THE INFLUENCE OF NORMAL, PATHOLOGICAL AND PSYCHOLOGICAL FACTORS ON POSTURAL REACTIONS TO MULTIDIRECTIONAL PERTURBATIONS

by

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ABSTRACT

This thesis constitutes a series of 4 studies which examined the physiological mechanisms involved in recovering balance from an unexpected perturbation to upright stance in humans. Postural reactions to unexpected falls are typically probed through manipulations of support surface characteristics, such as tilting rotations or sliding translations of the support surface. In the past, these perturbations have been applied almost exclusively in a single pitch (forward/backward) direction. However, outside of the laboratory we stumble, become shifted or bumped in many directions and not just in the pitch plane. The main objective of this thesis was to extend the current understanding of how humans recover from perturbations which might lead to falls in the pitch direction, to multiple directions which may mimic more normal postural challenges faced in daily life.

Our first study examined normal response characteristics of healthy young controls recovering from unexpected pitch and roll combinations of surface rotations in 16 different directions. The results revealed distinct muscle response characteristics of both early stretch and later balance correcting responses which were highly sensitive to the direction of perturbation. Trunk muscles in particular were found to provide early directionally sensitive proprioceptive information on roll perturbations. Trunk motion occurred earlier in the roll compared to the pitch direction. These findings verified the importance of examining postural reactions in multiple directions and highlighted the role of proximal muscles involved in control of the trunk and hip joint.

Previous studies examining the effects of either peripheral balance deficits such as vestibular loss or central disorders such as Parkinson's disease have had varied and inconsistent results. We hypothesized that the lack of agreement between studies and poor discriminatory ability of dynamic posturography to identify patients with balance deficits may have stemmed from the inability to observe roll directed instability in these populations. We performed two different studies to examine how bilateral peripheral vestibular loss and Parkinson's disease (PD) influenced postural reactions to perturbations in multiple directions. We have examined our results with two underlying themes. First to determine whether previous findings based on pure pitch plane research can be extended to directions other than the pitch plane. Second,

what new information can be yielded from multi-directional perturbations which is not available from observations restricted in the pitch plane.

In patients with compensated bilateral peripheral vestibular loss, we observed differences in amplitude modulation of both leg and trunk muscle balance correcting activity, and particularly abnormal control of the trunk in the pitch and roll directions which were not previously observed using only pitch plane perturbations. As a result we hypothesized that roll and pitch control is separately programmed by the central nervous system.

PD patients had impaired gain control of both stretch and subsequent balance correcting responses in lower leg, hip and trunk muscles. This was compounded by a loss of directional sensitivity in soleus and paraspinals, which led to co-contraction and stiffening of the ankle and trunk. Leg and trunk abnormalities were poorly compensated by protective arm movements which were reduced in amplitude and improperly tuned to the direction of the perturbation. Abnormalities in PD patients became most prominent when perturbations were backward and to the side. Although some of the abnormalities were clearly due to the disease itself, some may have also been related to medication effects and other factors such as increased fear of falling.

Previous studies have shown that fear of falling can influence other aspects of balance control including quiet standing, and anticipatory postural adjustments preceding a voluntary movement. The final study of the thesis was directed at identifying which components of a normal postural reaction are susceptible to a confounding influence of fear of falling. We found that both the amplitude of the balance correcting response as well as the directional sensitivity of some postural muscles was significantly influenced by an increase in postural threat. These alterations in muscle responses were expressed in significant changes in knee and trunk control as well as protective arm movements when standing under increased threat conditions.

In combination these studies provide important new evidence to suggest that multi-directional perturbations are necessary to fully explore aspects of both normal, pathological and psychological influences on postural reactions in man.

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CHAPTER 1

INTRODUCTION

What are the normal, pathological and psychological factors that contribute to generating a postural reaction of appropriate timing, magnitude and direction to prevent an unexpected fall? This has been a question which has driven extensive research over the past 25 years and formed the basis of the field of dynamic posturography, the study of postural reactions to unexpected perturbations to upright stance (Nashner et al. 1982). Although, a vast body of knowledge has been collected on how the CNS organizes muscular and biomechanical reactions to sudden perturbations, a great deal of inconsistency and controversy exists between researchers (for review refer to Horak et al. 1997; Dietz 1992; Allum and Shepard 1999; Allum et al. 1998; Horak and Macpherson, 1996).

The lack of consensus amongst researchers in the field of dynamic posturography may have resulted from differences in methodology. One major difference between studies is the way in which different groups have manipulated unexpected perturbations. There are a variety of methods that have been employed to deliver an unexpected perturbation to balance. The most common methods involve two types of movements of the support surface upon which the participant is standing, surface rotations about the ankle joint and horizontal translations in the sagittal plane (for review see Allum and Shepard, 1999). In addition, these two paradigms have also been combined to enhance or reduce stretch reflexes in triceps surae muscles. For example, combining backward translation with toe-up rotation maximizes the stretch reflex on triceps surae muscles. In contrast, using backward translations while controlling toe-down rotation to minimize ankle rotation, can effectively 'null' or reduce stretch reflexes in the triceps surae muscles (Allum et al. 1995; 1998, Bloem et al. 2000). Perturbations to balance have also been achieved by delivering a push or pulling force to the upper trunk or pelvis (Cresswell et al. 1994, Rietdyk et al. 1999, Gilles et al. 1999 Brown and Frank 1997, Matjacic et al. 2000).

The difficulty with such a wide range of protocols is that each elicits a distinctly different biomechanical challenge to the CNS which requires a unique balance response. For example,

toe-up rotations and backward translations both cause stretch of tricep surae muscles, but elicit oppositely directed displacements of the COM, and opposite polarities in balance correcting torques in the ankle, knee and hip joints after 150 ms (Allum et al. 1992). Therefore, studies using different perturbation types are in fact examining distinctly different balance response patterns.

Other characteristics of the platform perturbation have been shown to have significant influences on the ensuing postural responses. Different velocities and amplitudes of the initial platform displacement have been shown to influence the amplitude of the stretch and automatic triggered responses (Diener et al. 1984, Szturm and Fallang, 1998, Allum and Pfaltz, 1985) . In addition, the deceleration of the platform has been shown to also have a significant influence on the postural responses. McIlroy and Maki (1994a) demonstrated that participants not only respond to the deceleration of transient perturbation, but can also learn to anticipate the deceleration, and use this knowledge to alter the magnitude of the postural response.

Lack of common features between studies may have contributed to a divergence in opinions throughout the literature. For example, translational paradigms have, in general, supported a distal to proximal activation theory for postural control, in which a postural response is first triggered by ankle proprioceptive input, that radiates from distal to proximal joints (Horak et al. 1986; Nashner et al. 1982). In contrast, most studies using rotational perturbations or pushes to the upper trunk, have argued that postural reactions receive triggering input from more proximal centres in the knee, hip or trunk (Forsberg and Hirschfeld, 1994; Allum et al. 1998; Do et al. 1988; Rietdyk et al. 1999; Gilles et al. 1999; Horstmann and Dietz, 1990; Di Fabio, 1992).

Differences between studies also exist regarding different types of postural responses that are measured. Postural reactions have been divided into 'feet-in-place' responses, featuring reactions that do not require a change in base of support (for review see Horak et al. 1997; Allum and Honegger, 1998), and 'compensatory' responses which involve a change in base of support, such as taking single or multiple steps to recover balance (Maki and McIlroy, 1997; Nutt et al. 1993).

Reaching out with the arms has been shown to be another common compensatory postural response which is scaled to magnitude and direction of response (McIlroy and Maki, 1994b). However, arm movements are yet another factor which has not been consistently controlled between studies. For example, some studies have left arms free to move, while others require the arms to be crossed in front of the chest to facilitate motion analysis recording. Denying the ability to use normal arm responses may increase the need to compensate with other protective responses such as stepping. Conversely, fixing the feet in place, may increase the need to compensate with protective arm movements. In addition, the restriction of protective compensatory reactions, be it stepping or reaching movements may highlight or mask postural abnormalities in patients with different balance deficits. Therefore, it is important to gain a better understanding of how protective compensatory responses may influence other postural responses independently and in combination.

Other confounding factors have been shown to influence the automatic postural responses in standing subjects including prior knowledge (Horak et al. 1989; Maki and Whitelaw, 1993; Diener et al. 1991) expectation (Keshner et al. 1987, Sveistrup and Woollacott, 1997; Chong et al. 1999), pre-stimulus posture (Diener et al. 1983; Allum and Pfaltz 1985; Schieppati et al. 1995; Horak and Moore 1993; Beckley et al. 1991) and background activity (Bedingham and Tatton, 1984, Allum and Mauritz, 1984, Bloem et al. 1993). These factors have all been controlled or accounted for differently in different studies, thereby making meaningful comparisons between findings even more difficult.

The great variability in protocol and methodology in previous research may partly explain the limited success for pitch-plane dynamic posturography in diagnosing and discriminating balance disorders (Di Fabio 1995; Bronstein and Guerraz 1999, Bloem et al. 1992). Although more recently, greater success to discriminate between patient populations has been achieved using upper rather than lower-body responses to pitch plane rotations (Allum et al. 2001a), its fundamental utility to screen for more subtle balance disorders or recognize disease-specific information, such as the side of a lesion is questionable (Lipp and Longridge 1994; Furman 1995).

Adding further to the limited success of dynamic posturography to identify balance deficits has been the reliance on recordings from perturbations within a single unidirectional pitch plane. This has major drawbacks when concepts of normal and pathological balance control need to be generalized to multiple directions. Real life situations, such as an accelerating bus, pitching boat or rolling train, impose destabilizing forces which rarely act along a purely sagittal plane. When a fall does occur in older adults, they frequently occur in lateral as well as pitch directions (Holliday et al. 1990; Maki and McIlroy 1998). However, falls in the lateral direction may be more severe as they become the cause of hip and wrist fractures in elderly (Cummings and Nevitt, 1994; Greenspan et al. 1998). Lateral instability has been confirmed in both the pitch and roll planes in aging (Gill et al. 2001) and patients with balance disorders (Allum et al. 2000b) during both balance and locomotor tasks. Therefore, the ability to examine postural reactions in multi-directional perturbations may prove to be more useful in discriminating between groups with different balance deficits by using perturbation directions that are most destabilizing and threatening in these populations.

Sensory systems may also contribute to a balance correction differently depending upon the direction of perturbation. For example, vestibular receptors are most sensitive in the planes of the semi-circular canals (Tomko et al. 1981), while joint receptors (Rothwell, 1994) and stretch receptors provide directional information in multiple directions. Furthermore, the directions of maximum isometric stabilizing activity in neck (Keshner et al., 1988), trunk (Lavender et al., 1994) and elbow muscles (Buchanan et al., 1986) lie in multiple planes.

Recent studies have begun to examine postural reactions in multiple directions (Maki et al. 1994; Moore et al. 1988; Henry et al. 1998; Allum et al. 1998). These studies have yielded interesting new results which have shed new light on previously contentious issues. For example, perturbations in off-pitch directions elicit muscle responses in proximal muscles, such as erector spinae, tensor fascia latae and hip abdcuctors which have onsets as early or earlier that that in the distal ankle musculature (Maki et al. 1994; Henry et al. 1998). These observations provide convincing evidence to argue against a distal to proximal activation theory. Multi-directional perturbations also provides insight into the range of activation and

directional sensitivity of different muscles (Moore et al. 1988, Henry et al. 1998; Maki et al. 1994; Macpherson et al. 1988) which may prove to be susceptible to specific balance deficits. Finally, roll or lateral perturbations allows for analysis of trunk control in the frontal plane which has been shown to be unstable in gait and postural tasks in elderly (Gill et al. 2001) as well as patients with vestibular loss (Martin, 1965) and Parkinson's disease (Adkin et al. 2000).

To date, there have been no previous studies which have examined postural reactions to multi-directional perturbations delivered by rotations of the support surface. One benefit of using rotational perturbations, compared to translational perturbations, is that the stretch related information can be elicited in a muscle antagonistic to that generating the balance correcting response (Diener et al. 1983; 1984; Allum et al. 1992).

Therefore, the goal of the present thesis was to examine the normal, pathological and psychological factors that influence postural reactions from two new perspectives. The first goal was to determine if the present understanding of normal and pathological postural responses are applicable to perturbations in multiple directions, which may more accurately mimic events experienced in everyday life. The second goal was to determine what new information can be extracted from multi-directional perturbations that has not been previously evident using only pitch plane perturbations.

In the following chapters, these two goals will be addressed by examining the postural reactions in four distinct populations. First we have analyzed the response to 16 different directions in normal healthy young adults to gain a clear understanding of the normal postural response (Carpenter et al. 1999). Second we examined patients with a bilateral vestibular loss to determine the role of vestibulo-spinal interaction on controlling postural responses in multi-directions (Carpenter et al. 2001). Third we examined the effects of a more central balance deficit by comparing patients with idiopathic Parkinson's disease, both 'on' and 'off' their medication, to normal controls. Finally we examined the influence of a postural threat, in a group of young healthy controls to try and understand how increased threat and possibly fear

of falling may play a confounding role in the observed changes seen in patients with balance deficits.

REFERENCES

Adkin AL, Allum JHJ, Carpenter MG, Bloem BR. (2000) Clinical evaluation of postural instability in Parkinson's disease using quantifiable trunk sway measures in freely moving subjects. Movement Disorders 15 (Suppl 3): 77-78

Allum JHJ, Bloem BR, Carpenter MG, Honegger F (2001a) Differential diagnosis of proprioceptive and vestibular deficits using dynamic support-surface posturography. Gait & Posture (in press)

Allum JHJ, Bloem BR, Carpenter MG, Hulliger M, Hadders-Algra M (1998) Proprioceptive control of posture: a review of new concepts. Gait Posture 8:214-242

Allum JHJ, Held-Ziolkowska M, Adkin AL, Carpenter MG, Honegger F (2001b) Trunk sway measures of postural stability during clinical balance tests: Effects of a unilateral vestibular deficit. Gait and Posture (In Press)

Allum JHJ, Honegger F (1992) A postural model of balance-correcting movement strategies. J Vestib Res 2: 323-347

Allum JHJ, Honegger F (1998) Interactions between vestibular and proprioceptive inputs triggering and modulating human balance-correcting responses differ across muscles. Exp Brain Res. 121: 478-494

Allum JHJ, Honegger F, Acuna H (1995) Differential control of leg and trunk muscle activity by vestibulo-spinal and proprioceptive signals during human balance corrections. Acta Otolaryngol (Stockh) 115:124-129

Allum JHJ, Mauritz KH (1984) Compensation for intrinsic muscle stiffness by short-latency reflexes in human triceps surae muscles. J Neurophysiol 52:797-818

Allum JHJ, Pfaltz CR (1985) Visual and vestibular contributions to pitch sway stabilization in the ankle muscles of normals and patients with bilateral peripheral vestibular deficits. Exp Brain Res 58:82-94

Allum JHJ, Shepard NT (1999) An overview of the clinical use of dynamic posturography in the differential diagnosis of balance disorders. J Vestib Res 9: 223-252

Beckley DJ, Bloem BR, Remler MP, Roos RAC, Van Dijk JG (1991) Long latency postural responses are functionally modified by cognitive set. Electroenceph Clin Neurophysiol 81: 353-358

Bedingham W, Tatton WG (1984) Dependence of EMG responses evoked by imposed wrist displacements on pre-existing activity in the stretched muscles. Can J Neurol Sci 11: 272-280

Bloem BR, Allum JHJ, Carpenter MG, Honegger F (2000) Is lower leg proprioception essential for triggering human balance corrections? Exp Brain Res 130: 375-391

Bloem BR, Beckley DJ, van Dijk JG, Zwinderman AH, Roos RAC (1992) Are medium and long latency reflexes a screening tool for early Parkinson's disease? J Neurol Sci 113: 38-42

Bloem BR, van Dijk JG, Beckley DJ, Zwinderman AH, Remler MP, Roos RA (1993) Correction for the influence of background muscle activity on stretch reflex amplitudes J Neurosci Methods 46: 167-174

Bronstein AM, Guerraz M (1999) Visual-vestibular control of posture and gait: physiological mechanisms and disorders. Curr Opin Neurol 12:5-11

Brown LA, Frank JS (1997) Postural compensations to the potential consequences of instability: kinematics. Gait and Posture 6:89-97

Buchanan TS, Almdale DP, Lewis JL, Rymer WZ (1986) Characteristics of synergic relations during isometric contractions of human elbow muscles. J Neurophysiol 56: 1225-1241

Carpenter MG, Allum JHJ, Honegger F (1999) Directional sensitivity of stretch reflexes and balance corrections for normal subjects in the roll and pitch planes. Exp Brain Res 129: 93-113

Carpenter MG, Allum JHJ, Honegger F (2001) Vestibular influences on human postural control in combinations of pitch and roll planes reveal differences in spatiotemporal processing. Experimental Brain Research (In press)

Chong RKY, Horak FB, Woollacott MH (1999) Time-dependent influence of sensorimotor set on automatic responses in perturbed stance. Exp Brain Res 124: 513-519

Cresswell AG, Oddsson L, Thorstensson A (1994) The influence of sudden perturbations on trunk muscle activity and intra-abdominal pressure while standing Exp Brain Res 98: 336-341

Cummings SR, Nevitt MC. (1994) Non-skeletal determinants of fractures: The potential importance of the mechanics of falls. Osteoporosis International 1: S67-70

Di Fabio RP (1995) Sensitivity and specificity of platform posturography for identifying patients with vestibular dysfunction. Phys Ther. 75:290-305

Di Fabio RP, Graf B, Badke MB, Breunig A, Jensen K (1992) Effect of knee joint laxity on long-loop postural reflexes: evidence for a human capsular-hamstring reflex. Exp Brain Res 90: 189-200

Diener HC, Bootz F, Dichgans J, Bruzek W (1983) Variability of postural "reflexes" in humans. Exp Brain Res 52: 423-428

Diener HC, Dichgans J, Bootz F, Bacher M (1984) Early stabilization of human posture after a sudden disturbance: influence of rate and amplitude of displacement. Exp Brain Res 56:126-134

Diener HC, Horak F, Stelmach G, Guschbauer B, Dichgans J (1991) Direction and amplitude precuing has no effect on automatic posture responses. Exp Brain Res 84: 219-223

Dietz V (1992) Human neuronal control of automatic functional movements: interaction between central programs and afferent input. Physiol Rev. 72: 33-69

Do MC, Brenière Y, Bouisset S (1988) Compensatory reactions in forward fall: are they initiated by stretch receptors? Electroenceph Clin Neurophysiol 69: 448-452

Forssberg H, Hirschfeld H (1994) Postural adjustments in sitting humans following external perturbations: muscle activity and kinematics. Exp Brain Res 97: 515-527

Furman JM (1995) Role of posturography in the management of vestibular patients. Otolaryngol Head Neck Surg 112:8-15

Gill J, Allum JHJ, Held-Ziolkowska M, Carpenter MG, Honegger F, Pierchala K (2001)Trunk sway measures of dynamic equilibrium during clinical balance tasks: effects of age. J Gerontol Med Sci (in press)

Gilles M, Wing AM, Kirker GB (1999) Lateral balance organisation in human stance in response to a random or predictable perturbation. Exp Brain Res 124:137-144

Greenspan SL, Myers ER, Kiel DP, Parker RA, Hayes WC, Resnick NM (1998) Fall direction, bone mineral density, and function: risk factors for hip fracture in frail nursing home elderly. Am J Med 104: 539-545

Henry SM, Fung J, Horak FB (1998) EMG responses to maintain stance during multidirection surface translations. J Neurophysiol 80:1939-1950

Holliday PJ, Fernie GR, Gryfe CI, Griggs GT (1990) "Video recording of spontaneous falls of the elderly," slips, stumbles, and falls: Pedestrian footwear and surfaces, ASTM STP 1103, B.E. Gray, Ed., American Society for Testing and Materials, B.E. Gray (Ed.) Philadelphia: 7-16

Horak FB, Diener HC, Nashner LM (1989) Influence of central set on human postural responses. J Neurophysiol 62: 841-853

Horak FB, Henry SM, Shumway-Cook A (1997) Postural perturbations: New insights for treatment of balance disorders. Physical Therapy 77: 517-533

Horak FB, Macpherson JM. (1996) Postural orientation and equilibrium. In: Smith JL, ed. Handbook of Physiology, Section 12: Exercise: Regulation and Integration of Multiple Systems. New York, NY: Oxford University Press Inc: 255-292

Horak FB, Moore SP (1993) The effect of prior leaning on human postural responses. Gait Posture 1:203-210

Horak FB, Nashner LM (1986) Central programming of postural movements: Adaptation to altered support-surface configurations. J Neurophysiol 55: 1369-1381

Horstmann GA, Dietz V (1990) A basic posture control mechanism: the stabilization of the centre of gravity. Electroenceph Clin Neurophysiol 76: 165-176

Keshner EA, Allum JH, Pfaltz CR (1987) Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit. Exp Brain Res 69: 77-92

Keshner EA, Woollacott MH, Debu B (1988) Neck, trunk and limb muscle responses during postural perturbations in humans. Exp Brain Res 71(3): 455-466

Lavender S, Trafimow J, Andersson GB, Mayer RS, Chen ICH (1994) Trunk muscle activation. The effects of torso flexion, moment direction, and moment magnitude. Spine 19: 771-778

Lipp M, Longridge NS (1994) Computerised dynamic posturography: its place in the evaluation of patients with dizziness and imbalance. J Otolaryngol 23:177-183

Macpherson JM (1988) Strategies that simplify the control of quadrupedal stance. II. Electromyographic activity. J Neurophysiol 60: 218-231

Maki BE, McIlroy WE (1997) The role of limb movements in maintaining upright stance: the 'Change in Support' strategy. Phys Ther 77: 488-507

Maki BE, McIlroy WE (1998) Control of compensatory stepping reactions: Age related impairment and the potential for remedial intervention. Physiotherapy Theory and Practice 15: 69-90

Maki BE, McIlroy WE, Perry SD (1994) Compensatory responses to multi-directional perturbations. In: Taguchi K, Igarashi M, Mori S (eds) Vestibular and Neural Front, Elsevier Science, Amsterdam, pp 437-440

Maki BE, Whitelaw RS (1993) Influence of expectation and arousal on center-of-pressure responses to transient postural perturbations. J Vestib Res 3: 25-39

Martin JP (1965) Tilting reactions and disorders of the basal ganglia. Brain 88: 855-874

Matjacic Z, Voigt M, Popovic D, Sinkjaer T (2001) Functional postural responses after perturbations in multiple directions in a standing man revealed by net joint torques: A principle of decoupled control. J Biomech 34:187-196

McIlroy WE, Maki BE (1994a) The 'deceleration response' to transient perturbation of upright stance. Neurosci Lett 175: 13-16

McIlroy WE, Maki BE (1994b) Compensatory arm movements evoked by transient perturbations of upright stance. In: Taguchi K, Igarashi M, Mori S, editors. Vestibular and neural front. Amsterdam: Elsevier: 489-92

Moore SP, Rushmer DS, Windus SL, Nashner LM (1988) Human automatic postural responses: responses to horizontal perturbations of stance in multiple directions. Exp Brain Res 73:648-658

Nashner LM, Black FO, Wall C III (1982) Adaptation to altered support surface and visual conditions during stance: patients with vestibular deficits. J Neuroscience 2: 536-544

Nutt JG, Marsden CD, Thompson PD (1993) Human walking and higher-level gait disorders, particularly in the elderly. Neurology 43: 268-279

Rietdyk S, Patla AE, Winter DA, Ishac MG, Little CE (1999) NACOB presentation CSB New Investigator Award. Balance recovery from medio-lateral perturbations of the upper body during standing. J Biomech 32:1149-1158

Rothwell J (1994) Control of human voluntary movement, 2nd edn. Chapman Hall, London: 103-117

Schieppati M, Nardone A, Siliotto R, Grasso M (1995) Early and late stretch responses of human foot muscles induced by perturbation of stance. Exp Brain Res 105: 411-422

Sveistrup H, Woollacott MH (1997) Practice modifies the developing automatic postural response. Exp Brain Res 114: 33-43

Szturm T, Fallang B (1998) Effects of varying acceleration of platform translation and toes-up rotations on the pattern and magnitude of balance reactions in humans. J Vestib Res 8: 381-397

Tomko DL, Peterka RJ Schor RH (1981) Responses to head tilt in cat eigth nerve afferents. Exp Brain Res 41:216-221

CHAPTER 2

DIRECTIONAL SENSITIVITY OF STRETCH REFLEXES AND BALANCE CORRECTIONS FOR NORMAL SUBJECTS IN THE ROLL AND PITCH PLANES

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ABSTRACT

A large body of evidence has been collected which describes the response parameters associated with automatic balance corrections in man to perturbations in the pitch plane. However, perturbations to human stance can be expected from multiple directions. The purpose of the present study was to describe the directional sensitivities of muscle responses reestablishing disturbed stance equilibrium in normal subjects. The contributions of stretch reflex and automatic balance- correcting responses to balance control, and concomitant biomechanical reactions, were examined for combinations of pitch and roll perturbations of the support surface. More specifically, muscle responses, initial head accelerations and trunk velocities were analyzed with the intent to identify possible origins of directionally specific triggering signals and to examine how sensory information is used to modulate triggered balance corrections with respect to direction.

Fourteen healthy adults were required to stand on a dual axis rotating platform capable of delivering rotational perturbations with constant amplitude (7.5 deg) and velocity (50 deg/s) through multiple directions in the pitch and roll planes. Each subject was randomly presented with 44 support surface rotations through 16 different directions separated by 22.5 deg first under eyes open, and then, for a second identical set of rotations, under eyes closed conditions. Bilateral muscle activity from tibialis anterior, soleus, lateral quadriceps and paraspinals were collected, averaged across direction, and areas calculated over intervals with significant bursts of activity. Trunk angular velocity and ankle torque data were averaged over intervals corresponding to significant biomechanical events. Stretch reflex (intervals of 40-100, 80-120 ms) and automatic balance correcting responses (120-220, 240-340 ms) in the same muscle were sensitive to distinctly different directions. The directions of the maximum amplitude of balance-correcting activity in leg muscles were oriented along the pitch plane, approximately 180 deg from the maximum amplitude of stretch responses. Ankle torques for almost all perturbation directions were also aligned along the pitch plane. Stretch reflexes in paraspinal muscles were tuned along the 45 deg plane but at 90 deg to automatic balance corrections and 180 deg to unloading responses in the same muscle. Stretch reflex onsets in paraspinal muscles were observed at 60 ms, as early as those of soleus muscles. In contrast,

unloading reflexes in released paraspinal muscles were observed at 40 ms for perturbations which caused roll of the trunk towards the recorded muscle. Onsets of trunk roll velocities were earlier and more rapid than those observed for pitch velocities. Trunk pitch occurred for pure roll directions but not vice versa. When considered together, early stretch and unloading of paraspinals, and concomitant roll and pitch velocities of the trunk requiring a roll-and-pitch-based hip torque strategy, bring into question previous hypotheses of an ankle-based trigger signal or ankle-based movement strategies for postural balance reactions. These findings are compatible with the hypothesis that stretch, force and joint related proprioceptive receptors at the level of the trunk provide a directionally sensitive triggering mechanism underlying a, minimally, two stage (pitch-based leg and pitch-and-roll-based trunk) balance-correcting strategy. Accelerometer recordings from the head identified large vertical linear accelerations only for pitch movements and angular roll accelerations only during roll perturbations with latencies as early as 15 ms. Thus, it appears that balance corrections in leg and trunk muscles may receive strong, receptor dependent (otolith or vertical canal), and directionally sensitive amplitude modulating input from vestibulo-spinal signals.

Key words: Balance corrections, stretch reflexes, directional sensitivity, vestibulo-spinal input, proprioception, posturography, torque strategies.

INTRODUCTION

Falls occur in different directions and at different speeds depending primarily on the original direction and intensity of the perturbation and secondarily on the flexibility of the trunk in both the roll and pitch planes. The conversion of sensory information on the perturbation to appropriate balance corrections is a task the central nervous system (CNS) must rapidly initiate and accurately modulate if a fall is to be prevented. This is accomplished by the CNS initiating bursts of muscle activity with onset latencies around 100 to 120 ms at a number of body segments thereby providing the effective righting reaction (Allum and Honegger 1992, Allum et al 1994, Keshner et al 1987, 1988). Bursts of muscle activity appearing up to 100 ms after onset of the perturbation, generated by proprioceptive stretch reflexes, do not have sufficient strength to prevent a fall (Allum and Pfaltz,1985). From a neurophysiological viewpoint a number of general questions can be raised about these processes. For example, the question arises about the earliest and most directionally specific trigger signal for balance corrections, be it of vestibular or proprioceptive origin. Following initiation of a pattern of responses appropriate for the direction of falling, the responses must be scaled to correct the fall. Again the question is the nature of the underlying sensory signals contributing to this modulation of balance corrections and whether this scaling could be different for different directions of falling.

Previous work on balance corrections has almost been exclusively limited to the pitch plane. This work which mostly concentrated on lower-leg muscles, established that the latencies of balance corrections occur in a narrow time band of 90-120 ms (Nashner, 1977; Allum and Budingen, 1979; Diener et al., 1983). Following the terminology of Melville-Jones and Watt (1971) the observed balance corrections were initially classified as 'functional stretch reflexes', with ankle inputs seen as the primary sensory triggering signal (Nashner, 1976; 1977; Diener et al., 1983;1984). The term „functional stretch reflex" implies that automatic balance corrections are very similar to or even initiated by stretch of leg muscles. Based on this concept the question arises whether information on the directional sensitivity of balance corrections is best studied using either a constant amount of ankle rotation or other joint rotation.

One common approach used previously for pitch perturbations has been to manipulate different characteristics of the perturbation to destabilize the body while holding ankle rotation at a constant amplitude and observe resultant changes in the automatic responses of biomechanical and muscular variables. For example, translational versus rotational movements of the support surface yield very distinct postural responses regardless of the similarities in ankle joint rotation and stretch responses of ankle musculature (Nardone et al., 1990; Allum et al., 1990; 1992; 1993; Schieppati et al., 1995). Allum et al. (1989) observed postural responses to ankle dorsi-flexion rotations of the support surface which were consistent with a two segment 'stiffening strategy' whereas a backward translation elicited a multiple segment or 'multi-link' strategy of movement. Observation of stretch related information at the level of the neck (Keshner et al., 1988) and in intrinsic muscles of the foot (Schieppati et al., 1995) prior to stretch responses in triceps surae muscles provided additional evidence that stretch reflexes and balance corrections consist of distinct neurophysiological entities in contrast to the long-standing hypothesis of an ascending pattern of muscle activation triggered by stretch-related proprioceptive input from the ankle joint muscles (Nashner, 1977; Horak and Nashner, 1986). To provide further support for the concept that balance corrections can be triggered independent of ankle stretch input, Allum et al. (1995; 1998) utilized combinations of backward translations and plantarflexion rotations of the support surface. This combined perturbation provided a unique condition by nulling any proprioceptive input from ankle joint or lower-leg muscle stretch receptors. With this combination, Allum and colleagues were able to demonstrate that properly timed balance corrections in leg and trunk muscles of normal and vestibular loss subjects were still present in the absence of ankle input.

An alternative method commonly employed in searching for the origin of triggered postural responses has been to study the automatic responses of patient populations with 'lesions' of the hypothesized triggering sensory system. Observing changes associated with the absence of information from lower-leg proprioceptive systems may help to reveal the relative contributions of proprioceptive information to the triggering and modulation of automatic postural responses that comprise balance corrections. For example, Inglis et al. (1994) found significant differences in the onset latencies of postural responses between patients with selective proprioceptive loss at the level of the ankle and normal controls. However, recent

investigations by Bloem and colleagues (Allum et al. 1998, Bloem et al. 1999), which employed a more vigorous selection procedure to eliminate any confounding effects of muscle strength frequently accompanying selective proprioceptive loss, yielded no evidence of significant differences in onset latencies of automatic postural responses to pure rotation and combined translation/plantarflexion rotation perturbations with nulled ankle inputs. These findings, when considered with earlier evidence from the work of Keshner et al. (1988), Allum et al. (1995; 1998) and Schieppati et al. (1995), provide a substantial body of evidence supporting the position that lower leg stretch reflexes and subsequent balance corrections involve different neurophysiological mechanisms.

Additional directional characteristics of balance corrections in the pitch plane have been discovered. Following the observation of correct postural responses to changes in the forward and backward direction of platform perturbations within a single trial in cats, Rushmer et al. (1983) concluded that these triggered postural responses were sensitive to the direction of the perturbation. In addition, both the amount of information available prior to the perturbation and previous experience with similar perturbations in the pitch plane have been shown to significantly influence the magnitude of the automatic postural response (Horak et al., 1989; Beckley et al., 1991).

Although significant amounts of information regarding the characteristics of balance correcting responses have resulted from both altered perturbation and lesional studies, the general applicability of these findings to falls in several directions should be seriously questioned. In all of the aforementioned studies, regardless of the type of perturbation (i.e. translational, rotational or combination of both), each has limited the direction of the perturbation to a single plane, specifically the sagittal or pitch plane. However, joint receptors (Rothwell, 1994) and vestibular receptors (Tomko et al., 1981) provide directional information in planes other than the pitch plane. Furthermore, the directions of maximum isometric stabilizing activity in neck (Keshner et al., 1988), trunk (Lavender et al., 1994) and elbow muscles (Buchanan et al., 1986) lie in multiple planes. As perturbations to equilibrium can be expected, under normal conditions, to occur in directions other than that of a pure pitch orientation it seems essential to characterise the properties of the postural control system using perturbations in multiple

directions. Aruin and Latash (1995) have demonstrated that anticipatory postural reactions in postural leg muscles are modulated to the direction of fast voluntary arm movements. However, to date only a limited amount of research has been devoted to examining the sensitivity of postural leg and trunk muscle responses to unexpected perturbations in multiple directions. Some studies involving multi-direction platform displacements have been performed using human (Maki et al. 1994; Moore et al., 1988; Henry et al., 1998), and cat paradigms (Rushmer et al., 1988; Macpherson, 1988a;1988b; 1994). Experiments from both paradigms have produced evidence supporting the sensitivity of both muscular and biomechanical postural responses to perturbation direction. However, the findings of these multi-directional studies must be weighed against the limitations of the experimental design, including non-randomized and restricted directions and the choice of a single measurement interval overlapping both stretch-reflex and balance-correcting activity. Irrespective of any limitations in protocol, support surface translation studies may also lack the capability to independently observe directionally specific responses in stretch reflex and automatic balance correcting responses in the lower leg muscles unless the stretch reflex is first nulled out by simultaneous plantar-flexion of the support surface (Nashner et al. 1982, Allum and Honegger 1998). Support-surface translation, in contrast to rotation, has generally a slower rise time because of the large mass that must be moved. The slower rise time causes stretch reflex and balance correcting activity to coalese in lower leg muscles (Allum et al. 1993). However, in order to understand how directional proprioceptive information is used to trigger and/or modulate postural responses, the ability to distinguish between stretch reflex and triggered automatic balance-correcting responses seems vital.

The purpose of the present study was to examine the postural responses of normal healthy adults to support surface rotations in sixteen different, randomly presented, pitch and roll combinations. It was hypothesized that biomechanical and muscular activity of lower leg and trunk muscles with respect to stretch reflex and subsequent balance correcting responses would be sensitive to the direction of the perturbation. It was hoped that observed intramuscular differences in the directional sensitivity of response magnitudes would shed further light onto the relative contributions from the hip, knee and ankle proprioceptive inputs in triggering balance corrections and increase the understanding of how the CNS utilizes vestibular and

proprioceptive information to code and modulate responses to the direction of a postural perturbation.

METHODS

This study examined the effect of multi-direction rotations of the support surface on muscular and biomechanical responses in normal healthy young adults. Seventeen participants (8 male, 9 female; mean age=23 sd 2.6 yrs; height=1.73 sd 0.08 m; weight=69.5 sd 12.2 kg) volunteered for the study and gave witnessed prior informed consent to participate in the experiment after observing several multi-directional movements of the support surface. All subjects were free from any neurological or previous orthopaedic injuries as verified by self report and possessed normal balance function as verified by Unterberger and Romberg stance tests.

Subjects were positioned on the force-measuring platform with their feet lightly strapped across the instep to the support surface. Backward movement of the heel was prevented by an adjustable heel bar. This procedure ensured that the lateral malleoli were aligned with the platform's pitch axis of rotation for every trial and was identical to that used in our previous studies (Allum and Pfaltz 1985, Allum et al. 1993, 1994, 1995, 1996, Allum and Honegger 1998). The subjects were asked to assume their normal standing posture, with straight knees and arms hanging comfortably at their sides. Force-plate reaction forces were then reset to zero to establish a reference value for the individual's 'preferred stance' position.

Stimulus Parameters

The dual axis rotating force-platform was capable of delivering unexpected rotations through multiple directions in the pitch and roll planes. Platform rotations had a constant amplitude of 7.5 deg and angular velocity of 50 deg/s. A clockwise increasing notation, as viewed from above, was used to specify rotation direction. The 0 deg rotation direction represented a pure 'toes down' tilt of the platform, conversely, 180 deg direction represented a pure 'toes up' rotation. Pure roll movements were assigned angles of 90 deg to the right and 270 deg to the left. Combinations of pitch and roll rotations were used to denote 12 other directions, each separated by 22.5 deg. In order to record electromyographic activity, ankle torque and trunk

angular velocity two sequences of 8 directions (0, 45, 90, 135, 180, 225, 270, 315) and (22.5, 67.5, 112.5, 157.5, 202.5, 247.5, 292.5, 337.5) were presented on two separate days (on average, 3 days apart) under eyes open and eyes closed conditions on both occassions to 14 subjects (7 female and 7 male). Each series consisted of 44 randomly presented rotational stimuli. Responses to the very first stimulus in each series were ignored in the data analysis to reduce the effects of adaptation (Nashner et al., 1982; Keshner et al., 1987) entering the data, leaving 5-6 sets of responses per direction and condition in the subject average. The order of presentation of the two directional sequences was counterbalanced between subjects to minimize any ordering effects. Order of visual manipulation (eyes open vs. eyes closed) was not randomized to allow future comparisons between normal and subsequently collected patient population data. Although it is acknowledged that the non-random presentation of visual conditions may have invited confounding effects due to order (Keshner et al., 1987), the balance tasks had to be performed in a sequence of increasing difficulty to ensure the safety of patients in future studies. A five to ten minute rest period was provided between eyes open and eyes closed conditions to try to minimize any confounding effects due to order and/or fatigue. Biomechanical variables, and repeated measures of ankle torque and trunk angular velocity, were recorded in a separate session on average 19 days after the EMG recordings. For this session only the sequence of directions (0,45,90,135,180,225,270,315) was presented under eyes-open conditions to 9 of the subjects (5 females and 4 males) who had had EMG recordings and 3 female subjects without EMG recordings to have roughly equal numbers of subjects for EMG and biomechanical recordings.

Each perturbation was preceded by a random 5-20 s delay. During this delay period subjects were required to maintain anterior/posterior (A-P) ankle torque within a range of +/- 1 Nm from the 'preferred stance' reference value using on-line visual feedback from an oscilloscope placed at eye level approximately 1 m away from the subject. During the eyes closed condition two distinct auditory tones were substituted for visual feedback to monitor variations in A-P ankle torques prior to the stimulus onset. The 5-20 s interstimulus delay was initiated automatically once the platform had returned to its original pre-stimulus position and the subject regained his preferred vertical position.

In response to each rotational perturbation, the subject was instructed to recover their balance as quickly as possible. Handrails were located on the lateral borders of the platform apparatus in case of loss of balance and a spotter was present in close proximity to lend support in case of a fall. No stimuli caused the subject to touch the handrail or to need assistance by the spotter.

Biomechanical and EMG Recordings

All biomechanical and electromyographic (EMG) recordings were initiated 100 ms prior to the onset of the perturbation and had a sampling duration of 1 s. Support surface reaction forces were measured from two independent force-plates mounted on the moveable platform. Vertical forces were measured by strain gauges located under the corners of each plate. From these forces A-P and medial lateral (M-L) ankle torques were calculated (Allum and Honegger, 1998). Trunk angular velocity in the pitch and roll planes were collected using Watson Industries transducers (± 300 deg/s range) mounted to a metal plate which hung from shoulder straps at a level on the chest equivalent to the position of the sternum. Pitch and roll angles of the left lower leg were recorded with respect to vertical using a goniometer system consisting of two potentiometers oriented at 90 deg to one another and attached to a lightweight metal rod strapped to the lower leg just below the knee at 4 cm below the lateral condyle of the tibia. The left upper-leg pitch and roll angular velocities were measured with Watson Industries transducers (± 100 deg/s range, 0 to 50 Hz bandwidth). The transducers were mounted perpendicular to one another on a 20 cm long metal plate molded to the curvature of the upper leg. The plate was held firmly attached to the upper leg by means of an elasticated bandage. Left knee pitch and roll angular velocities were computed off-line from the difference of the upper- and lower-leg angular velocities after differentiating the lower leg angle digitally. All biomechanical data was sampled at 500 Hz and digitally low-pass filtered off-line at 25 Hz using a zero phase-shift 10th-order Butterworth filter.

Surface EMG electrodes were placed bilaterally, approximately 3 cm apart, along the muscle bellies of tibialis anterior, soleus, lateral quadricep (vastus lateralis) and paraspinal muscles. EMG amplifier gains were kept constant and pairs of electrodes and lead lengths assigned to individual muscles were not changed between subjects. EMG recordings were sampled at 1

KHz, band-pass filtered between 60-600 Hz, full wave rectified, and low pass filtered at 100 Hz as recommended by Gottlieb and Agarwal (1979).

Data Analysis

Following analog to digital conversion of the data, offline analysis was initiated by defining the zero latency point and averaging subject EMG and biomechanical signals for each perturbation direction (5-6 stimuli per direction). Zero latency was defined as the first inflexion of ankle rotation velocity and did not vary with direction or subject. For each trial background EMG activity of each muscle recorded 100 ms prior to stimulus onset was averaged and subtracted from the EMG signal before response areas for the trial were calculated. EMG areas were calculated using trapezoid integration within pre-determined time intervals associated with previously identified stretch (40-100, 80-120 ms from stimulus onset), balance correcting (120-220 ms), secondary balance correcting (240-340 ms), and stabilizing reaction (350-700) responses (Allum et al., 1993; 1994; 1996). Fixed intervals were used rather than alternative terminologies for averaging intervals such as medium (ML) and long latencies (LL) response intervals (Beckley et al. 1991, Diener et al. 1983, 1984) for two reasons. Often it is difficult to define separate ML and LL periods of activity in a muscle response (Allum et al. 1993) and secondly responses after early stretch reflexes were often active at several body segments simultaneously (see figures 4 and 6). All biomechanical and muscular profiles were averaged across each direction and subject averages were pooled to produce population average for each direction. Response latencies were measured on individual trials once it had been determined visually that a consistent activity pattern occurred for all responses from the subject for one direction. A latency was set if the activity pattern diverged for more than 40 ms at least one standard deviation away from the mean EMG activity 100 ms prior to stimulus onset.

RESULTS

Rotation of the support surface induced direction specific displacement of body segments during the first 150 ms from onset of the stimulus (Fig. 1). In general, the subsequent balance-corrections enhanced initial movements of the trunk (see Figs. 2, 3 and 5). Balance corrections consisted of multi-segmental, automatic, muscle activity with onsets in the range of 100 to 150 ms from stimulus onset (see Figs. 4 and 6). Roll displacements of the trunk occurred earlier

and were corrected earlier than pitch displacements (see Figs. 2, 3 and 5), even though balance correcting muscle activity did not appear to change in onset between pitch and roll. However, the depth of modulation of muscle activity changed considerably between pitch and roll (see Figs. 4 and 6) being stronger and more asymmetric in the trunk muscles, and weaker in the leg muscles for roll (see Figs. 7 to 10). The presence of stretch reflex and/or unloading activity after 39, 44 and 73 ms in paraspinal, soleus and tibialis anterior, respectively, was another significant muscle activity pattern change with direction (see Figs. 4 and 6). Our working hypothesis is that these phases of early stimulus induced and the later occurring, balance-correcting, muscle activity result from different neurophysiological phenomena underlying the postural response to the balance perturbations. Therefore our experimental observations have been presented within the concept of a sequence of such balance-related intervals which together, constitute a smoothly executed movement strategy.

