. Larger displacements of the trunk and pelvis were seen in the stroke subjects, while the displacements of the lower limb joints were not significantly different from the control subjects. Stroke subjects also used larger bilateral arm movements to assist in balance corrections. With light touch, the kinematic strategy in both groups of subjects was not altered but the movement trajectory of each body segment became smoother in the stroke subjects. Light touch benefited the stroke subjects more than the control subjects, as shown by the amount of peak-to-peak excursions of the trunk, pelvis and the shoulder joint of the freely hanging upper limb (not touching the plank) (Fig. 5.2B and 5.2C). While the tactile cue had minimal effects in the control subjects, the displacements of the trunk and pelvis, as well as the compensatory arm movements, were significantly reduced in stroke subjects.

Figure 5.3A shows an example of the sagittal trunk and pelvic coordination when balance adjustment was made during quiet stance. In the absence of light touch, the control subject made similar forward displacements of the trunk and pelvis in response to sudden surface tilt, and

restored the trunk and pelvis close to the initial position at the end of the trial. In contrast, the trunk and pelvic coordination was disrupted in the stroke subject with marked overshooting as the movements of the trunk and pelvis reversed. The stability of the trunk and pelvis, as measured by their RMS values, are shown in Figure 5.3B. The disrupted trunk-pelvic coordination seen in stroke subjects could be attributed to in the problem of stabilizing the trunk, rather than the pelvis, in both sagittal and frontal planes. Light touch assisted both groups of subjects in the control of trunk and pelvis such that their final positions were restored closer to the initial positions (Fig. 5.3A), but the effect of tactile cue was more prominent in the stroke subject, showing a smoother pattern with less overshooting. Similarly, trunk stability in both groups of subjects was increased with light touch (Fig. 5.3B). Light touch significantly reduced the RMS of sagittal plane trunk excursions in both groups of subjects ($p<0.001$) as well as the frontal ($p<0.05$) plane trunk excursion in stroke subjects.

5.4.1.2 The control of body CoM and CoP

Figure 5.4A contrasts the horizontal trajectories of the CoM and rCoP in response to a toes-up surface tilt during standing with and without light touch. Toes-up surface perturbation displaced the body's CoM backward. The CoP initially moved opposite to the direction of CoM movement before it followed and encompassed the movement of the CoM. In the absence of light touch, both CoM and CoP movements were jerky in the stroke subjects, as compared to the control subjects. Stroke subjects also showed difficulties in the control of M/L balance, as can be seen from the erratic and larger CoP trajectories in the M/L direction particularly near the final position. With light touch, minimal changes were observed in control subjects but the CoM and CoP movements were smoother with less CoP excursion in the stroke subjects. The average CoM and CoP displacements are shown in Figure

5.4B. Without light touch, despite larger CoM and CoP displacements in both A/P and M/L directions, there was no significant difference in the amount of CoM and CoP displacements between two groups of subjects. The CoM displacement was reduced with light touch in both groups of subjects but the reduction was not statistically significant. Similarly, stroke subjects showed a slight but insignificant decrease in CoP displacements due to light touch. Light touch, however, had a significant effect on the stability of the CoP, as shown by the significant decrease in RMS of CoP excursions in both A/P and M/L directions (Fig. 5.4C).

5.4.1.3 Muscle activities

The effects of light touch on EMG responses are shown in Figure 5.5A. EMG responses to toes-up perturbation began in the active response period (70-320 ms after perturbation onset). The control subjects maximally activated bilateral TAs to bring the body forward in resisting a toes-up tilt that displaced the CoM backwards. The activities of MG were brief and likely elicited by the sudden stretch induced by the surface tilt. Left (rear) RFM and TFL were activated to load the left leg, as the final position induced by a ramp perturbation required standing on an upslope surface. In contrast, muscle responses in the stroke subjects were under-activated on the paretic side (front foot) and over-activated on the non-paretic side, as shown by the average EMG integrals of the active period in Figure 5B. With light touch, the onset latency and the pattern of EMG responses were not altered, but the amplitude of EMG responses was significantly affected in the distal muscles and the effects were different between stroke and control subjects. With light touch, TA activation of the right (front) leg was significantly reduced in the control subjects ($p<0.001$) and unaltered in the stroke subjects. However, the MG in the non-paretic limb of stroke subjects, which was hyperactivated in the

absence of light touch, was significantly reduced in amplitude with light touch ($p<0.05$).

5.4.2 Effects of light touch on TPRs during locomotion

5.4.2.1 Kinematic responses

Figure 5.6A shows the kinematic responses with and without light touch when toes-up perturbation occurred during walking. In general, kinematic responses in both groups of subjects were smaller during walking as compared to quiet stance. The responses were mainly seen on the leading (right or paretic) limb as the trailing limb began its swing phase shortly after the perturbation, and all the weight was transferred onto the stance (right or paretic) limb. When a surface perturbation occurred during walking, healthy control subjects made the adjustments only at the ankle joint. In contrast, stroke subjects made the adjustments at the trunk, pelvis and all of the leading limb joints. Significant differences between the two groups of subjects were found at the trunk and pelvis levels. Stroke subjects also used larger arm movements to restore balance. Light touch had no effect on the kinematic response of control subjects during perturbed walking. However, light touch induced much smaller kinematic disturbances in most joints during perturbed walking in the stroke subjects. The average amount of peak-to-peak excursions of the trunk, pelvis and shoulder (of the unrestraint upper limb not touching the plank) shown in Figure 5.6B confirmed the overall reduction due to light touch in the stroke subjects. Significant reduction was found at the trunk ($p<0.05$) and shoulder ($p<0.01$) in the sagittal plane. The stability of the trunk in the sagittal ($p<0.05$) and frontal ($p<0.01$) planes and the pelvis ($p<0.05$) in the frontal plane was also increased with light touch in the stroke subjects (Fig. 5.6C).

5.4.2.2 The control of body CoM and CoP

The excursions of the body CoM and CoP during perturbed walking are shown in Figure 5.7A. In the control subjects, the CoM continued to move in the forward direction but the amount of forward displacement was slightly decreased during the toes-up tilt. The CoP was displaced backward and leftward during the perturbation. Stroke subjects displayed similar patterns of CoM and CoP excursions, but the amount of forward CoM displacement was smaller due to the decreased gait speed. Larger CoP displacements in the A/P and M/L directions were also evident in the stroke subjects (Fig. 5.7B). Although light touch reduced the displacement of both CoM and CoP in the stroke subjects, the effect was only significant for the reduction of CoP displacement in the M/L direction. Figure 7C demonstrates the stability of the body CoM and CoP during 350-1000 ms after perturbation onset, which was mainly the period of single limb support. In the absence of light touch, the RMS of CoM and CoP excursions in the stroke subjects was larger, indicating a problem of stability as balance was maintained with the paretic leg in single limb support. Whereas light touch had no significant effect on the stability of the CoM and CoP in the control subjects, the RMS of the CoP in stroke subjects showed a significant reduction in both A/P ($p<0.01$) and M/L ($p<0.001$) directions, indicating increased stability.

Figure 5.8A shows the CoM velocity in the A/P direction during walking in a control and a stroke subject with matching comfortable walking speeds. In the absence of light touch, the speed of forward progression as measured by the CoM velocity was slightly decreased in the control subject and markedly reduced in the stroke subject when walking was perturbed by a sudden toes-up surface tilt. Generally, the stroke subjects demonstrated an average of 60% decrease in the CoM velocity when walking was perturbed in the absence of light touch (Fig. 5.8B). While light touch did not affect the change in the forward progression of the control subjects during perturbed

walking, it significantly increased the speed of forward progression in all stroke subjects ($p<0.005$), even though stroke subjects still progressed slower than control subjects.

5.4.2.3 Lower limb muscle activities

Figure 5.9A contrasts EMG activation from the leading (right or paretic) limb of a control and a stroke subject, with and without light touch, in response to a sudden toes-up tilt during walking (right column) with the average background levels from unperturbed walking trials (left column) subtracted. In the absence of light touch, the control subject responded to a toes-up perturbation by increasing the activity of TA muscle on the leading stance leg to bring the body forward. The responses were brief and ended within 350 ms after the perturbation onset. Responses from the other muscles (MG, RFM and TFL) were negligible as compared to the TA activation. In contrast, the stroke subject showed co-activation of the paretic TA and MG. Increased activities of RFM and TFL were also seen in the stroke subject in response to a toes-up perturbation. The average EMG responses in figure 5.9B showed that in the absence of light touch, stroke subjects activated TA significantly less ($p<0.05$) than the control subjects, indicating weaker muscular force to bring the body forward. Large degree of MG, RFM and TFL activation in the stroke subjects also suggested that a co-contraction strategy was employed for balance as the weight was transferred onto the paretic limb during surface perturbation. Light touch had no effect on EMG responses triggered during perturbed walking in healthy controls, while the effect was more prominent in the distal muscles of the stroke subjects. TA responses were slightly increased, although not significant, with light touch. There was a significant reduction of the over-activated MG activity in the presence of light touch ($p<0.05$).

5.5 Discussion

Our hypothesis was supported in that a tactile cue provided in the form of light touch on a firm surface through the fingertip improved equilibrium control more in stroke subjects than healthy controls. Healthy subjects utilized tactile cue differently, depending on the task, such that the tactile cue improved postural control more during quiet stance than during walking. In contrast, stroke subjects benefited from the tactile cue during both tasks of standing and walking.