Stimulus Induced Responses:

Pitch plane rotations

During the 180 deg (toes up) rotational stimuli, the body was forced into movement as a two segment system (Fig. 1). This two-link motion dominated the subsequent balance correction (see Fig. 2), because the upper and lower legs moved as essentially one link. As observed in Fig. 3, within 50 ms following the onset of platform rotation the ankle was passively dorsi-flexed to reach a peak velocity of 60 deg/s (thinnest set of lines in ankle pitch velocity traces). Corresponding stretch reflexes in soleus muscles were observed (Fig. 4), with an onset latency of 44 ms (sd 3.7 ms). Stimulus onset marked the beginning of a passive backward rotation of the lower limb segment coupled with forward rotation of the upper leg thereby forcing the knee into hyperextension with a maximum knee angular-velocity of 20 deg/s. Forward flexion of the other major link, the trunk segment, was first observed at 50-60 ms which caused a stretch reflex in paraspinals with onset latency of 68 ms (sd 15 ms). Initial movements of the head were dominated by early upward accelerations at 15 ms followed by smaller horizontal accelerations in the backward direction. Upward accelerations of the head reached a maximum of 0.45 m/s^2 at a time to peak of 35 ms (Fig. 3). For the purposes of movement strategy conceptualisation, motion of the head as a separate link is not emphasised here (see Allum et al. 1997). These results did not differ from those obtained in our pitch rotation studies (see

figure 2, Allum and Honegger 1998) for which the feet were also strapped to the support surface.

As illustrated in Fig. 1, during the 0 deg (toes down) stimulus the body responded as a three-link system and again this mode of movement comprised the subsequent balance correction (see Figs. 1, 2 and 5). The ankles were initially pulled into plantar-flexion by platform rotation with a maximum angular velocity of 70 deg/s (Fig. 5). Stretched tibialis anterior muscles (see Fig. 6) demonstrated reflex responses with latencies of 73 ms (sd 12 ms). Forward rotation of the lower leg segment began at 100 ms. However, backward rotation of the upper leg caused flexion of the knee joint as early as 15 ms with increasing velocity until a maximum of 60 deg/s was reached at 200 ms (Fig. 5). Corresponding to this period of knee flexion, stretch reflexes were observed in quadricep muscles with latencies of 85 ms. Backward rotation of the trunk segment was first observed in angular velocity traces, unloading the paraspinal muscles as evidenced by a decrease in stimulus induced paraspinal EMG activity below pre-stimulus background levels clearly seen at 70 ms (see, for example, the thin traces in left paraspinals in Fig. 6). The onset latency of the unloading response was on average 45 ms (sd 12 ms). Trunk movement was preceded by early downward vertical linear accelerations of the head peaking at 30 ms and smaller anterior head accelerations at 50 ms (see Figs. 1 and 5). Maximum vertical accelerations of the head reached 0.6 m/s^2.

Roll plane rotations

For pure roll conditions to the right (with mirrored responses for left roll stimuli), the lower and upper leg segments initially rotated in the same direction as the platform rotation (see Figs. 1 and 3). Interestingly, stretch reflexes were observed in the left soleus muscle with an onset latency of ca 50 ms, similar to that observed in a pure toes up rotation but such early stretch reflexes were not observed in the right soleus (Fig. 4). The trunk segment demonstrated consistent early roll velocities at 20 ms in the direction opposite to that of the platform rotation (see Figs. 1 and 3). Between 100-150 ms the trunk demonstrated average roll velocities of 12 deg/s to the left (Figs. 1 and 3). Small stretch reflexes in the right paraspinal for right platform, left trunk roll, were observed at ca 60 ms while an unloading reflex, characterized by decreased EMG activity below background levels, was observed in left paraspinals with

latencies as early as 30 ms (Fig. 4). Very large head roll angular accelerations were recorded in the same direction as trunk rotation (to the left in response to a pure right roll stimuli) beginning at 40 ms and reaching peak accelerations of 200 deg/s^2 within 80 ms (Fig. 3). Notice however, that head vertical accelerations were of a small, almost negligible, amplitude over the first 150 ms (see Fig. 3).

Off-pitch, off-roll rotations

Stimulus induced movements were unique for each off-pitch and off-roll perturbation direction containing both pitch and roll characteristics, however, certain differences in trunk velocities and head accelerations were observed with respect to knee and ankle velocities. Generally, clear direction specific magnitude changes were seen in ankle and knee velocities that were not so well defined in trunk velocities and head accelerations. Generally roll of the support surface induced little pitch of the ankle and knee and vice versa for pitch displacements. Between these two extremes a step-wise progression in the amount of roll and pitch was noted, consistent with the direction of support-surface motion. This information on the direction of roll was not encoded into soleus stretch reflexes (see Fig. 8) nor clearly into tibialis anterior stretch reflexes (see Fig. 7) because the sensitivities of these reflexes were aligned along the pitch axis. In contrast, trunk velocities showed a strong pitch component for roll rotations of the support surface, but no trunk roll velocity was observed following pitch rotations of the support surface. Furthermore, the change in trunk velocity vector between the 67.5 and 112.5 directions of roll was encoded more in trunk pitch velocity (compare trunk roll and pitch velocity traces in Figs. 3 and 5). Perturbations with a right roll component (between 22.5 and 153 deg) induced trunk roll velocities to the left with latencies of 20-30 ms (see Figs. 3 and 6). Pitch rotations of the trunk followed with longer latencies of 60 ms and slower accelerations, pitching backward during toes down combinations (for directions in the range 270 to 90 deg), and forward in response to toes up combinations except 112.5 and 247.5 deg for which no early pitch occurred (see Figs. 3 and 5). The direction of maximum sensitivity of paraspinal stretch reflexes were observed for backward directions 45 deg from pure-pitch, with left and right paraspinal muscles having opposite directions (see upper left polar plots in Fig. 9). So for that matter were those of the unloading reflexes in paraspinals (not illustrated) albeit oriented 180 deg to the stretch reflexes. The earliest population average stretch reflex onsets in the right

paraspinals were observed for the 135 deg direction (63.4 ms, sd 12 ms) and the earliest average unloading response for the 315 deg direction (39 ms, sd 10 ms). Considering the mean onset for combined stretch reflexes in soleus and tibialis anterior muscles as one possible trigger source and the mean onset for paraspinal stretch/unloading interaction as another, comparison with a one-way analysis of variance revealed significantly lower onsets for the mean trunk compared to mean leg reflexes ($p<0.05$). Thus, despite the fact that trunk pitch velocities are observed even during roll perturbations, and limited variation in trunk roll velocities to near pure-roll perturbations, the interaction between early stretch and unloading paraspinal reflexes on opposite sides of the body provided discriminatory information regarding the direction and onset of trunk movements. Consistent with the observation of uniform trunk roll profiles for different directions, but different trunk pitch profiles, similar amplitudes of head roll angular acceleration profiles were observed for 135 and 90 deg, in contrast to the large change in head vertical linear acceleration profiles (see upper sets of traces in Fig. 3). Thus the direction of support surface rotation may also be accurately encoded from the combined head angular- and linear-acceleration profiles.

Balance Correcting Responses

Balance corrections were characterised biomechanically by a reversal from stimulus-induced responses in both A-P and M-L ankle torque records and by a second phase of body segment (lower-leg, upper-leg, trunk) velocity commencing at approximately 160 ms (Figs. 3 and 5). Ankle and knee joint pitch velocities rose to a second clearly defined peak around 240 ms for all perturbation directions (see Figs. 3 and 5). This phase in angular joint velocity occurred as a result of movement of the upper leg in the opposite direction to that induced by the stimulus (compare leg velocities in Figs. 1 and 2). The trunk, in contrast, generally continued to pitch forward or changed to pitching forward except for toe-down stimulus combinations between 315 and 45 degs. (compare upper and lower parts of Fig. 12). Roll velocities in the legs during balance corrections were of more limited amplitude than those during pitch movements presumably because of the limited joint motion possible in the roll direction. Ankle and knee joint roll velocities during balance corrections were, however always in the opposite direction to those induced by the stimulus and small compared to pitch velocities (Fig. 3). Trunk roll velocities reversed direction only after 220 ms (see Figs. 3 and 12). Thus, trunk motion is

fundamentally different during balance corrections from motion of the leg for all perturbation directions in one major aspect. The amplitudes of roll trunk velocities were similar to those of pitch. Due to this difference it might be expected that muscle response amplitudes of automatic balance corrections are highly directionally sensitive and differ in directional sensitivity between trunk and leg muscles. As shown in Figs. 4 and 6, amplitudes of muscle responses rather than muscle response onsets underlie these differences in trunk and leg movements to different perturbations.

Pitch plane rotations

Figs. 4 and 7 show that the largest responses in tibialis muscles over all directions were obtained between 120 and 220 ms when the support surface was tilted toes-up. Quadriceps also produced the largest burst of activity over this time period for the same direction (see Fig. 10). At the same time soleus showed a small amount of balance correcting activity (see Figs. 4 and 8) for this direction. Presumably this coactivated activity in the leg muscles is largely responsible for bringing the lower-legs forward and braking the rearward motion of the upper legs depicted schematically in Fig. 2. Interestingly, although the largest angular velocity of the trunk was observed for 180 deg pitch rotations of the support surface (the average velocity of the trunk between 240 and 300 ms equalled 19 deg/s in the pitch direction, see Fig. 2), the paraspinal activity was smaller than that obtained for roll perturbations that caused trunk motion towards the paraspinals recording side (e.g. left paraspinals unloaded by right roll support-surface motion).

In response to toes down perturbations, soleus produced the largest balance correcting responses of all muscles we recorded from. As with toe-up perturbations in tibialis anterior, responses in soleus were similar between eyes-open and -closed conditions, although responses were larger on average for eyes closed. Presumably, soleus activity was recruited to oppose the continuing forward motion of the lower leg (see Figs. 1 and 2). After motion of the support surface ceased at 150 ms, this forward motion of the lower leg led to a dorsi-flexion of the ankle joint which peaked with a velocity of 35 deg/s at 200 ms (Fig. 5). Quadriceps activity during the balance correcting period acted to decelerate backward motion of the upper leg (see Fig. 2). Thereby knee flexion peaked at 200 ms and came to rest at ca. 350 ms. Minimal

balance correcting activity for tibialis anterior and paraspinal muscles was observed during toe down perturbations (Figs. 6 and 9). We assume that muscles such as the abdominals, from which we did not record, were responsible for the double-peaked profile of trunk pitch angular velocity seen in response to toe-down support-surface rotations (see Fig. 5).

Roll plane rotations

The automatic balance-correcting responses in left and right muscles during the pure roll conditions were not symmetrical. The asymmetries were similar under eyes open and eyes closed test conditions. An asymmetrical balance-correcting response is unavoidable given that the trunk tilts in the direction opposite the support-surface movement and the uphill leg is flexed at the knee and the downhill leg is extended into a knee-locked position (see Fig. 2). Thus, in response to right down support-surface movements shown in Fig. 6, balance correcting activity in right tibialis anterior, quadriceps and soleus were involved in stabilizing the right ankle and knee joint to maintain a straightened position and resist the initial rightward rotation of the legs. The larger tibialis anterior and smaller soleus activity in the left leg (see lower left Figs. 6 and 8) preceded flexion of the knee and ankle which commenced at approximately 160 ms (see Fig. 3). Simultaneously, the knee was pulled slightly to the left, reflected by increased knee roll velocity which peaked at 190 ms. Following the initial stretch reflex of the right and unloading reflex of the left paraspinal muscles, large balance-correcting responses in the left paraspinals were observed accompanied by a weak contraction in the right paraspinal muscles (Fig. 6). This activity presumably was required to counteract ensuing trunk roll in the rightward direction. Average right roll velocities of the trunk to right down support-surface movements were thereby limited 2.4 deg/s between the measurement period of 240-340 ms (Figs. 2, 3 and 5).

Off-pitch, off-roll rotations

As roll and pitch perturbations were combined, the balance correcting responses associated with pure pitch and roll stimuli were superimposed to create unique directionally-specific postural responses. Balance correcting responses, like stretch reflexes described above, and subsequent stabilizing reactions described below, were influenced by perturbation direction. Tibialis anterior demonstrated balance responses through all backward directions with the

majority of activity observed in directions greater that 135 and less than 225 deg and maximal responses oriented just off 180 deg at 177 deg and 186 deg for left and right muscles, respectively (Figs. 4, 6 and 7). Balance correcting responses in soleus muscles were observed in all directions with maximum activity occurring in directions between 0 and 68 deg for the right and between 293 and 0 for the left muscle (Figs. 4, 6 and 8). Smaller amplitude soleus activity was observed in directions between 90-270 deg corresponding to coactivation with tibialis anterior activity. Minimum activity was recorded for pure roll directions. These two zones of soleus activity caused maximal activity vectors for soleus to be oriented slightly off the pitch axis at 332 and 35 deg for the left and right leg, respectively (Fig. 8). Quadriceps followed the trend exhibited by tibialis anterior with maximum activity vectors aligned along the toe-up (180 deg) direction, except that some activation was observed for roll and the toe-down directions. Quadriceps demonstrated larger mean values for the left than the right leg. Though this difference is unlikely to approach significance because large standard errors were associated with these means (see Fig. 10, lower right) the difference in means is nonetheless surprising given the precautions we took to avoid a left/right bias. This was the only muscle for which such differences were observed and could be associated with the fact that most subjects were right-footed. As observed in Fig. 9 from both the directions of maximum activity vectors and the range of activity, paraspinals responded best to roll backwards, consistent with the combined roll and pitch effect of the stimulus on the trunk (see Figs. 3 and 12). Maximal activity vectors for paraspinals were directed towards 142 and 225 deg for the left and right muscles with activation ranges between 90 and 225 deg and between 135 deg and 270 deg, respectively.

For all leg muscles we recorded from (tibialis anterior, quadriceps and soleus) maximum activity vectors for balance correcting responses were oriented along the pitch axis. Even for the lateral quadriceps muscle this finding is consistent with the action of leg muscles, being restricted by ankle and knee joint motion to the pitch plane. The stretch responses in leg muscles were oriented approx. 180 deg from the balance-correcting response in same muscle (compare polar plots in the left half of 7, 8 and 10). Paraspinal muscles, however, demonstrated maximum stretch reflexes activity in directions oriented 90 deg from the same muscle's maximum balance correcting response (see left half of Fig. 9). This evidence,

suggesting an underlying neurophysiological difference between the mechanisms responsible for eliciting stretch and subsequent balance correcting responses, can also be observed in Figs. 4 and 6. Directions which elicited strong stretch reflexes in a particular muscle were followed by proportionally smaller balance correcting responses. In addition, the inhibition or unloading reflex observed in left paraspinals during trunk motion caused by right roll perturbations, and likewise for right paraspinals during left roll perturbations, were succeeded by very prominent balance correcting responses. Maximum activity for the unloading reflexes were observed at 57 and 310 deg, for the left and right trunk muscles respectively. Maximum unloading reflex activity was oriented 90 deg from the maximal balance correcting response and 180 deg to maximal stretch reflexes in the same muscle.Thus in the trunk muscles the primary direction of the stretch reflex is not opposite that of the balance-correcting response, but phase shifted.

Joint torques

Our findings with respect to the A-P directionality of leg muscle maximum activity vectors was replicated in ankle muscle torques. This was done by examining the directionality of torque muscle responses during the period 160 to 260 ms, that is, in the period influenced by balance correcting responses acting over the time frame of 120 to 220 ms. The upper part of Fig. 11 shows the amplitudes of A-P and M-L ankle torque change calculated from the strain gauge measurements over 160 to 260 ms. Notice that the difference in the scales for the A-P and M-L torques in Fig. 11 signifies that the ankle torque is dominated by the A-P torque. As with the lower-leg muscle responses, eyes closed A-P torques were slightly larger than those obtained under eyes-open test conditions. The centre plot of the three polar plots in the upper part of Fig. 11 shows the direction of the movement forces for the right ankle torque vector. That is, the direction the body would move (viewed from above) if it could be modelled as an inverted pendulum rotating at the ankle joints. These directions are almost exclusively aligned along the slightly off-pitch direction of the tibialis anterior and soleus maximum activity vectors for the right foot for all perturbation directions. The alignment of the torque vectors was not different for eyes open and closed conditions (only eyes-closed torque directions are shown in Fig. 11).

Likewise our findings of a different directional sensitivity for trunk muscles could be confirmed by examining the directionality of average trunk velocity responses over the period 160-220 ms. This time frame encompasses the peak pitch velocity of the trunk backwards for all forward and roll support-surface rotations (see Fig. 5), the peak roll velocity of the trunk (see Figs. 3 and 5), and would presumably reflect the action of ankle and hip torques between 160 and 260 ms. The upper part of Fig. 12 shows how the amplitude of the average pitch and roll trunk velocity over 160-220 ms varies with perturbation direction. The centre plot of the three upper polar plots indicates that the direction of trunk motion is highly directionally tuned and symmetrical. That is, the trunk motion is always opposite the direction of support-surface perturbation. Differences between this directional sensitivity of trunk motion under eyes-open and eyes-closed conditions were not observed.

Secondary Balance Correcting Responses

A somewhat unexpected finding was an extension of the burst of balance-correcting activity for backwards perturbations compared to forwards and/or roll perturbations. It is possible that this additional activity is due to the greater instability of the body during backward compared to forward falls. This extension of activity lead to distinct burst of muscle activity at a latency of 220-240 ms in tibialis anterior, specifically for toe up (180 deg) directions (see Fig. 4). The mean latency of this burst has been shown to be 226 ms in previous studies (Allum and Pfaltz 1985, Figure 1). Furthermore the secondary balance correction in tibialis anterior is larger than normal in vestibular-loss subjects, whereas the primary balance correction is smaller (Allum and Honegger 1998, Figures 2, 6 and 7). These population differences in the pitch plane responses and observations of corresponding changes in time-parsed (160-260, 280-380 ms) ankle torque responses in all planes (Carpenter et al. 1999) provides a rationale for considering the muscle synergy of the secondary balance corrections as a distinct response. This secondary balance correcting activity following the decline of the initial automatic balance correcting response was generally absent for roll perturbations and much smaller for forwards perturbations. A comparison of the amplitudes of primary and secondary balance corrections in Figs. 7 and 8, shows that soleus secondary balance corrections were some 40 % of the main balance-correcting activity whereas that of tibialis anterior was of equal amplitude. Activity was also apparent in soleus and paraspinal muscles for toes-down rotations during the period

we designated as secondary balance correcting activity (240-340 ms), but did not have the same burst-like profile as seen with the toe-up rotation (compare Figs. 4 and 6). As observed in Figs. 7 and 10, secondary balance correcting activity in both tibialis anterior and quadriceps were oriented in the same directions as earlier automatic balance correcting responses (between 113 and 248 degrees) and share similar maximum activity directions at 180 deg. As shown in Fig. 9, paraspinal activity during this period is observed over a diverse range of directions, however the direction of maximum activity vector remained unchanged.

A-P directionality was also a feature of ankle torques associated with secondary balance corrections. The lower part of Fig. 11 shows the magnitude of the torque change generated for the right foot over the period 280-380 ms as the A-P torque approaches a minimum (Fig. 3). The torque change during this period was approximately one third of that during the previous balance correcting period (160-260 ms, see upper part of Fig. 11). The features noted for the earlier period were similar: resultant torque vectors were orientated just off the pitch axis, the change over from forward to backwards directed torque occured slightly backward of right roll and slightly forward of left roll for the right foot, and lastly eyes closed magnitudes were larger than those for eyes open.

Average trunk angular velocities computed during the period of the secondary balance correction displayed a strong pitch plane asymmetry and oppositely directed roll components to those observed during the main balance correction. The lower part of Fig. 12 illustrates both the amplitudes of the trunk pitch and roll velocities over the period 240-300 ms, as well as the direction of trunk motion. This measurement period includes the peak pitch velocity to toe-up (0 deg) displacement of the support surface, as well as the peak roll velocity opposite to that initially induced by the stimulus. It is apparent from the directional polar plot that the underlying torque profile at the trunk must have a stronger roll component than that at the ankle joint.

Stabilizing Reactions

Muscle activity between 350-500 ms reflects stabilizing reactions which fall well within the bounds of possible voluntary control (Allum et al. 1996). This tonic low-level activity is

required to maintain the new posture dictated by the biomechanical constraints of the new platform orientation and to control residual velocities of the trunk which are particularly prevalent in the pitch direction. Tibialis anterior and quadriceps had stabilizing activity which focused around 180 deg, reinforcing the automatic and secondary balance correcting activity (Figs. 7 and 10 lower right). Soleus activity was oriented to toes-down directions lateral of the pitch plane with maximum activity directed between 23 and 45 deg for the right side, similar to that of the automatic balance correcting response (compare Fig. 8 lower-left and lower-right panels). As expected from ongoing trunk pitch, paraspinals activity is still relatively high during the stabilizing period with activity ranges and maximum activity vectors directed to 225 and 135 deg for right and left muscles (Fig. 9 lower right).

DISCUSSION

Until recently, almost all hypotheses about the sensory mechanisms underlying human reactive postural control have been based on observations derived from uni-directional perturbations to equilibrium, specifically in the pitch plane (Nashner and McCollum, 1985; Dietz et al., 1992; Forssberg and Hirschfeld, 1994). These theories have mainly converged to describe human postural reactions as highly stereotyped patterns of muscle activity which are activated in an ascending distal-to-proximal order (Nashner and Horak, 1986) in leg and trunk muscles after being triggered by ankle inputs (Horak et al. 1990, Inglis et al. 1994). Leg-muscle balance-correcting activity, as we have demonstrated here, is almost exclusively pitch-plane directed. However, from the standpoint of external validity it is unreasonable to assume that under conditions of daily living a postural perturbation will be experienced along a purely pitch plane. As our findings demonstrate, the trunk moves readily in the roll plane even if leg movements are restricted by joint motion to the pitch plane.

Rushmer et al. (1983) and Allum et al. (1990) have shown that appropriate balance correcting responses are elicited within a single trial following directional change from forward to backward translations or rotations of the support surface. This finding highlighted the possible sensitivity of balance corrections to the pitch plane characteristics of the perturbation. Out of the pitch plane, COP displacements even for quiet stance in the A-P and M-L directions are controlled through different mechanisms (Winter et al. 1996). A-P displacements are achieved

through dorsi- and plantarflexion ankle torques, whereas M-L corrections are achieved through loading/unloading hip torques. Therefore, it might well be expected that multi-directional perturbations would provide insights on the CNS control of muscle co-ordination between ipsilateral and contralateral postural muscles to achieve appropriate directionally specific ankle, knee and hip torques to prevent a fall. The main purpose of the present study was to increase the understanding of postural control mechanisms through examination of muscular and biomechanical responses to postural perturbations in multiple planes. The focus of our investigation centered specifically upon three distinct characteristics of automatic postural responses. The first issue related to identifying the origin of directionally specific triggering information responsible for the initiation of muscle responses generating appropriate timing patterns for ankle and hip torques. Secondly, to understand how proprioceptive, vestibular and visual information could modulate triggered balance-correcting responses relative to the direction of the perturbations. Thirdly, to gain insights into the action of central pattern generators in executing appropriate ankle and hip torque movement strategies.

Possible Triggering Mechanisms

Moore et al. (1988) were the first to examine how changes in perturbation direction would influence variations in human muscle response patterns. Observation of discrete muscle responses corresponding to specific perturbation directions led to the conclusion that automatic postural responses were not components of a relatively small number of postural synergies (Nashner and McCullum, 1985; Horak and Nashner, 1986) but a complex process in which perturbation direction was a significant variable. Systematic variation of muscle responses to perturbation direction has also been observed in cats responding to multi-directional translations (Rushmer et al., 1988; Macpherson, 1988). However, the findings of these multi-directional studies must be weighed against the limitations of their design. To generate multi-directional perturbations, Moore et al. (1988) employed a uni-planar platform upon which the subject was turned to produce translations in 16 different directions relative to the subject. Therefore, unlike in the present study, the participant was capable of deducing that the upcoming perturbation would be directed in either the positive or negative direction of a given plane, based on their orientation relative to the platform's constant translational plane. Previous studies have shown that prior knowledge of the direction or magnitude of an

upcoming perturbation may influence the pre-stimulus posture of a subject, reflected by anticipatory changes in the mean position of the centre of pressure (COP) in healthy normals (Maki and Whitelaw, 1993) and patient populations (Diener et al., 1991). Anticipatory postural leaning supported through changes in the mean position of COP, significantly influences postural responses to unexpected surface translations (Nashner et al., 1985; Horak and Moore, 1993) and rotations (Diener et al., 1983; Allum and Pfaltz, 1985; Schieppati et al., 1995). The second limitation of the study by Moore et al. (1988) and Henry et al. (1998) was the restriction of muscle recordings to uni-lateral muscles, preventing any comparisons between bilateral muscle activity and asymmetries related to loading/unloading responses to perturbations with lateral components. Henry et al. (1998) attempted to improve upon the shortcomings of Moore et al. (1988) by investigating postural reactions under narrow and wide stance width to unexpected random translations of the support surface through both A-P and M-L planes. Their findings also supported different EMG synergies specific to the biomechanical constraints imposed by direction of the perturbation. Mechanical constraints were found to be imposed at the very initiation of the perturbation in the present study. As has been argued previously (Allum et al., 1993; Allum and Honegger, 1998), the number of links the body is forced into by the perturbation, plays a significant role in determining the subsequent movement strategy underlying the balance correction (see Figs. 1 and 2). We could demonstrate essentially three types of movement strategy and combinations thereof depending on whether the support surface moved toe-down, toe-up or into roll (see Figs. 2, 3 and 5). Interestingly, these movement strategies appeared to be generated by two types of muscle synergistic timing patterns that were simply modulated appropriately for the direction of perturbation. We termed these two types of activation pattern the primary and secondary balance-correcting activity (see Figs. 7-10). Given that two types of timing patterns are generated for all directions, with some predominance of the secondary correction for backwards perturbations, it would seem appropriate that a restricted number of trigger signals with directional information would initiate these pattern types.

The present findings support the notion proposed in previous work (Allum et al., 1995; Allum and Honegger, 1998) that proprioceptors responsive to early stretch and release of paraspinals (and muscle spindles in other muscles acting at the pelvis) provide the primary trigger signal

for balance corrections. We can now provide additional details concerning the directional specificity of this trigger signal. In a follow-up study we obtained even earlier stretch reflex responses in the lower hip muscles, gluteus medius, at 25 msec (Bloem et al 1999). These reflexes are also most active in roll directions. Such early proprioceptive reflexes in hip and pelvic are different on each side of the body providing an immediate indication of the lateral direction of the trunk motion. This indication appears as early as 25 msecs in hip and trunk muscles (see Allum et al 1999 as well as results reported here) presumably because the trunk roll motion, when the balance perturbation has any roll component, occurs earlier than pitch motion (Figs. 3 and 5). That is the trunk is more flexible in the roll direction. Such directionally specific information does not appear to be available in lower-leg muscles. For this reason it is surprising that Henry et al. (1998) still cling in their conclusions to an ankle-input triggered, distal-to-proximal activation of automatic postural responses in leg and trunk muscles, even though they observed early trunk flexor/extensor and tensor-fascia latae activity for perturbations in A-P and M-L directions, respectively, which consistently preceded lower leg muscle activation. Early proximal muscle activity could be triggered by muscle or joint proprioception underlying paraspinal stretch reflex activity with onset latencies equivalent to those observed in soleus muscles (Fig. 5) or by proprioceptive inputs underlying gluteus medius reflex activity at 25 ms (Allum et al 1999). All three sets of reflex responses are within the range of 40-70 ms which has been proposed as the latency limit for feedback information to facilitate an automatic postural response (Macpherson, 1994). Likewise, Moore et al. (1988) observed abdominal bursts of balance correcting activity in response to backward translations prior to activation of gastrocnemius, hamstring or paraspinal activity. Keshner et al. (1988) have reported balance-correcting activity in neck muscles prior to observed activity in ankle musculature, further challenging the theory of an ascending ankle-motion triggered synergy for postural reactions.

A growing body of evidence can be found which contradicts a proprioceptive trigger for postural reactions located at the ankle joint. Bloem et al. (1999) have demonstrated normal onset latencies of balance corrections in patients with selective proprioceptive loss of ankle stretch receptors in response to unexpected dorsiflexion rotations of the support surface. Allum and Honegger (1998) found normal latencies for automatic postural reactions when

plantarflexion rotations were combined with backward translations to null proprioceptive ankle input. Therefore, evidence of early activity in trunk muscles and tensor fascia latae muscles during multiple direction translations (Henry et al., 1998) and in paraspinal muscles in response to multi-directional rotations, as observed in the present study, focus on proprioceptive receptors at the level of the trunk or the hip as the most likely directionally specific triggering centres for automatic postural reactions. Interestingly, observation of trunk proprioceptive reflexes with the range of maximum activity along 45 deg axes highlights the necessity for off-pitch perturbations to accurately investigate trigger signals for balance corrections. The choice of a trigger signal aligned along the planes of the vertical semi-circular canals would presumably offer some simplification in the central processing of appropriate balance corrections using vestibular inputs. Paraspinal stretch and unloading reflexes provide another advantage for central processing because in contrast to leg and neck muscles (Allum and Honegger, 1998; Kanaya et al., 1995) these reflexes appear not to be modified by vestibular loss. That considerable central processing must occur in generating appropriate amplitudes for the bursts of muscle activity in the range of 90-120 ms has been emphasized by several authors (Nashner and Horak, 1986; Dietz, 1996; Forssberg and Hirschfeld, 1994). Concerning the use of trunk proprioceptive reflexes, we can add the information that this processing may well include a vectorial transformation from the planes of maximum activity of stretch reflexes to those of balance corrections. For this reason it seems crucial to consider balance corrections as generated by different neurophysiological processes from those generating stretch reflexes.

Platform rotations will elicit stretch reflexes in lower-leg muscles antagonistic to those used in balance correcting responses and act to further destabilize the body (Nashner, 1976; Diener et al., 1983; 1984). Alternatively, translational perturbations will elicit stretch reflexes and balance correcting responses in the same muscles (Allum et al., 1993). As observed in Fig. 4, the stretch reflex activity of the soleus muscle in response to toe-up perturbations does not subside until at least 80-90 ms following perturbation onset. Likewise, tibialis anterior stretch reflexes in response to toe-down perturbations begin at approximately 80 ms and diminish at 120 ms (Fig. 6). In the trunk muscles, balance- correcting activity to roll stimuli is asymmetric with the larger response preceded by an unloading response and the smaller response by a

stretch reflex (Figs. 4 and 6). Thus the time interval utilized by Moore et al. (1988) and Henry et al. (1999) to examine muscle responses, must have included portions of both stretch reflex and balance correcting responses and lead to an inability to separate stretch reflex and subsequent balance correcting responses from one another. For example, Henry et al. (1999) used an interval which began at 70ms after support surface movement and lasted 200 ms. Under these circumstances it is more difficult to reach conclusions about neurophysiological mechanisms underlying balance corrections. By examining stretch (or unloading) and balance correcting responses separately, as in the present study, an interesting, muscle specific, relationship between stretch and balance correcting responses in the same muscle becomes apparent. As shown in Figs. 7 and 8 maximum activity vectors for stretch reflexes for both tibialis anterior and soleus muscles are oriented approximately 180 deg from those associated with the balance correcting response in the respective muscle. Alternatively, paraspinal muscles demonstrated maximal balance correcting responses in directions approximately 90 degree from initial stretch reflexes (Fig. 7). This trade-off between stretch and subsequent balance correcting amplitude may at first glance suggest that automatic balance corrections may be based upon localized stretch responses in individual muscles. However, previous research has argued against such a local mechanism for postural control. Toe-upward rotation and backward translations of the support surface elicited similar ankle stretch, however, different timing patterns and response modulation of balance correcting activity was required in several muscles to respond to rotation in contrast to activity required during translation (Allum et al., 1993). In addition, observations of early arm movements with latencies similar to corrective activity in lower leg muscles despite a lack of prior stretch in shoulder and elbow muscles led McIlroy and Maki (1995) to conclude that balance correcting responses could not be related to simple localized reflexes.

As opposed to backward roll perturbations which elicit stretch reflex responses in paraspinal muscles at 63 ms, on average (Fig. 4, right paraspinal), forward roll perturbations are associated with unloading responses in the paraspinals, consistent with latencies at 39 ms, on average, (Fig. 6, left paraspinal). Similar to paraspinal stretch responses, maximum activity vectors for unloading responses are oriented 90 degrees to subsequent balance correcting responses. Other researchers have also reported observations of unloading responses in trunk

and neck muscles following unexpected postural perturbations. Hirschfeld and Forssberg (1994) observed postural reactions of lower leg and trunk muscles in seated infants following unexpected rotations and translations. Calculation of mean muscle activity (area) during the first 100 ms following both legs-up rotation and forward translation of the support surface, revealed reduced EMG activity below background levels associated with inhibition of neck, leg and trunk extensor muscles. Likewise, Kayana et al. (1995) observed inhibition of splenius/paraspinal muscles following unexpected pitch movements to seated healthy and labrinthine-defective patients. The inhibitory activity was observed with latencies of 20 ms and preceded a short muscle burst characterized by Kayana et al. as an unloading response. The latency of the inhibitory activity in splenius/paraspinal muscles following seated rotations is similar to the unloading of paraspinals observed in the present study (Fig. 3), and previously reported by Allum et al. (1995). Similar unloading responses have been reported in other skeletal muscles as well, including the hand (Marsden et al.,1983; Traub et al. 1980). In response to translations of the support surface at the 113 deg direction, Macpherson et al. (1988) observed significant decreases in muscle activity of hip, knee and ankle extensors in the unloaded limb of cats. Therefore, the unloading responses we observed in released paraspinal muscles following forward and roll perturbations is not a unique phenomenon. One receptor mechanism through which such muscle unloading may be coded to trigger postural reactions may be through afferent information received from force related Golgi tendon organs of the lower trunk and pelvic muscles. The abundancy of Golgi tendon organs in the muscle-tendon junction of most muscles, (approximately 1:2 ratio to stretch receptors), and low sensitivity threshold of approximately 0.1 gram (Rothwell, 1994) makes these receptors plausible candidates for postural triggering mechanisms. Usually considered to have an inhibitory role, feedback from Golgi tendon afferents via spinal interneurons has demonstrated a capability to modulate its reflex output, producing both inhibitory and excitatory signals during different phases of locomotion (Yang et al., 1990; Pearson, 1995). Based on the observation of compensatory EMG responses to platform translations and rotations of the support surface in vertical and supine orientations, Dietz et al. (1992; 1996) concluded that loading information, detected by Golgi tendon organs in extensor muscles, were responsible for activating postural reflexes. Therefore, based on recent evidence, it is very conceivable that unloading reflexes of

paraspinal and other trunk and hip muscles may provide directionally sensitive triggering information in parallel with muscle stretch information.

Attaching a high importance to early stretch and unloading responses in paraspinals focuses attention on the very early roll responses observed in the trunk segment. As demonstrated in Figs. 4 and 6 trunk roll occurs approximately 20 ms following perturbation onset and almost 40 ms prior to any pitching movements of the trunk. However, we cannot exclude other possible mechanisms through which early trunk movements, particularly in the roll direction, could be detected and integrated as a directionally sensitive triggering signal. Forssberg and Hirschfeld (1994) observed rotations of the pelvis as early as 10 ms following sudden rotations of the support surface beneath seated adults. They concluded that rotation of the pelvis may trigger a primary level of a central pattern generator (CPG) responsible for initial spatial and temporal activation of appropriate postural muscles. Following initial triggering of the postural response, a secondary system is required to modulate the magnitude of the response to correspond with the demands of the perturbation. As movements of the hip joint have been postulated to entrain the CPG for stepping during locomotion, it is possible that a similar CPG could tune postural responses to platform perturbations using similar directionally specific information for angular hip motion (Macpherson, 1988). Besides muscle spindle and Golgi tendon organs detecting stretch and unloading of paraspinal and hip muscles, other receptors at the level of the hip and trunk have been previously hypothesized as possible triggering mechanisms, such as joint receptors of the vertebral column (Gurfinkel et al., 1979; Horstmann and Dietz, 1990; Forssberg and Hirschfeld, 1994) and changes in abdominal pressure (Mittelstaedt, 1992; Do et al., 1988).

Vestibular signals may also be considered as a possible triggering mechanism for postural reactions. Directionally sensitive vertical accelerations were observed for pitch perturbations with latencies of 15 ms (Figs. 4 and 6); these could provide early stimulation to otolith receptors. During roll perturbations, semi-circular canal afferents would transmit angular roll acceleration information with latencies as early as 40 ms to the CNS (Figs. 3 and 5). Forssberg and Hirschfeld (1994) also reported early (10 ms) vertical accelerations of the head in seated adults following up and downward pitch plane rotations. By changing the location of the pitch

axis relative to the hip joint, these authors were able to induce vertical vestibular accelerations in different directions while maintaining constant rotation of the pelvis. Observations of no significant change in the activation patterns of postural muscle responses in leg and trunk muscles provided contradictory evidence against a vestibular trigger. Allum et al. (1994) and Horak et al. (1990) observed normal response latencies in patients with bilateral vestibular loss even under eyes-closed conditions following unexpected toe-up rotations and backward translations, supporting the notion of both a non-vestibular and non-visual origin for a triggering mechanism of postural reactions. Furthermore, the magnitude of postural leg balance correcting responses were significantly influenced by vestibular loss suggesting a modulatory role for the vestibular system.

Direction Specific Modulation of Balance-Correcting Responses

A directionally-specific modulatory role for the vestibular system has been further supported by recent studies in which unexpected translations and rotations were experienced by patients suffering from total body somatosensory loss (Horak et al. 1996, Allum et al. 1999). Although bursts of muscle activity in postural leg and neck muscles were delayed with respect to normal response latencies, the muscle activity was observed to be sensitive to the direction of the perturbation. These findings seem to converge with the two hypotheses we have developed above: the triggering mechanism for automatic balance correcting responses between 120-220 ms is tied to a trunk proprioceptive origin, and the directional sensitivity of postural responses must be modulated by vestibular information.

While there are obvious advantages to the concept that the early vertical linear accelerations and roll angular accelerations of the head may be coded as directionally-specific vestibular information which was used to modulate the magnitude of the balance correcting response, there are also disadvantages. Certainly the finding from the present study provides evidence of early vestibular directionally specific stimulation. As observed in Figs. 4 and 6 (top panels), vertical and angular roll accelerations provide very early (15 and 40 ms) directionally specific stimuli to both the otolith and the vertical semi-circular canal systems. Furthermore, muscle activity associated with automatic balance corrections are modulated with respect to the direction of the perturbation. Although this evidence alone does not confirm a vestibular

modulation of postural control it does collaborate with the findings of previous studies which make similar conclusions (Forssberg and Hirschfeld, 1994). The disadvantage of this concept is the permanent disability that results from loss of peripheral vestibular function. For example, Allum et al. (1994; 1985; 1998) observed similar latency, but changed amplitudes of EMG activity in tibialis anterior, soleus, and paraspinal muscles in response to unexpected rotations in the pitch plane measured from bilateral vestibular loss patients compared to healthy controls.

Ankle and Hip Torque Strategies

The modulation of muscle activity with the direction of perturbation has been shown to markedly influence both ankle torque generation and trunk angular velocities. As shown in Fig. 11, vectorial orientation of the resultant ankle torque was along one of two directions, similar to the „force constraint strategy" suggested by Macpherson (1988a;b). An exception to this strategy was observed at the change-over points from forward to backward-directed ankle torque close to the pure-roll perturbation direction (see Fig.11). In contrast, Henry et al. (1998) reported that orientation of ground reaction forces was dependent upon the direction of translational perturbations. This result may, however, be fortuitous, because these authors only perturbed in two sets of opposite directions, A-P and M-L, i.e. close to roll changeover points we observed. An interesting difference to our two-legged force constraint strategy at the ankle joint and that of Macpherson (1984a,1994) appeared. Cats generated ground reaction forces along one of two directions along the 45 deg plane, with a 90 deg shift of symmetry between paws for all perturbation directions. Only the amplitude of the force was changed as a function of direction as in our study. Differences between the directional orientation of the ankle torques we noted and those of the 'force constraint' strategy demonstrated by cats (Macpherson, 1994) may be explained by differences in the biomechanical constraints inherent to quadrapedal stance compared to bipedal stance. Macpherson et al. (1989) have shown that humans do demonstrate similar postural responses and torque profiles to cats when assuming a quadrapedal posture. These findings emphasize the need to take into account differences in the postural constraints between humans and animal models before attempting to parallel observations between the two. The interesting similarities between the results of the present study and that of Macpherson et al. (1988; 1994) is the consistent orientation of ankle vectors

slightly off-pitch for pure dorsi-flexion and plantarflexion rotations. Possible explanations include the biomechanical constraints associated with anatomical configuration of the foot, line of action of ankle dorsi-flexor and plantarflexor muscles and point of calcaneal insertion which may act independently or in concert to fix ankle torques to an off-pitch plane (Nichols et al., 1993; Bonasera et al., 1996). Certainly the off pitch-axis orientation of ankle torques is not unexpected in our results considering that maximum activity vectors of all leg muscles we recorded from favoured this orientation (see Figs. 7, 8 and 10).

It is an open question whether the ankle force-constraint strategy is also applicable to the hip joint, we suspect that this is not the case for two reasons. Firstly paraspinal, and presumably other trunk muscle maximum activity vectors, are not oriented along the pitch direction. Secondly, the flexibility of the trunk in the roll direction and the large roll velocities we observed in our study necessitates early roll torques prior to those in the pitch direction. Furthermore, we expect that from polar plots of trunk velocities (Fig. 12) and previous modelling studies (Allum and Honegger, 1992), that in contrast to ankle torques, hip torques will be oriented in a highly directional fashion and be triphasic in the roll direction, and biphasic in the pitch direction. All of these factors suggest that hip torques will be multi-directional in contrast to ankle torques. However, to answer the question definitively, two dimensional calculations of hip torques similar to those already performed in the pitch dimension (Allum and Honegger, 1992) are required. We assume that the results of these calculations will add more evidence for an at least three-stage vectorial transformation of sensory signals by the CNS to provide the appropriate modulation of joint torques via muscle activity to correct a postural disturbance from any direction. At one stage the appropriate hip-torque strategy is computed. At a second stage the „constrained" ankle torque is computed with an intermediate calculation of knee torques depending on whether this joint is forced into the locked position by the perturbation and at a third stage the stabilizing neck torques are worked out. This proximal to distal separation of torque strategies, coupled with the differences in sensitivity to roll and pitch of lower-leg, trunk and neck muscle responses indicates that balance corrections must be triggered and organised in other than a distal-proximal pattern.

In summary, we have established that stretch reflex, automatic balance-correcting, and subsequent balance and stabilizing reactions in trunk and leg muscles have different sensitivities to the direction of external perturbation. The observation of very early paraspinal stretch reflexes and unloading reflexes, coupled with early roll velocities of the trunk brings into question previously established theories regarding ankle-based triggering mechanisms. Perturbations containing roll characteristics are necessary to elicit maximal muscle responses, particularly in paraspinal muscles. In light of previous reports of enhanced sensitivity of otolith afferents, vestibular neurons, neck and vestibular reflexes to roll movements (Tomko et al., 1981; Schor et al., 1984; Wilson et al., 1986) it seems that multidirectional perturbations may prove to be a sensitive tool for assessing the contributions of vestibulo-spinal inputs to balance corrections.

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REFERENCES

Allum JHJ, Büdingen HJ (1979) Coupled stretch reflexes in ankle muscles: An evaluation of the contributions of active muscle mechanisms to human posture stability. In: Granit R, Pompeiano O. (eds). Reflex Control of Posture and Movement, Progress in Brain Research Elsevier/North-Holland Biomedical Press, Amsterdam. 50: 185-195.

Allum JHJ, Pfaltz CR (1985) Visual and vestibular contributions to pitch sway stabilization in the ankle muscles of normals and patients with bilateral peripheral vestibular deficits. Exp Brain Res 58(1): 82-94

Allum JHJ, Keshner EA, Honegger F, Pfaltz CR (1988) Organization of leg-trunk-head equilibrium movements in normals and patients with peripheral vestibular deficits. Progress in Brain Research 76: 277-290

Allum JHJ, Honegger F, Pfaltz CR (1989) The role of stretch and vestibulo-spinal reflexes in the generation of human equilibrating reactions. Prog Brain Res 80: 399-409.