5.5.1 Task specificity

When the perturbation occurred during quiet stance, light touch improved postural control in healthy subjects by increasing the sagittal plane trunk stability. This effect of tactile cue on the control of equilibrium in the healthy subjects during standing are consistent with previous findings (Holden et al., 1994; Jeka and Lackner, 1994; 1995). Jeka and Lackner (1994, 1995) suggested that an increase in body stability with the tactile cue was due to an additional precision in detecting the body orientation in space by the dense and sensitive cutaneous receptors in the fingertip and hand. Thus, tactile cue was able to detect body movement more precisely than the sensory information conveyed from the feet and ankles. In addition, proprioceptors from the muscles and joints of the finger and arm that touch the surface provided the information about the orientation of the body in relation to the ground surface.

Our finding that the front limb TA response decreased significantly when light touch was provided while quiet stance was disturbed by toes-up tilt suggests that muscle activation of the relatively loaded rear limb are sufficient to restore equilibrium on an inclined surface. The effect of tactile cue on distal

leg muscle is in accordance with a previous report, where the activation of peroneal muscles was reduced during balance maintenance in tandem stance (Jeka and Lackner, 1995). It is suggested that tactile cue recruits more activations from other sets of muscles such as the trunk so that the control was improved despite the reduction in leg muscle activation (Jeka, 1997). Trunk muscles were not evaluated in our study, but the fact that the trunk was more stable with tactile cue may indicate an increase in muscle control at the trunk level.

Despite the effectiveness of tactile cue on postural control during quiet stance, control subjects showed no change in postural responses during locomotion when tactile cue was provided. These findings suggest that the effect of tactile cue is task-specific, such that additional somatosensory information from the environment is not necessary when the body being transported in space. The differential effect of tactile cue between the static and dynamic task may be due to different postural control requirement between these two tasks. We have previously shown that balance demands are higher for the maintenance of quiet stance as compared to walking (Boonsinsukh et al., 2002, Fung et al. 2003). Only minimal postural adjustments were required to restore balance when external perturbations occurred during walking. Therefore, equilibrium during walking could be maintained with the sensory inputs available during normal walking and additional tactile cue was not needed. Another possible explanation may be that visual inputs are more prominent during goal-directed locomotion (Rossignol, 1996). With the presence of vision, the postural control system may not require a tactile cue to provide the information regarding the body orientation in the environment during walking.

5.5.2 Sensorimotor integration

The effect of tactile cue is more prominent in the stroke subjects, as tactile cue improved many components of postural responses in stroke subjects more than in healthy controls. It has been suggested that when tactile cue was provided, larger body sway can give rise to larger displacement and velocity between the contacted finger and the touched surface, hence, enhancing the sensory inputs (Rogers et al., 2001). This may explain the more prominent results due to light touch observed in the stroke subjects as compared to healthy controls in our study. Another explanation may lie at the sensorimotor integration process that involves the transduction of sensory stimuli into patterns of muscle activation (Cohen and Anderson, 2002). In healthy subjects, the sensorimotor integration process is intact and additional sensory information from a tactile cue may be redundant as there are already multiple sensory inputs from vision, vestibular apparatus and somatosensory receptors contributing to the control of posture. In contrast, sensorimotor integration can be impaired by stroke at several levels of the processing, such as sensory perception, internal representation of the body, neural integration circuit and motoneuronal recruitment. Loss of body sensation, such as touch discrimination, occurred in about 50% of subjects with stroke as a result of lesions in the somatosensory cortex (SI and SII) (Carey, 1995). Stroke could also damage the dorsal premotor area (Shen and Alexander, 1997) and the posterior parietal cortex (Cohen and Anderson, 2002) that contain the circuits for sensorimotor integration. As a result, impairment in the integration of somatosensory information from the lower limbs has been observed in stroke subjects during standing under sensory conflict conditions (Di Fabio and Badke, 1991).

An accurate internal representation of the body is essential for the appropriate perception of movement of the body in space (Gurfinkel et al., 1986). Neurons in the posterolateral thalamus are believed to inform the

position of the body in relation to the gravity (Karnath et al., 2000a), whereas right inferior-posterior parietal cortex, right premotor frontal cortex and the posterior and medial portions of the thalamus are involved in the perception of visual verticality (Fogassi et al., 1992; Galletti et al., 1989; MacKay and Riehle, 1992). Alteration in the internal representation of the body has been found in stroke subjects with hemineglect and pusher syndrome (Karnath, 1994; Karnath et al., 2000b). Tactile cue is believed to provide additional precision in the perception of the body in space (Jeka and Lackner, 1994; 1995). It is also found that tactile cue can suppress abnormal proprioceptive and motor signals induced by tendon vibration of the leg muscles, suggesting that tactile cue could substitute for impaired or altered sensory information from the feet (Lackner et al., 2000). Therefore, in the presence of sensorimotor integration problems, tactile cue may not be redundant, as seen in control subjects, but essential in providing the supplementary information that is required in the control of posture for stroke subjects. This may also explain why the stroke subjects benefited from the tactile cue provided during walking while no effect was seen in the control subjects.

5.5.3 Clinical implications

The success of tactile cue on the control of posture during quiet stance and locomotion in the stroke subjects suggests an alternative approach for rehabilitation of the subjects with balance problems. The use of tactile cue could be more advantageous for stroke rehabilitation as compared to conventional means of walking devices such as canes, commonly prescribed for the subjects with balance and mobility problems. With the use of a cane, a better balance during quiet stance in stroke subjects was due to compensatory function of the non-paretic side (Milczarek et al., 1993). Similarly, with the use of a cane during walking, gait parameters (Kuan et al.,

1999), gait symmetry and muscle activation patterns on the paretic side (Hesse et al., 1998) were not different from walking without walking aids. Thus, the use of a cane may not promote the recovery of the paretic side as the non-paretic side remains overused. In addition, therapists often observe a lateral tilt of the body during walking with a cane as the body leans towards the cane side. A lateral body tilting could steer the body CoM excursion away from a straight path, leading to veering or decreased forward progression velocity. In our study, we did not observe any lateral tilt of the trunk and pelvis with the use of tactile cue. In fact, with tactile cue, the trunk seemed to be more erected. Furthermore, tactile cue did not promote the increased function of the non-paretic limb. Therefore, the adverse effect of the cane can be overcome with the use of tactile cue.

The limitation of the tactile cue is that the stable surface must be within reach. This is not always possible in daily life and can be temporarily solved by touching an accompanying person when there is no surface available (Jeka, 1997). We suggest another solution for this surface problem by the implementation of a load sensor within a walking aid. Jeka et al (1996) demonstrated similar effect of tactile cue with a fixed surface and a cane. With less than 2N of force exerted on the cane, body sway was reduced in much the same way as lightly touching a fixed surface (Jeka et al., 1996).

5.6 Acknowledgement

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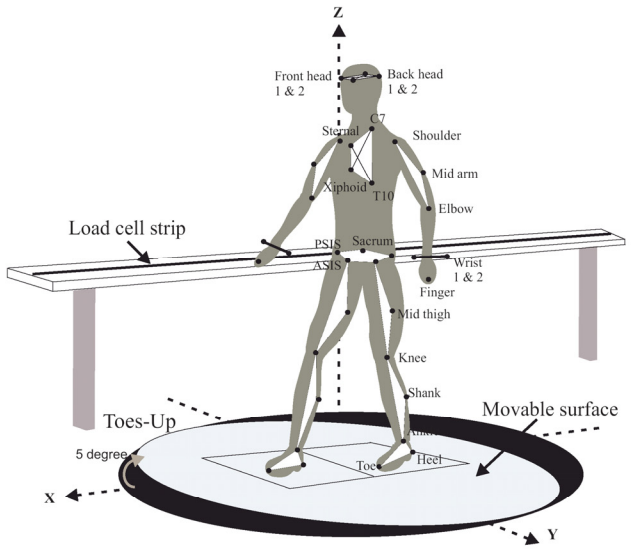
Figure 5.1. *A*: An example of the experimental setup showing that a subject was exposed to a sudden toes-up surface tilt during standing or walking. Each foot was placed on a separate force plate embedded within the surface of the movable platform, with the right or paretic foot in front of the left or nonparetic foot. Kinematic markers shown by filled circles were attached on the specified body landmarks. Tactile cue was given by touching the fingertip on the wooden plank mounted along the side of the walkway. The forces exerted (<4N) were measured by a strip of load sensors secured on top of the plank. *B*: Sample traces from a representative control subject during toes-up perturbation without light touch. The perturbation onset is the time measured at the initial inflection of the support surface position trace. The background period is 200 ms prior to perturbation onset for the standing task or average unperturbed walking cycle for the walking task. Postural responses are characterized by changes after perturbation onset from the background period during 0-1,000 ms for the standing task and 0-350 ms for the walking task. The EMG data were integrated over two windows after perturbation onset, 0-70 ms (E1: short latency reflex period) and 70-320 ms (E2: active response period).

A.