Allum JHJ, Honegger F, Keshner EA (1990) Head-trunk coordination in man: Is trunk angular velocity elicited by a support surface movement the only factor influencing head stabilization. In: The Head-Neck Sensory-Motor System: Evolution, Development, Disorders, and Neuronal Mechanisms. Ed. Berthoz A, Graf W, Vidal PP, Oxford University Press, London,

Allum JHJ, Honegger F (1992) A postural model of balance-correcting movement strategies. J Vestib Res 2(4): 323-347

Allum JHJ, Honegger F, Schicks H J (1993)Vestibular and proprioceptive modulation of postural synergies in normal subjects. J Vestib Res 3(1): 59-85

Allum JHJ, Honegger F, Schicks H (1994) The influence of a bilateral peripheral vestibular deficit on postural synergies. J Vestib Res 4(1): 49-70

Allum JHJ, Honegger F, Acuna H (1995) Differential control of leg and trunk muscle activity by vestibulo-spinal and proprioceptive signals during human balance corrections. Acta Otolaryngol (Stockh) 115(2): 124-129

Allum JHJ, Huwiler M, Honegger F (1996) Prior intention to mimic a balance disorder: Does central set influence normal balance-correcting responses. Gait and Posture 4: 39-51.

Allum JHJ, Gresty M, Keshner EA, Shupert C (1997) The control of head movements during human balance corrections. J Vestib Res 7: 189-218.

Allum JHJ, Honegger F (1998) Interactions between vestibular and proprioceptive inputs triggering and modulating human balance-correcting responses differ across muscles. Exp Brain Res. 121: 478-494.

Allum JHJ, Bloem BR, Carpenter MG, Hulliger M, Hadders-Algra M (1998) Proprioceptive control of posture: a review of new concepts.Gait and Posture 8: 214-242.

Allum JHJ, Bloem BR, Verschuuren JGM, Carpenter MG, Honegger F (1999) Influence of absent lower-body and reduced upper-body proprioceptive (stretch-reflex) inputs on the triggering and modulation of balance corrections. Abstract for the 14th Symposium of the Int Soc for posture and Gait Res.

Aruin AS, Latash ML (1995) Directional specificity of postural muscles in feed-forward postural reactions during fast voluntary arm movements. Exp Brain Res 103: 323-332.

Beckley DJ, Bloem BR, Remler MP, Roos RAC, Van Dijk JG (1991) Long latency postural responses are functionally modified by cognitive set. Electroenceph Clin Neurophysiol 81: 353-358.

Bloem BR, Allum JHJ, Carpenter MG, Honegger F (1999) Is lower-leg proprioception essential for triggering human balance corrections? Exp Brain Res 130: 375-391

Bonsera SJ, Nichols TR (1996) Mechanical actions of heterogenic reflexes among ankle stabilizers and their interactions with plantarflexors of the cat hindlimb. J Neurophysiol 75: 2050-2070.

Buchanan TS, Almdale DPJ, Lewis JL, Zev Rymer W (1986) Characteristics of synergic relations during isometric contractions of human elbow muscles. J Neurophysiol 56: 1225-1241.

Carpenter MG, Allum JHJ, Honegger F (1999) Vestibular influences on human postural control in combinations of pitch and roll planes (in preparation).

Diener HC, Bootz F, Dichgans J, Bruzek W (1983) Variability of postural „reflexes" in humans. Exp Brain Res 52: 423-428.

Diener HC, Dichgans J, Bootz F, Bacher M (1984) Early stabilization of human posture after a sudden disturbance: influence of rate and amplitude of displacement. Exp Brain Res 56: 126-134.

Diener HC, Horak F, Stelmach G, Guschbauer B, Dichgans J (1991) Direction and amplitude precuing has no effect on automatic posture responses. Exp Brain Res 84: 219-223.

Dietz V (1996) Interaction between central programs and afferent input in the control of posture and locomotion. J Biomechanics 29: 841-844.

Dietz V, Gollhofer A, Kleiber M, Trippel M (1992) Regulation of bipedal stance: dependency on „load" receptors. Exp Brain Res 89: 229-231.

Forssberg H, Hirschfeld H (1994) Postural adjustments in sitting humans following external perturbations: muscle activity and kinematics. Exp Brain Res 97: 515-527.

Gottlieb GL, Agarwal GC (1979) Response to sudden torques about ankle in man: Myotatic reflex. J Neurophysiol 42: 91-106

Gurfinkel VS, Lipshits MI, Popov KE (1979) On the origin of short-latency muscle responses to postural disturbances. Agressologie 20B: 153-154.

Henry SM, Fung J, Horak FB (1998) Control of stance during lateral and anterior/posterior surface translations. IEEE Trans Rehab Eng 6: 32-42.

Henry SM, Fung J, Horak FB (1999) EMG responses to maintain stance during multi-directional surface translations. J Neurophysiol (in press)

Hirschfeld H, Forssberg H (1994) Epigenetic development of postural responses for sitting during infancy. Exp Brain Res 97: 528-540.

Horak FB, Nashner LM (1986) Central programming of postural movements: Adaptation to altered support-surface configurations. J Neurophysiol 55: 1369-1381.

Horak FB, Diener HC, Nashner LM (1989) Influence of central set on human postural responses. J Neurophysiol 62: 841-853.

Horak FB, Nashner LM, Diener HC (1990) Postural strategies associated with somatosensory and vestibular loss. Exp Brain Res 82: 167-177.

Horak FB, Moore SP (1993) The effect of prior leaning on human postural responses. Gait and Posture 1: 203-210.

Horak FB, Lamarre Y, Macpherson JM, Jones C, Henry SM (1996) Postural control in a patient with total body somatosensory loss. Society for Neuroscience 22: 1632 (abstract).

Horstmann GA, Dietz V (1990) A basic posture control mechanism: the stabilization of the centre of gravity. Electroenceph Clin Neurophysiol 76: 165-176.

Inglis JT, Horak FB, Shupert CL, Jones-Rycewicz C (1994) The importance of somatosensory information in triggering and scaling automatic postural responses in humans. Exp Brain Res 101(1): 159-164.

Kanaya T, Gresty MA, Bronstein AM, Buchwell D, Day B (1995) Control of the head in response to tilt of the body in normal and labyrinthine-defective human subjects. Journal of Physiol (Lond) 489: 895-910.

Keshner EA, Allum JH, Pfaltz CR (1987) Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit. Exp Brain Res 69(1): 77-92

Keshner EA, Woollacott MH, Debu B (1988) Neck, trunk and limb muscle responses during postural perturbations in humans. Exp Brain Res 71(3): 455-466

Keshner EA, Campell D, Katz R, Peterson BW (1989) Neck muscle activation patterns in humans during isometric head stabilization. Exp Brain Res 75: 335-344.

Lavender S, Trafimow J, Andersson GBJ, Mayer RS, Chen I (1994) Trunk muscle activation. The effects of torso flexion, moment direction and moment magnitude. Spine 19: 771-778.

Macpherson JM (1988a) Strategies that simplify the control of quadrupedal stance. I. Forces at the ground. J Neurophysiol 60: 204-217.

Macpherson JM (1988b) Strategies that simplify the control of quadrupedal stance. II. Electromyographic activity. J Neurophysiol 60: 218-231.

Macpherson JM, Horak FB, Dunbar DC, Dow RS (1989) Stance dependence of automatic postural adjustments in humans. Exp Brain Res 78: 557-566

Macpherson JM (1994) The force constraint strategy for stance is independent of prior experience. Exp Brain Res 101: 397-405.

Maki BE, Whitelaw RS (1993) Influence of expectation and arousal on center-of-pressure responses to transient postural perturbations. J Vestib Res 3: 25-39.

Maki BE, McIlroy WE, Perry SO (1994) Compensatory responses to multi-directional perturbations. In: Taguchi K, Igarashi M, Mori S (eds). Vestibular and Neural Front, Elsevier, Amsterdam, 437-440.

Marsden CD, Obeso JA, Rothwell JC (1983) The function of antagonist muscle during fast limb movements in man. J Physiol (Lond) 335: 1-13.

McIlroy WE, Maki BE (1995) Early activation of arm muscles follows external perturbation of upright stance. Neuroscience Letters 184: 177-180.

Melvill-Jones G, Watt DGD (1971) Observations on the control of stepping and hopping movements in man. J Physiol (Lond) 219: 709-727.

Mittlestaedt H (1992) Somatic versus vestibular gravity perception in man. Annals of the New York Academy of Sciences 656: 124-139.

Moore SP, Rushmer DS, Windus SL, Nashner LM (1988) Human automatic postural responses: responses to horizontal perturbations of stance in multiple directions. Exp Brain Res 73: 648-658.

Nardone A, Corrà T, Schieppati M (1990) Different activations of the soleus and gastrocnemii muscles in response to various types of stance perturbation in man. Exp Brain Res 80: 323-332.

Nashner LM (1976) Adapting reflexes controlling the human posture. Exp Brain Res 26: 59-72.

Nashner LM (1977) Fixed patterns of rapid postural responses among leg muscles during stance. Exp Brain Res 30: 13-24.

Nashner LM, Black FO, Wall C III (1982) Adaptation to altered support surface and visual conditions during stance: patients with vestibular deficits. J Neuroscience 2: 536-544.

Nashner LM, McCollum G (1985) The organization of human postural movements: a formal basis and experimental synthesis. Behav Brain Sci 8:135-172.

Nichols TR, Lawrence JH III, Bonasera SJ (1993) Control of torque direction by spinal pathways at the cat ankle joint. Exp Brain Res 97: 366-371.

Pearson KG (1995) Proprioceptive regulation of locomotion. Current Opinion in Neurobiology 5: 786-791.

Rothwell J (1994) Control of Human Voluntary Movement. Second Edition. London: Chapman & Hall 103-117.

Rushmer DS, Russell CJ, Macpherson J, Phillips JO, Dunbar DC (1983) Automatic postural responses in the cat: Responses to headward and tailward translation. Exp Brain Res 50: 45-61.

Rushmer DS, Moore SP, Windus SL, Russell CJ (1988) Automatic postural responses in the cat: responses of hindlimb muscles to horizontal perturbations of stance in multiple directions. Exp Brain Res 71: 93-102.

Schieppati M, Nardone A, Siliotto R, Grasso M (1995) Early and late stretch responses of human foot muscles induced by perturbation of stance. Exp Brain Res 105: 411-422.

Shumway-Cook A, Horak FB (1986) Assessing the influence of sensory interaction on balance. Physical Ther 66: 1548-1550.

Tomko DL, Peterka RJ, Schor RH (1981) Responses to head tilt in cat eighth nerve afferents. Exp Brain Res 41: 216-221.

Traub MM, Rothwell JC, Marsden CD (1980) A grab reflex in the human hand. Brain 103: 869-884.

Winter DA, Patla AE, Frank JS (1990) Assessment of balance control in humans. Medical Progress through Technology 16: 31-51.

Winter DA, Prince F, Frank JS, Powell C, Zabjek KF (1996) Unified theory regarding A/P and M/L balance in quiet stance. J Neurophysiol 75(6): 2334-2343.

Yang JF, Stein R (1990) Phase-dependent reflex reversal in human leg muscles during walking. J Neurophysiol 63: 1109-1117.

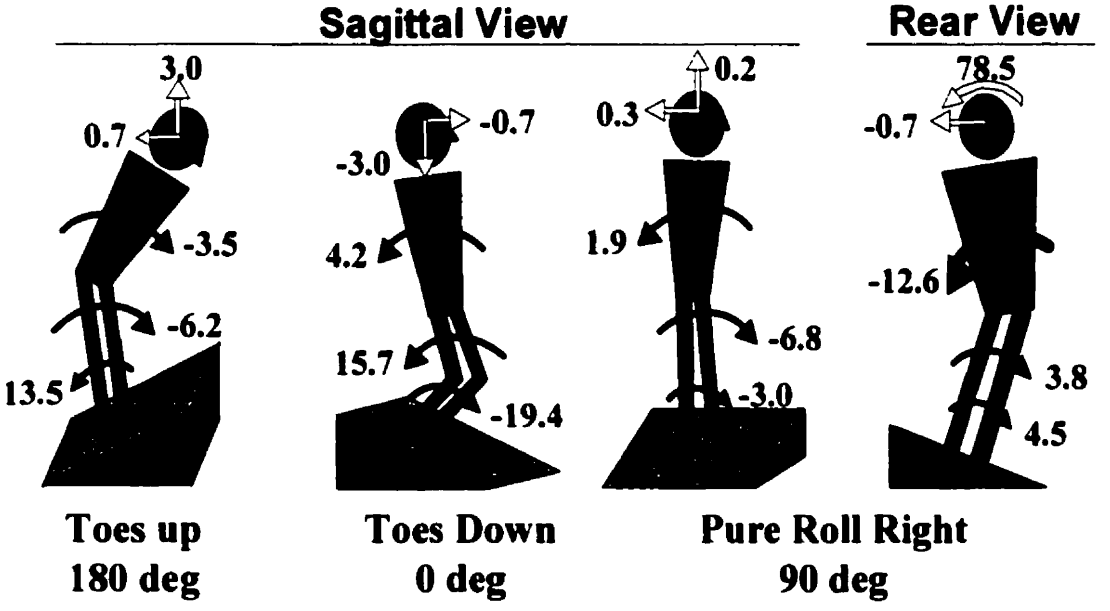
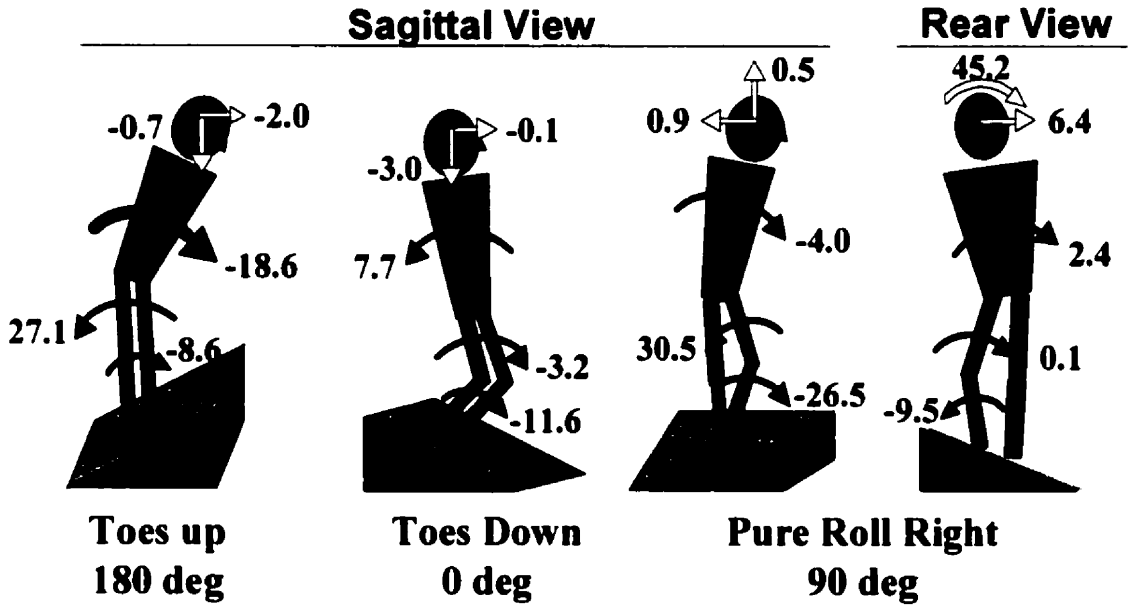


Figure 1. Initial stimulus induced link movements in response to rotational perturbations in pure pitch and roll directions. Curved filled arrows and corresponding values represent the direction and average (population) magnitude of trunk, upper leg and lower leg angular velocity in deg/s calculated over the period 0-150 ms. The thickened curved arrow indicates the fastest trunk velocity. Open arrows represent average linear (straight arrows, values in cm/s^2) and angular roll (curved arrows, values in deg/s^2) accelerations of the head between 40-90 ms. Toe up and roll perturbations elicit two segment, whereas toe down rotations elicit multi-link reactions.



Segment Movements during Automatic Balance Correcting Response

Figure 2. Biomechanical responses during of automatic balance correcting responses to rotational perturbations in pure pitch and roll directions. Curved filled arrows and corresponding values represent the direction and average (population) magnitude of trunk, upper leg and lower leg angular velocity in deg/s calculated between 240-300 ms. Open arrows represent average linear (straight in cm/s^2) and angular roll (curved in deg/s^2) accelerations of the head between 180-240 ms.

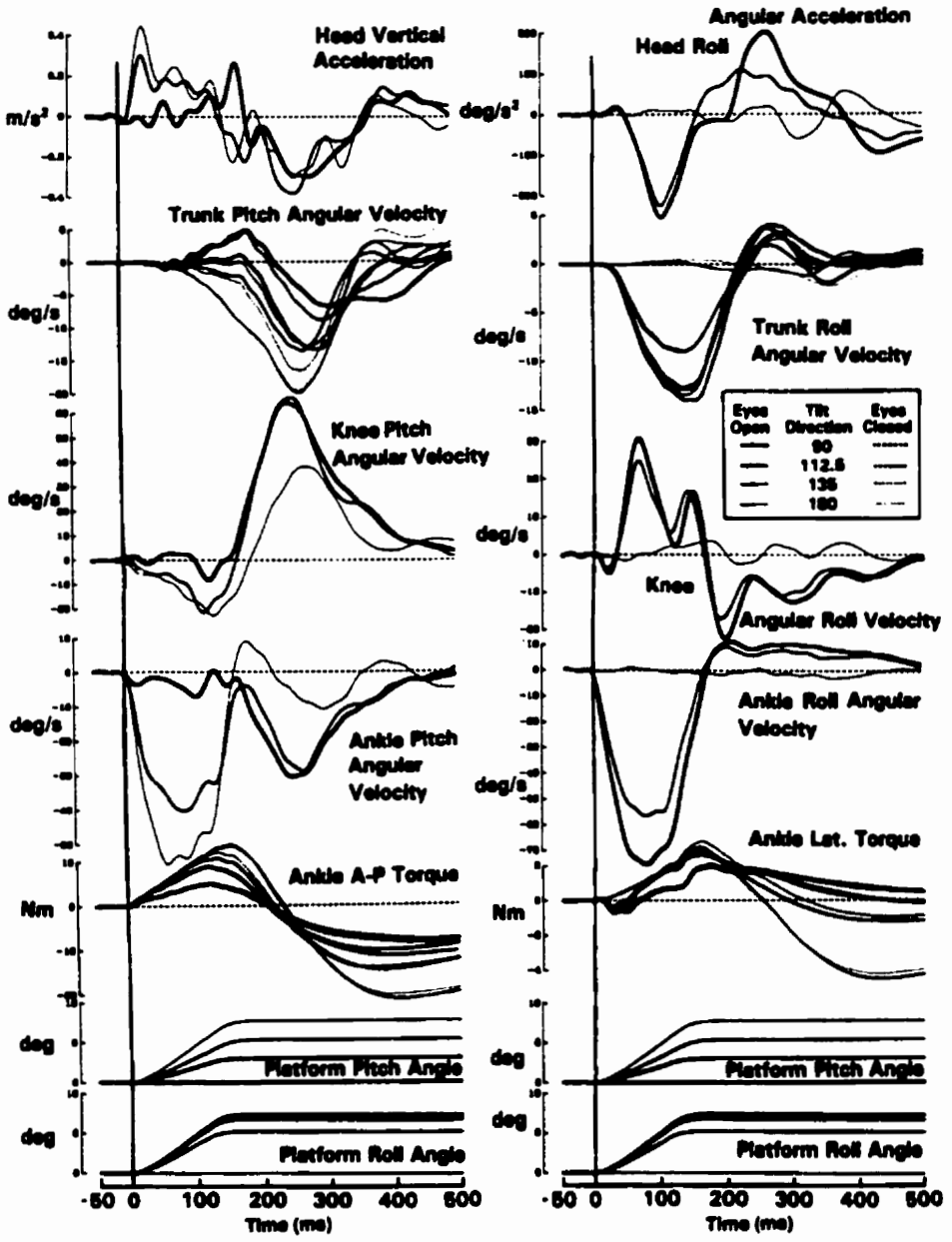


Figure 3. Biomechanical responses (average of 12 subjects) to rotational perturbations in multiple backward directions between 90 (pure roll right) and 180 (toe-up) directions for both eyes open (solid lines) and eyes closed (dashed lines) conditions. The black vertical line at 0 ms represents the onset of ankle rotation. For most recorded variables 4 traces are shown (for the directions of 90, 112.5, 135, 180 degs) in decreasing line thickness as the perturbation direction moves from pure roll to pure pitch. In the set of recordings for ankle and knee angular velocities, and head accelerations, only 3 traces are shown (for 90, 135 and 180 degs) because these variables were not recorded for 112.5 degs.

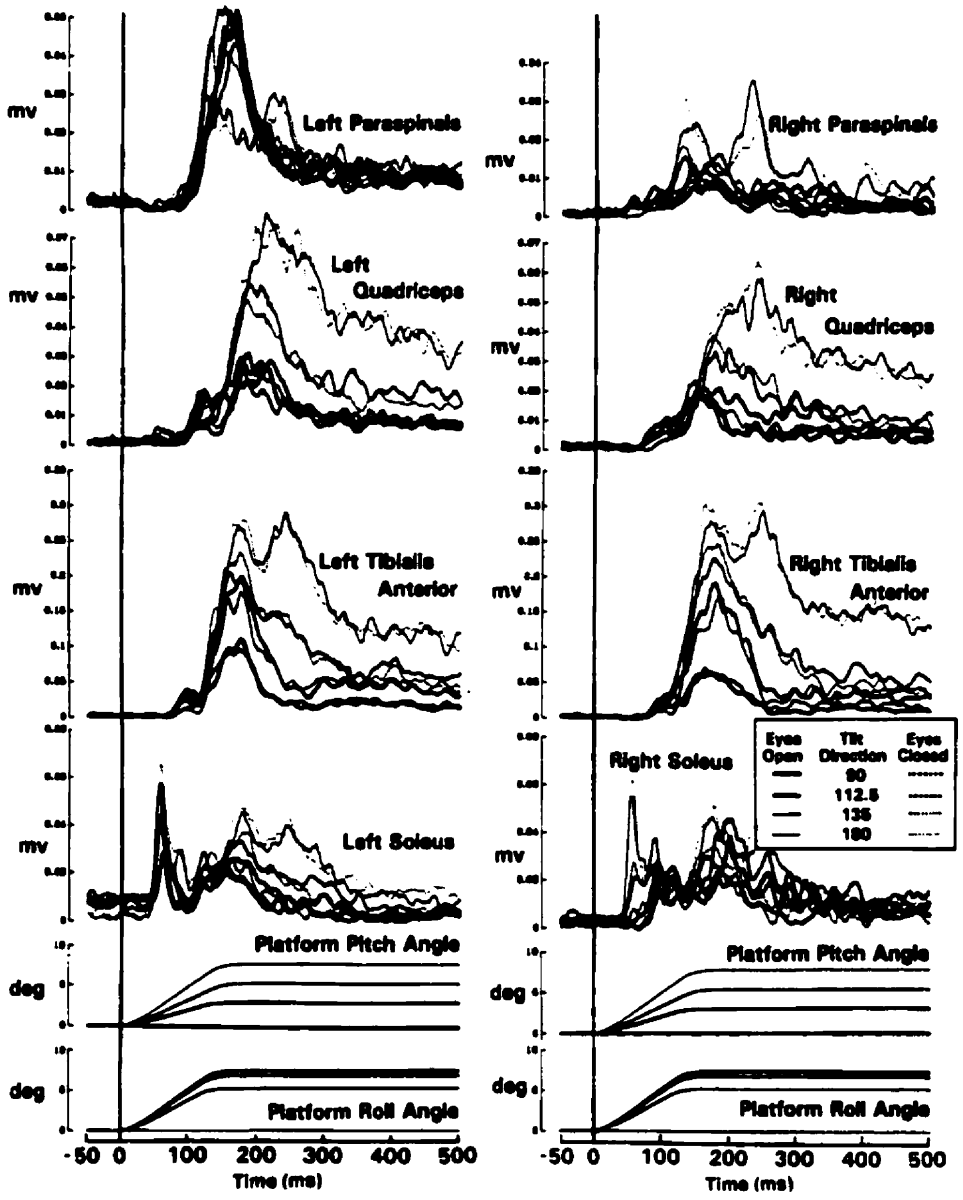


Figure 4. Muscle responses (average of 14 subjects) to rotational perturbations in multiple backward directions between 90 (pure roll right) and 180 (toe-up) directions for both eyes open (solid lines) and eyes closed (dashed lines) conditions. For each set of traces 3 or 4 recordings are shown increasing in line thickness with increasing roll component to the stimulus. For other details refer to Fig. 3.

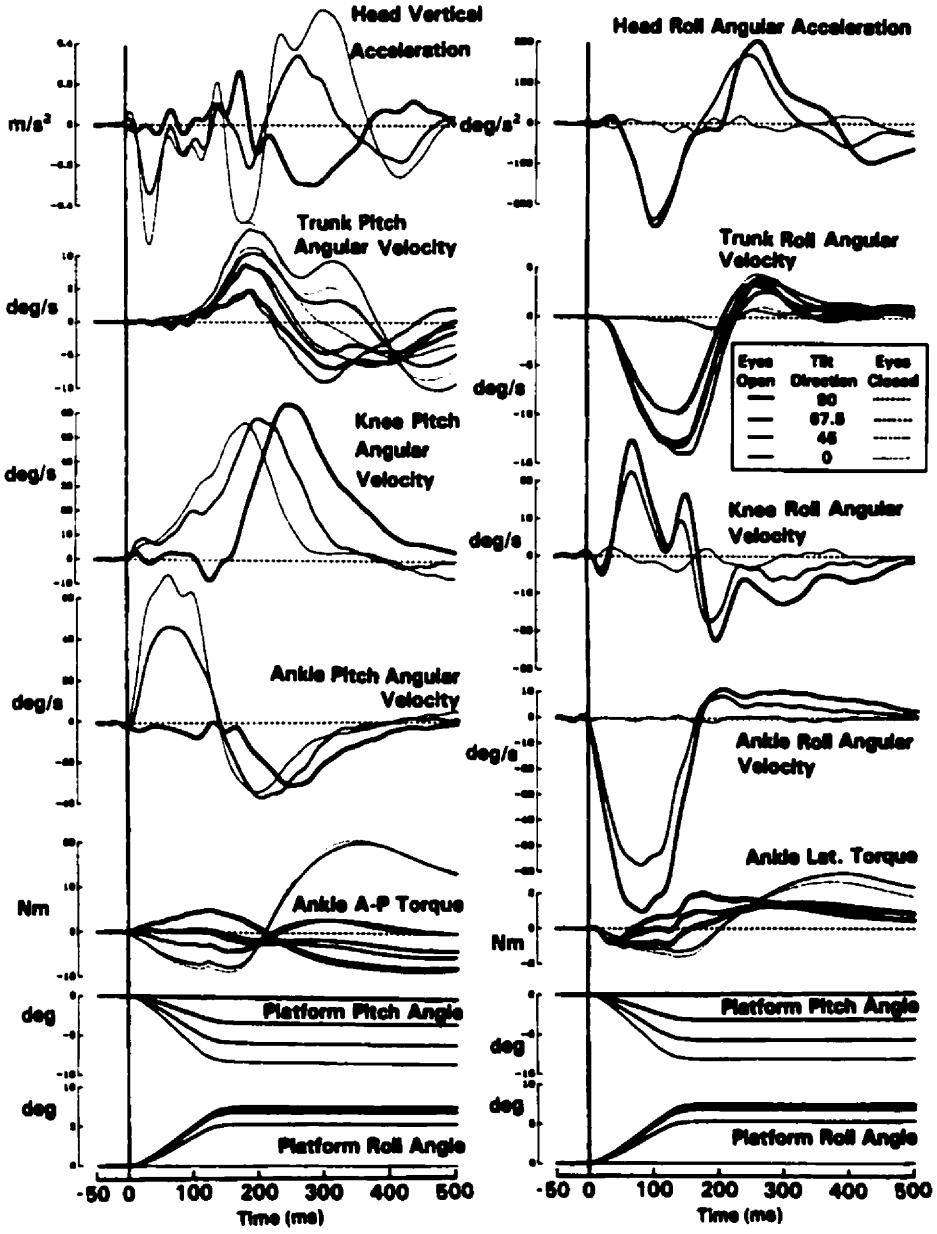


Figure 5. Population biomechanical responses to rotational perturbations in multiple forward directions between 0 (toes down) and 90 (pure roll right) directions for both eyes open (solid lines) and eyes closed (dashed lines) conditions. For details refer to Fig. 3.

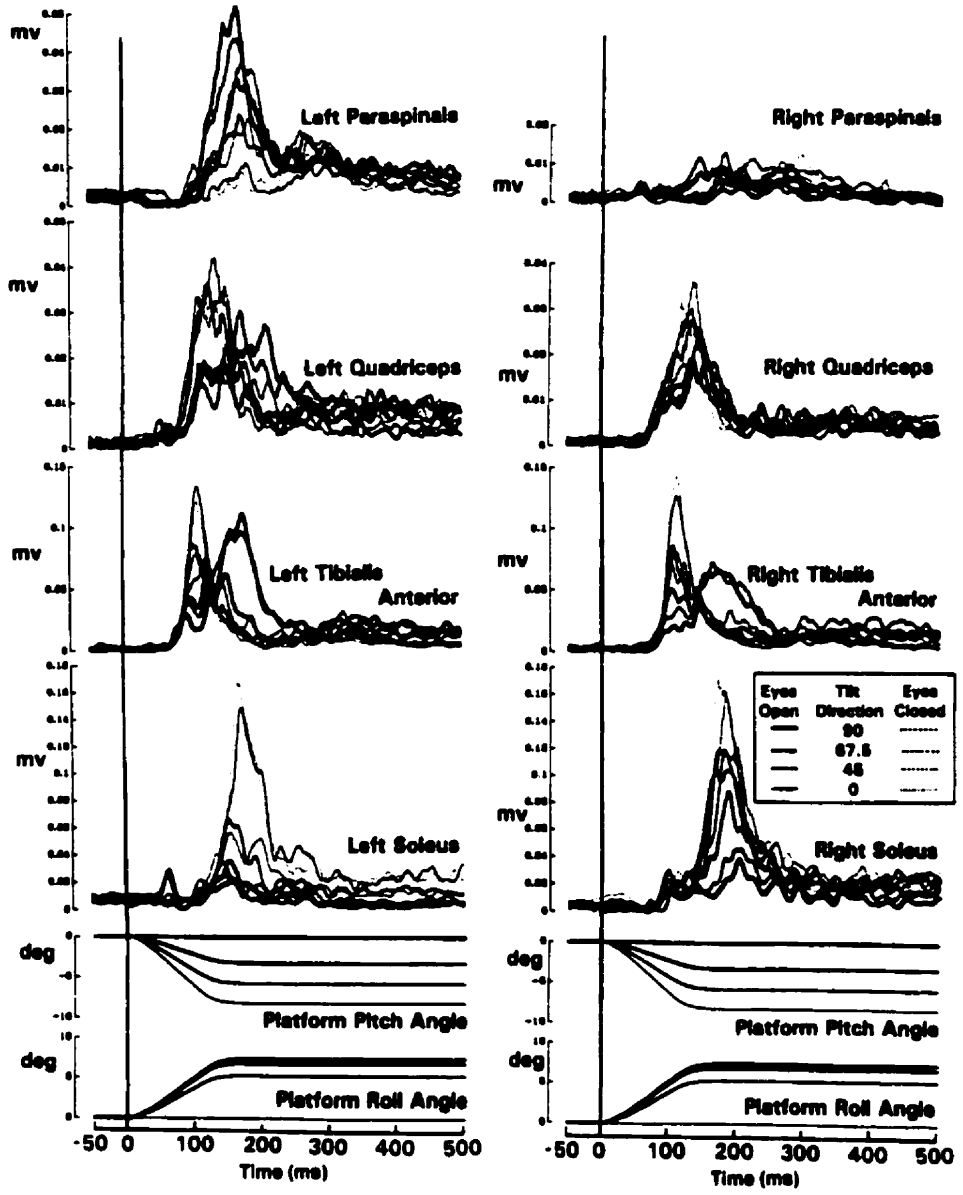


Figure 6. Population muscle responses to rotational perturbations in multiple forward directions between 0 (toes down) and 90 (pure roll right) directions for both eyes open (solid lines) and eyes closed (dashed lines) conditions. For details refer to Figs. 3 and 4.

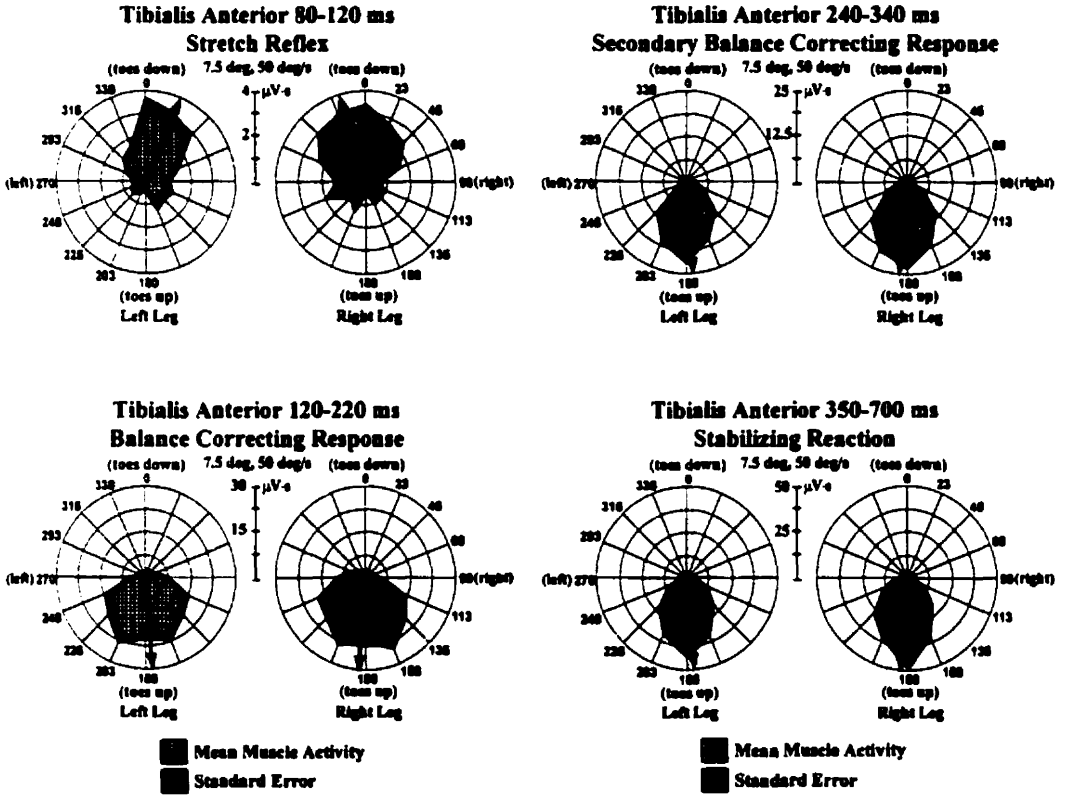


Figure 7. Polar plots for tibialis anterior EMG activity under eyes-open conditions averaged over four distinct time intervals representative of stretch, balance correcting, secondary balance correcting and stabilizing reactions. Each radial line or spoke represents the direction of platform rotation. For each direction mean muscle activity (pattern) and the mean plus one standard error (shade) of all subjects for eyes closed are plotted for left and right muscles separately. The amplitude is plotted as distance from the centre. The response amplitude represented by each of the concentric circles in the plot is scaled according to the vertical scale between the set of plots for the left and right recording sites. Black arrows represent the direction of calculated maximum activity vector for each averaging interval.

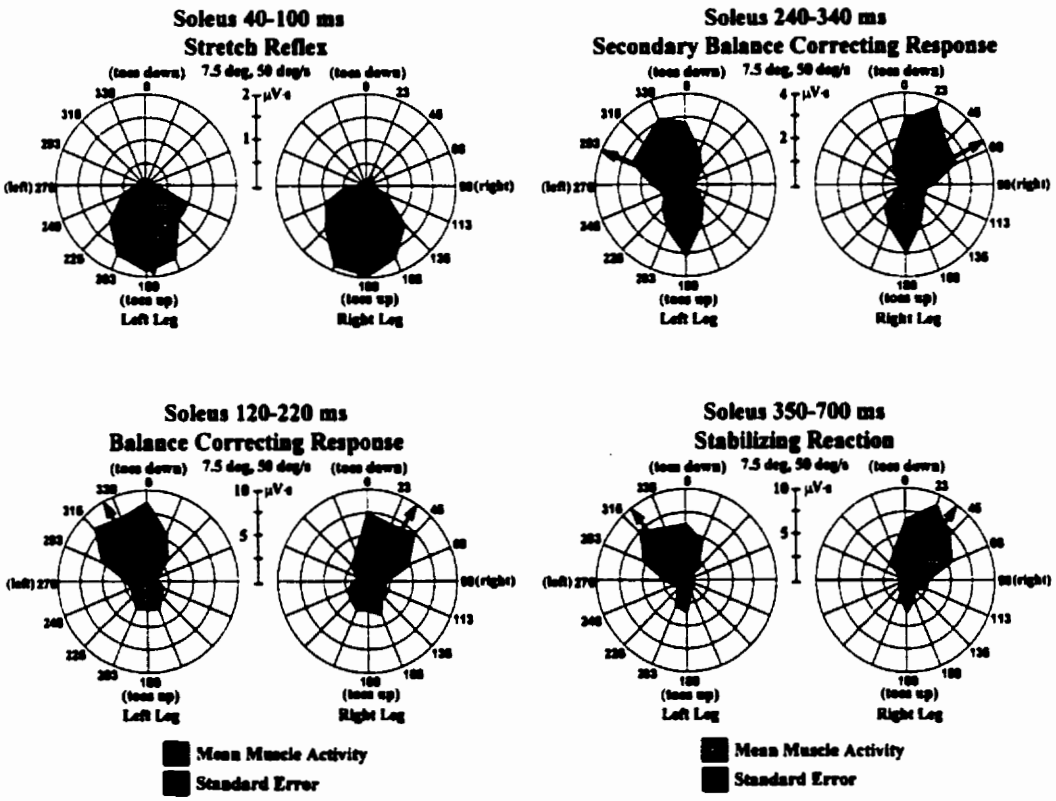


Figure 8. Polar plots for soleus EMG activity (eyes open) during four distinct time intervals representative of stretch, balance correcting, secondary balance correcting and stabilizing reactions. For details of the figure refer to the legend of Fig. 7.

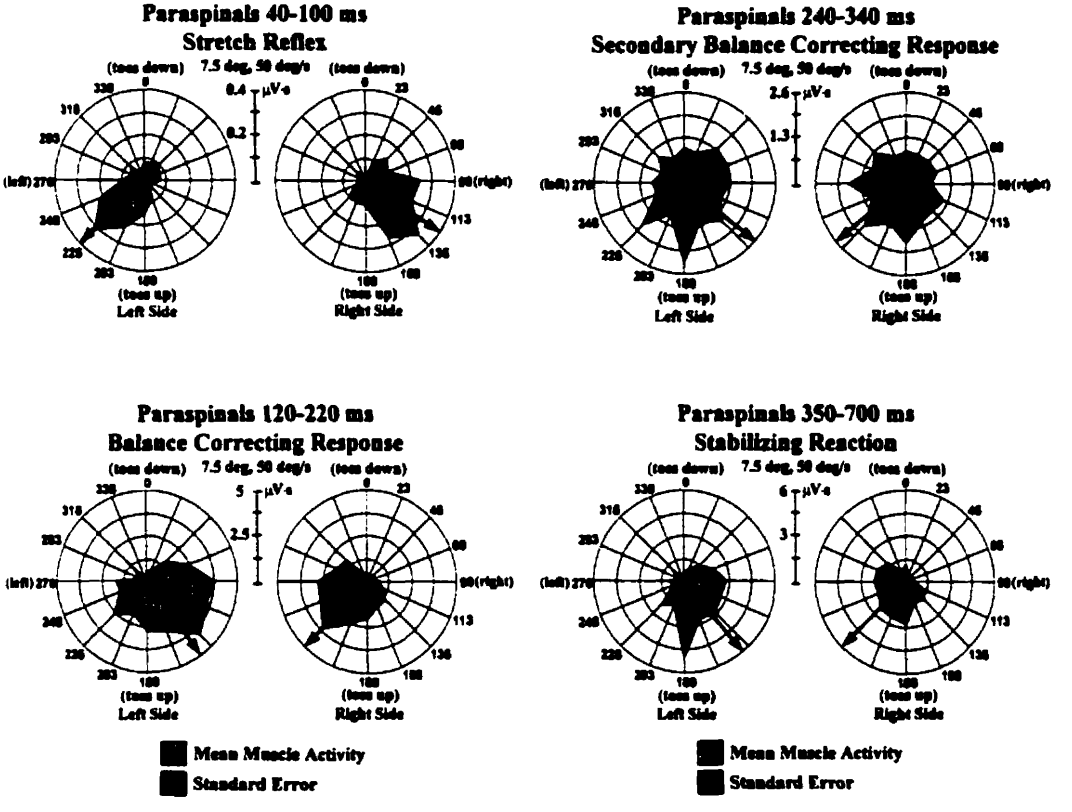


Figure 9. Polar plots for paraspinals EMG activity (eyes open) over distinct response intervals. For details of the figure refer to the legend of Fig. 7. Note the off-pitch-axis orientation of the maximum activity vectors.

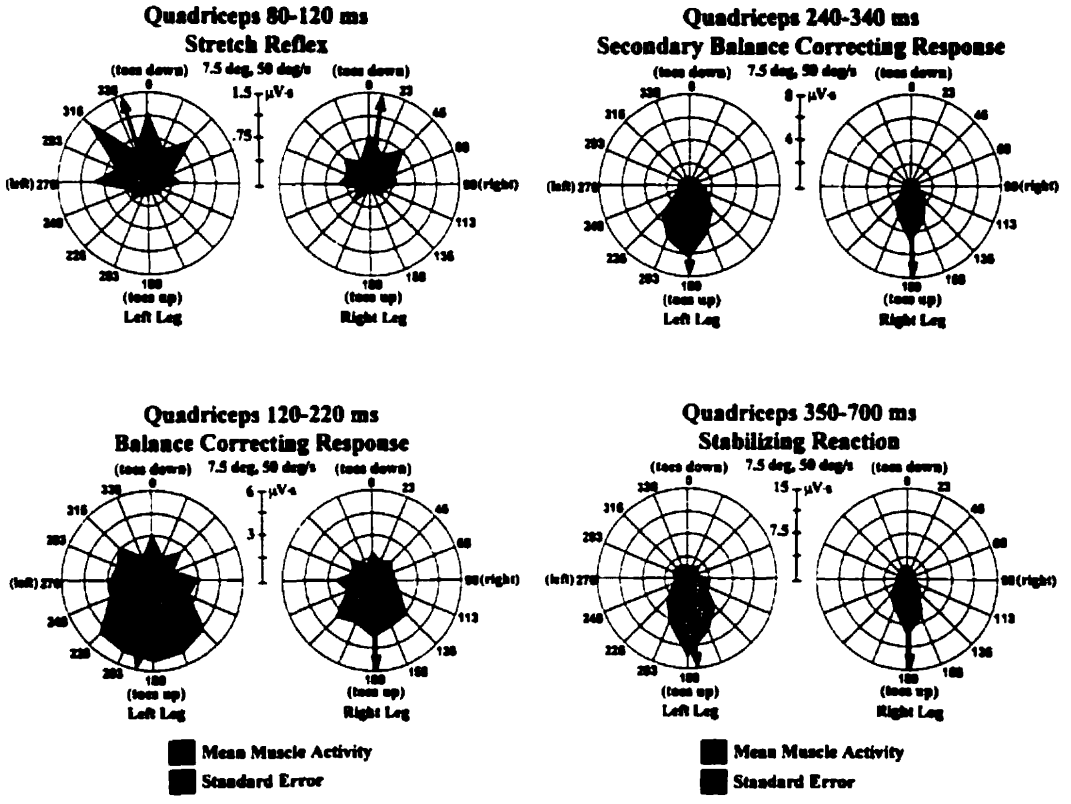


Figure 10. Polar plots for quadriceps EMG response activity (eyes open) over distinct time intervals. For details refer to the legend of Fig. 7.

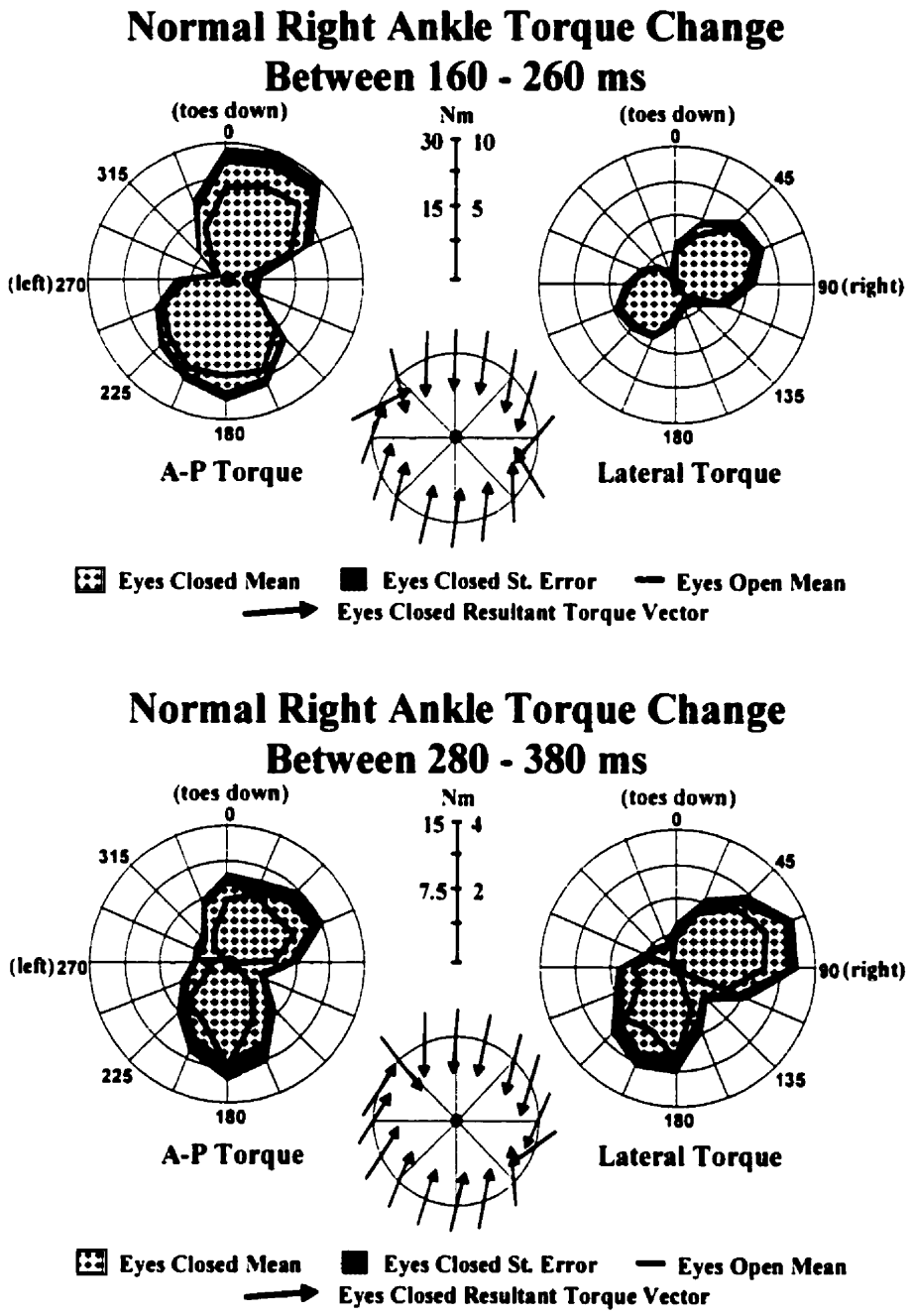
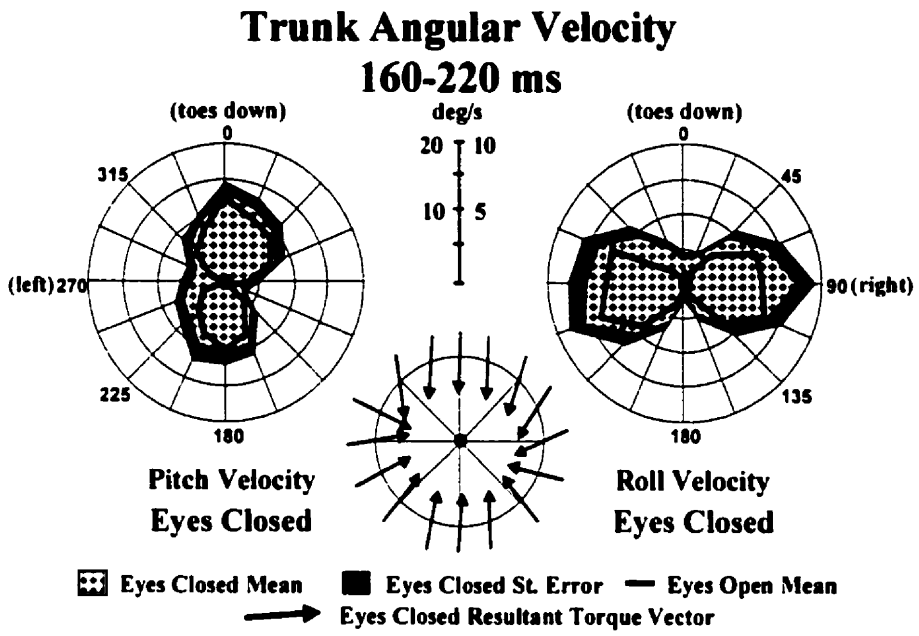


Figure 11. Polar plots of the calculated change in right ankle torque between 160-260 ms (upper plots) and between 280-380 ms (lower plots) under eyes-open and eyes-closed conditions. Each radial line or spoke represents the direction of platform rotation. The magnitude of the mean change (pattern) and mean standard error (shaded) in anterior-posterior (A-P) and lateral torque of all subjects are plotted for each direction on the left and right graphs respectively. Concentric circles indicate the amplitude of the torque change as shown by the vertical scale between the left and right pair of polar plots. Black arrows in the centre graphs illustrate the direction of the resultant vector calculated from A-P and Lateral torque for each perturbation direction with which the body would move assuming the body was simply an inverted pendulum.



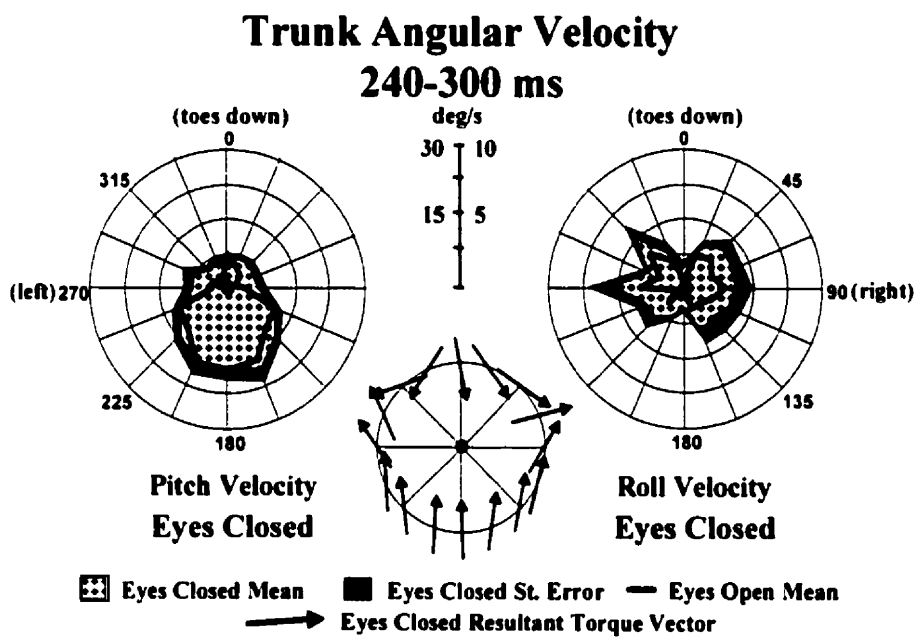


Figure 12. Polar plots of the average amplitude of trunk angular velocity between 160-220 ms and between 240-300 ms under eyes-open and eyes-closed conditions. The form of the plot is similar to that of Fig. 11. The black arrows in the centre graphs illustrate the resultant direction of the trunk movement during the time periods as viewed from above the subject.

CHAPTER 3

VESTIBULAR INFLUENCES ON HUMAN POSTURAL CONTROL IN COMBINATIONS OF PITCH AND ROLL PLANES REVEAL DIFFERENCES IN SPATIOTEMPORAL PROCESSING

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ABSTRACT

The present study examined the influence of bilateral peripheral vestibular loss in humans on postural responses to multi-directional surface rotations in the pitch and roll planes. Specifically, we examined the effects of vestibular loss on the directional sensitivity, timing and amplitude of early stretch, balance correcting and stabilizing reactions in postural leg and trunk muscles as well as changes in ankle torque and trunk angular velocity following multi-directional rotational perturbations of the support surface. Fourteen normal healthy adults and 5 bilateral peripheral vestibular loss (BVL) patients stood on a dual axis rotating platform which rotated 7.5 degrees at 50 deg/s through 8 different directions of pitch and roll combinations separated by 45 degrees. Directions were randomized within a series of 44 perturbation trials which were presented first with eyes open, followed by a second series of trials with eyes closed.

Vestibular loss did not influence the range of activation or direction of maximum sensitivity for balance correcting responses (*120-220 ms)*. Response onsets at approximately 120 ms were normal in tibialis anterior (TA), soleus (SOL), paraspinals (PARAS) or quadriceps (QUAD) muscles. Only SOL muscle activity demonstrated a 38-45 ms delay for combinations of forward (toe-down) and roll perturbations in BVL patients. The amplitude of balance correcting responses in leg muscles between 120-220 ms was, with one exception, severely reduced in BVL patients for eyes open and eyes closed conditions. Soleus responses were decreased bilaterally for toe-up and toe-down perturbations, but more significantly reduced in the downhill (load bearing) leg for combined roll and pitch perturbations. TA was significantly reduced bilaterally for toe-up perturbations, and in the downhill leg for backward roll perturbations. Forward perturbations, however, elicited significantly larger TA activity in BVL between 120-220 ms compared to normals, which would act to further destabilize the body. As a result of these changes in response amplitudes, BVL patients had reduced balance correcting ankle torque between 160-260 ms and increased torque between 280-380 ms compared to normals. There were no differences in the orientation of the resultant ankle torque vectors between BVL and normals, both of which were oriented primarily along the pitch plane. For combinations of backward (toe-up) and roll perturbations BVL patients had larger balance

correcting and stabilizing reactions (between 350-700 ms) in PARAS than normals and these corresponded to excessive trunk pitch and roll velocities. During roll perturbations, trunk velocities in BVL subjects after 200 ms were directed along directions different to those of normals. Furthermore, roll instabilities appeared later than those of pitch particularly for backward roll perturbations. The results of the study show that combinations of roll and pitch surface rotations yield important spatio-temporal information, especially with respect to trunk response strategies changed by BVL which are not revealed by pitch plane perturbations alone. Our results indicate that vestibular influences are earlier for the pitch plane and are directed to leg muscles, whereas roll control is later and focused on trunk muscles.

Key words: Balance control, vestibulo-spinal system, proprioceptive reflexes, vestibular loss.

INTRODUCTION

Pitch plane perturbations of the support surface (or dynamic posturography) have provided clinicians and scientists with an experimental paradigm to study normal and pathological characteristics of the CNS response to unexpected falling due to external perturbations. The most common form of this dynamic posturography involves tipping or translating the support surface forward or backward beneath the standing subject, while recording the ensuing muscular and biomechanical responses required to maintain upright equilibrium. Undeniably, a great amount of knowledge has been developed from this paradigm. For example, these uniplanar posturography studies have shown that movements of the support surface elicit automatically triggered patterns of balance correcting muscle activity across many body segments (Cordo and Nashner 1983; Allum et al. 1993; Horak et al. 1997), which are dependent upon the amplitude (Diener et al. 1984; 1991), velocity (Allum and Pfaltz 1985; Allum et al. 1993) and the direction (forwards or backwards) of perturbation (Rushmer et al. 1983; Allum et al. 1993). Pitch-plane dynamic posturography has also been used extensively to examine differences between healthy individuals and patients with selective sensory deficits. Based on these results, valuable information has been acquired to help understand the relative contribution and individual influences of different sources of sensory information on postural control including proprioception (Inglis et al. 1994; Horak et al. 1996, Bloem et al. 2000), vision (Nashner and Berthoz 1978, Timmann et al. 1994, DiFabio et al. 1998) and vestibular information (Allum and Pfaltz 1985; Keshner et al. 1987; Allum et al. 1994; 1998; Horak et al. 1990; Runge et al. 1998)

One major limitation inherent to all of the studies mentioned above is their reliance on recordings from perturbations within the pitch plane. This has several major drawbacks when concepts of normal and pathological balance control need to be generalized to multiple directions including the roll plane. First, falls in everyday life and particularly in older adults occur frequently in lateral directions (Maki and McIlroy 1998). Real life situations, for which surface perturbations are intended to mimic, such as an accelerating bus, pitching boat or rolling train, impose destabilizing forces which rarely act along a purely sagittal plane. Second, proprioceptive and vestibulo-sensory systems underlying balance control have sensitivities in

other than the pitch plane. Stretch receptors, for example, have preferred directions of activity along muscles. Furthermore, some central vestibular neurons show responses after transformation of afferent signals characteristic of canal plane responsiveness (Schor et al. 1984, Wilson et al. 1986) whereas others have different response properties for roll and pitch (Angelaki and Dickman 2000). Thus both sensory systems may contribute to a balance correction differently depending upon the direction of perturbation. Third, clinical observations of patients with balance disorders (Allum et al. 2001a) and aging individuals (Gill et al. 2001) reveal significant instability in both pitch and roll planes. The limited success that pitch-plane dynamic posturography has had in diagnosing and discriminating balance disorders (Di Fabio 1995; Bronstein and Guerraz 1999) clearly illustrates the limitation of pitch plane perturbations to capture the essential components of normal and pathological balance. Although more recent success to discriminate between patient populations has been achieved using upper rather than lower-body responses to pitch plane rotations (Allum et al. 2001b) its fundamental utility to screen for more subtle balance disorders or to recognize disease-specific information, such as the side of a lesion is questionable (Lipp and Longridge 1994; Furman 1995).

A shift to the use of multidirectional perturbations for understanding human postural control has provided new evidence that challenges the foundation of long standing postural control theories. Normal responses to multidirectional perturbations have been examined using surface translations and rotations in sagittal and frontal planes in quadripedal animals (Rushmer et al. 1988; Macpherson 1988a; 1988b; 1994) and humans (Moore et al. 1988; Maki et al. 1994; Henry et al. 1998a; 1998b; Carpenter et al. 1999), as well as perturbations delivered to the trunk and pelvis (Rietdyk et al. 1999; Gilles et al. 1999). Throughout this literature, two main themes emerge. First, postural responses are directionally sensitive and involve combinations of ankle, knee and hip responses which are different for roll and pitch directions. Second, directionally sensitive trigger information is available at the level of the hip and pelvis prior to, or simultaneously with more pitch-plane sensitive information received from the lower leg and ankles. These findings contend with previously developed concepts of human balance control based on unidirectional studies and highlight the need to investigate balance control under more rigorous parameters which challenge the multi-directional nature of the postural control system.

With a more comprehensive understanding of normal healthy responses to multidirectional perturbations, it is important to extend our research to investigate how different sources of sensory information may contribute to the triggering and modulation of directionally sensitive postural responses. The role of vestibular information on postural control has been studied extensively using unidirectional perturbations, with new studies beginning to shed new light (Allum and Honegger 1998; Runge et al. 1998) on previously conflicting results (Allum et al. 1994; Nashner et al. 1982; Horak et al. 1990). Only one study to date has examined the effects of vestibular loss on multidirectional perturbations. In this case, Inglis and Macpherson (1995) observed significant differences in amplitude, but not in timing or pattern of postural muscle responses in labrynthecomized cats during sudden unexpected multidirectional translations. Since the biomechanical constraints imposed by surface perturbations are different for quadripeds and bipeds (Macpherson et al. 1989), it is important to also examine the specific effects of vestibular loss in humans on triggering and modulation of postural responses to multidirectional perturbations.

Inherent to balance control comparisons between normal and vestibular loss subjects is the assumption that a vestibular deficit will lead to a permanent sensory deficit that cannot be ameliorated by switching to another sensory input for adequate directional information underlying balance commands (Allum and Honegger 1998). Although such a switching mechanism has been proposed (Horak et al. 1994; Nashner et al. 1982), it would appear that switching to other inputs only occurs for later stabilizing action, once the primary motor command to correct the imbalance has been issued (Allum and Shepard 1999). Another operating assumption of such a comparison must be that spinal stretch reflex mechanisms can be separately observed and are not altered by the absence of tonic or dynamic vestibular input, or that the alteration is not significant enough to change the pattern of subsequent balance corrections. For some perturbation paradigms, notably with translations of the support surface, an interaction between initial stretch reflex and subsequent balance corrections is difficult to avoid and this interaction is altered after vestibular and proprioceptive sensory loss (Allum and Honegger 1998; Bloem et al. 2000). For rotational support-surface paradigms a clearer distinction between early stretch reflex and balance corrections can be obtained (Diener et al.

1983; Allum et al. 1993; Carpenter et al 1999). Furthermore, it is known for pitch plane rotations, that influences of vestibular loss on stretch reflexes are small (Keshner et al. 1987; Allum and Honegger 1998). Under these assumptions the present study was dedicated to addressing *two*main goals using multi-directional rotations of the support surface. The first goal of the present study was to determine whether the current understanding of the effects of BVL on postural reactions, established with pitch plane perturbations, can be extended to perturbations which contain both pitch and roll components. The second goal of the experiment was to determine what new information is available from multidirectional (pitch and roll planes) perturbations, which might provide a framework for understanding the role of central transformations of vestibular inputs in generating motor programs that arrest falls in different directions.

METHODS

This study examined the effect of multi-directional rotations of the support surface on muscular and biomechanical responses in normal healthy young adults and subjects with bilateral peripheral vestibular loss acquired idiopathically as adults at least 2 years prior to these experiments. Fourteen normal controls (7 male, 7 female; mean age=22.71 sd 2.40 yrs; height=1.73 sd 0.08 m; weight=69.5 sd 11.7 kg) and 5 bilateral peripheral vestibular loss patients (4 male, 1 female; mean age=39.4 sd 6.18 yrs; height=1.72 sd 0.07 m; weight=74.2 sd 7.76 kg) volunteered for the study and gave witnessed prior informed consent to participate in the experiment after observing movements of the support surface. Normal subjects were free from any neurological or previous orthopaedic injuries as verified by extensive questioning. Normal vestibular function was further verified using Romberg and Unterberger stance tests. Bilateral peripheral vestibular loss was characterized by no response (slow phase velocity less than 2 deg/sec) to bithermal caloric irrigation (100 cc of water for 30 secs) of each ear and by horizontal vestibulo-ocular reflex responses to whole body rotations of 80 deg/ s^2 which were smaller than the lower 1% bound of normal reference values (Allum and Ledin 1999).

Subjects were positioned on the force-measuring platform with their feet lightly strapped to the support surface and the lateral malleoli aligned with the platform's pitch axis of rotation. The roll axis had the same height as the pitch axis and passed between the feet. The subjects were

asked to assume their normal standing posture, with knees locked and arms hanging comfortably at their sides. Offsets were added to force-plate readings so these readings were without a dc bias. These were then treated as the reference values for each individual's 'preferred-stance' position.

Stimulus Parameters

The dual axis rotating force-platform delivered unexpected rotations through 8 different directions in the pitch and roll planes. A clockwise increasing notation, as viewed from above, was used to specify rotation direction. The 0 deg rotation direction represented a pure 'toes-down' tilt of the platform, conversely, 180 deg direction represented a pure 'toes-up' rotation. Pure roll movements were assigned angles of 90 deg to the right and 270 deg to the left. Combinations of pitch and roll rotations were used to provide 4 other directions, each separated by 45 deg. Platform rotations had a constant amplitude of 7.5 deg and angular velocity of 50 deg/s. One series of 44 randomly presented directional stimuli was always performed first with eyes open. Following a 5 to 10 minute rest period to minimize any confounding effects due to order and/or fatigue a second series of 44 random stimuli were performed with eyes closed. Although it is acknowledged that the non-random presentation of visual conditions may have invited confounding effects due to order (Keshner et al. 1987), it was deemed necessary to maintain a constant presentation order of increasing difficulty to minimize anxiety and reduce the fear of falling of the vestibular-loss subjects. For each series of 44 stimuli, the very first stimulus was ignored in the data analysis to reduce the effects of adaptation (Nashner et al., 1982; Keshner et al. 1987) entering the data. Of the remaining 43 stimuli included in each data series, each of the 8 perturbation directions were presented randomly 5-6 times.

Each perturbation was preceded by a random 5-20 s delay. During this delay period subjects were required to maintain anterior/posterior (A-P) ankle torque within a range of +/- 1 Nm from the 'preferred-stance' reference value using on-line visual feedback from an oscilloscope placed at eye level (approximately 1 m away from the subject). During the eyes closed condition two distinct auditory tones were substituted for visual feedback to monitor variations in A-P ankle torques prior to the stimulus onset. The 5-20 s interstimulus delay was initiated

automatically once the platform had returned to its original pre-stimulus position and the subject regained and maintained his preferred vertical position as monitored by the A-P ankle torque reading. In response to each rotational perturbation, the subject was instructed to recover their balance as quickly as possible. Handrails were located on the lateral borders of the platform apparatus in case of loss of balance. Patients were instructed to grasp the handrails in the case of a fall. Two spotters were always arranged with one behind and one to the side of the vestibular-loss subjects to lend support in case of a fall.

Biomechanical and EMG Recordings

All biomechanical and electromyographic (EMG) recordings were initiated 100 ms prior to the onset of the perturbation and had a sampling duration of 1 second. Support surface reaction forces were measured from two independent force-plates, one for each foot, embedded within the rotating support surface of the moveable platform. Vertical forces were measured by strain gauges located under the corners of each plate. From these forces A-P and medial lateral (M-L) ankle torques were calculated (Allum and Honegger 1998). Trunk angular velocity in the pitch and roll planes were collected using Watson Industries transducers (+/- 300 deg/s range) mounted to a metal plate at a level of the sternum. The plate was strapped to the chest firmly with straps across the shoulders, back and waist. All biomechanical data was sampled at 500 Hz after second-order low pass filtering around 30 Hz. To avoid variations in analog low pass filtering occuring across different signals, all signals were digitally low-pass filtered off-line at 25 Hz using a zero phase-shift 10th-order Butterworth filter.

Surface EMG electrodes were placed bilaterally, approximately 3 cm apart, along the muscle bellies of tibialis anterior (TA), soleus (SOL), vastus lateralis (QUAD) and paraspinal (PARAS) muscles. Electrodes were mounted on the paraspinals at the L1-L2 region of the spine. EMG amplifier gains were kept constant and pairs of electrodes and lead lengths assigned to individual muscles were not changed between subjects. EMG recordings were band-pass analog filtered between 60-600 Hz, full wave rectified, and low pass filtered at 100 Hz as recommended by Gottlieb and Agarwal (1979) prior to sampling at 1 KHz.

Data Analysis

Offline analysis was initiated by averaging subject EMG and biomechanical signals for each perturbation direction (5-6 stimuli per direction). For this purpose zero latency was defined as the first inflexion of ankle rotation velocity. We had previously determined that this did not vary with direction or subject (Carpenter et al. 1999). The average level of single subject background EMG activity for each muscle response recorded 100 ms prior to stimulus onset was subtracted from the remaining EMG signal from the same response. EMG areas were then calculated using trapezoid integration within pre-determined time intervals associated with previously identified stretch (40-100, 80-120 ms from stimulus onset), balance correcting (120-220 ms), secondary balance correcting (240-340 ms), and stabilizing reaction (350-700) responses (Carpenter et al. 1999). Response latencies for balance correcting responses were determined semi-automatically based on the following criterion: later than 90 ms, burst longer than 40 ms and a continuous amplitude of at least 2 standard deviations above the mean activity level prior to the stimulus onset. All biomechanical and muscular profiles were averaged across each direction and subject averages were pooled to produce population averages for a single direction (as shown in figures 2, 3, 8 and 9). Average trunk angular velocity was calculated over 60 ms during time intervals between 160-220, 240-300, 470-530 ms. Torque changes were calculated between 160-260 and 280-380 ms. All EMG areas, average trunk velocities and ankle torque changes were analyzed in a 2 x 8 (group by direction) repeated measures ANOVA. Significant main effects were explored using paired t-tests with a level of significance set maximally at 0.05.

RESULTS

Our description of normal responses compared to BVL patients has been separated into three sections. First, we present the effect of BVL on the timing and pattern of the muscle responses for differently directed perturbations. This comparison provides information concerning the onset of activation of stretch and subsequent balance correcting activity as well as differences in intramuscular co-ordination with respect to normal responses. Second, we report on the influence of perturbation direction, vestibular loss and their interaction on the amplitude of triggered balance correcting responses and subsequent stabilizing reactions. Finally we describe the biomechanical consequences in the form of ankle torques and trunk motion to

alterations in muscle activation patterns and amplitude modulation associated with BVL as a function of perturbation direction.

Timing and Muscle Coordination

Platform rotations induced a cascade of muscle activation patterns that were highly dependent on the direction of perturbation. Varying the direction of perturbation selectively stretched or unloaded particular muscle groups as well as bilateral pairs of muscles differently, depending on their relative orientation with respect to the axis of rotation. In general, muscles that were stretched by the perturbation generated relatively small balance correcting responses following the stretch reflex in the same muscle. Such action has functional significance as activation of stretched muscles would act to further destabilize the body in the direction of the initial fall. In contrast, muscles which were unloaded or released by the initial stimulus movement displayed the most prominent balance correcting responses. For specific comparisons between normal and BVL subjects on the effects of timing and pattern of response we describe, in more detail, the muscle activation profiles associated with platform rotations in two directions, backward to the right (135 deg) and forward to the right (45 deg). A more detailed and comprehensive description of normal responses through 16 different directions can be found in Carpenter et al. (1999).

Backward to the right

When the platform tips backward and to the right, the body moves in a multi-link fashion (figure 1 left). The uphill leg (left) is driven upwards by the elevated side of the platform while the lower leg falls simultaneously backwards to the right. Consequently, the coupling action at the hip causes the trunk to roll first to the left starting at approximately 30 ms, then pitch forwards at 50 ms. The initial roll is rapid, but the pitch is only rapid after 100 ms (see figure 7). The uphill leg buckles during the rapid trunk roll, flexing at the knee and ankle joint (see figure 1 left). As illustrated in figure 2, the initial dorsiflexion of the ankles causes a prominent stretch reflex in soleus (SOL) muscles of normal subjects at a latency of 54 ms. Shortly thereafter, small amplitude stretch reflexes in the right paraspinals (PARAS) occurred at a normal latency of 63 ms. The stretch reflexes in PARAS and SOL were followed by relatively small levels of automatic balance correcting activity (120-220 ms) in the same muscles. In

contrast, muscles unloaded by the perturbation, including right and left tibialis anterior (TA), right quadriceps (QUAD) and left PARAS demonstrated dominant balance correcting responses to counter rotation of the ankle, knee and hip respectively (figure 2). During the stabilizing period elevated levels of muscle activity in right TA, SOL and QUAD and left PARAS were employed to stabilize the trunk and the ankle and knee joints of the downhill leg to compensate for the new tilted orientation of the support surface.

BVL patients did not differ in the timing or pattern of muscle activity for perturbations backwards to the right. As observed in figure 2, BVL patients demonstrated similar latencies to that of normals for stretch reflexes in SOL and PARAS muscles. In muscles unloaded by the perturbation (left TA, right QUAD and left PARAS), the normal pattern of an initial inhibition followed by a prominent balance correcting response was replicated in pattern and timing by BVL subjects (figure 2). However, differences in the magnitude of the balance correcting response were observed with BVL subjects. For example, BVL subjects in that they generated only negligble balance correcting activity in soleus after the initial stretch reflex compared to normals (figure 2). Distinct differences in amplitude modulation were also observed in the stretched PARAS muscles. In the right PARAS muscle, large bursts of activity were recorded following the initial stretch response in BVL subjects but not in the normals.

Forward to the right

Platform rotations forward to the right were associated with stimulus induced body movements and corresponding muscle activation patterns which were distinctly different from those for backward right perturbations. Both knee joints were flexed by the forward rotation of the platform, while the trunk was rotated backward to the left (see figure 1 right). The forward rotation of the platform also pulled the ankles into plantarflexion while platform roll movements caused eversion of the left and inversion of the right ankle simultaneously. Stretch reflexes in left TA and right QUADS were elicited in normals with latencies of approximately 80 ms (figure 3). A small stretch reflex in the right PARAS of normal subjects could also be observed. Stretch reflexes in TA and right PARAS muscles were followed by minimal balance correcting activity in normals as this activity would act to further destabilize the body. In contrast, stretch reflexes in the QUADS were followed by a significant balance correcting

response to resist further flexion of the knee. Left PARAS demonstrated an unloading response, characterized by decreased activity below background levels, with a latency of approximately 40 ms (preceding any other stretch responses we had observed in response to support surface movements). As noted for backward right perturbations, muscles released by the perturbation including, left PARAS and right SOL muscles, demonstrated the primary balance correcting responses in normals (figure 3).

Vestibular loss subjects did not differ from normals in the onset of stretch reflexes or unloading reflexes (figure 3). BVL subjects exhibited distinct pattern differences during balance-correcting periods in both stretched and unloaded muscles. The balance correcting activity in the unloaded SOL muscles was not only reduced in amplitude, but was also delayed in onset for vestibular loss patients. Statistical comparisons using t-tests for onset latencies in right soleus between normals and BVL subjects revealed significant delays in BVL subjects for all forward directions. Onsets latencies of normal balance correcting responses in right SOL were 136.2+/- 15.8 ms for 0 deg, 140.6+/-23.7 ms for 45 deg and 140.9+/-27.2 ms 315 deg perturbations. Compared to normals, BVL subjects had significantly delayed balance correcting onsets in right SOL with average latencies of 181.2+/-19.4 ms for 0 deg ($p< 0.001$), 183.6+/-25.1 ms for 45 deg ($p< 0.013$) 179.1+/-18.5 ms for 315 deg ($p<0.022$). Similar differences were observed in the left soleus. In the stretched left and right TA and right PARAS muscles, BVL subjects had strong bursts of activity during the balance correcting period (120-220 ms) which was absent in normals. Such TA responses are clearly destabilizing by continuing the forward rotation of the lower leg (figure 3). BVL subjects had a normal pattern and magnitude of balance correcting response following stretch of the right QUADS. BVL subjects also demonstrated normal balance correcting activity in the unloaded left PARAS muscles.

Amplitude Modulation

Stretch reflexes (responses occuring between 40-120 ms)

There were no significant effects of *BVL* on the amplitude of stretch reflexes over the period we analysed (40-120 ms) in any of the postural muscles. There was a significant main effect for direction on stretch reflex amplitude for all muscles. As observed in the polar plots of the

stretch reflex amplitude in figure 4 and the responses in figures 2 and 3, different muscles were selectively stretched by perturbations of different directions. Stretch reflexes for right TA ($F(7,119)=37.7$, $p<0.0001$) were activated by directions ranging between 225 deg to 135 deg (clockwise notation) with a maximum activity vector at 338 deg for both normals and BVL patients. Right SOL ($F(7,119)=23.5$, $p<0.0001$) was stretched by toe up rotations, ranging between 135 and 225 deg with maximum activity vectors oriented close to 180 deg for normals and vestibular loss patients. PARAS were stretched by perturbations that caused pitch of the trunk forward and roll of the trunk away from the side of the PARAS muscle. Therefore, for the right PARAS ($F(7,119)=7.3$, $p<0.0001$) perturbations between 45 and 180 deg caused stretch reflexes with maximum stretch vectors calculated at ca 135 for normal and BVL subjects. Right QUADS ($F(7,119)=12.4$, $p<0.0001$) were stretched by toes-down perturbations causing flexion of the knee, with maximum activity at approximately 0 degrees for both groups. Similar significant effects were found for left sided muscles, with activation ranges and directions of maximum activity vectors which mirrored those reported above for right muscles (figure 4).

Balance correcting activity (responses occurring between 120-220 ms)

The amplitude of balance correcting activity measured between 120-200 ms was also significantly influenced by the direction of the perturbation with each muscle having clearly defined ranges of activation. Balance correcting activity in SOL, TA and QUADS muscles was oriented 180 deg from directions that elicited stretch reflexes in both normal and BVL subjects (figure 4).

In addition to the significant main effect of direction, balance correcting activity in TA (*right* $F(7,119)=7.44$, $p<0.0001$; left $F(7,119)=7.85$, $p<0.0001$) and SOL (*right* $F(7,119)=4.22$, $p<0.0003$; left $F(7,119)=2.65$, $p<0.0138$) muscles were significantly influenced by the interaction between perturbation direction and vestibular loss, independent of vision. That is, the effect of BVL on the amplitude of the balance correcting response in both SOL and TA muscles was dependent upon the direction of the perturbation (figures 4 and 5). The amplitudes for five directions and their standard deviations have been plotted on horizontal bar representations in the upper and lower part of figure 5 for TA and SOL, respectively, to

highlight the significant interaction between BVL and perturbation direction for these muscles. As observed in figure 5, for pure toe-up rotation (180 deg), balance correcting activity in TA was significantly lower for BVL patients compared to normals for both left and right muscles ($p<0.05$), and significantly lower in right TA when the perturbation was backwards to the right (135 deg). Likewise, the same trend of reduced amplitude response in BVL subjects was observed for left TA when the perturbation was backward to the left (figure 4). In contrast, when perturbations are composed of pure rotations to the right (90 deg) and forward roll right (45 deg), normal and BVL patients have similar amplitudes of balance correcting activity in TA. One unexpected finding was the significant differences between the responses of normal and *BVL* patients during the balance correcting period for 0 deg toes down perturbations. BVL patients showed significantly larger responses between 120-220 ms compared to normals, in both left and right TA ($p<0.05$) for the 0 deg direction (figure 5). It is of note that TA activity is minimal over the balance correcting period in normals during forward perturbations. Therefore the increased activity in BVL subjects would act, in addition to stretch reflex activity, to further destabilize the body in the direction of the perturbation. Soleus balance correcting activity was also influenced by a significant interaction between vestibular loss and perturbation direction. As observed in figure 5, the magnitude of this interaction was different for right and left muscles for rightward perturbations. For the right SOL (downhill leg), vestibular loss patients had signficantly lower balance correcting activity for all directions ($p<0.05$). Less significant differences were observed in the left SOL muscle on the uphill leg (figures 4 and 5). The largest differences between normals and vestibular loss patients were for toes-down and forward roll conditions, when the muscle has been initially unloaded by the perturbation ($p<0.01$). It must be noted that the decreased amplitude seen in SOL for forward perturbations may be partially explained by the delayed onset of this muscle. However, despite the delay, the peak response for both normals and BVL patients has been captured within the predetermined time window (120-220 ms) used for calculating the balance correcting response. Furthermore, comparisons between the peak amplitudes of the balance correcting responses in figure 3, confirms the reduced amplitude response in BVL patients which is independent of alterations in timing. For perturbations which initially stretch the SOL muscle (135 and 180 deg), vestibular loss patients also had significantly less activity in the right soleus compared to normals ($p<0.05$). Notably, in normals this activity is much smaller than for toe-down

perturbations. For the 0 deg direction, the left SOL, just as the right, was significantly reduced in vestibular loss patients compared to normals ($p<0.05$). Overall SOL responses were the most reduced of all balance correcting responses we measured. The maximum activity direction vector was associated with considerable variance in BVL subjects because of the effect of the reduced response amplitudes. Therefore the differences observed in figure 4 with respect to the directions of normal subjects were not significant.

Stabilizing reactions (responses occurring between 350-700 ms)

Consistent with both stretch and balance correcting responses, stabilizing reactions were significantly influenced by the direction of the perturbation. As observed in figure 6, stabilizing reactions in TA (right $F(7,119)=45.8$, $p<0.0001$; left $F(7,119)=28.22$, $p<0.0001$) had activation ranges and maximum activity vectors that correspond to earlier balance correcting responses. Soleus responses were similar in this respect (right $F(7,119)=30.3$, $p<0.0001$; left $F(7,119)=27.46$, $p<0.0001$). However, stabilizing reactions were also influenced by a three way interaction between perturbation direction, BVL and vision, for TA (right $F(7,119)=6.03$, $p<0.0001$; left $F(7,119)=3.80$, $p<0.0009$), QUADS (right $F(7,119)=5.06$, $p<0.0001$; left $F(7,119)=3.89$, $p<0.0008$) and PARAS (right $F(7,119)=2.93$, $p<0.0072$) (upper part figure 6). For normals, there is no difference between stabilizing reaction amplitudes for eyes open and eyes closed as perturbation direction changes for any of the muscles. However, vision does significantly affect stabilizing reactions in BVL subjects differently for different directions. For example, in both right TA (figure 6) and QUADS, as perturbation direction moved from forward right to backward right directions, BVL subjects had greater stabilizing amplitudes compared to normals (figure 6) and these responses were larger for eyes closed compared to eyes open conditions. However, for the pure toes up (180 deg) perturbation, there was a change in the BVL pattern, in which larger stabilizing reactions were observed during the eyes open compared to the eyes closed condition. For the right PARAS, BVL patients standing with eyes open demonstrated the largest stabilizing responses compared to eyes closed and normal responses. This effect remained for all perturbation directions contralateral to the right PARAS muscle (upper right figure 6). The net result of the changed amplitudes of stabilizing reactions and foregoing balance corrections in BVL subjects is shown in the lower half of figure 6. Trunk sway at 500 ms in BVL subjects was an order of magnitude larger than

for normals and roll perturbations yielded a backward rather than forward instability compared to normals as shown by the differently directed resultant velocity vectors.

Biomechanical Consequences

Ankle torques

Differences in ankle torque profiles between normals and BVL patients were primarily related to timing and rate of change for active torque responses. For backward perturbations, A-P ankle torque in normals initially dorsiflexes, then begins to plantarflex, beginning at 150 ms and reaching peak plantarflexion at 350 ms. BVL patients have a similar onset of plantarflexion compared to normals, however the rate of change is decreased. In addition, BVL patients do not reach peak plantarflexion torque until after 500 ms (figure 7). In forward perturbations, normal ankle torque is initially plantarflexing, followed by rapid dorsiflexion beginning at 120 ms and peaking at ca 375 ms (figure 8). For identical perturbations, BVL patients have a slightly extended period of dorsiflexion, followed by a decreased rate of plantarflexion which does not reach a peak before 500 ms. These differences can be easily observed in figure 9, where the ankle torque change between two different time periods is plotted for each perturbation direction. During the early period between 160-260 ms, significant interaction effects were found between group and direction for A-P ($F(7,119)=3.59$, $p<0.0015$) and M-L ($F(7,119)=2.30$, $p<0.0313$) ankle torque change. During this period, BVL patients had reduced A-P torque for perturbations with a pitch component, and reduced M-L torque for perturbations with a roll component (figure 9, upper right). A significant interaction between group and direction was also observed between 280-380 ms for A-P ($F(7,119)=4.25$, $p<0.0003$) and M-L ($F(7,119)=2.13$, $p<0.0457$) ankle torque change. During this latter period of time, normal torques have reached a plateau, whereas BVL torques continue to change. This results in relatively larger A-P torque changes in pitch directions and larger M-L torque changes in roll directions for BVL compared to normals (figure 9, upper left). Although the magnitude of torque change was altered in BVL patients compared to normals, the directional sensitivity of the torque response was maintained (see resultant vector plot in figure 9), with no differences in direction of the resultant torque vectors. The resultant torques remained oriented mainly in the pitch plane.

Trunk velocity

A common element of the trunk response, regardless of perturbation direction was an initial movement in a direction opposite to that of the support surface (figure 1 and figure 9, lower left). However, in response to perturbations with a roll component, initial trunk roll movements were observed 30 ms prior to any detected movements in the pitch direction (figure 7 and 8). Platform rotation to the right caused trunk roll to the left with peak velocities reaching 10 deg/s by 120 ms in normals. After this time, trunk roll slowed and the angular velocity changed direction after crossing zero velocity at 200 ms (figure 7 and 8). Initial roll velocities in BVL patients were slightly smaller in magnitude compared to normals, but took longer to bring under control, crossing zero around 300 ms. After 300 ms, BVL patients experienced large roll velocities in the opposite direction to initial platform induced trunk movements instead of near zero velocities of normal subjects (figures 6, 7 and 8). That is, the BVL patients tended to fall in the direction of platform movement.

All perturbations, including pure roll perturbations induced pitch movements of the trunk but pure pitch perturbations did not induce noticeable roll movements. For backward perturbations, initial forward pitch velocities were similar in magnitude for BVL and normals, however, BVL patients peaked earlier and changed direction earlier than normals (figure 7). BVL patients also had very large residual pitch velocities after 350 ms, which were opposite in direction to the initial pitch velocity, but in the same direction as platform movement (see figures 6 and 9). For forward perturbations, both the magnitude and timing of the trunk pitch velocity profile was different for BVL patients. For these perturbations, BVL patients experienced backward trunk velocities almost two times greater and peaked 80 ms later than normals. Once the backward rotation of the trunk was arrested it was followed by large overcorrecting 'stabilizing', response in BVL patients (figure 6) also in the direction of the initial platform motion, however without a falling tendency. As shown in figure 9, differences between BVL and normals extended to all perturbation directions. During the period between 160-220 ms, which primarily measures the stimulus induced rotation of the trunk, BVL patients had only slightly larger average trunk pitch velocities for perturbations with pitch and roll combinations (figure 9 lower left). During the later period between 240-300 ms, BVL patients had significantly larger ($F(1,17)=8.82$, $p<0.0086$) pitch velocities across all

perturbation directions (figure 9 lower right). Significant group by direction interaction effects were observed during the later period between 470-530 ms for both pitch trunk velocity ($F(7,119)=3.45$, $p<0.0021$) and trunk roll velocity ($F(7,119)=4.69$, $p<0.0001$). During this later period, BVL subjects had larger trunk pitch velocities for pitch oriented perturbations, and larger trunk roll velocities for roll oriented perturbations (figure 6, lower half).

Unlike the resultant direction of the ankle torque vectors which were oriented primarily along the pitch plane, the resultant trunk velocity vectors during the early period between 160-220 ms were oriented opposite to the direction of platform movement for both normal and BVL subjects (figure 9, lower left). The resultant trunk velocity vectors took on a slightly greater pitch orientation later during the measurement period 240-300 ms as indicated in figure 9. However, during this latter period, the orientation of the BVL vectors deviated from normal for pure roll and forward roll perturbations. Finally, over the stabilizing period when a steady state combined roll and pitch hip torque must be imposed to keep the trunk upright, pelvic torque was clearly insufficient for BVL subjects as they had continued motion in the perturbation direction (figure 6). Furthermore, for roll directions the orientation of trunk motion was still different from normal.

DISCUSSION

From its fastest (skiing) to most elegant (dancing) forms, human motion is constantly disturbed in multiple directions either by changes in surface orientation or external forces acting on the body. In all cases, the ability to roll the trunk and counter roll the legs by flexing the knees provides a crucial element of stability for motion. Previous research on human balance control has largely ignored this element by limiting analysis to a single planar model moving only in the pitch plane. Although important information has been discovered using single plane perturbation models, the results represent only a flat snapshot of the balance phenomenon without important three-dimensional detail, thereby preventing application to real life situations. Therefore, previous findings which have proven highly consistent in the pitch plane, may not represent normal responses to perturbations which occur in off-pitch planes. The first goal of the present study was to determine whether the current understanding of the effects of

BVL on postural reactions, established with uni-planar perturbations, can be applied to perturbations which contain both pitch and roll components.

The second goal of the experiment was to determine what new information, if any, is available with multidirectional perturbations, which would provide insights on how the CNS develops motor programs based on vestibular inputs when arresting falls in different directions. Due to the directional sensitivity of vestibular receptors, particularly the off-pitch orientation of the vertical semi-circular canals, it was hypothesized that a multi-directional posturographic paradigm would provide new insights about the focus of vestibular and proprioceptive contributions to balance control using the patient model of bilateral peripheral vestibular loss. Furthermore the different dynamics of some central vestibular neurons for roll and pitch perturbations (Angelaki et al. 2000) might be matched to the different dynamics of the trunk in these directions (Carpenter et al. 1999). With onsets as early as 15-20 ms, Carpenter et al. (1999) observed vertical linear and angular roll accelerations of the head, with magnitudes exceeding known vestibular thresholds (Benson et al. 1989; Gianna et al. 1996). In addition, the magnitude of the vertical and angular accelerations were dependent upon the direction of platform rotation and/or trunk motion. Information from otolith afferents (Tomko et al. 1981) and semi-circular receptors (Graf et al. 1993; Wilson et al. 1995) is integrated into a single directional signal which acts to drive ocular and cervical motor responses. Directional integration of information for balance control may also occur in higher neural centres such as the vestibular nuclei (Schor et al. 1984) and the cerebellum (Pompeiano et al. 1997). When directionally specific head acceleration information is not available, as is the case for bilateral peripheral vestibular loss, the behavioural deficits observed in dynamic postural control may be used to hypothesize the specific role of the vestibular system in triggering and/or modulating appropriately scaled, directionally- sensitive balance responses. If central vestibular neurons play a major role in coordinating roll and pitch movements of the trunk, the loss or reduced effect of the neural control exercised by these neurons can be expected to lead to uncoordinated trunk control.