Z
Front head 1 & 2
Back head 1 & 2
C7
Sternal
Shoulder
Mid arm
Xiphoid
T10
Elbow
Load cell strip
PSIS
Sacrum
ASIS
Wrist 1 & 2
Finger
Mid thigh
Knee
Shank
Toes-Up
Movable surface
5 degree
Toe
Heel
X
Y

B.

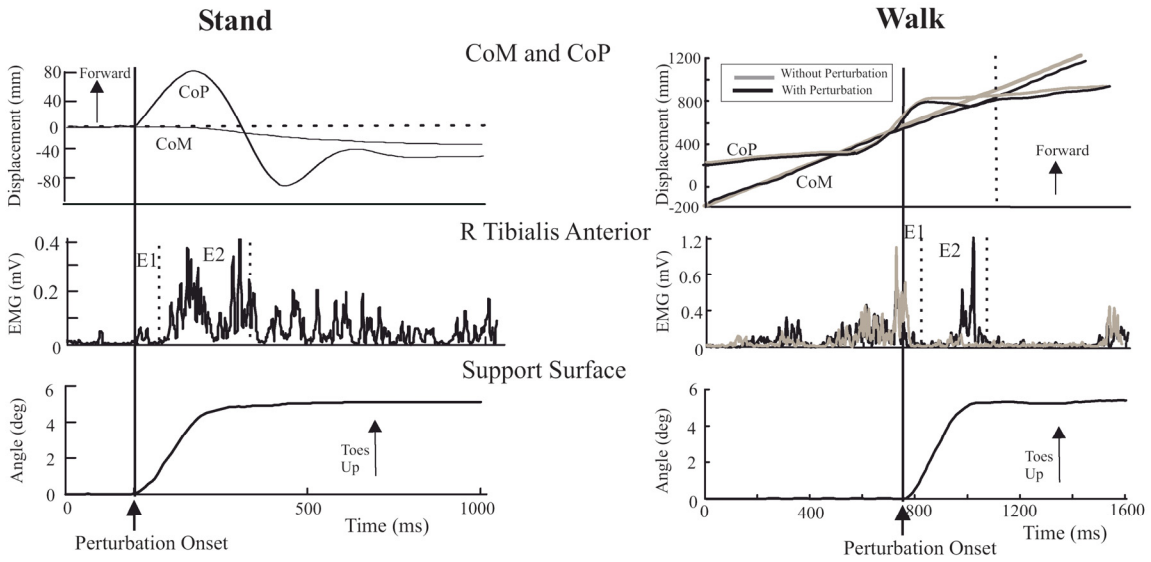
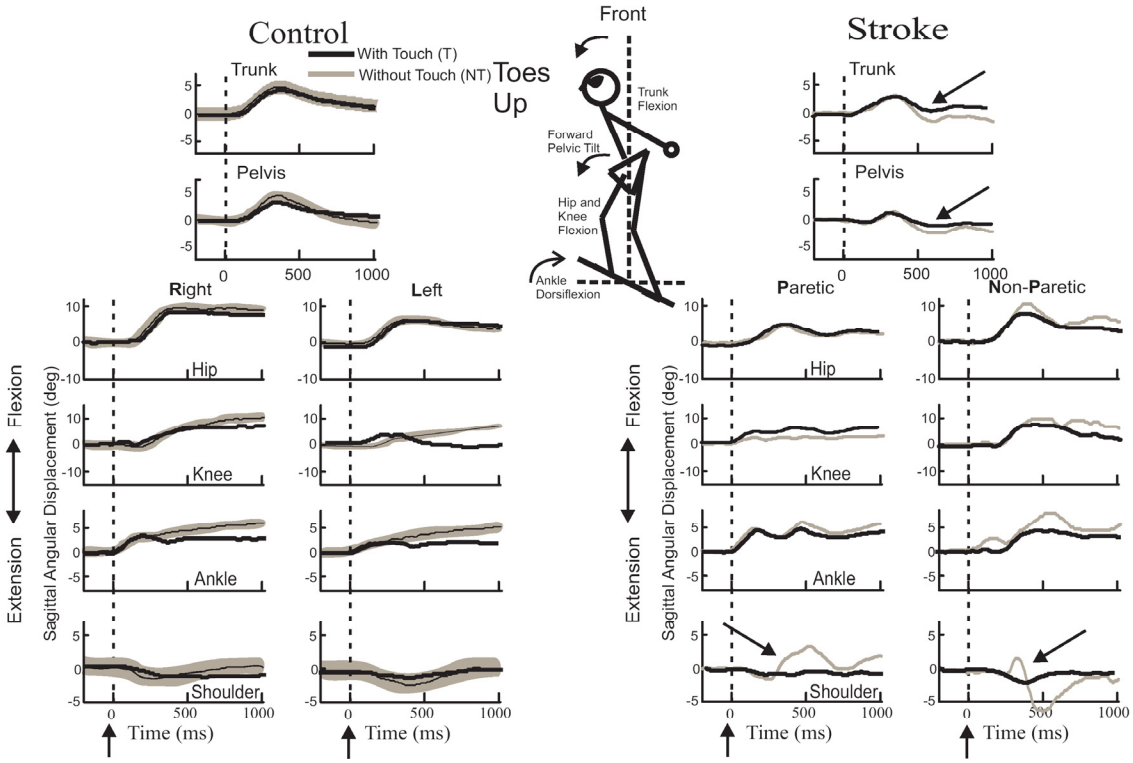
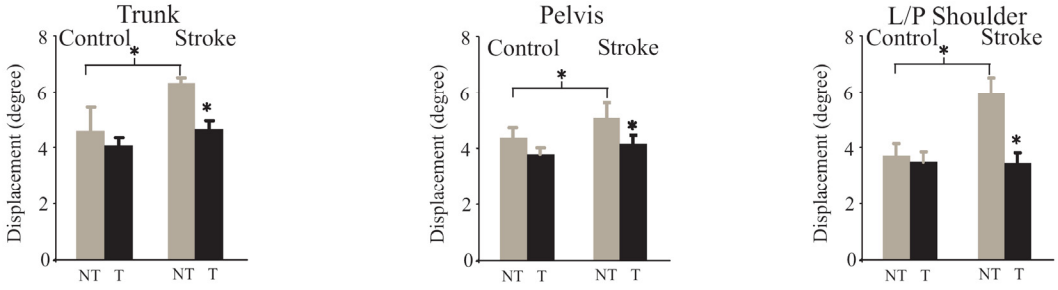

Stand
Walk
CoM and CoP
Displacement (mm)
Forward
CoP
CoM
Without Perturbation
With Perturbation
R Tibialis Anterior
EMG (mV)
E1
E2
Support Surface
Angle (deg)
Toes Up
Perturbation Onset
Time (ms)

Figure 5.2. *A*: Traces representing the sagittal plane kinematic strategies in response to a toes-up perturbation occurring during quiet stance with and without tactile cue for a control and a stroke subject. The control traces are the average of 8 control subjects (thin black) with the 95% confident intervals (grey shade), whereas the stroke traces are from a representative stroke subject. The perturbation onset is indicated by an arrow at time 0. *B and C*: Average (±1 SE) peak-to-peak amplitude of kinematic responses during quiet stance with and without light touch in the sagittal and frontal planes from 0-1,000 ms after perturbation onset in 8 control and 11 stroke subjects. The asterisk-over-solid-line indicates significant difference between groups and the asterisk indicates significant difference between the conditions of tactile cue.

A. Kinematic responses during quiet stance



B. Sagittal plane peak-to-peak excursions



C. Frontal plane peak-to-peak excursions

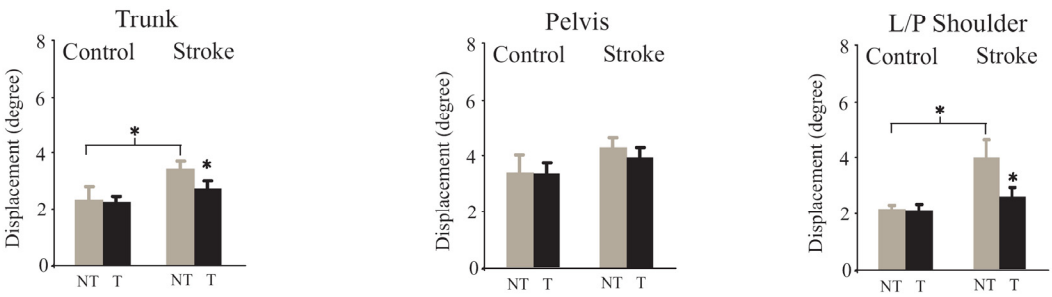
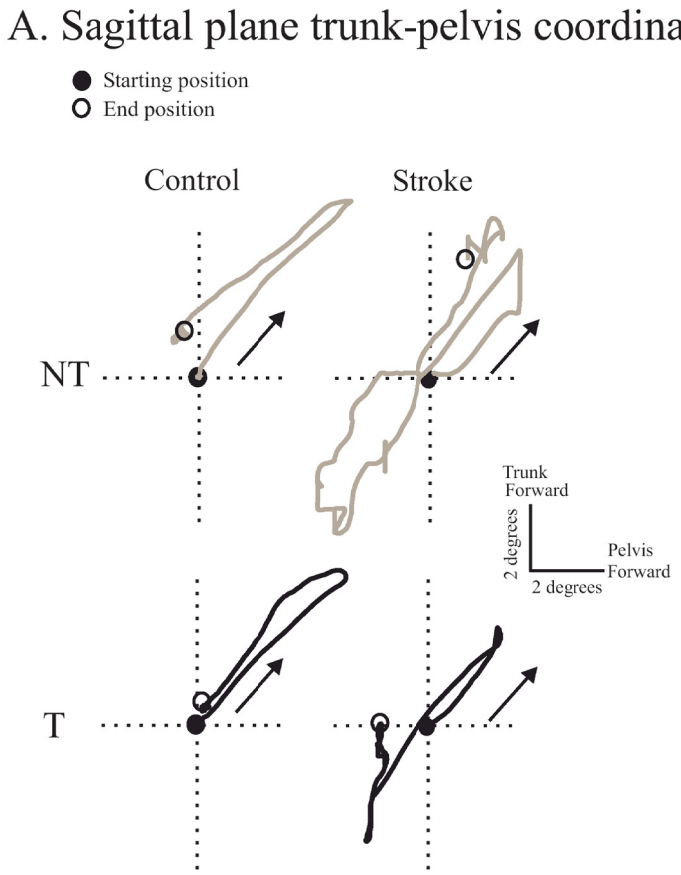