Previous posturography experiments using only pitch plane perturbations have uncovered consistent differences in balance control between patients with BVL and normals. BVL

patients have normal onset latencies of both stretch reflexes and automatic balance correcting responses to pitch directed translation (Runge et al. 1998; Horak et al. 1990; Allum et al. 1994), rotation (Allum and Pfaltz 1985; Allum et al. 1994), and combined translation/rotation (Allum and Honegger, 1998) of the support surface. Despite normal onset, the amplitude of automatic balance corrections between 120-220 ms in TA, SOL and QUAD muscles was found to be significantly decreased in BVL (Allum and Pfaltz 1985; Keshner et al. 1987; Allum et al. 1994; Allum and Honegger 1998; Runge et al. 1998). Slower rate of change in A-P ankle torque generation in BVL patients (Allum and Pfaltz 1985, Keshner et al. 1987) has been shown to be correlated with the decreased amplitude of lower leg automatic responses in BVL patients. Decreased balance correcting responses in postural leg muscles are typically followed by excessive activity in paraspinal muscles after 200 ms. (Allum et al. 1994, Runge et al. 1998). The increased trunk activity during this later period corresponds to a significantly larger trunk pitch velocity in BVL which persist longer than that of normals.

We have determined that the findings uncovered by pitch plane perturbations do in fact apply to perturbations which include both pitch and roll components. Specifically this applies to leg muscle responses whose direction of maximum activation lies primarily along the pitch plane. We have observed similar effects in BVL to that of previous uni-directional studies for perturbations in both pure pitch, as well as pitch and roll directions. The timing and amplitude of early stretch reflexes are normal in BVL across all directions (figures 2, 3 and 5). As observed in figures 2 and 3, there were no observable differences in the timing or pattern of the balance correcting responses between BVL patients and normals, with the exception of SOL. For soleus, muscle activity was significantly delayed by 38-45 ms for all toe-down perturbations. The amplitude of balance correcting activity in TA and SOL was significantly reduced in BVL patients for both pitch and pitch/roll directions (figure 4 and 5). This attenuation of lower leg balance correcting activity was followed by a decreased rate of A-P *ankle* torque production, and longer time to peak in BVL compared to normals for both pitch and off-pitch perturbations. Also similar to uni-directional perturbations, BVL patients demonstrated excessive muscular activity during the stabilizing period between 350-700 ms in TA, QUADS and PARAS, for pitch and off pitch perturbations (figures 2, 3 and 6). Finally, BVL patients experienced significantly larger average trunk pitch velocities compared to

normals as early as 240-340 ms following perturbation onset, which persisted between 470-530 ms, when normal subjects experience small residual trunk motion (figure 7). Inglis and Macpherson (1995) also observed normal timing and pattern of muscle activation. Their responses, however, were accompanied by increased response amplitudes of postural muscles in labrynthecomized cats following multi-directional translations. At first this observation seems contradictory to our observations of decreased amplitude balance correcting activity in lower leg muscles with BVL. However, considering that translational perturbations stretch the same muscle responsible for the balance correction, these findings, do in fact coincide with our observations of increased destabilizing activity over the balance-correcting measurement period in TA muscles that were initially stretched by the perturbation (figures 3 and 5).

There are a number of other similarities and differences in findings between the present and other multidirectional studies for normals which should be highlighted as they may influence the interpretation of results pertaining to the effects of bilateral vestibular loss (BVL). The range of activation of erector spinae and vastus medialis reported by Henry et al. (1998a) is similar to the ranges we observed in left paraspinals and left vastus lateralis (quadriceps) responding to rotational directions that would elicit comparable body sway as that induced by a translational perturbation. A preponderance of pitch oriented lower leg muscle activity, specifically noted in SOL and TA (see figure 4), was also observed by Henry et al. (1998a) and Moore et al. (1988). However, there are differences between the present and previous findings concerning the direction of maximum activity vectors. Henry et al. (1998a) report maximal activity in TA and medial gastrocnemius muscles to be oriented at approximately 60 and 300 degrees respectively, whereas in the present study, maximal balance correcting activity in right TA and SOL was more pitch-oriented at 186 and 35 degrees, respectively (see figure 4) very similar to the direction of balance correcting torque for the same foot (figure 9). Since the use of translational perturbations induces stretch and balance correcting activity in the same muscle (Diener et al. 1983; Allum et al. 1993), the constant long time-frame (70-270 ms after platform onset) which was used to observe integrated EMG areas by Henry et al. (1998a) must have captured components of both early stretch and later triggered balance correcting responses. In contrast, rotational studies such as the present study, elicit stretch and triggered balance correcting responses in antagonistic muscles for a single perturbation (Diener

et al. 1983; Allum et al. 1993). This approach, coupled with the use of consecutive time intervals to measure stretch (40-100 ms) and balance-correcting responses (120-220 ms) permits stretch reflexes to be observed in TA and SOL muscles which were oriented approximately 180 degrees to balance correcting activity in the same muscle (compare upper and lower plots in left side of figure 4). In contrast, PARAS balance correcting activity is oriented 90 degrees to the most sensitive directions for stretch reflexes and the amplitude of the PARAS stretch reflex compared to balance correcting activity is smaller than in lower muscles (Carpenter et al, 1999).

A non-vestibular and non-lower leg proprioceptive origin of a postural trigger for balance corrections was originally proposed by Forsberg and Hirschfeld (1994). This finding has recently been verified by observation of unaltered balance correcting response latencies to 'nulled ankle input' responses of healthy normals to combined backward translation and downward rotation, and patients with either selective lower leg proprioceptive loss (Bloem et al. 2000) or bilateral vestibular loss (Allum and Honegger 1998). The absence of a vestibular based postural trigger was extended to all pitch and roll directions of rotation in the present study with the important exception of the soleus response to forward pitch and roll rotations. It is interesting to note that SOL, the only muscle to date for which a change in latency and the largest change in amplitude has been observed following vestibular loss, is also a muscle which plays a major role in arresting a vertical fall (Greenwood and Hopkins 1976; Melvell Jones and Watt 1971; Wicke and Oman 1982). Vertical falling, while either sitting or standing, is associated with an initial early startle response in all muscles, followed by a second burst of activity, between 70-120ms which is confined to lower-limb extensors in triceps surae muscles in falling humans (Greenwood and Hopkins 1976), baboons (Lacour et al. 1978; Lacour et al. 1983) and cats (Watt 1976). The amplitude of the second burst has been shown to be modified by bilateral vestibular loss (Lacour, 1978) as well as experience. The response amplitude decreases with multiple exposure (Lacour 1978). Similar modulating characteristics with respect to vestibular loss (Allum and Pfaltz 1984; Allum et al.1994), and experience (Horak et al. 1989; Beckley et al. 1991) have been shown in leg muscles to unexpected movements of the support surface, providing a common ground from which shared neural mechanisms may be inferred. According to Watt (1981), the vestibulo-spinal reflexes, observed in falling studies,

would be suitable to contribute to ankle extensor muscles during locomotion, and presumably during postural reactions. However, it must be acknowledged that unlike the selective activation of vertically oriented otoliths affected in falling studies, our rotational perturbations involve very early (15-20 ms onset), vertical linear and roll angular accelerations of the head which are sensitive to both direction of perturbation (Carpenter et al. 1999) and reduced stimulus velocity (Carpenter et al. unpublished *observations) that will* simultaneously activate a variety of receptors at the head including semi-circular canals, otoliths and proprioceptive receptors in the neck. As off-pitch components are added to the perturbation direction, head vertical accelerations are decreased and roll angular accelerations are increased. For example, when the platform rotates forward to the left, head vertical acceleration is directed downward and head roll acceleration is directed to the right both in normals and BVL subjects (Carpenter et al. 1999, Bloem et al. 2001). Other authors have also reported early, and directionally discriminating, head accelerations measured during pitch plane rotations (Allum and Pfaltz 1985; Forssberg and Hirschfeld 1994) and translations (Allum et al. 1993; Runge et al. 1998) of the support surface. However, none of these previous experiments have measured head linear and angular accelerations along several axes as we have done (Carpenter et al. 1999, Bloem et al. 2001) in order to parse out those accelerations showing the greatest sensitivity to perturbation direction. In searching for possible control mechanisms by which vestibular-based modulation of muscles may be achieved especially by head roll accelerations, important clues may be drawn from studies examining postural reactions in subjects with unilateral vestibular loss (Carpenter et al. unpublished observations).

Our observations of combined pitch and roll surface rotation in BVL subjects support the notion that automatic balance correcting movements characterized by flexion of the contra-lateral *"uphill"* leg (generated by several muscles including tibialis anterior) and extension of the ipsilateral *"downhill"* stance-bearing leg to platform rotation (assisted by soleus activity) are driven by vestibulo-spinal inputs induced by head roll and linear accelerations. This movement pattern is not quite consistent with that associated with vestibulo-spinal reflexes in the cat elicited when the head is rolled to the side (Wilson et al. 1986). In the cat vestibulo-spinal reflexes involve extension of the ipsilateral limbs to head roll, and flexion of the contralateral limbs (Wilson and Melville-Jones 1979), such that when the head is rolled to the

left, with right ear up in relation to the body, the left limbs are extended while the right limbs are flexed. Extension of the ipsilateral limbs is achieved by facilitory input from the lateral vestibular nucleus to ipsilateral extensor muscles with simultaneous inhibition of ipsilateral flexors via the medial reticulo-spinal neurons (Lund and Pompeiano 1968). Cervico-collic reflexes act in opposition to vestibulo-spinal reflexes, so that, when the head is rolled to the left, with right ear up in relation to the body, the right limbs are extended and the left limbs are flexed. In both these cases, it may be hypothesized that a loss of vestibular input would have the most pronounced effects on the amplitude modulation of ipsilateral extensor muscles in response to unexpected rotation of the platform. The results of the present study seem to be correlated with expected behaviour predicted from neurophysiological cat experiments as long as one takes into account a major hinging at the pelvis that occurs in man when the body is rolled via a support surface. As observed in figure 5, the most dramatic effects of decreased balance correcting activity in soleus due to BVL occurs for muscles that are contra-lateral to the side of head rotation, but ipsilateral to pelvis rotation.

Our results have succeeded in demonstrating that the effects of BVL on postural control in leg muscles observed with pitch plane perturbations comprise elements of postural control required in each leg when support surface tilts contain both pitch and roll directions. These results are directed towards our second goal which was to determine what additional information, if any, might be yielded from a multi-directional paradigm which can be used to expand our present understanding of the effects of BVL on postural reactions. Similar to differences observed for A-P ankle torque in pitch directed perturbations, significant differences in lateral ankle torque change for BVL were observed for perturbations which contained a roll component. As observed in figure 9, lateral torque was significantly smaller in BVL for roll directions between 160-260 ms, and significantly larger in roll directions between 280-380 ms. Although changes in the magnitude of ankle torque change was altered by BVL, the relative contribution of A-P and lateral torque to a given perturbation was maintained, as observed by the normally oriented resultant ankle torque vectors (figure 9). Based on the assumption that the CNS controls pitch and roll torques separately (Winter et al. 1996; Matjacic et al. 2000), our observations would indicate that the co-ordination between these separate control systems is maintained in BVL for ankle torques but not for hip torques for

reasons described below.

Significant differences in trunk roll velocities between normals and BVL were also revealed exclusively by roll directed perturbations (figure 6 and 9). When roll components were added to the perturbation, BVL patients experienced smaller (although statistically insignificant) initial trunk roll velocities compared to normal between 160-220 ms, followed by significantly larger trunk roll velocities during the periods between 240-300 ms and 470-530 ms. We would hypothesize that this may be due to insufficient amplitudes in early hip muscular activity (From which we have yet to record – it is readily acknowledged by the authors that other hip muscles, from which we have not recorded, may also be influenced by BVL) but more prominantly due to excessive trunk muscle activity during the later stabilizing period, as we have shown in the present study (figures 2, 3, 6) and in previous pitch plane experiments (Allum et al. 1994). These findings suggest that, while patients are able to achieve directionally modulated, although delayed, trunk corrections in the pitch plane with respect to normal, there is a diminished capacity to maintain appropriate control of direction, timing and magnitude of trunk movements and corresponding hip torques in the roll plane. These findings in roll perturbations collaborate with well known clinical findings of lateral instability in BVL patients performing clinical balance tasks which require lateral control of the center of mass with lateral hip torques such as tandem walking, standing on one leg or walking while rotating the head (Allum et al. 2001 a).

Roll instability of the trunk bears directly on the issue that has been unresolved by previous pitch plane studies concerning the relationship between vestibular loss and control of postural hip movements. Horak et al. (1990) postulated an inability of vestibular loss patients to generate hip movement strategies while standing on a narrow support surface, possibly related to alterations in the timing metrics associated with hip torque generation (Allum et al. 1992;1997). In contrast, both Allum et al. (1992; 1997) and Runge et al. (1998) have demonstrated that in the pitch plane, vestibular loss subjects are able to generate appropriately sized hip torque amplitudes even for high perturbation velocities. It is the timing metrics of the pitch plane torques, being progressively delayed throughout the responses in vestibular loss subjects (Allum and Honegger 1992; Allum et al. 1997), which cause these subjects to have

excessive velocities and to fall. The results of the present study suggest that trunk roll movements associated with BVL are delayed and excessive too but with different metrics than the pitch delays. This and previous studies (Allum et al. 1994; Allum and Honegger 1998) have provided evidence that vestibular modulation of trunk responses is predominantly later than modulation in the leg muscles (figures 2 and 3) and trunk roll modulation is even later than that of pitch. One reason for this could well be linked to the early biomechanical response of the trunk in roll compared to pitch (Carpenter et al, 1999) and possibly the marked response differences of central vestibular neurons to different directions of head tilt (Angelaki and Dickman 2000). Balance corrections probably can influence those in the pitch direction as these are occurring, but only partially brake those in roll. Another reason for this may be due to the inhibitory nature of trunk roll control via paraspinal muscles. We assume that in BVL subjects, the excessive activity in paraspinal muscles ipsilateral to platform tilt direction is the result of an absence of inhibitory control by vestibulo-spinal pathways. This excessive activity causes the body to be "pulled" downhill following the tilt of the support surface. During the stabilizing period, between 470-530 ms, normal subjects experience small residual trunk motion following backwards support-surface rotations with a roll component. BVL patients however, exhibit excessive backward pitch and roll velocities (figure 7) related to hip roll torque generation as a result of further excessive paraspinal activity needed in uphill muscle during later stabilizing periods (figures 2 and 6) to avoid falling. Noticably this activity is larger when visual input are present (figure 6). Thus when roll components are added to the perturbation, BVL patients experience initially similar roll velocities to those of normals followed by significantly larger average roll velocities between the period of 240-300 ms and 470-530 ms because paraspinal muscle responses with backwards roll directions of maximum activity are enhanced due to a lack of inhibitory vestibulo-spinal control.

By recording responses under both eyes-open and eyes-closed conditions we were in a position to investigate whether BVL subjects can better utilize visual information to compensate for lack of balance related vestibular information. Normals did not demonstrate any significant differences between eyes open and eyes closed conditions in any direction for either onset or amplitude of stretch, balance correcting or later stabilizing reactions for any of the postural muscles recorded. These findings are consistent with previous research that has shown similar

responses to unexpected perturbations in normals when standing with eyes open and closed (Vidal et al. 1982). Vestibular loss patients also demonstrated, with the exception of soleus, similar onset and amplitude of stretch and balance correcting activity in all muscles and directions for eyes open compared to eyes closed conditions. However, during the stabilizing period between 350-700 ms, vision significantly interacted with direction in BVL patients. For pure roll and backward roll perturbations, BVL patients had stabilizing reactions in TA and QUADS which were reduced in eyes open compared to eyes closed conditions. In contrast, during pure toe-up perturbations, BVL patients demonstrated larger stabilizing activity in TA and QUADS for eyes open compared to eyes closed conditions (figure 6). The modulatory effect of vision on vestibular induced postural responses has been previously demonstrated in studies on falls (Vidal et al. 1979), and the present results suggest that BVL patients attempt to use vision to compensate for earlier consequences of absent vestibular input. However, the direction-dependence for use of vision in BVL has not been previously demonstrated and may pose interesting questions on the differential use of visual inputs for pitch and roll control which require further investigation. It might be hypothesized that roll and backward roll perturbations, which are associated with significant angular roll and lateral head accelerations (Carpenter et al. 1999), would require an intact and multi-directional acting VOR reflex to maintain multi-dimensional gaze on a fixation point and provide useful information to make a visually based compensation to postural response. In this regard, BVL patients would not be able to accurately maintain gaze on a visual target and make appropriate postural adjustments based on visual input. Thus, lower stabilizing responses may be expected in the eyes open compared to eyes closed conditions. In contrast, pure toe-up perturbations do not induce significant head roll or lateral head accelerations (Carpenter et al. 1999) that would complicate estimation of head movement in BVL patients using visual and neck proprioceptive inputs. Interestingly, right PARAS demonstrated increased activity for eyes open compared to eyes closed conditions for all directions except pure roll to the right (figure 6). Such an observation may suggest a greater role of trunk muscle proprioceptors in establishing appropriate head-trunk co-ordination in the roll plane. These possibilities are only speculative at best and definitely require further investigation.

In conclusion, roll directed disturbances to equilibrium, provided by multidirectional perturbations, are necessary to fully comprehend the extent to which bilateral vestibular loss influences normal postural reactions. Multi-directional perturbations were used to identify observable differences in muscle activation profiles, and particularly differences in trunk and ankle torque control with bilateral vestibular loss that were not previously observed using only pitch plane perturbations. Part of the reason for this may be due to fundamentally earlier hinging of the trunk around the pelvis which occurs with roll compared to pitch. Not only does this have consequences with respect to sensing of centre of mass motion by vestibular sensors, but also with respect to the need for different response dynamics of trunk roll and pitch motion in order to regain upright stance. It is perhaps for these reasons that leg muscle control by vestibulo-spinal system in man appears to be different from that of the trunk where inhibitory vestibulo-spinal effects seem to dominate. Future neurophysiological research should be dedicated to extending the implications of these observations on balance control of the trunk in the roll plane and examining the contribution of central and peripheral mechanisms to the different dynamics of balance control in the roll and pitch planes.

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REFERENCES

Allum JHJ, Pfaltz CR (1984) Influence of bilateral and acute unilateral peripheral vestibular deficits on early sway stabilizing responses in human tibialis anterior muscles. Acta Otolaryngol (Stockh) 406:115-119

Allum JHJ, Pfaltz CR (1985) Visual and vestibular contributions to pitch sway stabilization in the ankle muscles of normals and patients with bilateral peripheral vestibular deficits. Exp Brain Res 58:82-94.

Allum JHJ, Honegger F (1992) A postural model of balance-correcting movement strategies. J Vestib Res 2(4): 323-347

Allum JH, Honegger F, Schicks H (1993) Vestibular and proprioceptive modulation of postural synergies in normal subjects. J Vestib Res 3:59-85.

Allum JHJ, Honegger F, Schicks H (1994) The influence of a bilateral peripheral vestibular deficit on postural synergies. J Vestib Res 4:49-70

Allum JHJ, Huwiler M, Honegger F (1996) Prior intention to mimic a balance disorder: Does central set influence normal balance-correcting responses. Gait and Posture 4: 39-51

Allum JHJ, Gresty M, Keshner EA, Shupert C (1997) The control of head movements during human balance corrections. J Vestib Res 7: 189-218

Allum JHJ, Honegger F (1998) Interactions between vestibular and proprioceptive inputs triggering and modulating human balance-correcting responses differ across muscles. Exp Brain Res 121:478-494

Allum JHJ, Ledin T (1999) Recovery of vestibulo-ocular function in subjects with acute peripheral vestibular loss. J Vest Res 9: 135-144

Allum JHJ, Shepard NT (1999) An overview of the clinical use of dynamic posturography in the differential diagnosis of balance disorders. J Vestib Res 9:223-52

Allum JHJ, Held-Ziolkowska M, Adkin AL, Carpenter MG, Honegger F (2001a) Trunk sway measures of postural stability during clinical balance tests. Gait and Posture (in press)

Allum JHJ, Bloem BR, Carpenter MG, Honegger F (2001b) Differential diagnosis of proprioceptive and vestibular deficits using dynamic support-surface posturography. Gait and Posture (in press)

Angelaki DE, Dickman JD (2000) Spatio temporal processing of linear acceleration: primary afferent and central vestibular neuron responses. J Neurophysiol 84: 2113-2132

Beckley DJ, Bloem BR, Remler MP, Roos RAC, Van Dijk JG (1991) Long latency postural responses are functionally modified by cognitive set. Electroenceph Clin Neurophysiol 81: 353-358

Benson AJ, Hutt ECB, Brown SF (1989) Thresholds for the perception of whole-body angular movement about a vertical axis. Aviat Space Environ Med 60:205-213

Bloem BR, Beckley DJ, van Dijk JG (1999) Are automatic postural responses in patients with Parkinson's disease abnormal due to their stooped posture? Exp Brain Res 124:481-488

Bloem BR, Allum JH, Carpenter MG, Honegger (2000) Is lower leg proprioception essential for triggering human automatic postural responses? Exp Brain Res. 30:375-91

Bloem BR, Allum JHJ, Carpenter MG (2001) Triggering of balance corrections and compensatory strategies in a deafferented patient. Submitted to Exp Brain Res

Bronstein AM, Guerraz M (1999) Visual-vestibular control of posture and gait: physiological mechanisms and disorders. Curr Opin Neurol 12:5-11

Carpenter MG, Allum JHJ, Honegger, F (1999) Directional sensitivity of stretch reflexes and balance corrections for normal subjects in the roll and pitch planes. Exp Brain Res 129:93-113

Cordo PJ, Nashner LM (1982) Properties of postural adjustments associated with rapid arm movements. J Neurophysiol 47:287-302

Diener HC, Bootz F, Dichgans J, Bruzek W (1983) Variability of postural "reflexes" in humans. Exp Brain Res 52: 423-428

Diener HC, Dichgans J, Bootz F, Bacher M (1984) Early stabilization of human posture after a sudden disturbance: influence of rate and amplitude of displacement. Exp Brain Res 56:126-134

Diener HC, Horak F, Stelmach G, Guschlbauer B, Dichgans J (1991) Direction and amplitude precuing has no effect on automatic posture responses. Exp Brain Res 84:219-223

Di Fabio RP (1995) Sensitivity and specificity of platform posturography for identifying patients with vestibular dysfunction. Phys Ther. 75:290-305

Di Fabio RP, Emasithi A, Paul S. (1998) Validity of visual stabilization conditions used with computerized dynamic platform posturography. Acta Otolaryngol (Stockh) 118:449-54.

Forssberg H, Hirschfeld H (1994) Postural adjustments in sitting humans following external perturbations: muscle activity and kinematics. Exp Brain Res 97: 515-527

Furman JM (1995) Role of posturography in the management of vestibular patients. Otolaryngol Head Neck Surg 112:8-15

Gianna C, Heimbrand S, Gresty M (1996) Thresholds for detection of motion direction during passive lateral whole-body acceleration in normal subjects and patients with bilateral loss of

labyrinthine function. Brain Res Bull. 40:443-7

Gill J, Allum JHJ, Carpenter MG, Held-Ziolkowska M, Adkin AL, Honegger F, Pierchala K (2001) Trunk sway measures of postural stability during clinical balance tests: effects of age. J Gerontol A Biol Sci Med Sci. (In Press)

Gilles M, Wing AM, Kirker GB (1999) Lateral balance organisation in human stance in response to a random or predictable perturbation. Exp Brain Res 124:137-144

Gottlieb GL, Agarwal GC (1979) Response to sudden torques about ankle in man: Myotatic reflex. J Neurophysiol 42: 91-106

Graf W, Baker J, Peterson BW. (1993) Sensorimotor transformation in the cat's vestibuloocular reflex system. I. Neuronal signals coding spatial coordination of compensatory eye movements. J Neurophysiol 70:2425-2441

Greenwood R, Hopkins A (1976) Muscle responses during sudden falls in man. J Physiol Lond 254:507-518

Henry SM, Fung J, Horak FB (1998a) EMG responses to maintain stance during multidirection surface translations. J Neurophysiol 80:1939-1950

Henry SM, Fung J, Horak FB (1998b) Control of stance during lateral and anterior/posterior surface translations. IEEE Trans Rehabil Eng 6:32-42

Hirschfeld H, Forssberg H (1994) Epigenetic development of postural responses for sitting during infancy. Exp Brain Res 97: 528-540

Holliday PJ, Fernie GR, Gryfe CI, Griggs GT (1990) Video recording of spontaneous falls of the elderly. In: Gray BE (ed) Slips, Stumbles and Falls: Pedestrian Footwear and Surfaces, American Society for Testing Materials, Philadelphia: pp 7-16

Horak FB, Nashner LM (1986) Central programming of postural movements: Adaptation to altered support-surface configurations. J Neurophysiol 55: 1369-1381

Horak FB, Diener HC, Nashner LM (1989) Influence of central set on human postural responses. J Neurophysiol 62:841-853

Horak FB, Nashner LM, Diener HC (1990) Postural strategies associated with somatosensory and vestibular loss. Exp Brain Res 82: 167-177

Horak FB, Moore SP (1993) The effect of prior leaning on human postural responses. Gait and Posture 1: 203-210

Horak FB, Shupert CL, Dietz V, Horstmann G (1994) Vestibular and somatosensory contributions to responses to head and body displacements in stance. Exp Brain Res 100:93-106.

Horak FB, Lamarre Y, Macpherson JM, Jones C, Henry SM (1996) Postural control in a patient with total body somatosensory loss. Society for Neuroscience 22: 1632 (abstract).

Horak FB, Henry SM, Shumway-Cook A (1997) Postural perturbations: new insights for treatment of balance disorders. Phys Ther 77:517-533

Inglis JT, Horak FB, Shupert CL, Jones-Rycewicz C (1994) The importance of somatosensory information in triggering and scaling automatic postural responses in humans. Exp Brain Res 101(1): 159-164

Inglis JT, Macpherson JM. (1995) Bilateral labyrinthectomy in the cat: effects on the postural response to translation. J Neurophysiol 73:1181-91.

Keshner EA, Allum JH, Pfaltz CR (1987) Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit. Exp Brain Res 69: 77-92

Lacour M, Xerri C, Hugon M (1978) Muscle responses and monosynaptic reflexes in falling monkey. Role of the vestibular system J Physiol (Paris) 74:427-438

Lacour M, Vidal PP, Xerri C (1983) Dynamic characteristics of vestibular and visual control of rapid postural adjustments. Adv Neurol 39:589-605

Lipp M, Longridge NS (1994) Computerised dynamic posturography: its place in the evaluation of patients with dizziness and imbalance. J Otolaryngol 23:177-183

Lund S, Pompeiano O (1968) Monosynaptic excitation of alpha motoneurones from supraspinal structures in the cat. Acta Physiol Scand 73:1-21

Macpherson JM (1988a) Strategies that simplify the control of quadrupedal stance. I. Forces at the ground. J Neurophysiol 60: 204-217

Macpherson JM (1988b) Strategies that simplify the control of quadrupedal stance. II. Electromyographic activity. J Neurophysiol 60: 218-231

Macpherson JM, Horak FB, Dunbar DC, Dow RS (1989) Stance dependence of automatic postural adjustments in humans. Exp Brain Res 78: 557-566

Macpherson JM (1994) The force constraint strategy for stance is independent of prior experience. Exp Brain Res 101: 397-405.

Maki BE, Whitelaw RS (1993) Influence of expectation and arousal on center-of-pressure responses to transient postural perturbations. J Vestib Res 3: 25-39

Maki BE, McIlroy WE, Perry SD (1994) Compensatory responses to multi-directional perturbations. In: Taguchi K, Igarashi M, Mori S (eds) Vestibular and Neural Front, Elsevier Science, Amsterdam, pp 437-440

Maki BE, McIlroy WE (1996) Postural control in the older adult. Clinics in Geriatric Medicine 12:635-658

Maki BE and McIlroy WE (1998) Control of compensatory stepping reactions: Age-related impairment and the potential for remedial intervention. Physiotherapy Theory and Practice 15:69-90

Matjacic Z, Voigt M, Popovic D, Sinkjaer T (2000) Functional postural responses after perturbations in multiple directions in a standing man revealed by net joint torques: A principle of decoupled control. J Biomech (In press).

Melvill Jones G, Watt DGD (1971) Muscular control of landing from unexpected falls in man. J Physiol Lond 219:729-737

Moore SP, Rushmer DS, Windus SL, Nashner LM (1988) Human automatic postural responses: responses to horizontal perturbations of stance in multiple directions. Exp Brain Res 73:648-658

Nashner L, Berthoz A (1978) Visual contribution to rapid motor responses during postural control. Brain Res. 150:403-7

Nashner LM, Black FO, Wall C III (1982) Adaptation to altered support surface and visual conditions during stance: patients with vestibular deficits. J Neuroscience 2: 536-544

Nashner LM, McCollum G (1985) The organization of human postural movements: a formal basis and experimental synthesis. Behav Brain Sci 8:135-172

Pompeiano O, Andre P, Manzoni D (1997) Spatiotemporal response properties of cerebellar purkinje cells to animal displacement: a population analysis. Neuroscience 81:609-626

Rietdyk S, Patla AE, Winter DA, Ishac MG, Little CE (1999) NACOB presentation CSB New Investigator Award. Balance recovery from medio-lateral perturbations of the upper body during standing. J Biomech 32:1149-1158

Runge CF, Shupert CL, Horak FB, Zajac FE (1998) Role of vestibular information in initiation of rapid postural responses. Exp Brain Res 122:403-412

Rushmer DS, Russell CJ, Macpherson J, Phillips JO, Dunbar DC (1983) Automatic postural responses in the cat: responses to headward and tailward translation. Exp Brain Res 50:45-61

Rushmer DS, Moore SP, Windus SL, Russell CJ (1988) Automatic postural responses in the cat: responses of hindlimb muscles to horizontal perturbations of stance in multiple directions. Exp Brain Res 71:93-102

Schieppati M, Nardone A, Siliotto R, Grasso M (1995) Early and late stretch responses of human foot muscles induced by perturbation of stance. Exp Brain Res 105: 411-422

Schor RH, Miller AD, Tomko DL (1984) Responses to head tilt in cat central vestibular neurons. I. Direction of maximum sensitivity. J Neurophysiol 51:136-146

Timmann D, Belting C, Schwarz M, Diener HC. (1994) Influence of visual and somatosensory input on leg EMG responses in dynamic posturography in normals. Electroencephalogr Clin Neurophysiol 93:7-14.

Tomko DL, Peterka RJ Schor RH (1981) Responses to head tilt in cat eigth nerve afferents. Exp Brain Res 41:216-221

Vidal PP, Lacour M, Berthoz A (1979) Contribution of vision to muscle responses in monkey during free-fall: visual stabilization decreases vestibular dependent responses. Exp Brain Res 37:241-252

Vidal PP, Berthoz A, Millanvoye M (1982) Difference between eye closure and visual stabilization in the control of posture in man. Aviat Space Environ Med 53:166-170

Watt DGD (1976) Responses of cats to sudden falls: an otolith originating reflex assisting landing. J Neurophysiol 39:257-265

Watt DG (1981) Effect of vertical linear acceleration on H-reflex in decerebrate cat. I. Transient stimuli. J Neurophysiol 45:644-655

Wicke RW, Oman CM (1982) Visual and graviceptive influences on lower leg EMG activitz in humans during brief falls. Exp Brain Res 46:324-330

Winter DA, Prince F, Frank JS, Powell C, Zabjek KF (1996) Unified theory regarding A/P and M/L balance in quiet stance. J Neurophysiol 75:2334-43

Winter DA, Patla AE, Prince F, Ishac M, Gielo-Perczak K (1998) Stiffness control of balance in quiet standing. J Neurophysiol 80:1211-1221.

Wilson VJ and Melvill Jones G (1979) Mammalian Vestibular Physiology, Plenium, New York pp 239-245

Wilson VJ, Schor RH, Suzuki I, Parks BR (1986) Spatial organization of neck and vestibular reflexes acting on the forelimbs of the decerebrate cat. J Neurophysiol 55:514-526

Wilson VJ, Boyle R, Fukushima K, Rose PK, Shinoda Y, Sugiuchi Y, Uchino Y (1995) The vestibulocollic reflex. J Vestib Res 5:147-170

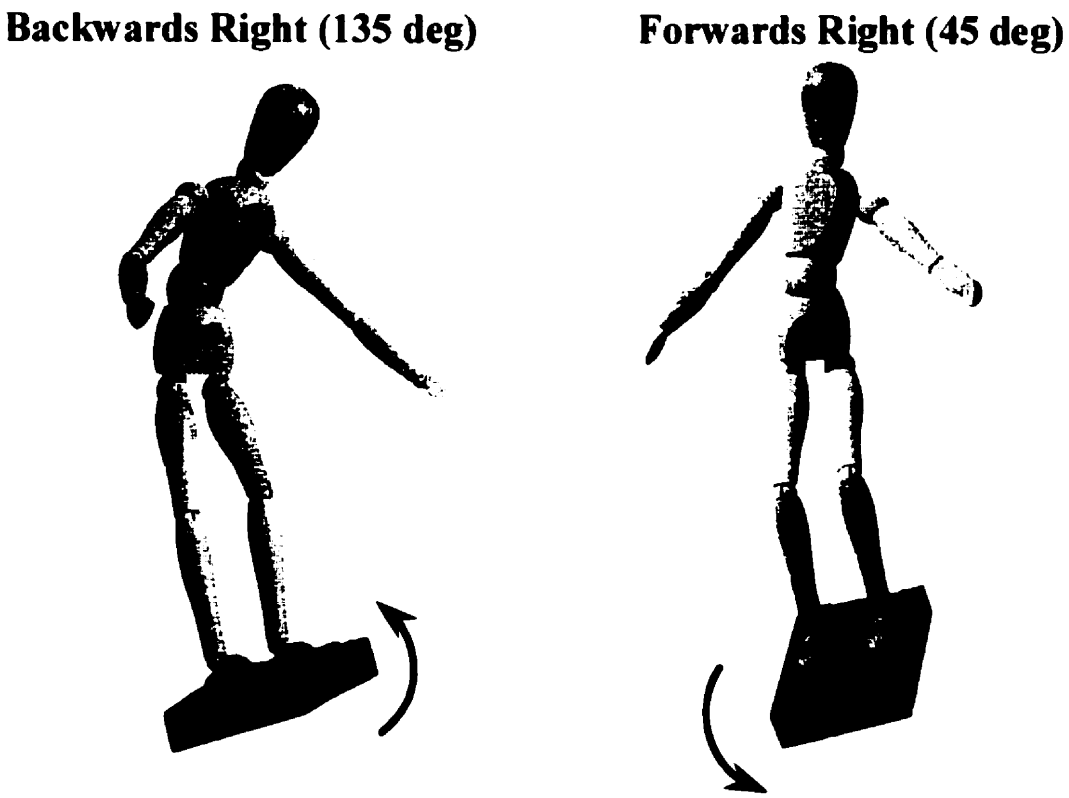

Figure 1 – Graphical schematic representation of stimulus induced movements of the head, trunk and leg segments in response to unexpected support surface rotations directed backwards to the right (135 deg) and forwards to the right (45 deg)

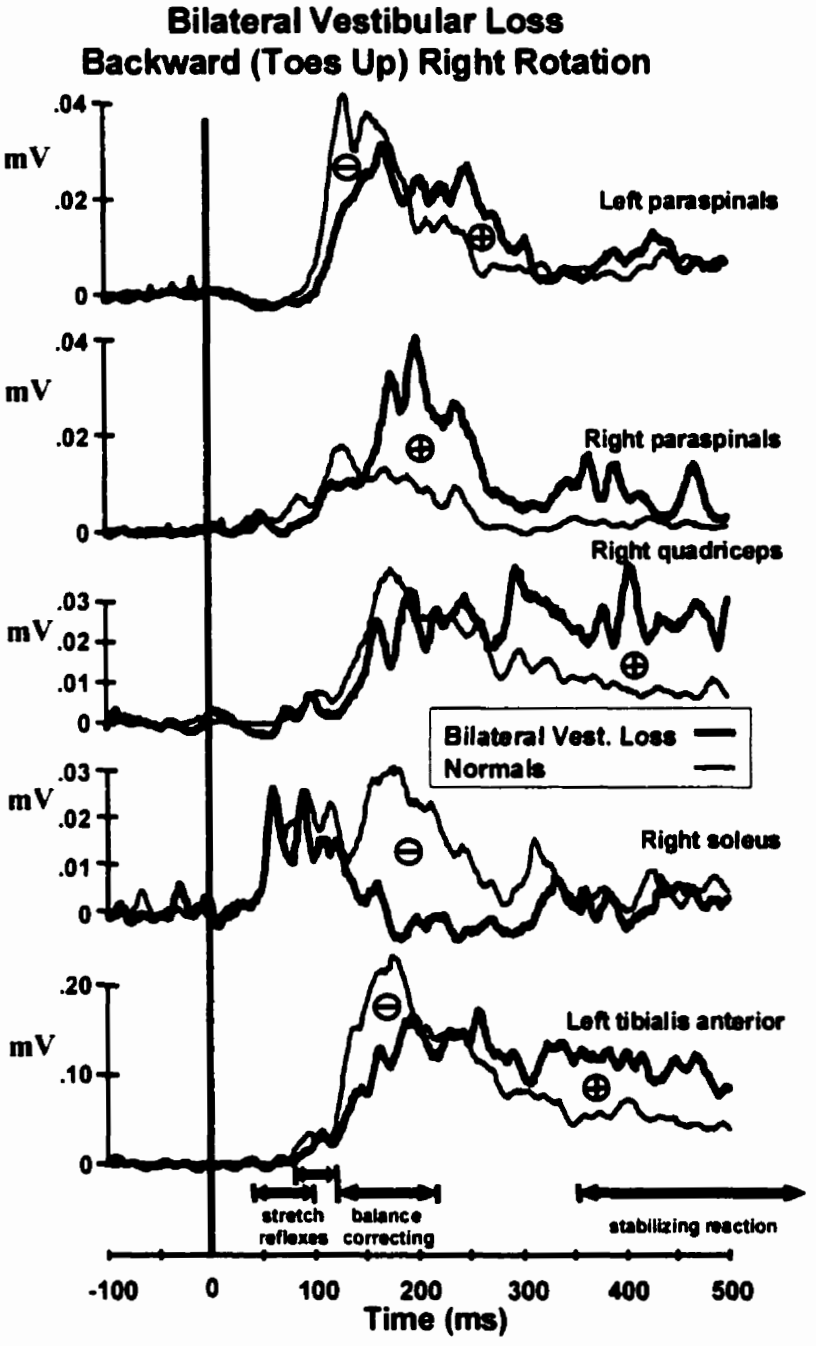

Figure 2 – Average EMG traces from 14 normal subjects (thin lines) and 5 bilateral vestibular loss patients (thick lines), measured during eyes closed trials in response to unexpected surface rotations directed backwards to the right (135 deg). The black vertical line at 0 ms represents the onset of ankle rotation. Gray arrows represent predetermined time intervals selected for calculation of stretch reflex (40-100 ms or 80-120 ms depending on the occurrence in the muscle), balance correcting (120-220 ms) and stabilizing (350-700 ms, only the first 150 ms is shown) responses.

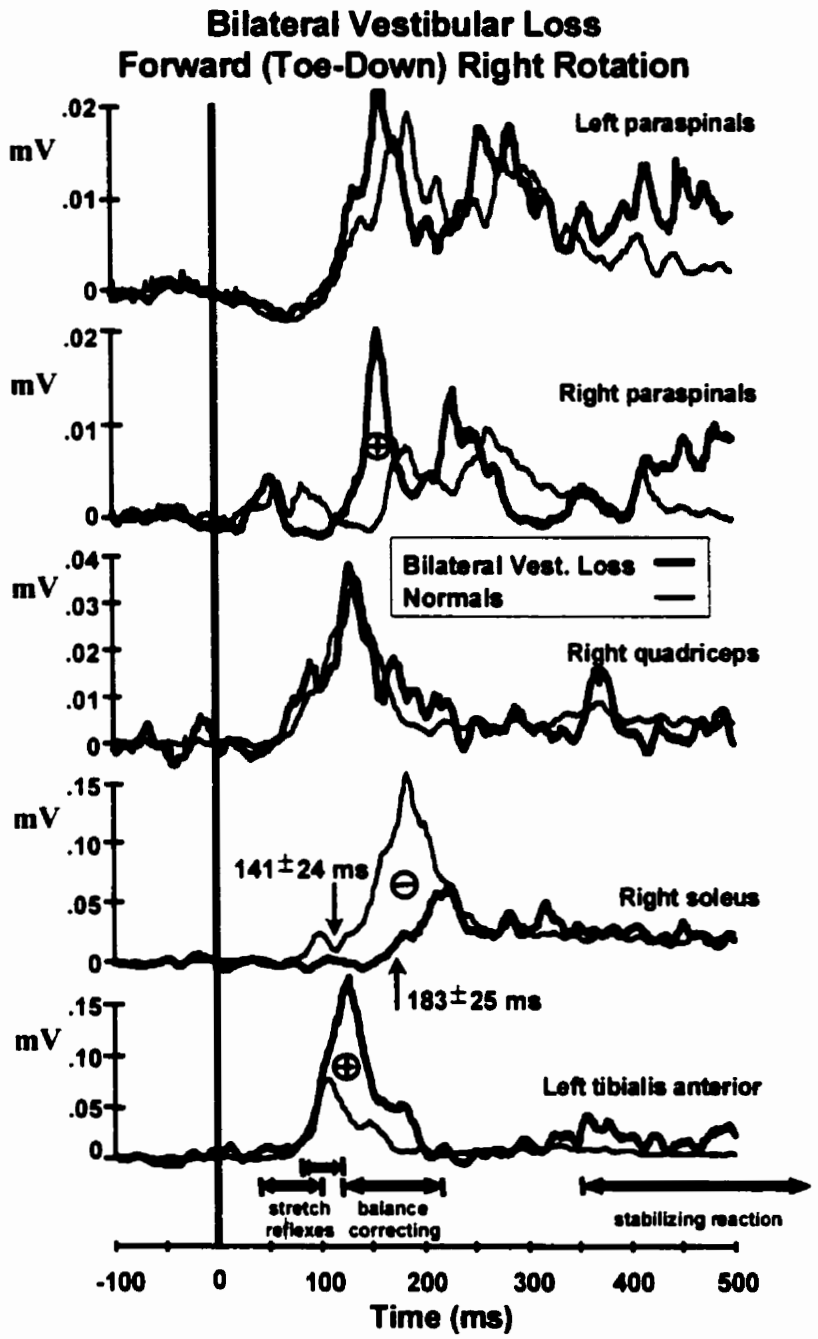

Figure 3 - Average EMG traces from 14 normal subjects (thin line) and 5 bilateral vestibular loss patients (thick line), measured during eyes closed trials in response to unexpected surface rotations directed forwards to the right (45 deg). The black vertical line at 0 ms represents the onset of ankle rotation. Mean onset of normal and vestibular loss soleus responses are marked with arrows. The mean and standard deviation are also listed next to the arrows. Gray arrows represent predetermined time intervals selected for calculation of stretch reflex (40-100m or 80-120 ms depending on the occurrence in the muscle), balance correcting (120-220 ms) and stabilizing (350-700 ms) responses.