Figure 5.3. A: Sagittal plane trunk-pelvis coordination during quiet stance from 0-1,000 ms after perturbation onset when provided with and without tactile cue in a representative control and stroke subject. The arrow indicates the initial direction of trunk-pelvic excursion. *B*: Average (±1 SE) RMS of the trunk and pelvic excursion in the sagittal and frontal planes from 350-1,000 ms after perturbation onset during quiet stance when provided with and without tactile cue. The asterisk-over-solid-line indicates significant difference between groups and the asterisk indicates significant difference between the conditions of tactile cue.


A. Sagittal plane trunk-pelvis coordination
Starting position
End position
Control
Stroke
NT
T
Trunk Forward
2 degrees
Pelvis Forward
2 degrees

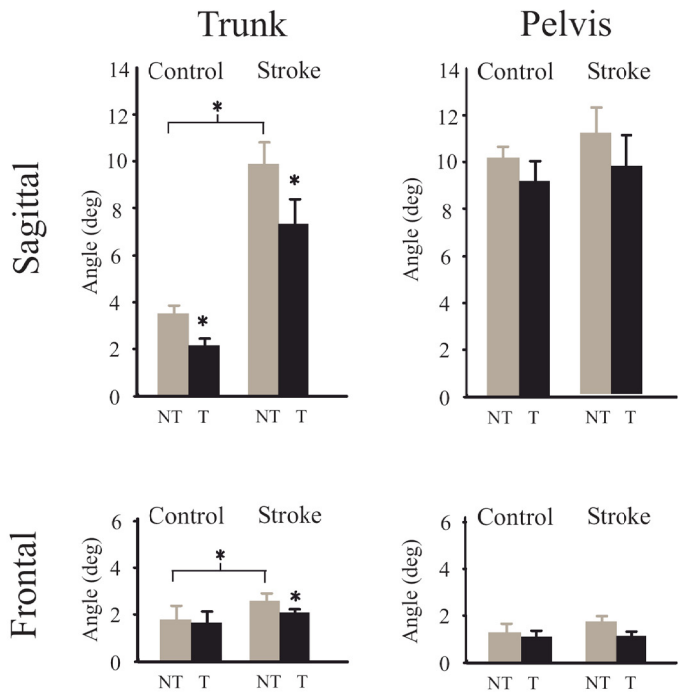
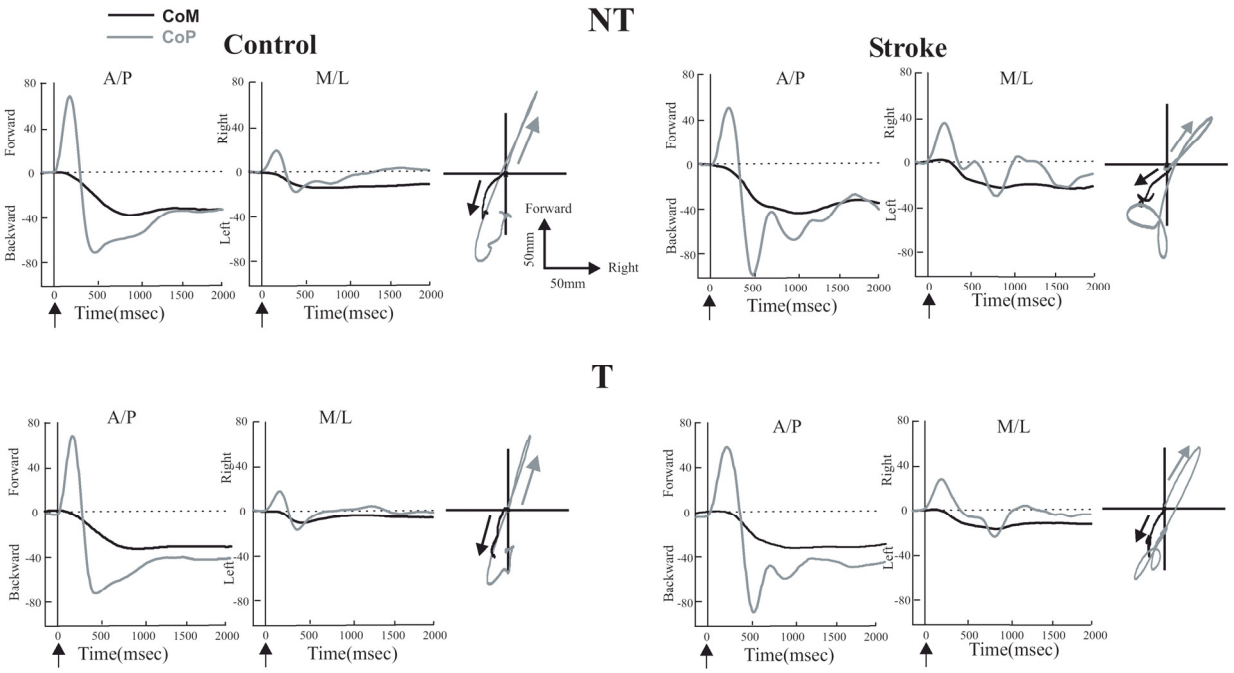
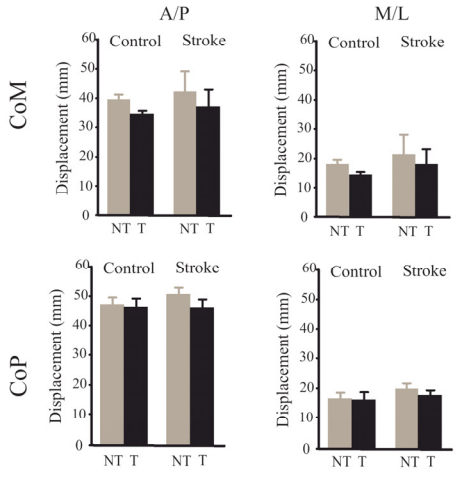

B. Trunk-Pelvis stability
Trunk
Pelvis
Sagittal
Frontal
Control
Stroke
Angle (deg)
NT T NT T

Figure 5.4. A: Horizontal displacements of the CoM and CoP during toes-up perturbation with and without tactile cue in a representative control and stroke subject. The black and grey arrows indicate the initial direction of CoM and CoP displacements, respectively. Each horizontal displacement traces is derived from the vectorial summation of the sagittal (A-P) and frontal (M-L) displacements. *B*: Average (±1 SE) peak-to-peak amplitude of the CoM and CoP displacements during quiet stance with and without tactile cue from 0-1,000 ms after perturbation onset in the A/P and M/L directions of 8 control and 11 stroke subjects. *C*: Average (±1 SE) RMS of CoM and CoP from 350-1,000 ms after perturbation onset in the A/P and M/L direction of 8 control and 11 stroke subjects during quiet stance with and with tactile cue. The asterisk-over-solid-line indicates significant difference between groups and the asterisk indicates significant difference between the conditions of tactile cue.

A. CoP and CoM displacements



B. Group CoM and CoP p-p excursions



C. Group CoM and CoP stability

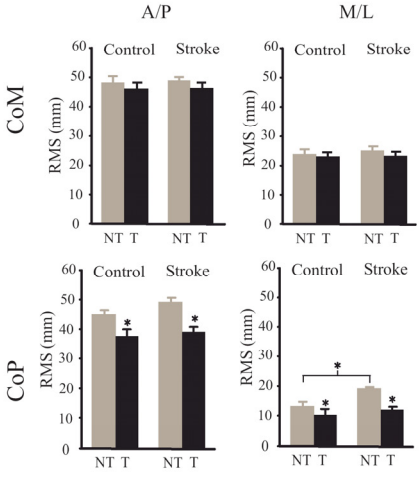


Figure 5.5. A: EMG traces from bilateral tibialis anterior (TA), medial gastrocnemius (MG), rectus femoris (RFM) and tensor fascia latae (TFL) muscles in a representative control and a stroke subject when quiet stance was perturbed by sudden toes-up surface tilt, with and without light touch. The vertical solid line indicates the perturbation onset. The grey zone indicates the 2nd integral window; the active response period (70-320 ms). *B*: Average ($\pm$1 SE) EMG integrals in the active response period of four bilateral lower limb muscles in 8 control and 11 stroke subject when quiet stance was perturbed with and without light touch. EMG responses (with background subtracted) are expressed as the percentage of the maximal response during perturbed stance from the same muscle in control subjects or from the non-paretic side of stroke subjects. The asterisk indicates significant difference between the conditions of tactile cue.

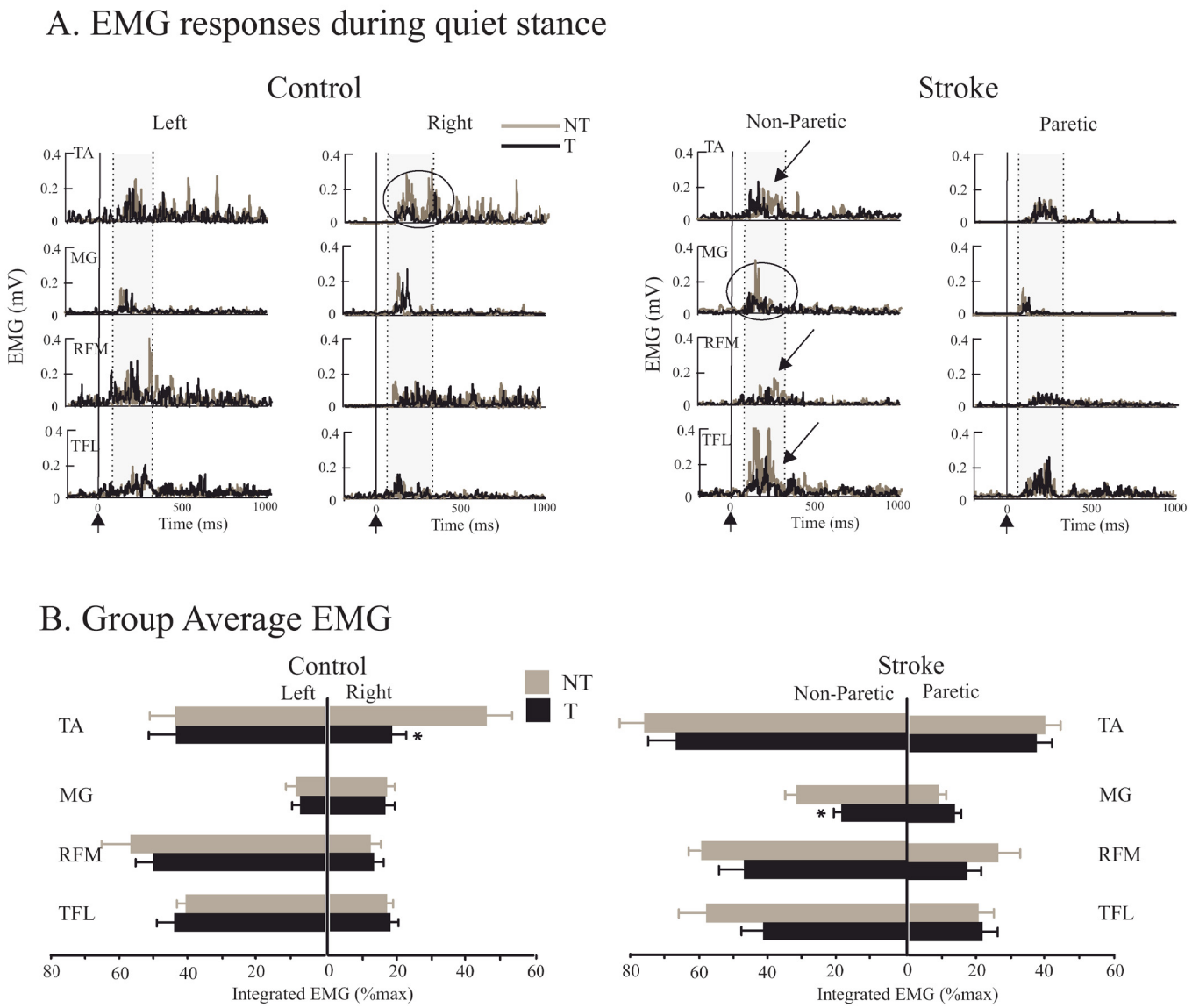

A. EMG responses during quiet stance
Control
Stroke
Left
Right
Non-Paretic
Paretic
NT
T
TA
MG
RFM
TFL
EMG (mV)
Time (ms)
B. Group Average EMG
Integrated EMG (%max)

Figure 5.6. A: Kinematic responses to toes-up perturbation during walking with and without light touch, normalized to 100% gait cycle in a representative control and stroke subject. The control traces are the average of 10 walking trials without perturbation (thin black) with the 95% confident intervals (grey shade). Kinematic profiles during perturbation without light touch are represented by the dash line while the perturbed traces with light touch are represented by the solid line. The arrow indicates the initial contact of the right or paretic leg. The thick vertical line indicates the perturbation onset and the thin vertical line indicates the 350 ms after perturbation onset. *B*: Average (±1 SE) maximum displacement of the trunk, pelvic and shoulder responses (from 0-350 ms after perturbation onset, unperturbed walking subtracted) in the sagittal and frontal planes when perturbation occurred during walking with and without light touch in 8 control and 11 stroke subjects. *C*: Average (±1 SE) RMS of trunk and pelvic responses (from 350-1000 ms after perturbation onset, unperturbed walking subtracted) with and without light touch from 8 control and 11 stroke subjects in the sagittal and frontal planes. The asterisk-over-solid-line indicates significant difference between groups and the asterisk indicates significant difference between the conditions of tactile cue.

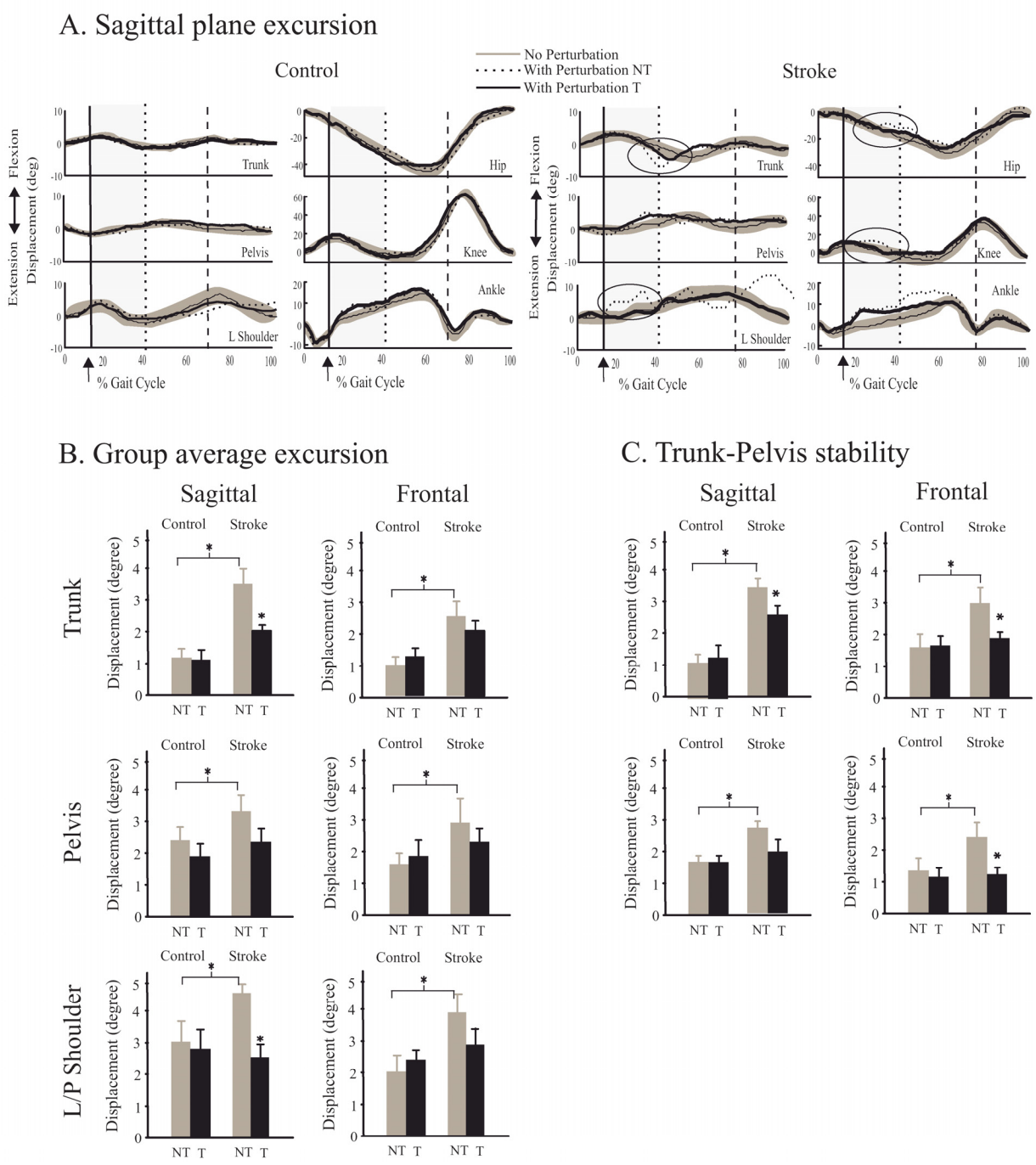
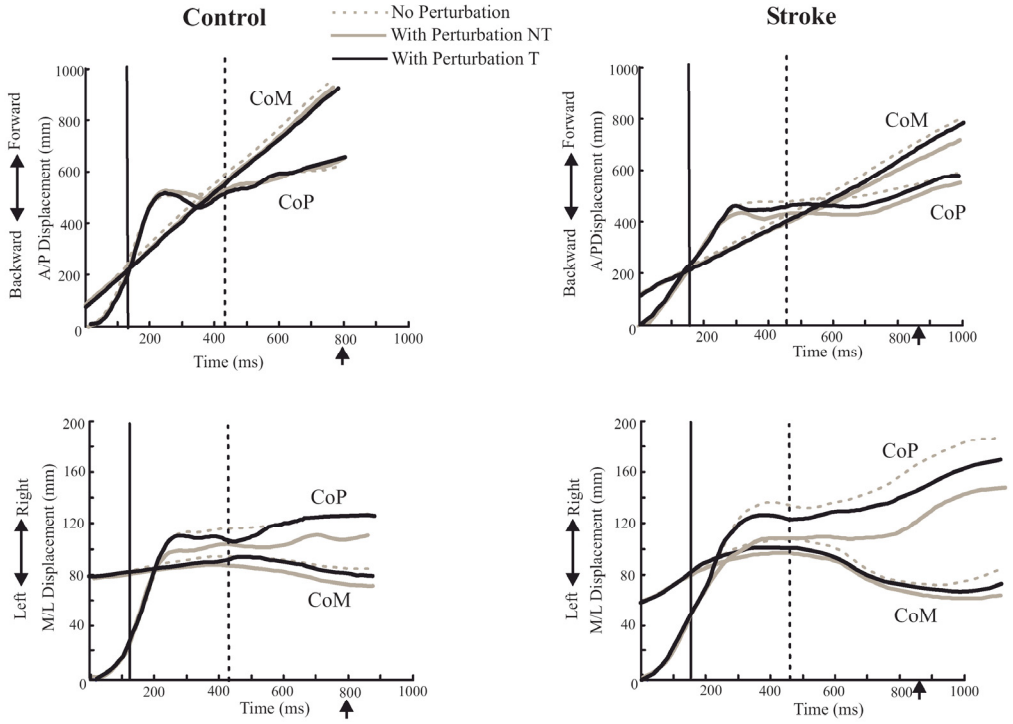