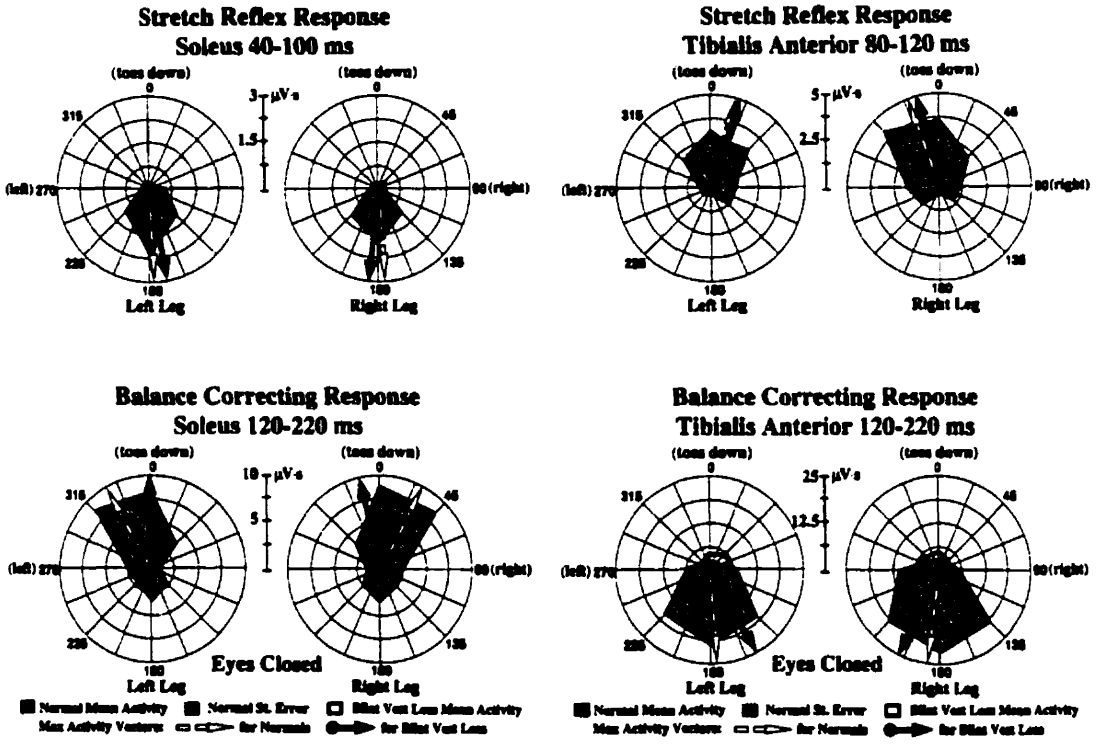

Figure 4 – Polar plots for tibialis anterior (left panel) and soleus (right panel) muscles under eyes closed conditions averaged over three distinct time intervals representative of stretch (40-100; 80-120 ms) and balance correcting (120-220 ms) responses. Each radial line or spoke represents one of eight platform directions. For each direction, mean muscle activity for normals (diagonal hatch filled), normal mean plus one standard error (cross-hatched border) and bilateral vestibular loss (unfilled, thick line as border) for right and left muscles separately. The response amplitude represented by each of the concentric circles in the plot is scaled according to the vertical scale between each set of plots for left and right recording sites. White and black arrows represent the direction of calculated maximum activity vector for each averaging interval for normals and vestibular loss patients respectively.

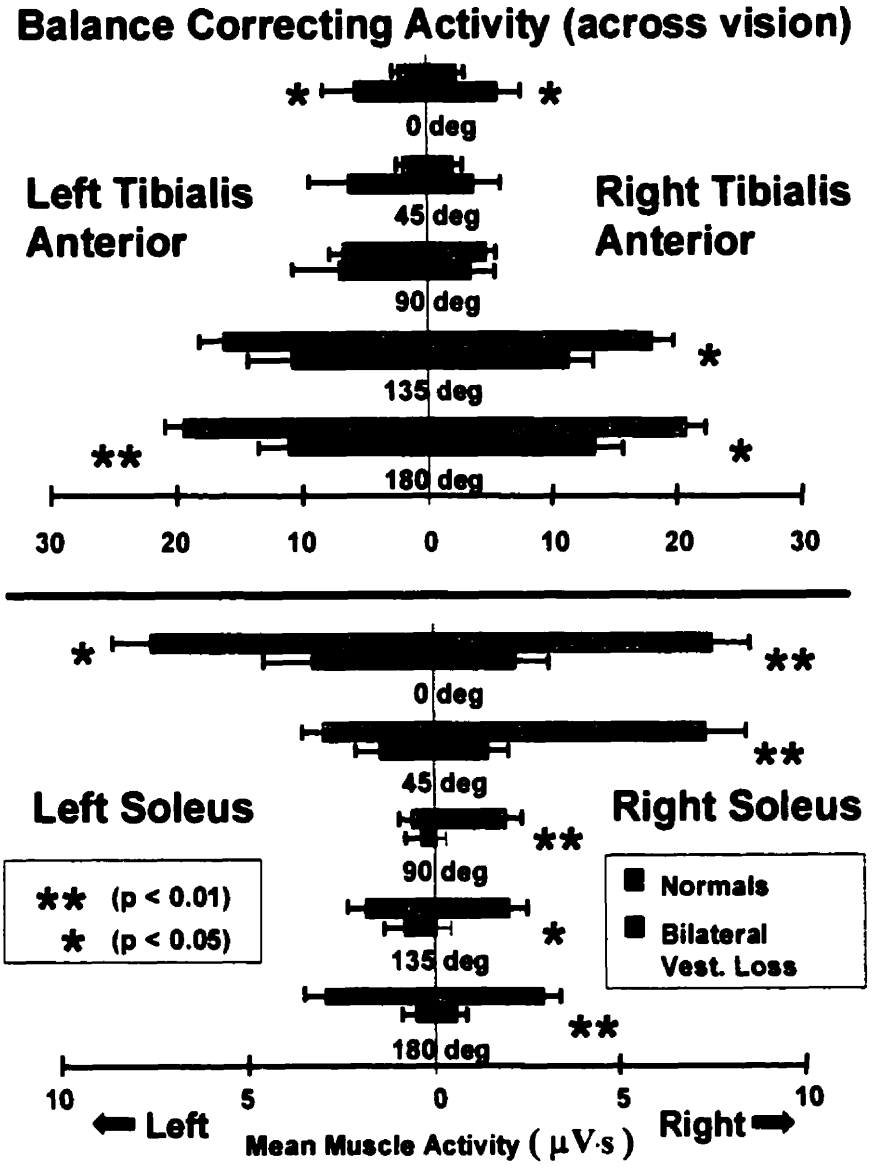

Figure 5 - Mean amplitudes and standard error bars (eyes open and eyes closed combined) for balance correcting response (120-220 ms) in upper half left and right tibialis anterior muscles and lower half left and right soleus muscles. Normal amplitudes are shown as white bars and bilateral vestibular loss patients as black bars for pure pitch, and roll right combinations of platform rotations. The maximum activity direction in normals is 35 deg in right soleus and 177 deg in left tibialis anterior and mirror-imaged for the opposite sided muscles.

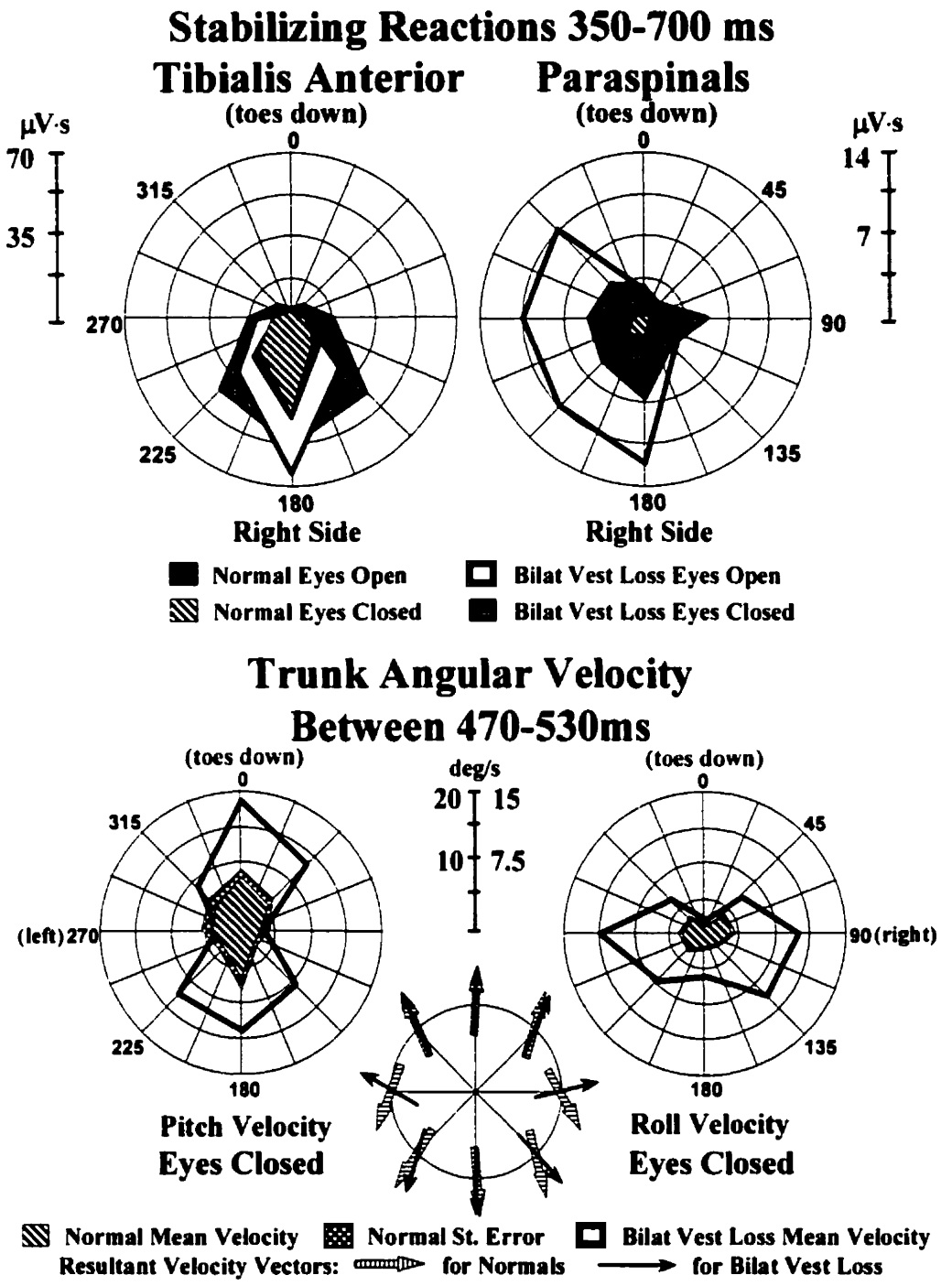

Figure 6 – *Upper half* - Polar plots of average EMG activity during the period between 350-700 ms for normals with eyes open (filled, black) and eyes closed (filled with diagonal hatch lines), and bilateral vestibular loss eyes patients with eyes open (filled white) and eyes closed (filled grey, black border) measured in right tibialis anterior and right paraspinals muscles. *Lower half* - Mean absolute values for average trunk angular velocity calculated between 470 - 530 ms. The larger polar plots to the left and right of each panel represent pitch velocity and roll velocity respectively. The lower centered polar plot represents the calculated direction of the resultant trunk angular velocity vector for each perturbation direction for normals (thick hatched arrow) and bilateral vestibular loss patients (thin black arrow).

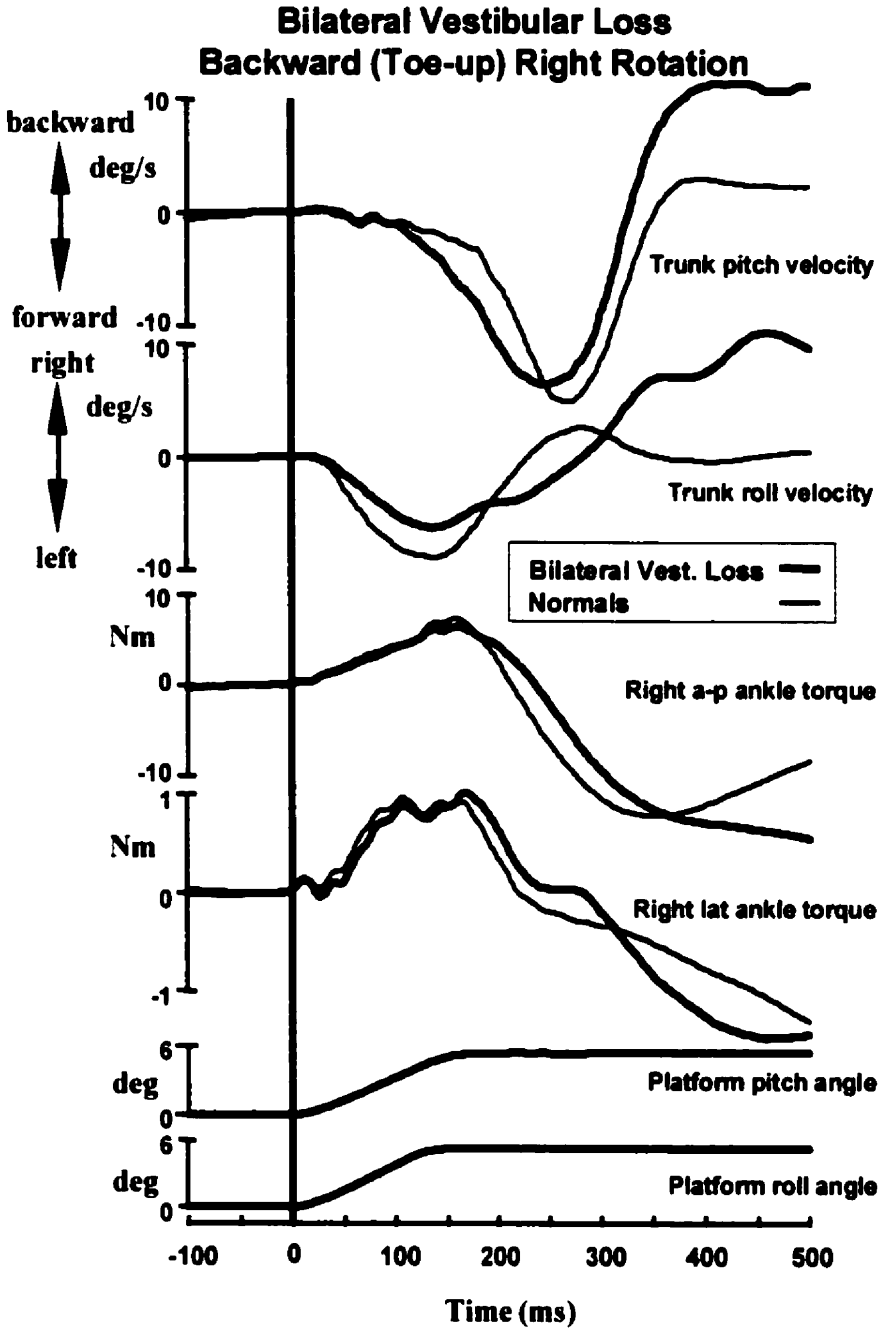

Figure 7 – Average biomechanical traces for normals (thin lines) and bilateral vestibular loss patients (thick lines), measured during eyes closed trials in response to unexpected surface rotations directed backwards to the right (135 deg). Refer to fig. 3 for details.

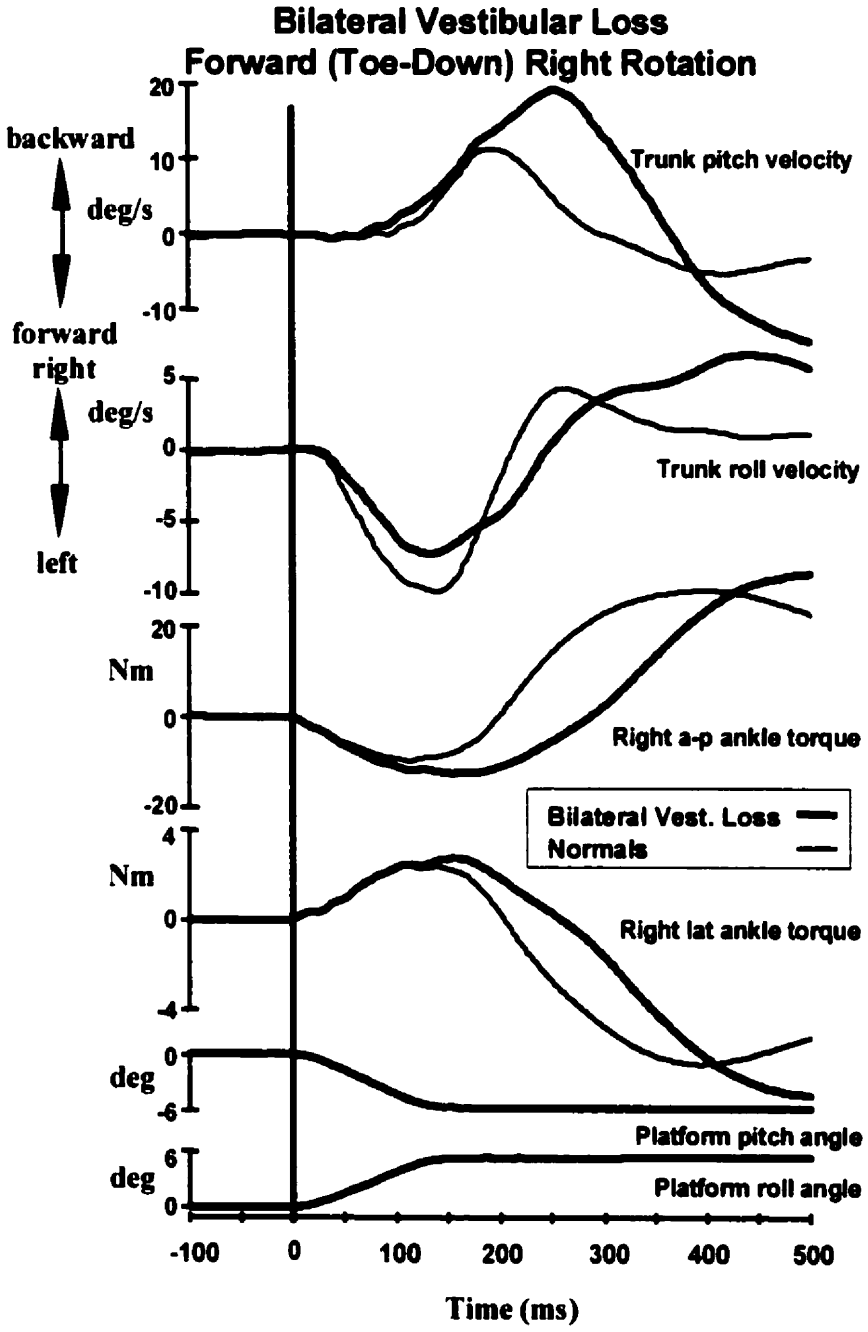

Figure 8 – Average biomechanical traces for normals (thin line) and bilateral vestibular loss patients (thick line), measured during eyes closed trials in response to unexpected surface rotations directed forwards to the right (45 deg). Refer to fig. 3 for details.

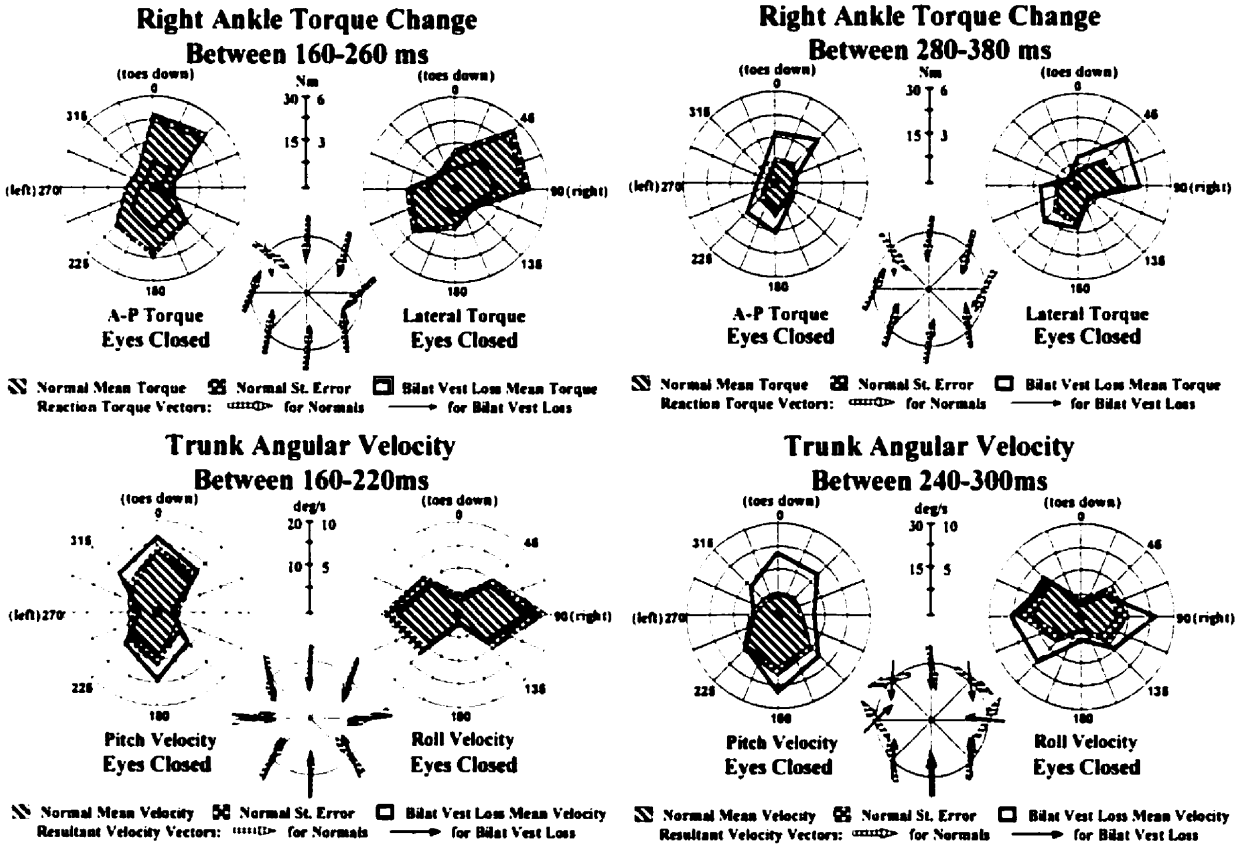

Figure 9 – *Upper half* – Mean absolute values of right ankle torque change calculated on the left between 160-260 ms and on the right for 280-380 ms. The larger polar plots to the left and right of each panel represent A-P torque and M-L torque respectively. The lower centered plot in each panel represents the calculated direction of the resultant reaction torque vector for each perturbation direction for normals (thick hatched arrow) and bilateral vestibular loss patients (thin black arrow).
Lower half – Mean absolute values for average trunk angular velocity calculated on the left between 160-220 ms and on the right 240-300 ms. Refer to fig. 6 for details.

CHAPTER 4

POSTURAL ABNORMALITIES TO MULTIDIRECTIONAL STANCE PERTURBATIONS IN PARKINSON'S DISEASE

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ABSTRACT

If and why patients with Parkinson's disease (PD) have a directional preponderance for falls is unclear. To answer these questions, we studied automatic postural responses to randomly mixed perturbations of stance in multiple directions. We examined trunk control, protective arm movements and associated muscle responses. These questions were addressed in 10 PD patients (mean age 64 years) tested during their best clinical condition ('ON') and in 11 healthy controls (mean age 68 years). To examine the effects of antiparkinson medication, we performed additional tests (randomised with the 'ON' tests) in 7 patients after overnight withdrawal of their medication ('OFF'). In all experiments, upright-standing subjects received support surface rotational perturbations (7.5 degrees amplitude) that were randomly delivered in 6 different directions and at 2 different velocities (30 or 60 deg/s). Outcome measures included electromyographical and biomechanical responses of the lower legs, pelvis, trunk and arms.

Patients revealed several distinct postural abnormalities: *(1)* excessive medium latency activity (80-120 ms) and later balance correcting responses (120-220 ms), not only in distal (lower leg) muscles but also in more proximal (hip and trunk) postural muscles; *(2)* a loss of directional sensitivity for soleus and paraspinal muscles, leading to co-contraction and stiffness ('inflexibility') of the ankles and trunk, particularly for backward and roll-oriented falling; and *(3)* early activated but functionally inadequate protective arm responses, again particularly for backward and roll-oriented falling. Antiparkinson medication gave little improvement of these postural abnormalities. Velocity scaling was normally preserved in patients.

Our results suggest that postural instability in PD is not a 'negative' phenomenon of basal ganglia dysfunction, characterised by a loss of postural responses, but is rather a 'positive' phenomenon characterised by abnormally high muscle activity and co-contraction of postural responses, resulting in excessive stiffness. The backward and laterally directed preponderance for falls, combined with inappropriately executed arm movements, may clarify why hip fractures appear more common in PD than wrist fractures. Our findings also help to elucidate why balance impairment and falls in PD are often resistant to dopaminergic medication, and underscore the need to develop strategies to improve trunk and arm control in PD.

INTRODUCTION

Balance impairment and falls are an important feature of Parkinson's disease (PD) [Rogers 1996;Bloem *et al.* 2001a]. Studies using stance perturbations (dynamic posturography) have helped to clarify the pathophysiology of these balance impairments in PD. Several distinguishing postural abnormalities included: *(1)* abnormally sized automatic postural responses, in particular an increased amplitude of 'medium latency' stretch responses in lower leg muscles; *(2)* 'inflexibility' of automatic postural responses, *i.e.* an inability to modulate the response magnitude according to the demands of the actual postural task at hand; *(3)* delayed initiation or underscaling of voluntary postural responses; *(4)* abnormal execution of compensatory stepping movements; and *(5)* inadequate anticipatory postural responses (for reviews, see Horak and Frank 1996;Bloem 1994;Bloem *et al.* 2001b). Although such postural abnormalities are clearly present in patients as a group, they do not appear to provide reliable diagnostic indicators [Bloem *et al.* 1992]. Furthermore, many postural abnormalities do not correlate well to clinically-rated impairments [Schieppati and Nardone 1991;Waterston *et al.* 1993;Bloem *et al.* 1998a].

It is possible that the poor correlation between posturography results and clinical balance measures is due to shortcomings in previous study designs. Most previous studies analysed postural control in PD using a series of identical perturbations in only one direction, particularly the pitch plane [Schieppati and Nardone 1991;Bloem *et al.* 1996;Horak *et al.* 1992, 1996]. Hence, the postural perturbations became directionally predictable though repetition. This predictability could lead to habituation of postural responses, and such habituation effects may mask abnormalities in PD patients, which are most pronounced for unpredictable perturbations [Bloem *et al.* 1998a;Smithson *et al.* 1998]. Furthermore, use of unidirectional and predictable perturbations prevents insight into natural circumstances, where falls are typically unpredictable and may occur in any given direction. Analysis of automatic postural responses to a randomly applied mix of perturbations in multiple directions might provide better insight into the mechanisms underlying falls in daily life in PD patients. Assessing postural responses to multidirectional perturbations could particularly help to clarify if and why there is a directional preponderance to falls in PD. Anecdotal reports suggest that PD

patients fall more easily backward than forward [Nutt *et al.* 1992;Bloem *et al.* 2001b], but this observation needs to be studied objectively.

Most postural studies of PD have focused largely on electromyographical (EMG) responses in the lower legs [Scholz *et al.* 1987;Beckley *et al.* 1991;Schieppati and Nardone 1991;Horak *et al.* 1992]. Only few studies have studied trunk control in PD patients, either while sitting [Martin 1965;Schenkman *et al.* 2000], standing upright [Bloem *et al.* 1996;Schenkman *et al.* 2000] or while turning around in a recumbent position [Lakke 1985;Bridgewater and Sharpe 1998]. Studies of trunk movement in standing PD patients are interesting, for several reasons. First, simultaneous evaluation of trunk motion and trunk muscle activation could clarify whether postural instability in PD is truly a 'negative' manifestation of basal ganglia disease, characterised by loss of normal postural responses [Martin 1965], or whether other mechanisms might play a more important role. Second, because an abnormal trunk position (as reflected by the characteristically stooped posture) is a core feature of PD, analyses of trunk motion and muscle activation patterns in proximal leg or trunk muscles may yield an increased diagnostic identification of balance problems in PD. Indeed, assessment of these variables yields a good diagnostic utility for the classification of patients with vestibular or lower leg proprioceptive disorders [Allum *et al.* 2001a]. Use of perturbations in multiple directions are particularly useful in this respect due to a need to process pitch and roll motion separately to maintain balance. This spatio-temporal coupling is deficient in vestibular loss patients [Carpenter *et al.* 1999a], but has not been studied in PD.

We were also interested in studying protective arm movements in PD. When postural perturbations are large enough to threaten balance, normal subjects initiate protective arm movements so rapidly that they are considered to be 'automatic' [McIlroy and Maki 1995]. An alternative explanation is that arm movements represent early 'learned' voluntary reactions. These protective arm movements apparently constitute an important defensive strategy against falls [Nutt *et al.* 1993]. It is conceivable that protective arm movements are delayed or even absent in PD. There is some evidence that initiation of automatic and later, more voluntarily initiated postural corrections are delayed in PD [Scholz *et al.* 1987;Allum *et al.* 1988;Bloem *et al.* 1996;Burleigh-Jacobs *et al.* 1997]. If protective arm movements are (partially) under

voluntary control, then the akinesia and bradykinesia of PD patients would result in these being delayed in initiation and execution, respectively. Protective arm movements could also be lost entirely due to basal ganglia dysfunction [Martin 1965]. A demonstration of abnormal arm movements could help clarify anecdotal reports that wrist fractures are relatively rare in PD patients, compared to the incidence of hip fractures [Johnell *et al.* 1992;Sato *et al.* 1999;Bloem *et al.* 2001b]. To our knowledge, protective arm movements have never been examined in PD while standing.

In an attempt to clarify the issues outlined above, we have studied postural responses to multidirectional postural perturbations in PD. To reduce predictability, we randomly administered perturbations at two different velocities and in six different directions. This also permitted us to examine whether PD patients can scale their postural responses to velocity. Our primary focus was to examine if and why PD patients have preponderance for falls in specific directions. A second goal was to examine postural abnormalities in the pelvis, trunk and arms, and to compare these to previously described lower leg abnormalities. These two goals were addressed in patients tested during their "best" clinical condition ('on' medication). To examine whether antiparkinson medication could correct any of the observed postural abnormalities, we performed additional tests (randomised with the 'on' tests) in a subgroup of patients who consented to a withdrawal of their usual medication for over12 hours. This might help to elucidate why balance impairment and falls in PD are often resistant to dopaminergic medication [Bonnet *et al.* 1987;Koller *et al.* 1989;Bloem *et al.* 1996].

SUBJECTS AND METHODS

Subjects (Table 1)

Subjects included 10 patients who fulfilled the criteria for idiopathic PD of the Brain Bank of the United Kingdom Parkinson's Disease Society [Hughes et al. 1992]. All patients sustained a clear and lasting beneficial response to treatment with levodopa and/or a dopamine agonist. Controls included 11 healthy elderly subjects. Subjects with other neurological or non-neurological causes of balance impairment (including visual, vestibular and orthopaedic disorders) were excluded. We also excluded patients with dementia, a considerable postural tremor and significant dyskinesias (score >2 on the Modified Dyskinesia Rating Scale) [Goetz

et al. 1994]. All patients were examined during their best clinical condition (termed 'ON' in this paper), about 1 hr after intake of their antiparkinson medication.

Seven of these patients were also tested after overnight withdrawal of all antiparkinson medication ('OFF'). All patients had predictable end-of-dose wearing off. Some patients reported occasional random on-off fluctuations in daily life, but none occurred during the present experiments. The interval between start of the experiments and intake of the last medication was at least 12 hrs. Although it may be necessary to withdraw antiparkinson medication for several days to entirely eliminate treatment effects, this approach allows for assessment of parkinsonian manifestations in a fairly stable 'off' state [Blin *et al.* 1991]. The order of the 'ON' and 'OFF' experiments was counter-balanced across subjects.

Before the experiments, patients (both 'ON' and 'OFF') and controls were clinically examined using the modified Hoehn and Yahr stages, the Unified Parkinson's Disease Rating Scale (UPDRS) [Lang 1995], the Tinetti Mobility Index [Tinetti 1986], a quantified balance screening protocol based on trunk sway [Gill *et al.* 2001] and the Activities-specific Balance Confidence (ABC) scale [Powell and Myers 1995]. A fear of falls was more common among patients than controls, and patients had lower ABC scores. All patients had clinical balance impairment, as reflected by the Tinetti Mobility Index. Posture and balance were unremarkable in controls. One control had recently fallen, due to environmental circumstances. Clinical signs (including balance and gait scores) were more severe during the OFF condition than during the ON condition (Table 1).

All subjects gave witnessed informed consent according to the declaration of Helsinki. The Institutional Review Boards of the hospitals where the subjects were outpatients (Basel and Leiden) approved the study.

Outcome Measures

We obtained EMG and biomechanical outcome measures. To record EMG signals, pairs of silver-silver chloride electrodes were placed approximately 3 cm apart along the muscle bellies of left tibialis anterior, left soleus, and bilaterally on gluteus medius, paraspinals at the L1-L2

level of the spine, and medial deltoid muscles. EMG amplifier gains were kept constant and pairs of electrodes and lead lengths assigned to individual muscles were not changed between subjects. Support-surface reaction forces of the left foot were measured from strain gauges embedded within the rotating support. The strain gauges were located under the corners of the plate supporting the left foot. From these forces, the AP and mediolateral ankle torques were calculated for the left foot [Carpenter *et al.* 1999b]. Trunk angular velocity in the pitch and roll planes was collected using Watson Industries transducers (+/- 300 deg/s range) mounted to a metal plate that hung at the level of the sternum from shoulder straps that wrapped around the shoulders back and chest. Two similar Systron-Donner angular velocity transducers (Inglewood, Calif., USA) measured movements of the left upper arm in the pitch and roll directions. These transducers had a range of +/- 200 deg/sec and noise specification of 0.04 deg/sec (average standard deviation). The transducers were attached to a 10-cm long metal plate curved to the radius of the arm. The plate was strapped to the lateral aspect of the left upper arm using an elasticised bandage. To measure lower leg angle in the pitch plane a lightweight metal rod was fixed with an adjustable strap to the lateral aspect of the left tibia, about 4 cm below the level of the lateral condyle. The rod was connected to a potentiometer located on the pitch axis of the platform.

Procedure

The subject's feet were lightly strapped into heel guides fixed to the top surface of the dual-axis rotating platform. The guides were adjusted in the AP direction to ensure that the ankle joint axis was aligned with the pitch axis of the rotating platform. The roll axis had the same height as the pitch axis and passed between the feet. Just prior to the experiment, subjects were asked to assume their 'preferred' standing posture with the arms hanging comfortably at their sides. At each individual's 'preferred-stance' position, we measured the low pass filtered (5Hz) AP torque from two strain gauge systems embedded in the surface of the rotating platform. This was then treated as the reference value for 'preferred-stance' for the remainder of the experiment.

The experiment consisted of two series of 44 perturbations each. The first trial of each series was excluded from further analysis to reduce habituation effects [Keshner *et al.* 1987;Bloem *et*

al. 1998b]. The remaining 86 perturbations consisted of randomised combinations of six different perturbation directions and two different perturbation velocities (either 30 deg/s or 60 deg/s), all at a constant amplitude of 7.5 deg. The six perturbation directions included two that were purely in the pitch plane (forward or 0 deg; and backward or 180 deg in our notation). For the four additional perturbation directions, pitch stimuli were combined with leftward and rightward roll components to form 'forward right' (45 deg), 'backward right' (135 deg), 'backward left' (225 deg) or 'forward left' (315 deg) perturbations. Each of the 12 different combinations of perturbation direction and velocity were randomly presented 7 or 8 times throughout the two series of perturbations. Each perturbation was preceded by a random 5-20 s delay. During this period, subjects were asked to monitor an oscilloscope, which was located at eye level, approximately 1 m in front. This oscilloscope displayed online the low pass filtered AP torque, which was measured as described above. Using this visual feedback, subjects were required to maintain AP ankle torque within a range of +/- 1 Nm from their 'preferred-stance' reference value. The 5-20 s interstimulus delay was initiated automatically once the platform had returned to its original pre-stimulus position and the subject had regained and maintained his preferred vertical position as monitored by AP ankle torque reading. In response to each rotational perturbation, subjects were instructed to recover their balance as quickly as possible. Three handrails (80 cm high) were located at a distance of 40 cm to the sides and to the front of the platform centre. Subjects were informed they were allowed to grasp the handrails if needed. Two assistants (one behind and one to the side of the subjects) were present to lend support in case of a fall. To minimise fatigue, all participants were given a 2-3 minute seated rest after the 15th and 30th trial of each series. A longer seated rest period (5 minutes) was provided between each series.

Data Analysis

All EMG and biomechanical recordings were initiated 100 ms prior to perturbation onset and had a sampling duration of 1 s. EMG recordings were band-pass analog filtered between 60-600 Hz, full wave rectified, and low pass filtered at 100 Hz prior to sampling at 1 KHz. All biomechanical data were sampled at 500 Hz and digitally low-pass filtered off-line at 25 Hz using a zero phase-shift 10th-order Butterworth filter. Angular velocities were integrated off-line using trapezoid numerical integration to yield angular displacement.

Following analog to digital conversion of the data, all biomechanical and EMG signals were averaged offline across each perturbation direction and velocity. Zero latency was defined as the first inflexion of ankle rotation velocity and did not vary with direction or subject. Subject averages were pooled to produce population averages for a single direction and velocity combination (as shown in figures 2, 3, 8 and 9). Average trunk angular velocity was calculated over time intervals between 160-220, 240-300 and 470-530 ms. Ankle torque changes were calculated between 160-260 and 280-380 ms. Angular displacements of the arm were calculated relative to the trunk by subtracting the arm position from the trunk position.

Onset latencies were analysed for directions that elicited primary balance correcting responses in each particular muscle. For each subject, the average EMG traces for all directions and muscles were displayed on screen. EMG latencies were determined using a semi-automatic computer algorithm that selected the first point at which the average profile exceeded and remained longer than 50 ms above a threshold greater than 2 standard deviations above background muscle activity (BGA), calculated over the 100 ms period preceding perturbation onset. Each latency was first selected by the algorithm and then approved or manually corrected following inspection by the operator. This was particularly important in patients who frequently had enlarged, so called 'medium latency' (ML) responses (interval between 80-120 ms) which blended with the later balance correcting responses. The same operator checked all latencies to maintain consistency across trials.

Effects of prestimulus BGA may confound between-group comparisons for stretch reflex and automatic balance correcting amplitudes [Bedingham and Tatton 1984;Allum and Mauritz 1984]. Therefore, EMG areas were corrected by subtracting the average amount of BGA (measured over a 100 ms period prior to perturbation onset) from the overall response amplitude. This approach largely eliminates influences of prestimulus BGA [Bloem *et al.* 1993]. Corrected EMG areas were calculated using trapezoid integration within pre-determined time intervals associated with stretch reflex (40-100 from stimulus onset), ML responses (80-120 ms) and balance correcting responses (120-220 ms) [Carpenter *et al.* 1999b]. We also analysed secondary balance correcting responses (240-340 ms) and stabilising

responses (350-700), but these showed no changes in patients. Therefore, this paper will be restricted to the earlier postural responses.

We were interested to see how symmetrically muscles were activated for perturbations containing a roll component. For this purpose, we calculated an 'asymmetry index' as the ratio between left-sided and right-sided activity. Purely symmetrical responses in left and right muscles have a ratio equal to 1, whereas smaller or larger ratios indicate asymmetrical activation.

Because all 10 patients were tested during their best clinical condition, our primary analyses concerned the between-groups comparison of PD-ON patients and controls. To examine differences between patients and controls, between different fall-directions and between different velocities, we used a mixed ANOVA model (group x direction x velocity) for both EMG and biomechanical data. Distributions of EMG response areas and BGA were skewed, even after log transformation, and were therefore analysed statistically following rank-transformation. To determine the effect of medication on postural responses, the mixed-model ANOVA was applied to repeated measures for the seven patients who were tested both ON and OFF. Significant main and interaction effects were further explored using post-hoc comparisons, suing t-tests for parametric data (biomechanics and onset latencies) and non-parametric tests (Mann-Whitney and Wilcoxon tests) for EMG amplitudes. Significance levels were set at 0.05.

RESULTS

Lower Leg Control

Normal responses

The normal response to backward left perturbations consists of multi-segmental body displacements in both the pitch and roll plane. The toe-up rotation of the platform drives the ankles into dorsiflexion and the lower legs backward. As shown in the left panel of figure 1, the left lower leg pitch angle increased immediately following perturbation onset, and reached an initial peak backward displacement at ca 200 ms. After this time point, the lower leg displacement levelled off, before falling still further backward at 300 ms. AP plantar-flexing

torque increased with the initial upward rotation of the platform until ca 200 ms, at which point it reversed and was dominated by an active dorsi-flexion torque which peaked at 400 ms.

Backward perturbations elicited a short latency reflex in the stretched soleus muscle at ca 50 ms. This stretch reflex in soleus was followed by a relatively small balance correcting response. The major balance correcting response occurred in the antagonist tibialis anterior muscle, which generated a dorsiflexion torque to counteract the backward COM displacement that follows these toe-up perturbations. Onsets for balance correcting responses in left tibialis anterior were 132.8 +/- 18.7 ms for backward left perturbations, 132.1 +/- 11.2 ms for purely backward perturbations, and 126.4 +/- 12.2 ms for backward right perturbations.

For forward left perturbations, the left lower leg initially fell slightly backward over the first 150 ms (figure 1, right panel). This was followed by a large forward displacement, which reached a peak forward angular displacement of ca 3 deg at 275 ms. Initially dorsiflexing ankle torque was observed during the first 100 ms as the front of the platform dropped away from the feet. This was followed by an actively generated plantar-flexion torque that reached a peak at 500 ms. At the same time, the knees flexed while the trunk pitched backward. Forward perturbations pulled the ankle joint into plantarflexion, and thus elicited a stretch response in tibialis anterior muscles at ca 80 ms. This was followed by a large balance correcting response in the antagonist soleus muscle (which was initially unloaded by the perturbation). Onset latencies for balance correcting activity in left soleus were 182.4 +/- 18.2 ms for forward left perturbations, 178.3 +/-20.3 ms for purely forward perturbations, and 147.3 +/- 36.9 ms for forward right perturbations.

Parkinson patients ('ON' condition)

PD-ON patients demonstrated segmental movements and muscle response profiles similar to those of controls, with the exception of clear bursts of ML activity. For backward left perturbations, the lower leg angle was displaced backward with an identical onset and velocity compared to controls (figure 1, left panel). However, the lower leg angle had a smaller peak backward displacement, which remained less than that of controls throughout the trial. Initial plantar-flexing torque induced by platform rotation did not differ between PD-ON patients and

controls. However, the active dorsi-flexing torque profile was weaker after 200 ms in PD-ON patients compared to controls. BGA in soleus and tibialis anterior was higher in patients than controls, but these differences were not significant. Initial stretch reflexes in soleus had normal onset latencies and amplitudes. Onset latencies for balance correcting activity in left tibialis anterior did not differ from controls for any backward direction, with mean values of 143.8+/-24.7 ms (backward left), 129.2+/-17.7 ms (purely backward) and 131.5+/-34.2 ms (backward right). In contrast, ML responses and balance correcting responses in tibialis anterior did differ between patients and controls. Because these differences are best appreciated from the polar plots (figure 2), we shall discuss these amplitude changes below for all perturbation directions.

For forward left perturbations, PD-ON patients had a larger initial backward displacement of the lower legs, followed by a more rapid forward angular leg displacement (figure 1, right panel). Initial dorsi-flexion torques were normal in patients, but active plantar-flexing torques after 150 ms were generated at a lower rate and had a reduced peak magnitude. Onset latencies and amplitudes of stretch reflexes in left tibialis anterior were similar between PD-ON patients and controls. Onset latencies of balance correcting responses in left soleus of patients were 176.9+/-23.0 ms (forward left) and 149.7+/-9.8 ms (forward right), and this did not differ from controls. Onset latencies in soleus could not always be determined reliably for purely forward perturbations because there was often no clear distinction between balance correcting and earlier ML responses.