A. Sagittal plane excursion
Control
Stroke
No Perturbation
With Perturbation NT
With Perturbation T
Flexion
Extension
Displacement (deg)
Trunk
Hip
Pelvis
Knee
Ankle
L Shoulder
% Gait Cycle
B. Group average excursion
C. Trunk-Pelvis stability
Sagittal
Frontal
Trunk
Pelvis
L/P Shoulder
Displacement (degree)
Control
Stroke
NT T

Figure 5.7. A: Traces of CoP and CoM displacements, from a representative control and stroke subjects, in the A/P and M/L direction during unperturbed walking and when walking was perturbed by toes-up perturbation, with and without light touch. The solid vertical line indicates the perturbation onset and the dash vertical line indicates 350 ms after perturbation onset. *B*: Average (±1 SE) maximum CoM and CoP displacements from 8 control and 11 stroke subjects (changes from unperturbed walking during 0-350 ms after perturbation onset) in the A/P and M/L directions, with and without light touch. *C*: Average (±1 SE) RMS of CoM and CoP displacements from 8 control and 11 stroke subjects (changes from baseline walking during 350-1,000 ms after perturbation onset) in the A/P and M/L directions, with and without light touch. The asterisk-over-solid-line indicates significant difference between groups and the asterisk indicates significant difference between the conditions of tactile cue.

A. CoM and CoP displacements during walking



B. Group CoM and CoP displacement

C. Group CoM and CoP stability

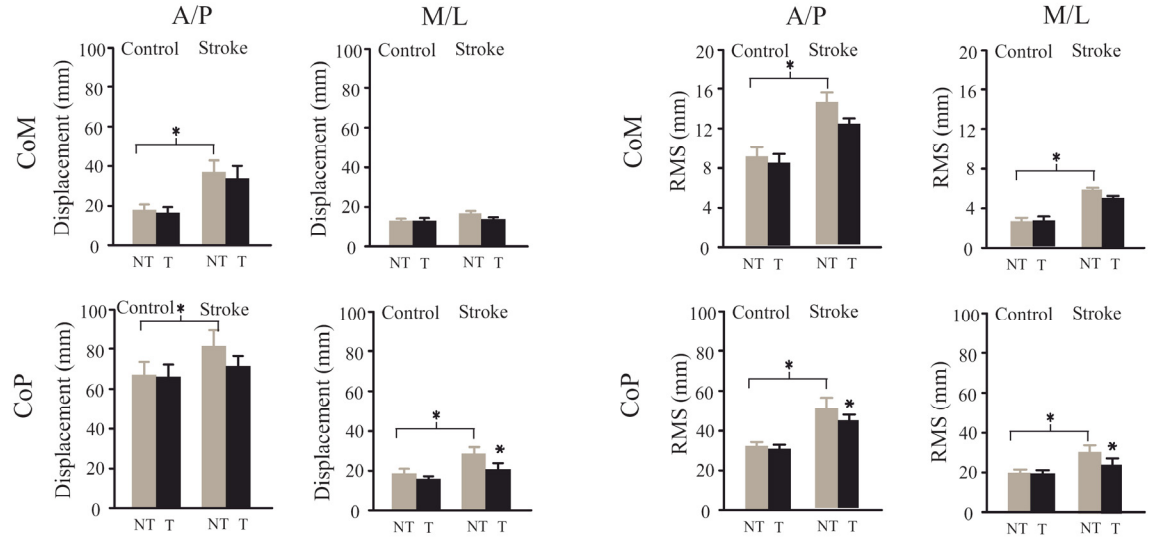


Figure 5.8. A: Traces of CoM velocity displacement in the sagittal plane normalized to 100% gait cycle during unperturbed walking and when walking was perturbed by toes-up surface tilt, with and without light touch,in a representative control and stroke subject. The no-perturbation trace is the average of 10 walking trials without perturbation (thin black) with the 95% confident intervals (grey shade) while each perturbation trace is a single walking trial with perturbation. The solid vertical line indicates the perturbation onset and the dash vertical line indicates the 350 ms after perturbation onset. *B*: Average (±1 SE) change in CoM velocity in the first 350 ms (expressed as a percentage of the comfortable unperturbed walking velocity) when walking was perturbed by toes-up surface tilt, with and without light touch, from 8 control and 11 stroke subjects. The asterisk indicates significant difference between the conditions of tactile cue.