Illustrating the EMG areas on a polar plot, with median amplitudes plotted along axes that correspond to different perturbation directions, allows for an easy visualisation of the magnitude and directional sensitivity of responses from different postural muscles. The polar plots of figure 2 illustrate the median amplitude and directional sensitivity of ML and balance correcting responses for left tibialis anterior and soleus across all perturbation directions. In tibialis anterior, both ML responses (significant interaction effect between group and perturbation direction; $F(5,430) = 2.46$; $p<0.05$) and balance correcting responses (significant interaction effect between group and perturbation direction; $F(5,430) = 2.68$; $p<0.05$) were increased in PD-ON patients compared to controls. Figure 2 reveals enlarged ML responses for all forward and right perturbations (upper left panel) and enlarged balance correcting responses

in tibialis anterior of patients for all backward perturbations (lower left panel). The maximal directional sensitivity for tibialis anterior ML responses were oriented along the pitch axis (7.3 deg) in controls, which is similar to that reported for stretch reflexes at this latency in young normals (Carpenter et al. 1999b). In contrast, directional sensitivity for ML responses in PD-ON patients was more roll oriented at 35.9 deg . The directional sensitivity of the balance correcting responses in tibialis anterior was normally maintained in PD-ON patients, with maximal directional activity vectors at 173.5 deg and 176.1 deg for controls and PD-ON patients respectively.

Figure 2 also shows that PD-ON patients had increased ML responses in soleus across all perturbation directions (significant group effect; $F(1,19) = 9.88$; $p<0.01$). Furthermore, soleus balance correcting activity was significantly larger in PD-ON patients compared to controls across all perturbation direction (significant group effect; $F(1,19) = 10.43$; $p<0.005$). Similar to tibialis anterior, the directional sensitivity of soleus responses was markedly changed in PD-ON patients. This was particularly clear for ML responses in soleus, which (unlike controls) showed prominent activity for backward and both roll directions in patients. Overall, figures 1 and 2 show PD-ON patients had increased amplitudes of balance correcting responses in tibialis anterior and soleus that were appropriately directed but were preceded by abnormal activation and direction of ML activity in antagonist muscles. For example, for backward right perturbations, enlarged balance correcting responses were preceded by a distinct earlier ('medium latency') burst in tibialis anterior activity that was not observed in controls and accompanied by large soleus co-contracting activity. This excessive activity in both ankle muscles presumably resulted in a significantly reduced AP torque generation in PD-ON patients, not only for backward and lateral directions, but also for forward falling (significant interaction effect for ankle torque between group and direction ($F(5,95)=5.20$, $p<0.001$).

Effect of antiparkinson medication

The pattern and timing of postural responses in lower leg muscles was very similar between PD-OFF and PD-ON patients (figure 1). For all recorded responses, onset latencies did not differ between PD-OFF and PD-ON patients. Response amplitudes were not further increased in PD-OFF patients compared to PD-ON patients. If anything, antiparkinson medication

appeared to aggravate the postural abnormalities in PD, because ML responses (in tibialis anterior) and balance correcting responses (both in tibialis anterior and in soleus) were larger in PD-ON patients compared to PD-OFF patients (figure 1). For example, balance correcting responses in tibialis anterior were larger in PD-ON patients compared to PD-OFF patients (ANOVA, significant interaction effect between group and perturbation direction; $F(5,288) = 5.91$; $p<0.0001$).

Hip, Trunk and Head Control

Normal responses

Backward left perturbations caused the trunk to rotate forward, *i.e.* in the opposite direction to pitch rotation of the platform and lower legs, and leftward (figure 3, left panel). Forward pitch rotation occurred first at ca 100 ms, reaching a peak pitch angle at 350 ms. The trunk was then slowly returned to a position with slightly less forward lean by 700 ms. Trunk roll in controls had a negligble rightward roll displacement (<0.2 deg on average) beginning at ca 30 ms, then oscillated around 0 deg (vertical) until 150 ms. This was followed by a rapid rotation of the trunk to the left (the same direction as platform roll) which reached an initial peak at 300 ms, then fell further to the left to an angle of >2 deg by 700 ms.

For forward left perturbations, the trunk pitched backward at ca 50 ms and reached a peak backward displacement of 2 deg at ca 300 ms (figure 3, right panel). After 300 ms, the trunk began to return to a near upright position, which was reached by about 700 ms. In the roll direction, the trunk initially moved negligbly to the right at ca 30 ms. After 125 ms the trunk moved rapidly leftward and reached a maximum displacement by 300 ms, where it remained in this position for the remainder of the trial.

Proximal muscle activity was highly sensitive to perturbation direction, with larger balance correcting responses for perturbations that initially unloaded the muscle. For example, leftward roll perturbations initially generated an unloading response in the left gluteus medius that was followed by a large balance correcting response (figure 3). Purely forward or rightward perturbations elicited smaller responses in left gluteus medius muscles.

Paraspinals were stretched by perturbations that tilted the support surface towards the muscle. For example, left roll perturbations caused the trunk and pelvis to roll in opposite directions (right and left respectively) and stretched the left paraspinal muscle (figure 3). Similar to gluteus medius muscles, controls exhibited larger balance correcting activity in paraspinals that were initially unloaded by the perturbation (i.e. rightward perturbation for the left paraspinal muscle). Thus, controls had relatively smaller balance correcting responses in paraspinals for directions that initially stretched this muscle (i.e. left roll for left paraspinals).

Parkinson patients ('ON' condition)

Distinct changes were observed in the pattern and amplitude of proximal muscle responses of PD-ON patients. Onset latencies of gluteus medius muscles did not differ significantly between PD-ON patients and controls (figure 3). BGA in gluteus medius activity was higher in PD-ON patients compared to controls (ANOVA, significant group effect; $F(1,19) = 8.59$; $p<0.01$). For all perturbation directions, PD-ON patients showed bursts of gluteus medius activity at ca 80 ms, which were not present in controls. However, after correcting for BGA, the amplitude of ML activity in gluteus medius, did not differ significantly between both groups. Figure 3 also shows that amplitudes of balance correcting responses in gluteus medius were increased in PD-ON patients compared to controls. This amplitude difference is best appreciated from the polar plots (figure 4A), which show that balance correcting responses in gluteus medius were increased in PD-ON patients for all perturbation directions (ANOVA, significant group effect, $F(1,19) = 5.28$; $p<0.05$). However, directional sensitivity (with the largest balance correcting responses for directions that initially unloaded gluteus medius) was preserved in PD-ON patients. Maximum activity vectors were oriented at 255.5 deg in controls and 238.3 deg for PD-ON patients. Preserved directional sensitivity in gluteus medius was further supported by analysis of the 'asymmetry index' (ratio between left-sided and right-sided activity), which reflects how asymmetrically both muscles are activated (figure 4B). Both groups showed similar ratios of asymmetry for backward left perturbations (ratio >1) and backward right perturbations (ratio <1). Ratios between both gluteus medius muscles did not differ significantly between PD-ON patients and controls, which suggests that the relative contribution of left and right muscles to balance corrections was similar in both groups.

In paraspinals BGA was about 42% higher in PD-ON patients compared to controls, but this difference was not significant. Both the timing and amplitude of stretch reflexes in paraspinals were normal in PD-ON patients. For all roll directions, PD-ON patients developed a burst of paraspinal activity at ca 80 ms that was not present in controls (plotted for two leftward perturbations in figure 3). However, the difference in responses between patients and controls over this ML measurement period was not significant. For directions that stretched (not unloaded) the paraspinals, balance correcting activity was larger in PD-ON patients compared to controls. Group comparisons for all eight perturbation directions (plotted in figure 4) showed that balance correcting activity in paraspinals was particularly enlarged in PD-ON patients for those perturbations that normally elicit small responses in controls (ANOVA, significant interaction effect between group and perturbation direction, $F(5,430) = 10.69$; $p = 0.0001$). For example, balance correcting responses in left paraspinals were significantly increased compared to controls for left roll perturbations, which stretched the left paraspinal muscle and caused minimal activity in controls. In contrast, balance correcting activity was decreased for directions that unloaded the paraspinals and caused large responses in controls (backward right, figure 4, top right). Maximum activity vectors were more roll oriented at 143.5 deg for controls and were more pitch oriented at 165.4 deg in PD-ON patients. PD-ON patients consequently lacked the normal left-right asymmetry in their paraspinal responses, which were co-contracted. This was reflected by the asymmetry index, which showed that controls had very asymmetrical activation amplitudes for left and right paraspinals (figure 4B). In contrast, ratios between left and right paraspinals were significantly different from controls (significant interaction effect between group and perturbation direction ($F(5,218)=3.48$; $p<0.005$). PD-ON patients did not have asymmetrical responses (as seen in controls) but instead had more symmetrical activation of bilateral paraspinals, as evidenced by asymmetry ratios that were closer to one (figure 4B).

The global pattern of trunk movement in patients was similar to that of controls. However, quantitative analysis revealed various changes in trunk movements of PD-ON patients, both in the pitch and the roll plane. Following backward left perturbations, there was no difference in onset and time to peak forward displacement of trunk pitch (figure 3, left panel). However, for backward tilts the velocity of forward trunk pitch was slower, and resulted in a smaller peak

forward displacement in PD-ON patients (figure 5). The reduced peak trunk pitch angle was followed by a reduced final position at 700 ms in PD-ON patients. In contrast, the backward trunk pitch angle differed little between PD-ON patients and controls following forward left perturbations (figure 3, right panel). This discrepancy between forward and backward directed perturbations is further illustrated in figure 5, which shows the group comparisons for all eight perturbation directions. Peak trunk pitch angle at 300 ms was significantly influenced by a interaction effect between group and perturbation direction ($F(5,80)=2.81$, $p<0.05$). PD-ON patients had a significantly smaller forward pitch angular displacement of the trunk for backward perturbations (particularly the purely backward rotations). In contrast, peak backward trunk angular displacement did not differ between PD-ON patients and controls for forward perturbations.

Trunk roll movements were also abnormal in PD-ON patients. Trunk roll angle was delayed in PD-ON patients, particularly for forward roll tilts. When the trunk did roll (to the left for leftward roll perturbations), the displacement was rapid (particularly when combined with backward perturbations) and reached a peak roll angle by 700 ms that was almost two times greater than in controls. Controls consistently demonstrated a larger early trunk roll angle at 300 ms compared to PD-ON patients (figure 5B)but this difference was not statistically significant.

Effect of antiparkinson medication

There were no major differences between PD-ON and PD-OFF patients for hip or trunk control. Balance correcting responses in gluteus medius were somewhat larger in PD-OFF compared to PD-ON patients (figure 3). Withdrawal of antiparkinson medication further increased the amplitudes of balance correcting responses in paraspinals. The loss of normal asymmetry for paraspinal balance correcting responses was equally present in both PD-OFF and PD-ON patients. Trunk angular displacement was slightly more impaired in PD-OFF compared to PD-ON patients. However, none of these differences were significant.

Protective Arm Movements

Normal responses

EMG recordings from both medial deltoid muscles revealed responses for all perturbation directions. Onset latencies in the left medial deltoid muscle were 124.1+/-18.1 ms (backward left), 140.4+/-12.3 ms (purely backward) and 133.6+/-18.2 ms (backward right perturbations). Muscle activity appeared to be bilateral, because deltoid responses were observed in both arms regardless of whether the perturbation was to the left or right (figure 6). However, some directional sensitivity of arm responses was observed for forward versus backward perturbations. Smaller arm responses were observed for forward perturbations, with almost negligible activity for purely 'toes down' (0 deg) perturbations. Larger responses were elicited for directions with either a 'toes up' or a roll component (figure 7).

The biomechanical analyses (measured from the left arm) revealed that arm movements were initiated in the same pitch direction as the initial trunk movements (figure 6). Controls thus moved their arms into the direction of the impending trunk instability. For backward left perturbations, the trunk pitched forward, and rapid shoulder flexion occurred with a peak angular displacement of 6 deg by 350 ms. Similarly, for forward left perturbations, the trunk pitched backward and the arm was simultaneously brought back (shoulder extension) to reach a peak amplitude relative to the trunk at ca 300 ms (figure 6). Generally, backward roll perturbations elicited larger absolute arm angular displacements compared to forward roll perturbations in controls (figure 6 and 7). Arm roll movements were also in the same direction as trunk roll movements in controls. For backward left perturbations (which eventually roll the trunk to the left), controls abducted the left arm away from the trunk, reaching a peak angle of 4 deg by 700 ms. Alternatively, when the platform rolled rightward, causing the trunk to roll in the same direction, the left arm was adducted towards the trunk.

Parkinson patients ('ON' condition)

The onset of medial deltoid responses was often earlier in PD-ON patients compared to controls. Onset latencies for deltoid responses in patients were earlier for purely backward perturbations (119.6+/-22.1 ms; t-test, $p<0.05$) and backward right perturbations (114.3+/-19.1 ms; t-test, $p<0.05$), but in the normal range for backward left perturbations (116.6+/-28.7 ms; t-test, $p = 0.53$),. Furthermore, the amplitude of balance correcting deltoid responses tended to

be increased in PD-ON patients (figure 7) compared to controls (interaction effect between group and perturbation direction, $F(5,430)=1.87$; $p = 0.09$).

The biomechanical analyses revealed directionally dependent changes in arm responses of PD-ON patients compared to controls, similar to the trunk abnormalities described above. Abnormalities were seen in both the pitch and roll planes. For backward left perturbations, patients had less peak forward arm pitch angle than controls, and brought the arms back rapidly to near the pre-stimulus position by 700 ms. In contrast, PD-ON patients had a normal amplitude of peak backward arm pitch angle for forward left perturbations. This was again followed by a quick return of the arms to a slightly forward (flexed) position. The relative arm pitch angle was reduced in patients compared to controls for all backward perturbations, with less difference between groups for forward directions. However, the overall group differences were not significant.

In the roll plane, there was a significant group by direction interaction for relative arm movement at 300 ms ($F(5,80)=2.35$, $p<0.05$), suggesting directionally specific abnormalities in PD. Leftward roll perturbations elicited left arm movements in PD-ON patients that, after 150 ms, were oppositely directed to those of controls (figure 6 and 7). For leftward roll perturbations, PD-ON patients adducted the left arm (bringing it closer to the trunk), then abducted the left arm after 300 ms. Patients even adducted their arms for purely backward perturbations, when practically no arm roll movement occurred in controls. Left arm movements for right roll directions showed similar, but smaller, differences in trajectories between PD-ON patients and controls (figure 6 right). These differences were not significant in amplitude at 300 ms.

Effect of antiparkinson medication

Compared to PD-ON patients, PD-OFF patients had delayed onset latencies for purely backward perturbations (133.2+/-10.7 ms; t-test, $p<0.05$) but comparable latencies for backward left perturbations (107.0+/-15.8 ms; t-test, $p = 0.52$) and backward right perturbations (117.5+/-19.1 ms; t-test, $p=0.71$). Amplitudes of left medial deltoid responses did

not differ between PD-OFF and PD-ON patients. There were no significant differences in peak amplitude of arm angles between PD-ON and PD-OFF patients.

Scaling Effects

Figure 8 shows that control subjects appropriately scaled their balance correcting responses in leg and trunk muscles to different perturbation velocities (significant main effect for velocity, $p<0.05$). Patients also scaled their balance correcting response in leg and trunk muscles to the same degree as (or even greater than) controls when perturbation velocity was increased (no interaction between velocity and group, or between velocity, group and direction). There were no scaling differences between PD-ON and PD-OFF patients.

DISCUSSION

Multidirectional stance perturbations revealed four distinct postural abnormalities in PD. First, patients had excessive activity over the ML period prior to onset of balance correcting activity in the antagonist muscle. Second, excessively large balance correcting responses were observed in both distal (lower leg) and proximal (hip and trunk) postural muscles. PD thus appears to be characterised by global overactivity of automatic postural responses. Third, the spatio-temporal coupling was also changed. Medium latency activity in leg muscles changed their overall pitch sensitivity, while balance correcting responses in paraspinals lost their roll sensitivity and were bilaterally co-activated. This was associated with stiffness of the ankles and trunk, particularly for backward and laterally directed falling. Fourth, patients had early but functionally inadequate compensatory arm responses. Antiparkinson medication gave little improvement of these postural abnormalities. These observations will be discussed below, with two underlying themes. First, what new information on postural abnormalities in PD was obtained using multidirectional perturbations with pitch and roll components, over and above previously identified changes using pure pitch plane perturbations. Second, how can analysis of trunk control and protective arm movements improve our understanding of clinical balance deficits and fall-related injuries in PD. We will also consider various factors that may affect the interpretation of the present results.

Enhanced Activation of Medium Latency and Balance Correcting Responses

Posturography studies using pitch plane perturbations reported impaired gain control of ML responses in lower leg muscles of PD patients [Scholz *et al.* 1987;Dietz *et al.* 1988;Schieppati and Nardone 1991;Horak *et al.* 1992;Bloem *et al.* 1996]. This was reflected by abnormal (usually increased) response amplitudes and, in particular, an inability to adapt postural responses to the environmental demands ('postural inflexibility'). Others reported similar gain changes in leg muscles of seated subjects [Berardelli *et al.* 1983;Diener *et al.* 1987] and upper limb muscles [Tatton and Lee 1975;Cody *et al.* 1986]. The present study extends these observations to a multidirectional environment and to different muscles. We observed a global elevation of ML activity in PD, not only in muscles that showed ML activity in controls (such as tibialis anterior), but also in muscles that normally show little ML activity. Indeed, healthy subjects generally avoid strong activity over the ML period in in muscles such as soleus, presumably because this would counteract later balance correcting responses. This is not the case, in muscles such as gastrocnemius and tibialis anterior which have weak or absent stretch activity at 50 ms, but have large responses with onsets at 80 ms. [Nardone *et al.* 1990; Allum *et al.* 1998]. Thus, a novel observation in the current study was the presence of distinct ML bursts in soleus, gluteus medius and paraspinals of patients that were not present in controls. Interestingly, enhanced ML responses in patients occurred even when muscles were unloaded, rather than stretched. It is possible that the higher BGA levels of patients facilitated this appearance of ML activity in gluteus medius and paraspinal muscles, but not in the lower leg muscles. Taken together, our findings suggest that PD is associated with a global impairment of ML gain control in any muscle, independent of stretching. Impaired ML gain control (particularly for muscles that normally show little ML activity) may contribute to balance impairment in PD, because it leads to co-contraction just before normal balance correcting activity in antagonist muscles.

Enhanced amplitudes (presumably reflecting impaired gain control) were also observed for balance correcting responses. Previous studies using rotational or translational perturbations in the pitch plane reported abnormal amplitudes of balance correcting ('long latency') responses in tibialis anterior [Allum *et al.* 1988;Dietz *et al.* 1988;Schieppati and Nardone 1991;Beckley *et al.* 1993]. These abnormalities were associated with a reduced stabilising torque and

appeared to contribute to balance impairment in PD [Bloem *et al.* 1996;Horak *et al.* 1996]. The present study extends these observations. Patients had an increased response gain in soleus and tibialis anterior for both pure toe-up rotations and perturbations that combined pitch and roll components. Theoretically, these higher response amplitudes should have increased the stabilising torque about the ankle joint with respect to controls [Keshner *et al.* 1987]. However, this was not the case (patients actually had weaker ankle torques), because the antagonist muscles showed enlarged ML responses and excessive activity over the balance correcting period (see below), leading to counteractive torques. We also observed an increased gain for balance correcting responses in proximal muscles (gluteus medius and paraspinals), and, because of the roll sensitivity of these muscles, this was most clearly seen for off-pitch stimuli. Thus, balance correcting responses in gluteus medius were markedly increased for backward left perturbations, but no abnormalities were seen using purely toe-down perturbations. Such observations provide a first indication that multidirectional perturbations are needed to fully comprehend balance abnormalities in PD. However, particularly relevant information was obtained regarding the directional sensitivity of balance correcting responses, as will be discussed next.

Reduced Directional Sensitivity and Postural Stiffness

Use of multidirectional perturbations showed that the directional sensitivity of postural responses in leg and trunk muscles is changed in PD. In the lower legs, controls primarily had large balance correcting activity in soleus for toe-down perturbations that initially unloaded the muscle. This directional sensitivity was altered in PD, because patients had globally enhanced soleus responses across all backward perturbation directions. This change of directional sensitivity presumably offset the normal agonist-antagonist relationship with tibialis anterior. Thus, the overactivity of soleus for backward perturbations, coupled with normally directed balance correcting response in tibialis anterior, led to increased active co-contraction in patients. This co-contraction might be expected to increase stiffness of the ankle joint, and reduce net joint torques. The latter was indeed revealed by the biomechanical analyses. PD patients had a smaller lower leg angular displacement and a decreased change in active AP ankle torque between 280-380 ms. Other investigators also identified ankle stiffness and weaker ankle torques in PD [Hufschmidt *et al.* 1991;Bloem *et al.* 1996;Horak *et al.* 1996]. A

new observation is that ankle torque changes appear to result from enlargement of both ML and later balance correcting activity, leading to co-contraction in tibialis anterior and soleus. Opposing effects in antagonist muscles explains why ankle torques were weaker, even though muscle response amplitudes were increased. Significantly, this co-contraction was seen in particular for backward directed perturbations. This may be one of the mechanisms underlying a directional preponderance for falls in daily life. Note that co-contraction has also been observed in PD under various other circumstances, including gait [Dietz *et al.* 1995], maintaining balance against external perturbations [Beckley *et al.* 1991;Dietz *et al.* 1993;Horak *et al.* 1996] and self-initiated postural adjustments [Lee *et al.* 1995].

A similar loss of directional sensitivity was noted for muscular and biomechanical responses of the trunk. In controls, the normal paraspinal response to roll perturbations is a reciprocal relationship between stretched and unloaded muscles. For example, backward left perturbations elicited large balance correcting responses in the unloaded right paraspinal muscle, with minimal activity in the left paraspinal muscle. In contrast, PD patients demonstrated more symmetrical activation of paraspinals, with near-equal activation of each muscle for both backward left and backward right perturbations. An increased BGA level in paraspinals compounded this reduced asymmetry. As expected, the co-contraction and high BGA levels of paraspinals were associated with increased trunk stiffness, as reflected by the biomechanical responses of the trunk in the pitch and roll planes. For backward left perturbations, the rate and maximum amplitude of forward trunk pitch was reduced in PD patients. Similar observations (a reduced rate of trunk angular acceleration and an earlier reversal in trunk acceleration) were made in PD patients recovering from pure toe-up perturbations [Allum *et al.* 1988]. Furthermore, the peak trunk pitch angle at 300 ms was significantly reduced for backward directions. Interestingly, there were no differences in trunk pitch angle for forward perturbations. Together with our observations on ankle muscle co-contraction, these results suggest that PD patients predominantly experience postural problems for backward and lateral falling.

PD patients also had a reduced rate of trunk roll angle compared to controls, suggesting additional stiffness in the roll plane. The high BGA levels in hip and trunk muscles, as

observed here and by others [Horak *et al.* 1996], might partially explain this roll stiffness by providing a stiffer trunk. Indeed, we have observed an even more radically altered trunk pitch and roll profile in a patient with severe proprioceptive deficits, and this stiffness was apparently brought about by excessive BGA levels in gluteus medius and paraspinals [Bloem *et al.*, submitted]. Some additional stiffness may be explained by the increased ML and balance correcting responses in gluteus medius. An additional problem for both patients and elderly controls is that their initial trunk roll is negligible compared to younger subjects [Allum *et al.* 2001b]. Thus, both elderly controls and PD patients had initial trunk roll (over the first 150 ms) in the same direction as the platform perturbation. In contrast, young controls have a 'hinging' response, so that the upper trunk rolls in the opposite direction to platform roll and thus moves the COM away from the downhill side [Carpenter *et al.* 1999b]. Therefore, it is possible that some aspects of trunk stiffness in PD (particularly in the roll plane) may be compounded by age-dependent alterations in *e.g.* viscoelastic properties of muscles, joints or ligaments.

Our observations shed new light on the pathophysiology underlying trunk instability in PD. It has long been believed that balance deficits in PD are 'negative' phenomenon, characterised by loss of normal postural responses. For example, when seated patients with postencephaletic parkinsonism are tilted laterally, righting responses of the trunk seem absent because patients fall passively sideways into the direction of tilt, without making compensatory movements [Martin 1965]. This absence of postural trunk responses seemed to be caused by defective central processing of vestibular feedback, because patients with labyrinthine defects showed a similar lack of trunk movements. However, this observation has puzzled subsequent investigators who failed to observe vestibular deficits in PD (reviewed by Bloem 1994). We have now observed a similar absence of compensatory trunk movements in the roll plane, yet this was not caused by lack of postural activity, but rather by excessively large and co-contracting responses. Note that Martin did not record EMG of postural responses to tilt, but only used visual inspection of cinematograph records to analyse trunk movements. To the naked eye, his Parkinsonian patients may certainly have looked similar to vestibular loss patients. However, we are aware that the pathophysiology of trunk instability is different in the two groups [Carpenter et al. 2001], with excessive co-contraction occurring in Parkinsonian

patients. As such, the postural trunk deficits in PD would be better classified as a 'positive' phenomenon of basal ganglia dysfunction.

Protective Arm Movements

Arm movements are an important defence strategy against unexpected balance perturbations [Nutt *et al.* 1993;Maki and McIlroy 1997]. Protective arm movements would be particularly vital for PD patients to compensate for the above-described abnormalities of automatic postural responses in leg and trunk muscles. Controls had onset latencies in deltoid muscles that ranged between 124-140 ms, *i.e.* very similar to onset latencies for balance correcting responses in distal and proximal postural muscles. In contrast to our prediction, PD patients had significantly earlier deltoid responses (range 114-119 ms) than controls. This unexpected observation can be explained in several ways. First, it is possible that the reduced onset latency in patients is caused by an early occurring 'startle' reaction that precedes and blends with the normally timed balance correcting response. Startle reactions have rarely been studied for somatosensory stimuli, but if there is any resemblance to the acoustic startle reaction, then the observed onset latencies would be appropriately timed to represent such startle responses [Rothwell 1994]. Acoustic startle reactions normally habituate rapidly, and this also occurs (even in PD) for postural 'startle-like' responses evoked by unexpected platform movements [Bloem *et al.* 1998b]. However, habituation of postural responses is diminished when subjects are prepared to execute a motor task [Valls-Sole *et al.* 1997], as was the case in our experiments. Furthermore, it is possible that habituation was reduced by the postural threat of our randomly mixed and multidirectional perturbations. Note that most of our patients were fearful and had low balance confidence scores. Interestingly, others have observed significantly earlier onset latencies for posterior deltoids during large compared to small translational perturbations (90 ms and 106 ms, respectively) [McIlroy and Maki 1994]. The shorter latencies were perhaps associated with an increased postural threat or startle.

A second possibility is that the early responses in PD represent some form of triggered or even 'over-learned' voluntary responses [McIlroy and Maki 1995]. Indeed, both healthy subjects and PD patients can activate arm muscles much earlier than under normal volitional control when movement is accompanied by a startling (acoustic) stimulus [Valldeoriola *et al.* 1998].

Interestingly, the motor cortex projects not only to contralateral deltoid muscles (using fast-conducting corticomotoneuronal projections), but also projects to left and right deltoids via somewhat slower bilateral connections [Colebatch *et al.* 1990]. This bilateral projection could well be involved in the bilateral arm movements seen in our study. The possibility of an early voluntary response is supported by the further reduction in onset latencies following intake of antiparkinson medication, because this reduces bradykinesia and akinesia. However, akinesia should result in delayed onset latencies for PD-OFF patients, with perhaps a return to more normal latencies in PD-ON patients, but this was not observed.

A third possibility is that a distinct ML response was elicited in PD patients that blended with a normally timed, later balance correcting response. Indeed, closer inspection of the deltoid muscle traces reveals an initial peak in PD patients, which is not present in controls. This early response could be similar to the increased ML responses seen in leg and trunk muscles of patients, and would thus represent yet another muscle affected by abnormal ML gain control. Our observations of earlier and larger deltoid responses in muscles for PD-ON patients compared to PD-OFF patients are consistent with the similarly enlarged ML responses in tibialis anterior and soleus for PD-ON patients.

The larger and earlier arm muscle responses raises the question whether patients inadequately executed their protective arm movements. The biomechanical analyses provide the impression of inappropriate arm responses. In the pitch direction (backward falls), PD patients had decreased forward arm movements compared to controls. Moving the arms forward normally moves the body's COM forward and thus acts to counter the backward body displacement following toe-up perturbations ('counterweight' function). In addition, arm raising creates reaction moments at leg and trunk joints that may aid or disrupt stability, depending on the direction of arm acceleration or deceleration [Eng *et al.* 1992]. Interestingly, backward arm movements (elicited by forward perturbations) did not differ between PD patients and controls. This observation again emphasises the increased difficulty of PD patients to compensate backward falls, relative to forward falls.

In the roll direction, controls abducted their arms relative to the trunk. Others made similar observations in sitting [Martin 1965] and standing subjects [Maki and McIlroy 1997]. Because the trunk fell in the same direction as the platform movement, these abduction movements of the arms in controls were likely protective in nature and served to either grasp the rail or brace for a fall. PD patients had distinctly different arm roll responses compared to controls. After 150 ms, they initially adducted their arms (bringing them closer to the trunk) for all perturbation directions, including pure toes-up perturbations (which elicit negligible responses in controls). After 300 ms, the arm movements were reversed and abducted, similar to controls, but still remained adducted. Taken together, our findings suggest that (despite early and large muscle responses) protective arm responses are poorly executed in PD patients.

Note that we could not test stepping reactions, which represent another important protective postural response [Nutt *et al.* 1993;Maki and McIlroy 1997]. The feet of our subjects were strapped to the platform for safety reasons (in view of the small support surface), and to maintain constant foot placement and body orientation between and within subjects. Denying stepping responses may have increased the need to compensate with arm responses and have highlighted the abnormalities in PD. Interestingly, others have previously shown that protective stepping responses are abnormal (delayed and reduced in amplitude) in PD [Burleigh-Jacobs *et al.* 1997]. PD patients are therefore saddled with a particularly unfortunate combination of inadequate 'reactive' postural responses and poorly executed defensive responses.

Velocity Scaling

Velocity scaling of ML and balance correcting responses was preserved in PD. In both groups, response amplitudes were larger for fast compared to slow velocities for all muscles tested. These findings confirm previous observations of normal velocity scaling in PD using unexpected translations [Horak *et al.* 1996]. In contrast, scaling to different perturbation amplitudes is impaired in PD [Beckley *et al.* 1993]. It thus appears that the basal ganglia are responsible for response scaling to perturbation amplitude. In contrast, the basal ganglia apparently play a minor role in 'online' scaling of postural responses to perturbation velocity, for which the cerebellum seems more important [Timmann and Horak 1997].

Effects of Antiparkinson Medication

Antiparkinson medication generally yielded little improvement of postural abnormalities in PD. Minor improvements included a decreased BGA and reduced amplitude in gluteus medius balance correcting responses. The lack of improvement to postural reactions with medication was not caused by an overall treatment failure, because UPDRS scores and Tinetti Mobility Index were significantly better in PD-ON compared to PD-OFF patients.

Some postural abnormalities even appeared to be aggravated by antiparkinson medication. For example, we observed increased BGA levels in PD-ON patients, particularly in lower leg muscles. This finding contrasts with previously described reductions in muscle tone in PD-ON versus PD-OFF patients [Burleigh *et al.* 1995]. A possible explanation for the high BGA levels in PD-ON patients might be increased voluntary activation due to reduction in bradykinesia. It is unlikely that the changes in BGA with medication were due to excessive dyskinesias, because patients with scores >2 on the Modified Dyskinesia Rating Scale [Goetz *et al.* 1994] were excluded. Fatigue, learning effects and changes in electrode positions or impedance (which we tried to keep constant across test conditions) are also unlikely explanations, because the testing order was counter-balanced across PD-ON and PD-OFF patients.

The biomechanical analyses neither showed significant improvement in PD-ON compared to PD-OFF patients. The ankle torque remained weaker than controls. Furthermore, there were no improvements in trunk stiffness, as reflected by roll and pitch flexibility, or protective arm movements with medication. These findings corroborate previous studies which found little or no improvement of postural responses with antiparkinson medication [Bonnet *et al.* 1987;Blin *et al.* 1991;Bloem *et al.* 1996].

Possible Confounding Factors

Various factors may have influenced the observed differences between patients and controls. High BGA levels were observed in most muscles of PD patients, including tibialis anterior, gluteus medius and paraspinals. Similar findings were reported by others [Scholz *et al.* 1987;Schieppati and Nardone 1991;Bloem *et al.* 1993;Burleigh *et al.* 1995;Horak *et al.* 1996].

BGA markedly affects the amplitude of short latency and ML stretch responses, and differences in BGA can partially explain 'changes' in response amplitudes of patients and controls [Bedingham and Tatton 1984;Allum and Mauritz 1984;Bloem *et al.* 1993]. However, it is unlikely that the observed amplitude differences of ML activity and balance correcting responses were attributable to high BGA alone, for three reasons: *(a)* BGA was corrected using a subtraction method, a technique that adequately removes the confounding influence of BGA [Bloem *et al.* 1993]; *(b)* ML and balance correcting responses were significantly enlarged in soleus, which did not have elevated BGA levels; and *(c)* no group differences were observed for early stretch reflexes (which are very sensitive to BGA changes [Bedingham and Tatton 1984; Allum and Mauritz 1984]), suggesting that the correction for BGA was effective.

A second possible confounding influence is the stooped posture of PD patients. As noted above, patients were purposely studied in their preferred stance, hence initial posture likely differed between patients and controls. Indeed, the pattern in patients of high BGA in tibialis anterior and normal BGA in soleus also occurs in healthy subjects who assume a stooped posture [Bloem *et al.* 1999]. However, this study did not report the amplitude changes we have observed for ML and balance correcting responses. It remains possible that a stooped posture partially explained the stiffening observed in this study, and further studies are required to disentangle the primary ('disease related') postural abnormalities and the secondary (compensatory) changes due to *e.g.* a stooped posture. Such information could help guide the development of new treatment strategies for secondary changes, such as physiotherapy to improve posture and reduce trunk inflexibility.

Clinical Implications

These findings may help clarify some clinical features observed in PD, and offer opportunities for new treatment strategies. Our study is one of the first to provide a pathophysiological explanation why PD patients might fall particularly backward and sideways. Co-contraction and concomitant stiffness of the ankles and trunk seemed to play a major role in causing these falls. In terms of stability, it is unimportant whether this stiffness was primarily disease-related or a secondary manifestation because, irrespective of its cause, co-contraction impairs postural responses to multidirectional perturbations. Trunk stiffening caused by tonic co-contraction

may help to reduce sway under static conditions, but can be deleterious if phasically applied to unexpected postural perturbations because the trunk action is forced to 'follow' the direction of the fall. Indeed, young healthy subjects whose trunk was 'artificially' stiffened by a rigid corset had similarly directed roll movements as PD patients and were severely unstable on a moving platform [Grüneberg *et al.* 2001]. An abnormal trunk movement in roll could explain the high incidence of hip fractures in PD, which mostly occur after falls sideways onto the affected side [Greenspan *et al.* 1998]. This notion may have particular implications for the prevention of hip fractures, *e.g.* by using physiotherapy to reduce co-contraction and stiffness of the ankles and trunk [Bridgewater and Sharpe 1997].

The absence of abduction movements of the arms (as healthy persons do in an attempt to cushion the fall) could explain why wrist fractures are relatively rare in PD. The potential association with startle and fear of falls may have therapeutic implications. Reduction of fear, perhaps by cognitive training or physical therapy, could help to improve balance control.

Finally, our results suggest that antiparkinson medication gave little improvement of the observed postural abnormalities. This finding highlights the need for development of alternative treatments. As mentioned earlier, one promising approach is the use of rehabilitative strategies, such as physiotherapy to reduce co-contraction and therefore trunk stiffness [Bridgewater and Sharpe 1997]. Another option is stereotactic deep brain surgery (stimulation or lesions), that often leads to marked alleviation of parkinsonian manifestations in the extremities [Bloem *et al.* 2001b]. The effects on axial features of PD are less well studied, but we are currently investigating whether bilateral stimulation of the subthalamic nucleus can be used to improve trunk and arm control in PD.

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REFERENCES

Allum JHJ, Bloem BR, Carpenter MG, Honegger F (2001a) Differential diagnosis of proprioceptive and vestibular deficits using dynamic support-surface posturography. Gait & Posture (in press)

Allum JHJ, Carpenter MG, Honegger F, Adkin AL, Bloem BR (2001b) Age-dependent variations in the directional sensivity of balance corrections. In: Duysens J, Smits-Engelsman BCM, Kingma H, editors. Control of Posture and Gait. Maastricht: Symposium of the International Society for Postural and Gait Research 912-915

Allum JHJ, Honegger F (1998) Interactions between vestibular and proprioceptive inputs triggering and modulating human balance-correcting responses differ across muscles. Exp Brain Res 121:478-494

Allum JHJ, Keshner EA, Honegger F, Wuetrich R. Disturbance of posture in patients with Parkinson's disease (1988) In: Amblard B, Berthoz A, Clarac F, editors. Posture and gait: development, adaptation and modulation. Amsterdam: Elsevier 245-257

Allum JHJ, Mauritz K-H (1984) Compensation for intrinsic muscle stiffness by short-latency reflexes in human triceps surae. J Neurophysiol 52: 797-818

Beckley DJ, Bloem BR, Remler MP (1993) Impaired scaling of long latency postural reflexes in patients with Parkinson's disease. Electroenceph Clin Neurophysiol 89: 22-28.

Beckley DJ, Bloem BR, van Dijk JG, Roos RAC, Remler MP (1991) Electrophysiological correlates of postural instability in Parkinson's disease. Electroenceph Clin Neurophysiol 81: 263-268.

Bedingham W, Tatton WG (1984) Dependence of EMG responses evoked by imposed wrist displacements on pre-existing activity in the stretched muscles. Can J Neurol Sci 11: 272-280

Berardelli A, Sabra AF, Hallett M (1983) Physiological mechanisms of rigidity in Parkinson's disease. J Neurol Neurosurg Psychiatry 46: 45-53

Blin O, Ferrandez AM, Pailhous J, Serratrice G (1991) Dopa-sensitive and dopa-resistant gait parameters in Parkinson's disease. J Neurol Sci 103: 51-54

Bloem BR (1994) Postural reflexes in Parkinson's disease. Delft: Eburon

Bloem BR, Beckley DJ, van Dijk JG, Zwinderman AH, Remler MP, Roos RAC (1996) Influence of dopaminergic medication on automatic postural responses and balance impairment in Parkinson's disease. Mov Disord 11: 509-521

Bloem BR, Beckley DJ, van Dijk JG, Zwinderman AH, Roos RAC (1992) Are medium and long latency reflexes a screening tool for early Parkinson's disease? J Neurol Sci 113: 38-42

Bloem BR, Beckley DJ, van Hilten JJ, Roos RAC (1998a) Clinimetrics of postural instability in Parkinson's disease. J Neurol 245: 669-673

Bloem BR, Grimbergen YAM, Cramer M, Willemsen MD, Zwinderman AH (2001a) Prospective assessment of falls in Parkinson's disease. J Neurol (in press)

Bloem BR, van Dijk JG, Beckley DJ (1999) Are automatic postural responses in patients with Parkinson's disease abnormal due to their stooped posture? Exp Brain Res 124: 481-488

Bloem BR, van Dijk JG, Beckley DJ, Zwinderman AH, Remler MP, Roos RAC (1993) Correction for the influence of background muscle activity on stretch reflex amplitudes. J Neurosci Methods 46: 167-174

Bloem BR, van Vugt JPP, Beckley DJ (2001b) Postural instability and falls in Parkinson's disease. Adv Neurol 87: 209-223

Bloem BR, van Vugt JPP, Beckley DJ, Remler MP, Roos RAC (1998b) Habituation of lower leg stretch reflexes in Parkinson's disease. Electroenceph Clin Neurophysiol 109: 73-77

Bonnet AM, Loria Y, Saint-Hilaire MH, Lhermitte F, Agid Y (1987) Does long-term aggravation of Parkinson's disease result from nondopaminergic lesions? Neurology 37: 1539-1542

Bridgewater KJ, Sharpe MH (1997) Trunk muscle training and early Parkinson's disease. Physiother Theory Pract 13: 139-153

Bridgewater KJ, Sharpe MH (1998) Trunk muscle performance in early Parkinson's disease. Phys Ther 78: 566-676

Burleigh-Jacobs A, Horak FB, Nutt JG, Obeso JA (1997) Step initiation in Parkinson's disease: influence of levodopa and external sensory triggers. Mov Disord 12: 206-215

Burleigh AL, Horak FB, Nutt JG, Frank JS (1995) Levodopa reduces muscle tone and lower extremity tremor in Parkinson's disease. Can J Neurol Sci 22: 280-285

Carpenter MG, Allum JHJ, Honegger F (1999b) Directional sensitivity of stretch reflexes and balance corrections for normal subjects in the roll and pitch planes. Exp Brain Res 129: 93-113

Carpenter MG, Allum JHJ, Honegger F (1999a) Influence of acute unilateral vestibular loss on postural responses in multiple directions. Gait & Posture 9 (Suppl.1): S2

Cody FWJ, Macdermott N, Matthews PBC, Richardson HC (1986) Observations on the genesis of the stretch reflex in Parkinson's disease. Brain 109: 229-249

Colebatch JG, Rothwell JC, Day BL, Thompson PD, Marsden CD (1990) Cortical outflow to proximal arm muscles in man. Brain 113: 1843-1856

Diener HC, Scholz E, Guschlbauer B, Dichgans J (1987) Increased shortening reaction in Parkinson's disease reflects a difficulty in modulating long loop reflexes. Mov Disord 2: 31-36

Dietz V, Berger W, Horstmann GA (1988) Posture in Parkinson's disease: impairment of reflexes and programming. Ann Neurol 124: 660-669

Dietz V, Zijlstra W, Assaiante Ch, Trippel M, Berger W (1993) Balance control in Parkinson's disease. Gait & Posture 1: 77-84

Dietz V, Zijlstra W, Prokop T, Berger W (1995) Leg muscle activation during gait in Parkinson's disease: adaptation and interlimb coordination. Electroenceph Clin Neurophysiol 97: 408-415

Eng JJ, Winter DA, MacKinnon CD, Patla AE (1992) Interaction of reaction moments and centre of mass displacement for postural control during voluntary arm movements. Neurosci Res Commun 11: 73-80

Gill J, Allum JHJ, Held-Ziolkowska M, Carpenter MG, Honegger F, Pierchala K (2001) Trunk sway measures of dynamic equilibrium during clinical balance tasks: effects of age. J Gerontol Med Sci (in press)

Goetz CG, Stebbins GT, Shale HM (1994) Utility of an objective dyskinesia rating scale for Parkinson's disease: inter- and intrarater reliability assessment. Mov Disord 9: 390-394

Greenspan SL, Myers ER, Kiel DP, Parker RA, Hayes WC, Resnick NM (1998) Fall direction, bone mineral density, and function: risk factors for hip fracture in frail nursing home elderly. Am J Med 104: 539-545

Grüneberg C, Allum JHJ, Honegger F, Bloem BR (2001) The influence of artificially increased trunk stiffness on automatic balance correcting responses. In: Duysens J, Smits-

Engelsman BCM, Kingma H, editors. Control of Posture and Gait. Maastricht : Symposium of the International Society for Postural and Gait Research 359-364

Horak FB, Frank JS. Three separate postural systems affected in parkinsonism (1996) In: Stuart DG, Gurfinkel VS, Wiesendanger M, editors. Motor control, volume 7. Tucson: Motor Control Press 343-346

Horak FB, Frank JS, Nutt JG (1996) Effects of dopamine on postural control in parkinsonian subjects: scaling, set, and tone. J Neurophysiol 75: 2380-2396

Horak FB, Nutt JG, Nashner LM (1992) Postural inflexibility in parkinsonian subjects. J Neurol Sci 111: 46-58

Hufschmidt A, Stark K, Lucking CH (1991) Contractile properties of lower leg muscles are normal in Parkinson's disease. J Neurol Neurosurg Psychiatry 54: 457-460

Hughes AJ, Daniel SE, Kilford L, Lees AJ (1992) Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study of 100 cases. J Neurol Neurosurg Psychiatry 55: 181-184

Johnell O, Melton ILJ, Atkinson EJ, O'Fallon WM, Kurland LT (1992) Fracture risk in patients with parkinsonism: a population based study in Olmsted County, Minnesota. Age Ageing 21: 32-38

Keshner EA, Allum JHJ, Pfaltz CR (1987) Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit. Exp Brain Res 69: 77-92

Koller WC, Glatt S, Vetere-Overfield B, Hassanein R (1989) Falls and Parkinson's disease. Clin Neuropharmacol 2: 98-105

Lakke JPWF (1985) Axial apraxia in Parkinson's disease. J Neurol Sci 69: 37-46

Lang AE (1995) Clinical rating scales and videotape analysis. In: Koller WC, Paulson G, editors. Therapy of Parkinson's disease. New York: Marcel Dekker 21-46

Lee RG, Tonolli I, Viallet F, Aurenty R, Massion J (1995) Preparatory postural adjustments in parkinsonian patients with postural instability. Can J Neurol Sci 22: 126-135

Maki BE, McIlroy WE (1997) The role of limb movements in maintaining upright stance: the 'Change in Support' strategy. Phys Ther 77: 488-507

Martin JP (1965) Tilting reactions and disorders of the basal ganglia. Brain 88: 855-874

McIlroy WE, Maki BE (1994) Compensatory arm movements evoked by transient perturbations of upright stance. In: Taguchi K, Igarashi M, Mori S, editors. Vestibular and neural front. Amsterdam: Elsevier 489-492

McIlroy WE, Maki BE (1995) Early activation of arm muscles follows external perturbations of upright stance. Neurosci Lett 184: 177-180

Nardone A, Corrà T, Schieppati M (1990) Different activations of the soleus and gastrocnemii muscles in response to various types of stance pertubation in man. Exp Brain Res 80: 323-332

Nutt JG, Hammerstad JP, Gancher ST (1992) Parkinson's disease: 100 maxims. London: Edward Arnold

Nutt JG, Marsden CD, Thompson PD (1993) Human walking and higher-level gait disorders, particularly in the elderly. Neurology 43: 268-279

Powell LE, Myers AM (1995) The activities-specific balance confidence (ABC) scale. J Gerontol Med Sci 50A: M28-M34

Rogers MW (1996) Disorders of posture, balance, and gait in Parkinson's disease. Clin Geriatr Med 12: 825-845

Rothwell JC (1994) Control of Human Voluntary Movement. London: Chapman & Hall

Sato Y, Manabe S, Kuno H, Oizumi K (1999) Amelioration of osteopenia and hypovitaminosis D by 1 alpha-hydroxyvitamin D3 in elderly patients with Parkinson's disease. J Neurol Neurosurg Psychiatry 66: 64-68

Schenkman M, Morey M, Kuchibhatla M (2000) Spinal flexibility and balance control among community-dwelling adults with and without Parkinson's disease. J Gerontol Med Sci 55: M441-M445

Schieppati M, Nardone A (1991) Free and supported stance in Parkinson's disease. Brain 114: 1227-1244

Scholz E, Diener HC, Noth J, Friedemann HH, Dichgans J, Bacher M (1987) Medium and long latency EMG responses in leg muscles: Parkinson's disease. J Neurol Neurosurg Psychiatry 50: 66-70

Smithson F, Morris ME, Iansek R (1998) Performance on clinical tests of balance in Parkinson's disease. Phys Ther 78: 577-592

Tatton WG, Lee RG (1975) Evidence for abnormal long-loop reflexes in rigid Parkinsonian patients. Brain Res 100: 671-676

Timmann D, Horak FB (1997) Prediction and set-dependent scaling of early postural responses in cerebellar patients. Brain 120: 327-337

Tinetti ME (1986) Performance-oriented assessment of mobility problems in elderly patients. J Am Geriatr Soc 34: 119-126

Valldeoriola F, Valls-Sole J, Tolosa E, Ventura PJ, Nobbe FA, Marti MJ (1998) Effects of a startling acoustic stimulus on reaction time in different parkinsonian syndromes. Neurology 51: 1315-1320

Valls-Sole J, Valldeoriola F, Tolosa E, Nobbe F (1997) Habituation of the auditory startle reaction is reduced during preparation for execution of a motor task in normal human subjects. Brain Res 751: 155-159

Waterston JA, Hawken MB, Tanyeri S, Jantti P, Kennard C (1993) Influence of sensory manipulation on postural control in Parkinson's disease. J Neurol Neurosurg Psychiatry 56: 1276-1281

Table 1. Baseline clinical characteristics. Data are displayed as mean ± standard deviation or as the number of persons, as well as number of persons for which information was available (percentage between parentheses).