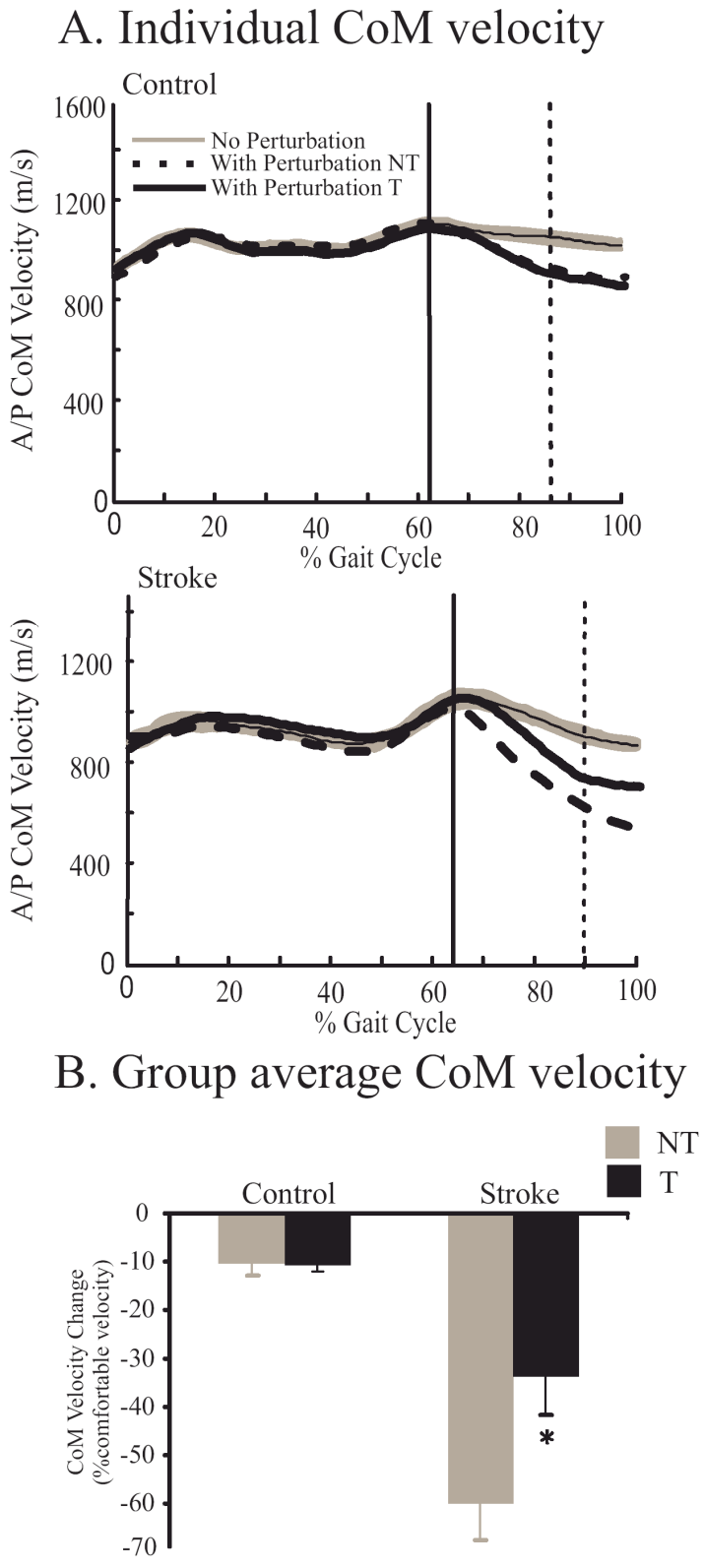

A. Individual CoM velocity
Control
No Perturbation
With Perturbation NT
With Perturbation T
A/P CoM Velocity (m/s)
1600
1200
800
400
0
0
20
40
60
80
100
% Gait Cycle
Stroke
A/P CoM Velocity (m/s)
1200
800
400
0
0
20
40
60
80
100
% Gait Cycle
B. Group average CoM velocity
NT
T
Control
Stroke
CoM Velocity Change (%comfortable velocity)
0
-10
-20
-30
-40
-50
-60
-70
*

Figure 5.9. A: EMG traces from the right/paretic TA, MG, RFM and TFL in a representative control and a stroke subject during walking without perturbation (left column) and with toes-up perturbation (right column). The right column showed EMG responses (changes from unperturbed walking) when walking was perturbed by toes-up surface tilt, with and without light touch. The arrow indicates the perturbation onset and "IC" indicates the initial ground contact of the right or paretic leg. The grey zone represents the 2nd integral window; active response period (70-320 ms after perturbation onset). *B*: Average (±1 SE) EMG integrals in the active response period of 4 muscles of the right/paretic lower limb in 8 control and 11 stroke subject during perturbed walking with and without light touch. The EMG responses (with unperturbed background subtracted) are expressed as a percentage of the maximal EMG response during perturbed stance from the same muscle in control subjects or from the non-paretic muscle of stroke subjects. The asterisk-over-solid-line indicates significant difference between the groups, "NS" indicates no significant difference between groups and the asterisk indicates significant difference between the conditions of tactile cue.

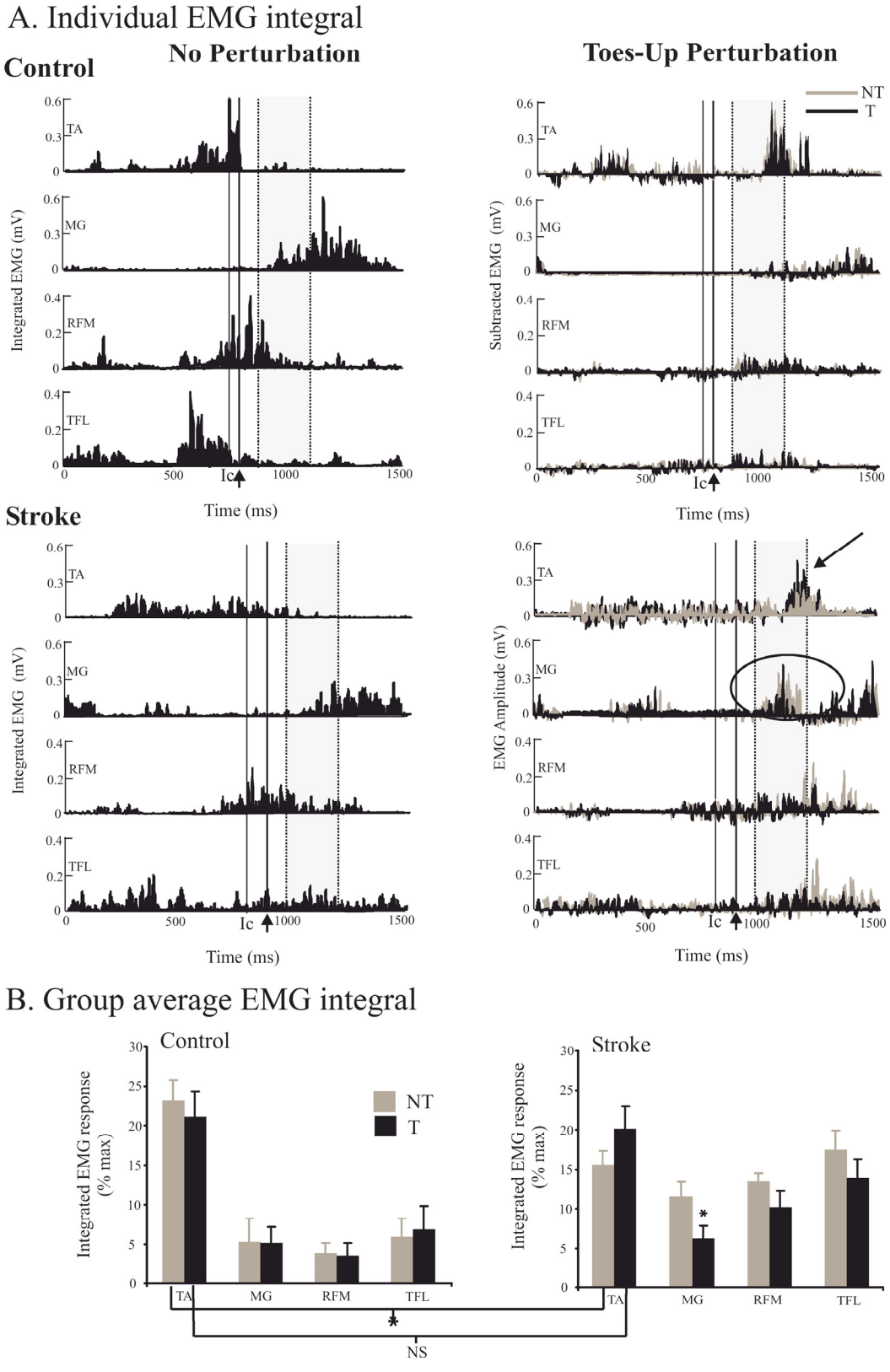

A. Individual EMG integral
No Perturbation
Toes-Up Perturbation
Control
Stroke
TA
MG
RFM
TFL
Integrated EMG (mV)
Subtracted EMG (mV)
EMG Amplitude (mV)
NT
T
Ic
Time (ms)
B. Group average EMG integral
Control
Stroke
Integrated EMG response (% max)
NT
T
TA
MG
RFM
TFL
*
NS

Table 5.1: Subject Characteristics

Stroke subjects	Age	Gender	Side of paralysis	Time since stroke (months)	Timed up and go (s)	Chedoke-McMaster Impairment score* (Leg/Foot/Postural Control)
1	54	F	L	8	14.90	6/5/5
2	60	M	R	2	10.16	6/6/6
3	63	M	R	0.5	11.15	6/6/6
4	64	M	R	3	12.27	6/3/5
5	64	F	R	2	17.66	6/5/5
6	65	F	R	1	11.63	6/6/6
7	66	M	R	4	29.31	5/3/5
8	66	M	L	2	14.66	5/5/5
9	70	M	R	12	14.31	5/4/5
10	79	F	R	8	19.42	5/5/5
11	80	M	R	3	19.53	5/4/5
Stroke (range)	54-80	7M/4F	9R/2L	0.5-12	10-29	5-6/4-6/5-6
Healthy (range)	53-79	4M/4F	N/A	N/A	5-7	7/7/7

N/A=Not Applicable

*** Maximum score for each component = 7**

CHAPTER 6

SUMMARY AND CONCLUSIONS

Impairments of the central nervous system such as those found in a cerebrovascular accident (CVA) lead to the disruption of posture and balance. Up to 73% of stroke patients fall at least once in the first six months following discharge, and as many as 50% may fall at least twice (Foster and Young 1995). The understanding of how patients with CVA react to the unexpected changes in the support surface are essential not only for allowing clinicians to target rehabilitation strategies more effectively in order to improve equilibrium control, but also for the prevention of falls in the stroke patients. Furthermore, the understanding of the relationship between the control of postural responses triggered (Triggered Postural Responses; TPRs) by a surface movement during static and dynamic conditions, provide clinicians with the insight on whether the ability to control equilibrium could transfer across tasks. The findings could be used to guide the clinicians in planning balance training programs that allow the patients to maintain equilibrium in a variety of tasks.

The research presented in this study explored the postural strategies employed by the stroke subjects to maintain balance during unexpected changes of the support surface in the pitch and roll planes during static and dynamic tasks, as compared to the healthy subjects of similar age and gender. Results from this study demonstrated that, with the intact central nervous system (CNS), the postural control system adjusts TPRs appropriately to the task demands and the context of the disturbances. The close functional connection of the postural control and locomotor centers was also verified in this study. However, these characteristics of the postural control system were disrupted following the injury of the CNS as in a stroke.

Decreased supraspinal activation to the motoneuron pool may give rise to the under-activated postural responses on the paretic limb seen in this study, whereas overactivity in the non-paretic side may be due to an adaptive or compensatory mechanism of the nervous system. The effectiveness of light touch from the fingertip in improving the control of posture during both static and dynamic tasks in the stroke subjects was presented in this research. Thus, the use of a fingertip cue in balance rehabilitation for stroke patients is proposed.

This doctoral thesis research has made some original contributions to our fundamental knowledge in the control of balance and mobility functions following stroke, as well as the development of a novel intervention strategy with the use of a tactile cue in the form of light touch with the fingertip on a firm support surface. The following conclusions and original insights can be drawn from this thesis research:

1. The demands of postural adjustment are higher during a quasi-static task such as quiet stance as compared to a dynamic task such as locomotion.

The ability to maintain equilibrium during static position and dynamic movement is crucial for performing daily activities. However, it is still unclear on the relationship between static and dynamic equilibrium maintenance, as the goal of equilibrium control during these two tasks is not similar (Winter 1987). This thesis research is the first study to provide the experimental evidence obtained in human beings, directly comparing the postural control strategies between static and dynamic tasks. Direct comparison is possible as the postural adjustments in the two tasks were observed during similar limb geometry. The findings demonstrate that postural adjustments triggered by unexpected external perturbations are normally modulated according to the requirement of the task, such that postural responses are tuned down during

walking, as compared to standing. These findings also suggest that balance requirements depend on the task goals such that the requirement is less during walking. This statement holds true when there is an injury to the central nervous system injury resulting from CVA. Stroke subjects showed less difficulty in maintaining equilibrium during walking than standing. This finding could encourage therapists to start the postural control and balance training in the stroke patients during walking earlier than waiting for the patients to regain better control of posture in standing, as done conventionally. Differences in balance requirements between static and dynamic tasks could help explain why the ability to maintain equilibrium in one task does not transfer to the same ability in the other tasks.

2. Following stroke, the ability to modulate postural adjustment to the task demand and to the direction of the perturbation is absent.

Previous studies have been shown that TPRs are disrupted following a stroke (Badke and Duncan 1983; Di Fabio et al. 1986; Dietz and Berger 1984). Nevertheless, all of those findings were obtained when the perturbation was introduced in the sagittal plane, such as forward/backward translation of the support surface, during static posture only. With only EMG recordings, those studies did not provide a complete picture of the impact of stroke on triggered postural control. This doctoral thesis is the first to provide a comprehensive understanding of the postural adaptations and compensations used by the stroke patients to maintain equilibrium when the external disturbances occur in different directions or tasks. The results demonstrated that TPRs in stroke subjects were not tuned to the axes of rotation or task demands. Stroke subjects showed asymmetrical TPRs, characterized by an under-activation of muscles and force generations in the paretic lower limb, and hyperactivity in the non-paretic side. Paretic muscles of the lower limb revealed a delay in activations, especially during roll plane

perturbations. These impairments gave rise to instability in the frontal plane as measured by the variability of center of pressure excursion. In this study, the ability to select the motor program is not likely to be affected by CVA, as most stroke subjects were able to initiate similar trunk, pelvic and lower limb responses to surface rotation during standing. However, they had difficulty in restoring and maintaining the position of the trunk and pelvis, which could be resulted from reduced sensory inputs and muscle recruitments of the trunk, pelvis and lower limbs or impaired internal representation of the body following CVA. Stroke subjects also used larger arm movements as a compensatory strategy to assist in the control of equilibrium. Our findings suggest that CVA disrupts the process of sensorimotor integration needed to regulate the TPRs. The overcompensation from the non-paretic side may be disadvantageous as it could eventually delay the recovery of the paretic side.

3. The ability to react to sudden external perturbations while walking is impaired following stroke.

A conventional way of assessing the locomotor capability of stroke patients is to investigate the change in walking velocity when performing another concurrent cognitive task, such as talking (Bowen et al. 2001; Haggard et al. 2000). This doctoral thesis expands the previous findings that stroke affects the performance of dual task when the concurrent task is not only a cognitive task, but also a non-cognitive task such as the control of equilibrium. Stroke patients showed a marked reduction in walking velocity, with the largest reduction in speed during toes-up perturbations which directly opposed the forward progression. This finding suggests that the capacity of the central nervous system to process simultaneously equilibrium responses and locomotion is reduced as a result of stroke. The pattern of change in gait speed was related to the functional level of the stroke subjects. For instance, the lower functional group of stroke subjects terminated walking to regain

equilibrium, whereas the higher functional group continued walking with an extensive reduction in the speed of forward progression.

4. Tactile cueing in the form of light touch from the fingertip is a promising strategy for balance rehabilitation

A large body of researches reported the effectiveness of light touch from the fingertip in improving the stability of the body in healthy subjects during quiet stance (Holden et al. 1994; Jeka and Lackner 1994; Jeka and Lackner 1995). It has been suggested from those studies that light touch provide precise somatosensory and proprioceptive inputs regarding body orientation with respect to external environment. This doctoral research is the first to assess the effect of light touch on the control of balance during the dynamic task. The findings in this study expand those previous findings in that not only healthy but also stroke subjects also benefited from the use of fingertip cue. In fact, the effect of light touch was more prominent in the stroke subjects, possibly due to the different degree of redundancy in available sensory information in both subject groups. The stability of the trunk and center of pressure improved during both standing and walking with the fingertip cue in the stroke subjects, leading to a reduction in hyperactivity of the non-paretic lower limb muscle and in the use of compensatory arm movement to assist in balance correction. The speed of forward progression that was affected by the toes-up rotation in the stroke subjects was significantly increased with light touch. Task-specific effect of light touch was first reported in this doctoral thesis in which the healthy subjects utilized the fingertip cue differently depending on the task demands. The differential effect of light touch between the two tasks may be due to a lesser degree of triggered postural requirement or increased visual reliance during walking.

Based on the above results, this research proposed the use of light touch from the fingertip as a tool to assist in equilibrium maintenance in the

stroke subjects. As tactile cue did not promote lateral tilting of the trunk, it may be preferable to the traditional use of walking aids such as canes. To solve the problem of limited fixed surface available for the use of light touch during walking, this study suggested the use of walking aids in a similar way (i.e., by lightly pushing on the walking aid) that a blind person uses the cane to assist in orientating and navigating through space.

Limitations of the study and future directions

The speed of walking can be a potentially confounding factor in the data analysis procedure. The average walking speed in the healthy subjects is faster than in the stroke subjects. Nevertheless, we are confident that the speed of walking does not affect the differences in triggered postural responses between healthy and stroke subjects found in this study. The comparison of the individual data from two stroke subjects and a healthy control walking at the same gait speed revealed the same contrast in TPRs. Moreover, in the static task where the speed of walking has no effect on the outcome measure, impairments of triggered postural control in the stroke subjects are similar as compared to walking, even though the deficits are larger during standing. Therefore, the findings in this study during the walking task are not influenced by the speed of walking.

The present study focuses on the stroke subjects with mild to moderate problems in mobility. Stroke subjects at other functional levels may not respond to the surface rotation or benefit from the light touch in the same way as our participants did. Therefore, results from this study may not be generalized to the stroke populations with a large range of postural control dysfunctions. Furthermore, this study investigates triggered postural responses in the step stance posture and during the double support phase of walking. Thus, results will be limited to explain TPRs only in similar body postures and task context. Somatosensory information from light touch is

given continuously by touching the wooden plank throughout the experimental trial. It is not known whether continuous fingertip cues given in this study will represent how a person uses sensory cues in every day life. In daily circumstances, more intermittent instead of continuous somatosensory cues may be utilised, as the person may not always need the somatosensory information from the fingertip and this information may be required only when balance is threatened. With regard to limitations in the present study, future research should be directed to examine postural control across various functional levels of stroke subjects, during diversity of task requirements and during several types of perturbation. Moreover, future studies should be conducted to find the optimal methods in utilising light touch or other somatosensory cues on postural control in the patient population with stroke.

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APPENDIX

HÔPITAL JUIF DE RÉADAPTATION
JEWISH REHABILITATION HOSPITAL
3205 Place Alton Goldbloom • Laval, Qué., H7V 1R2 • (450) 688-9550 • (450) 688-3673

RESEARCH ETHICS COMMITTEE

DECLARATION OF ACCEPTANCE

The present is to certify that the Research Ethics Committee of the Jewish Rehabilitation Hospital composed of:

- R. Becker – Chair and Representative of Research Review Committee
- J. Fung – Director, Research Centre
- M. Nadon – Director of Nursing
- B. Mazer – Methodologist
- F. Kaizer – Clinical Coordinator of Neurology
- B. Kroll – Spiritual Representative
- N. Mousseau – Chief of Medical Archives
- I. Shanefield – Lay Representative

Has studied the following Research proposal:

"Effects of Multi-Directional Surface Perturbations on the Triggered Postural Responses in Hemiplegic Subjects during Standing and Walking"

submitted by:

Rumpa Boonsinsukh

And found to be acceptable from an ethical standpoint.

R. Becker, M.D.
Chairperson, Research Ethics Committee
RB:vr

June 5/07
Date