	Patients (N = 10)	Controls (N = 11)	Significance
Age (years)	63.7 ± 6.1	68.1 ± 4.8	p = 0.08
Women	5 (50%)	6 (55%)	p = 0.60
Height (m)	1.7 ± 0.1	1.7 ± 0.1	p = 0.84
Weight (kg)	70.4 ● 12.6	76.2 ± 13.1	p = 0.32
Duration of disease (years)	10.4 ± 7.2	—	—
Fallers (≤ 3 months)	6 (60%)	1 (12.5%)	p = 0.07
Fear of falling	6 (60%)	1 (12.5%)	p = 0.07
ABC scale	6.2 ± 1.9	8.9 ± 1.1	p < 0.01
Hoehn & Yahr stage [a]			
'On' condition	2.6 ± 0.6	—	—
'Off' condition [b,c]	2.9 ± 0.6	—	—
UPDRS motor score			
'On' condition	31.8 ± 12.8	0.2 ± 0.4	p < 0.001
'Off' condition [b,d]	39.4 ± 14.3	—	
Tinetti Mobility Index, total score			
'On' condition	7.7 ● 5.1	0.0 ± 0.0	p < 0.005
'Off' condition [b,e]	10.3 ± 6.3	—	
Medication			
Levodopa / carbidopa	9 (90%)	—	
Dopamine receptor agonist	8 (80%)	—	
Amantadine	8 (80%)	—	
Anticholinergic	4 (40%)	—	
Other			
Benzodiazepine	4 (40%)	—	
Clozapine	4 (40%)	—	
Cisapride	1 (10%)	—	
Omeprazol	1 (10%)	1 (17%)	
Diuretics	0 (0%)	1 (17%)	

[a] Individual Hoehn and Yahr stages were stage 1.5 (n = 1), stage 2.5 (n = 6), stage 3 (n = 2) and stage 4 (n = 1) for the ON condition, and stage 2.5 (n = 4), stage 3 (n = 2) and stage 4 (n = 1) for the OFF condition; [b] seven patients were tested during the OFF condition; controls were only tested once; [c] p = 0.06 (ON versus OFF); [d] p < 0.05 (ON versus OFF); [e] p = 0.07 (ON versus OFF)

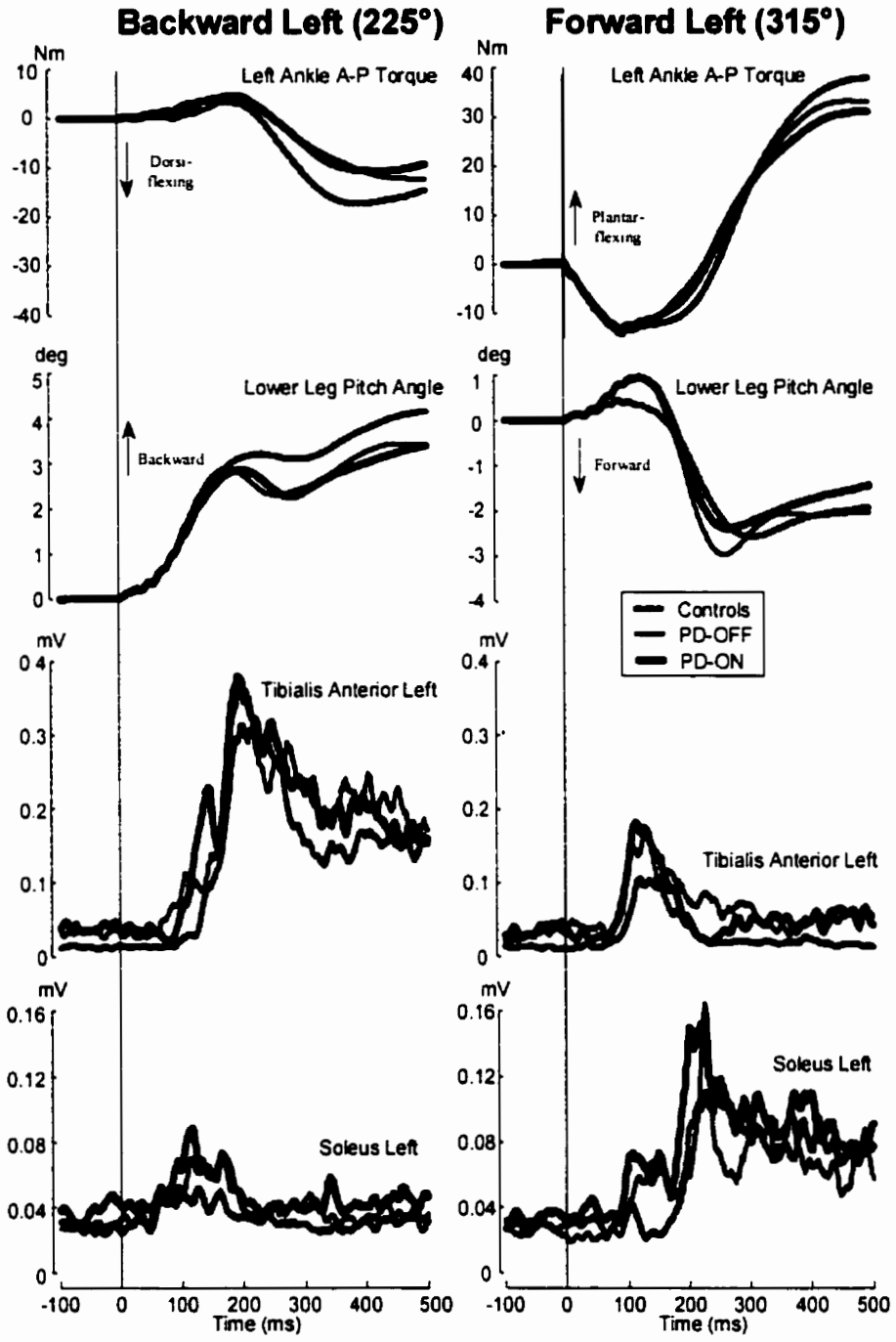

Figure 1. Average lower leg biomechanical and EMG traces for patients (both ON and OFF) and controls. Responses are shown for surface rotations of 7.5 deg at 60 deg/s, directed backward left (225°) or forward left (315°). The black vertical line at 0 ms represents the onset of ankle rotation.

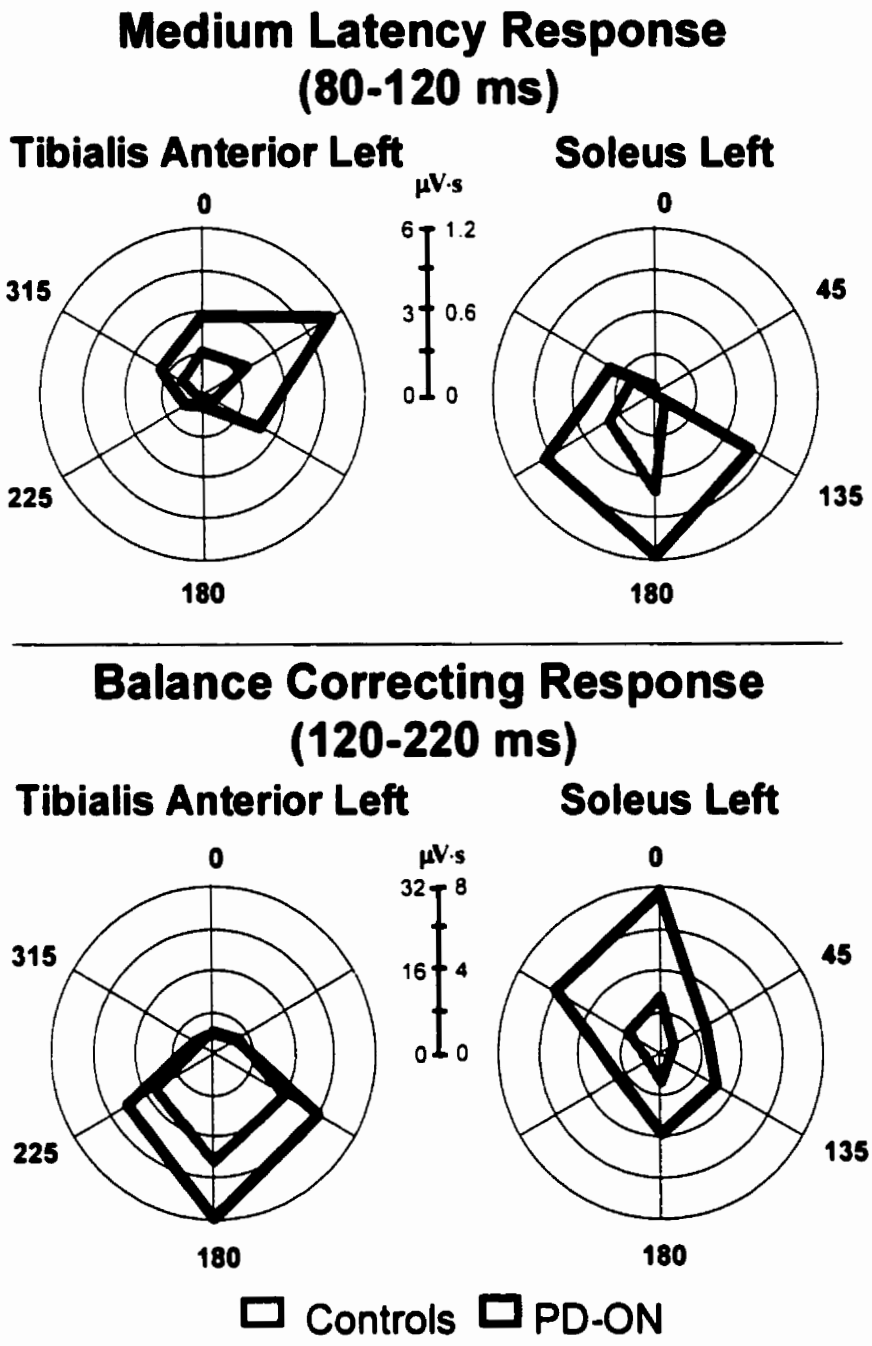

Figure 2. Polar plots for medium latency (top panel) and balance correcting responses (bottom panel) for left tibialis anterior (left plots) and left soleus (right plots). Each radial line ('spoke') represents one of six platform directions (0°, 45°, 135°, 180°, 225°, 315°, in a clockwise notation). For each direction, mean muscle activity is plotted for PD-ON patients and controls. The response amplitude represented by each of the concentric circles in the plot is scaled according to the vertical scale between each set of plots.

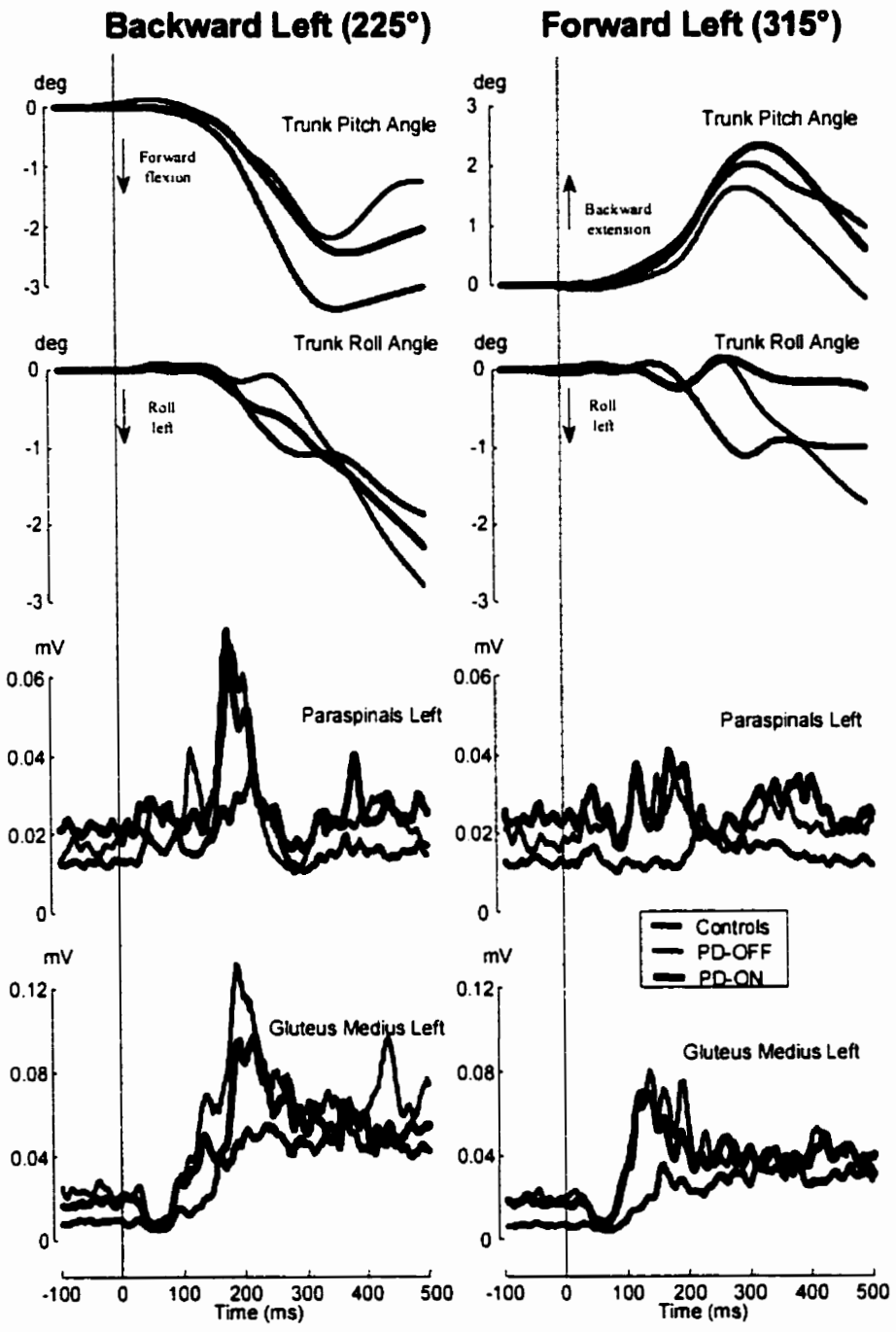

Figure 3. Average biomechanical and EMG traces from the hip and trunk for patients (both ON and OFF) and controls. Responses are shown for surface rotations of 7.5 deg at 60 deg/s, directed backward left (225°) or forward left (315°). The black vertical line at 0 ms represents the onset of ankle rotation.

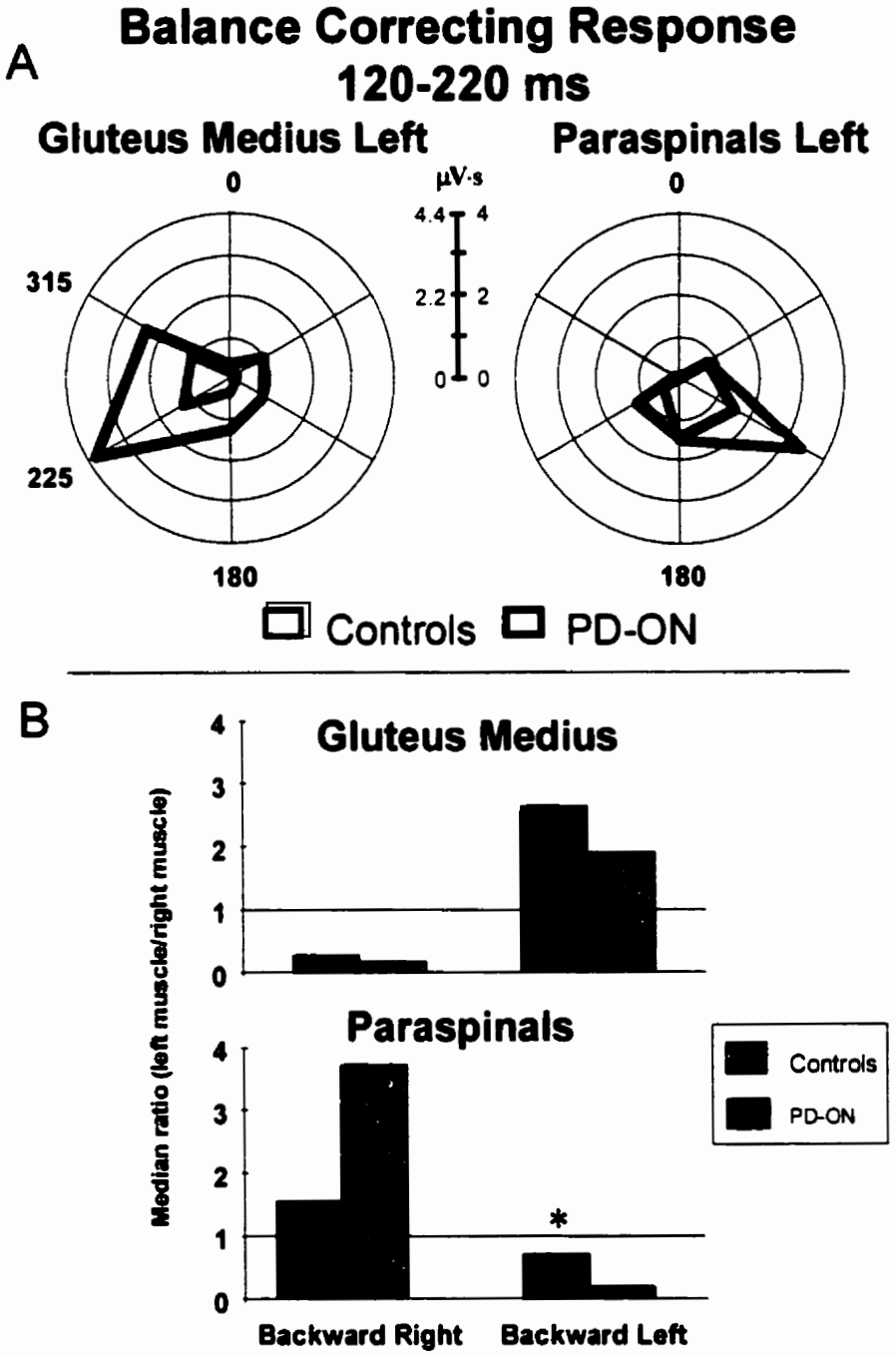

Figure 4. (A) Polar plots for balance correcting responses for left gluteus medius (left plot) and left paraspinal (right plot). Each radial line ('spoke') represents one of six platform directions (0°, 45°, 135°, 180°, 225°, 315°, in a clockwise notation). For each direction, mean muscle activity is plotted for PD-ON patients and controls. The response amplitude represented by each of the concentric circles in the plot is scaled according to the vertical scale between each set of plots. (B) Asymmetry index (ratio between median amplitude of left muscle divided by median amplitude of right muscle), illustrated for gluteus medius and paraspinal muscles for backward right (135°) and backward left (225°) directions.

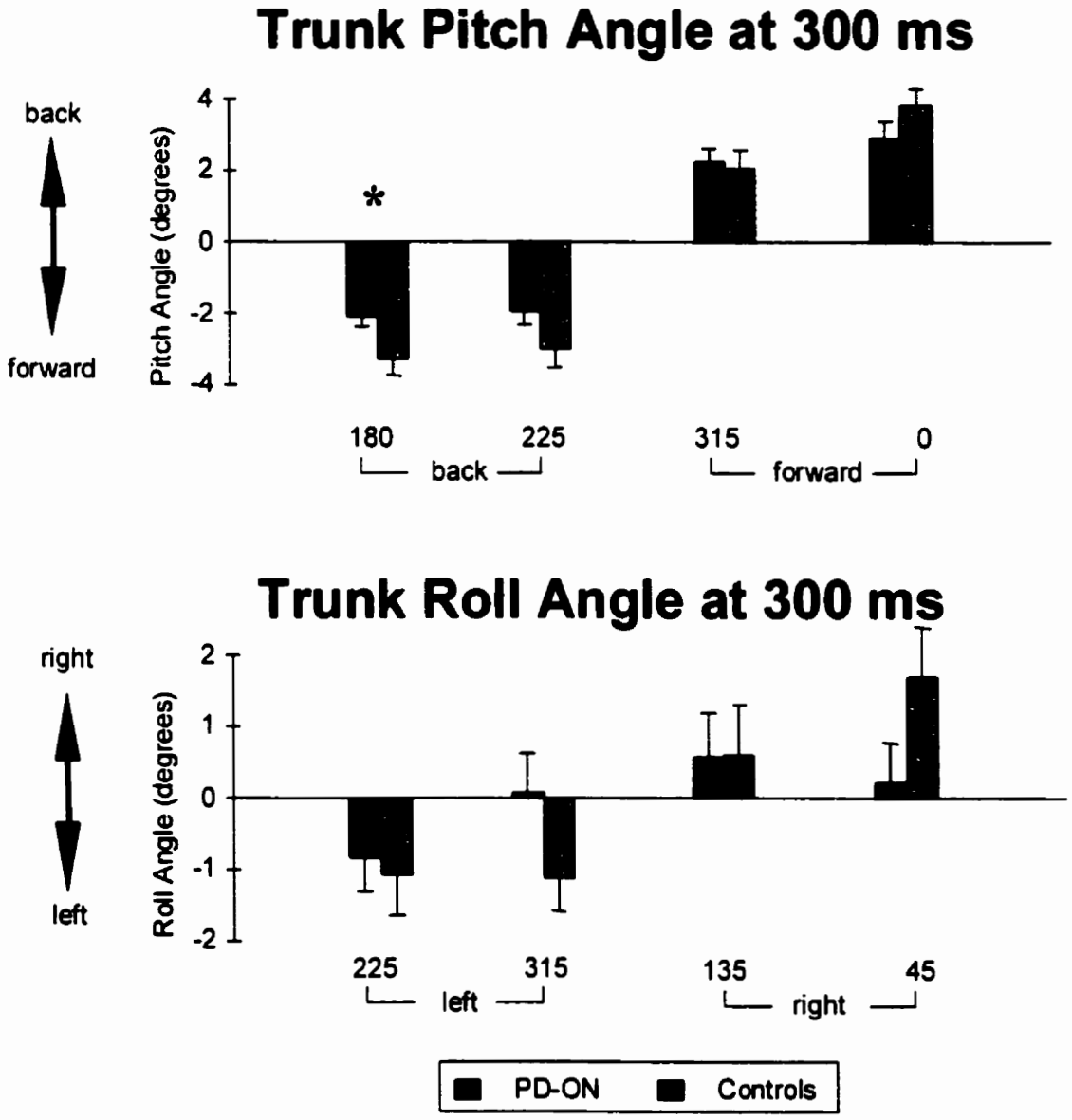

Figure 5. Mean angles of trunk pitch (top panel) and trunk roll (bottom panel), measured at 300 ms for PD-ON patients and controls. Error bars indicate the standard error of the mean. The direction of the support-surface rotation (7.5 deg amplitude at 60 deg/s) is shown in the plot abscissa.

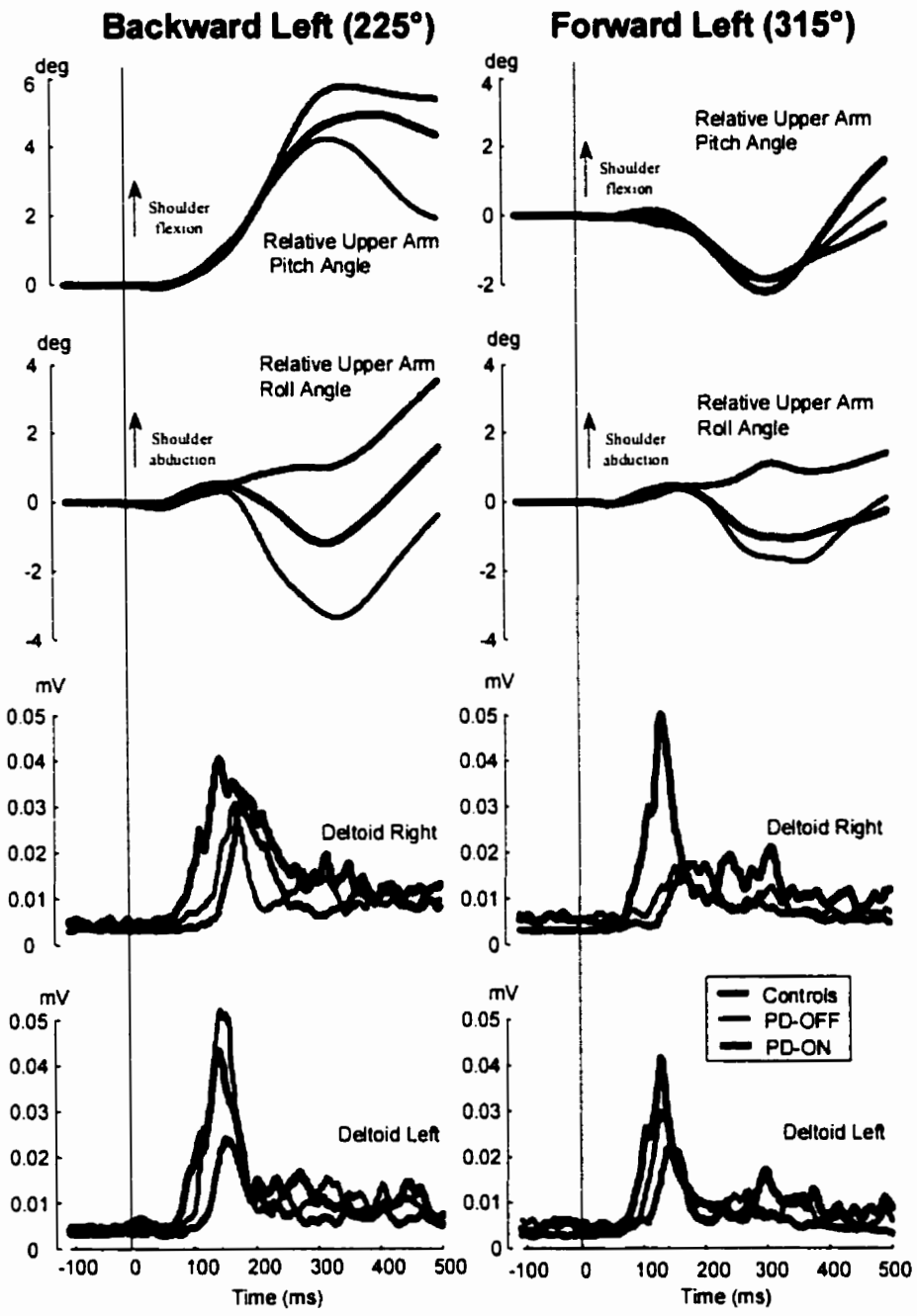

Figure 6. Mean angles of arm pitch and arm roll (relative to the trunk segment) and EMG traces from medial deltoids are shown for patients (both ON and OFF) and controls. Responses in the left panel are for surface rotations directed backward left (225°); responses in the right panel represent rotations directed forward left (315°). The black vertical line at 0 ms represents the onset of ankle rotation.

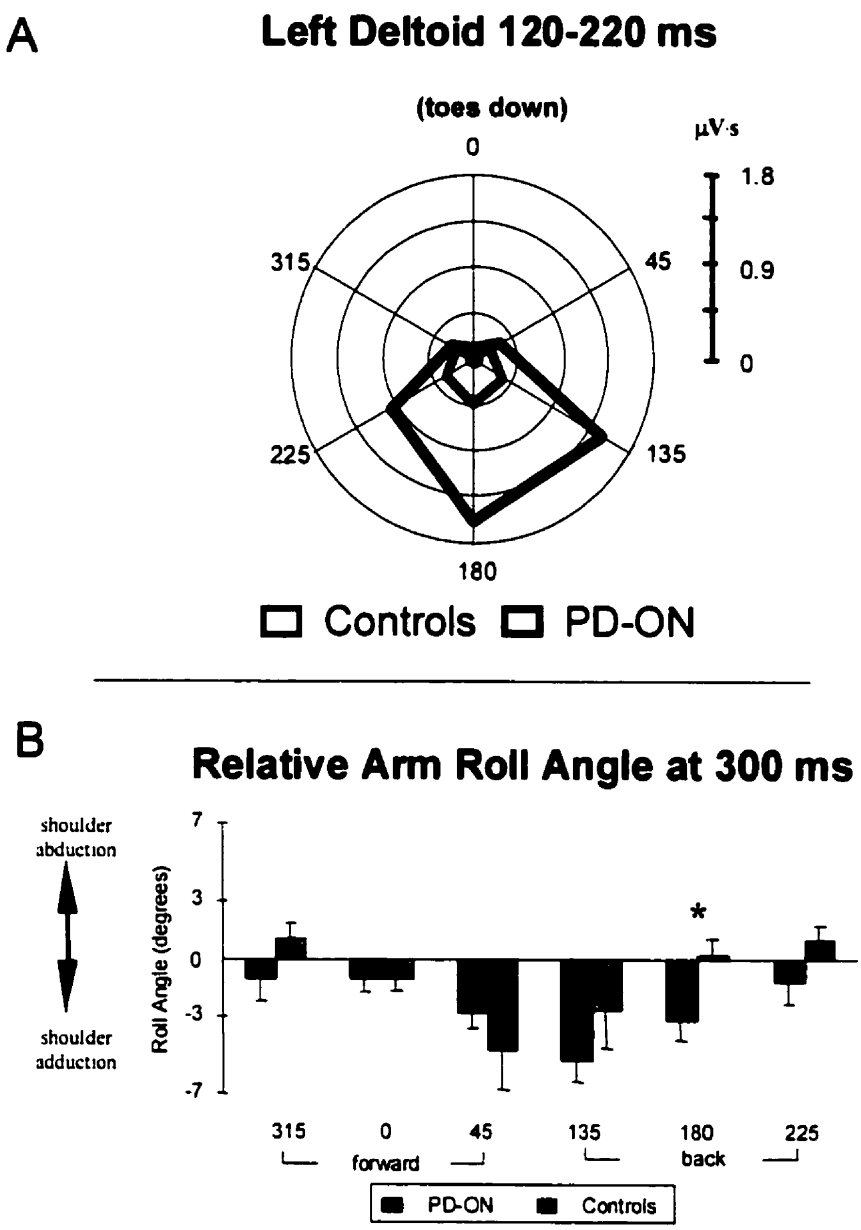

Figure 7. (A) Polar plots for balance correcting responses in left medial deltoid muscle. Each radial line ('spoke') represents one of six platform directions (0°, 45°, 135°, 180°, 225°, 315°, in a clockwise notation). For each direction, mean muscle activity is plotted for PD-ON patients and controls. The response amplitude represented by each of the concentric circles in the plot is scaled according to the vertical scale on the left side of the plot. (B) Mean arm roll angle (relative to the trunk) measured at 300 ms, for PD-ON patients and controls. Error bars indicate the standard error of the mean. The direction of the support-surface rotation (7.5 deg at 60 deg/s) is shown in the plot abscissa.

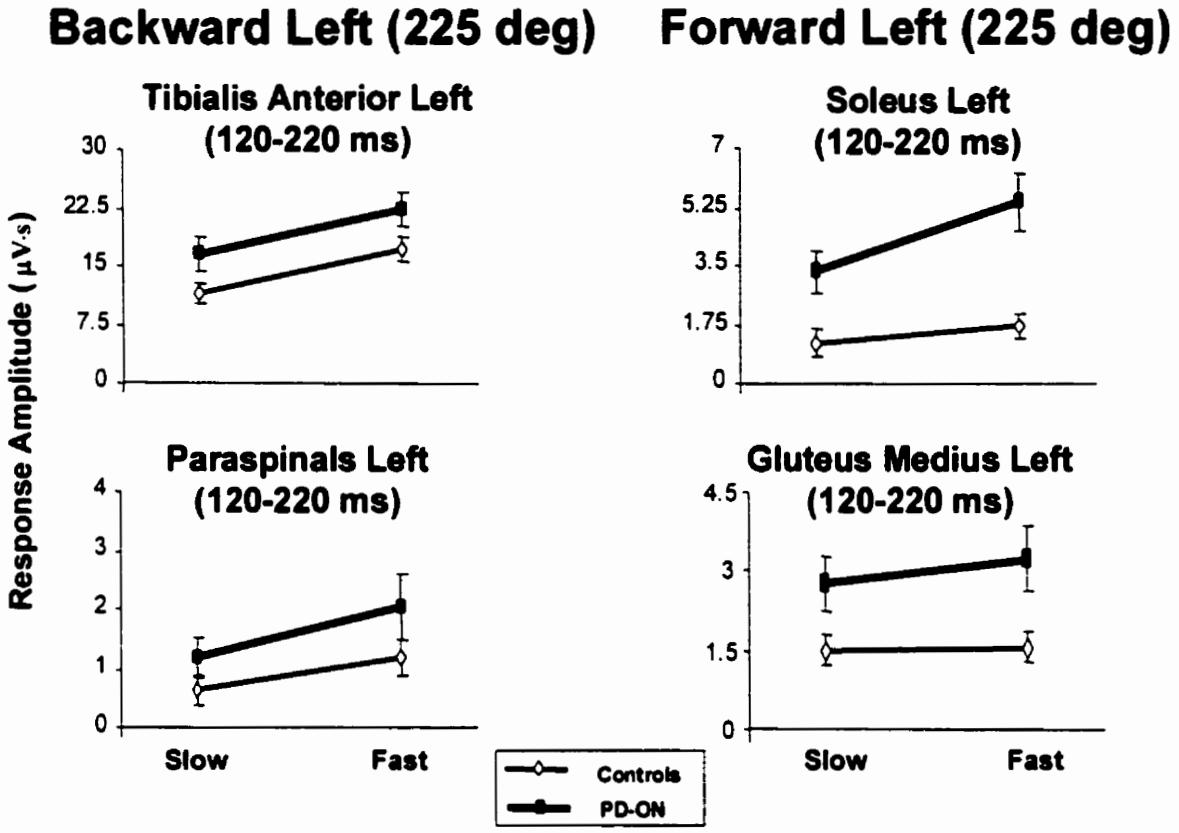

Figure 8. Mean balance correcting activity induced by slow (30 deg/s) and fast (60 deg/s) platform velocities is shown for PD-ON patients and controls. Error bars indicate the standard error of the mean. The left panel shows response amplitudes for backward left (225°) perturbations; the right panel shows response amplitudes for forward left (225°) perturbations.

CHAPTER 5

INFLUENCE OF POSTURAL THREAT ON POSTURAL REACTIONS TO MULTI-DIRECTIONAL SURFACE ROTATIONS

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ABSTRACT

Previous studies have shown significant effects of increased postural threat in even healthy young individuals when standing, performing voluntary postural tasks or recovering from an unexpected push from behind. However, all of these studies examined postural control in only the sagittal plane. The present study examined how increased postural threat influenced postural reactions to unexpected surface rotations in multiple directions.

Ten healthy young adults (mean age 25.5, range 22-27 years) were required to recover from unexpected rotations of the support surface (7.5 deg amplitude, 50°/s velocity) delivered in six different directions while standing in a low postural threat (surface height 60 cm above ground) or high postural threat (surface height 160 cm above ground) condition. From full body kinematics, joint angular displacements and total body centre of mass (COM) displacement and velocity was calculated. Electromyographical data from 10 different postural leg, hip and trunk muscles was collected simultaneously.

Increased postural threat caused significant increases in the automatic balance correcting responses (120-220 ms after perturbation onset) in all postural muscles. Despite this increase, the directional sensitivity of balance correcting activity was preserved in all muscles except in two muscles. Altered pitch sensitivity in biceps femoris and roll sensitivity in paraspinals was observed. Peak anterior-posterior displacements of COM were reduced when postural threat was increased, which was likely achieved through co-contraction of leg and trunk muscles and increased arm movements. Increased postural threat was also related to significant reductions in perceived balance confidence and perceived stability and increases in perceived anxiety. In conclusion, postural threat has been shown to significantly increase muscle reactions and reduce biomechanical responses comprising responses to an unexpected perturbation and should be strongly considered as a potentially confounding variable when using dynamic posturography to diagnose patients in whom fear of falling may be prevalent.

INTRODUCTION

Fear of falling has been shown to have a significant relationship with balance control and falling, especially in the aging population. Approximately 50% of older adults who have previously fallen have reported having a fear of falling (Arfken et al. 1994; Niino et al. 2000; Tinetti et al. 1994; Vellas et al. 1997; Walker and Howland; 1991). Fear of falling does not only occur following a fall, since over 30% of older individuals who have never fallen report a fear of falling (Downton and Andrews, 1990). Fear of falling has been shown to be related to an increased risk of falls (Cumming et al. 2000) as well as impairments to balance and gait control (Tinetti et al. 1994; Vellas et al. 1997; Meyers et al. 1996; Hill et al. 1996).

Recent efforts have been made to determine how fear of falling may contribute to specific aspects of balance control. It has been established that increased postural threat can cause significant changes in postural sway, muscle tone and ankle stiffness during quiet standing, which are indicative of a tighter control of the COM, both in young healthy adults (Carpenter et al. 1999a; 2001a; Adkin et al 2000) and in elderly (Carpenter et al. 1999b). Increased postural threat also influences the preparatory postural adjustments and subsequent voluntary control of a rise-to-toes task (Adkin et al. 2001a).

In all of the aforementioned studies, postural threat was manipulated by altering the height at which the participant stood above the ground (Carpenter et al. 1999a; 2001a; Adkin et al. 2000; 2001a). Alterations of surface height, using an elevated maze, also is commonly used in studies with mice to examine anxiety related behaviour to different pharmacological or genetic manipulations of anxiety (Lepicard et al. 2000). In humans, increasing the surface height on which an individual stands has been shown to significantly alter their perceived balance confidence, perceived anxiety, as well as cause physiological changes such as increased blood pressure (Carpenter et al. 1999b) and increased skin conductance (Adkin et al. 2001a).

However, most falls in the elderly do not occur during quiet stance, but instead are a result of environmental conditions or unexpected perturbations to balance (Holliday et al. 1990). Therefore, a more interesting question is how increased fear of falling can influence automatic postural reactions to unexpected perturbations to stance. Fear of falling frequently has been

implied as a possible confounding factor which may influence postural reactions to unexpected perturbations (Bloem et al. 2001; Maki and McIlroy, 1996; Maki and Whitelaw, 1993). However, there have been few studies that have examined the extent to which fear of falling may influence postural reactions. It has been found that fear of falling and decreased balance confidence are prevalent in patients with balance deficits, such as vestibular loss, proprioceptive loss (Yardley and Hallam, 1996) and Parkinson's disease (Adkin et al 2001b; Carpenter et al. 2001b). Therefore, any interaction between fear of falling and postural reactions may play an important role as a confounding factor when trying to use dynamic posturography to diagnose or discriminate between those patients in whom fear of falling may be prevalent, and those with the same disease state, but without a fear of falling.

Brown and Frank (1997) were the first to examine the influence of increased postural threat on postural reactions to unexpected perturbations. Participants were required to recover from unexpected perturbations delivered to the back while standing under different conditions of postural threat, manipulated through changes in surface height. The results revealed significant changes in the peak magnitude and time to peak velocity of COM displacement which were consistent with a stiffening control strategy when standing in conditions of increased postural threat. However, the limitations of the study by Brown and Frank (1997) were that the perturbations were delivered in a constant direction (relative to the location of postural threat) and caused predictable falls in a single pitch plane. It is unclear to what extent findings established in the purely pitch plane can be applied to other directions of falling, which may occur more frequently in natural conditions. For example, lateral falls can account for up to 23-43% of reported falls (Maki and McIlroy, 1998) and represent the greatest threat for hip and wrist fractures (Cummings and Nevitt, 1994). It might be hypothesized that falls which contain a lateral component may pose a greater risk of injury and thus may elicit more distinct changes in postural control than pitch directed falls when standing in conditions of increased postural threat.

Differences for postural control in pitch and roll planes to conditions of increased postural threat can be better examined using postural reactions which include those to multi-directional perturbations. Other balance characteristics in normal (Carpenter et al. 1999c; Henry et al.

1998, Maki et al. 1994a) and pathological populations (Carpenter et al. 2001b, 2001c) have been recently discovered using multidirectional perturbations which were previously undetected by pitch plane perturbations.

Therefore, the goal of the present study was to examine the influence of increased postural threat on postural reactions to unexpected surface rotations in combinations of both the pitch and roll planes. We have utilized different surface heights on which a person stands as a method of manipulating postural threat in otherwise healthy young adults. We have examined muscle activation patterns as well as segment and COM displacements to determine how both neuromuscular responses and resulting movement strategies may be influenced by an increase in postural threat.

METHODS

Subjects

Six male and four female university students (mean age 25.5 +/- 5.3 years) volunteered to participate in the study and provided informed consent in accordance with guidelines outlined by the Human Ethics Committee, University of Waterloo. Each participant was free from any neurological or othopaedic disorder as verified by self report. Prior to the experiment, anthropometric measures were recorded, including height (mean 174.5 +/- 9.9 cm), weight (73.4 +/- 11.7 kg) and leg length (86.7 +/- 6.7 cm). Subjects were tested barefoot, and wore tight fitting clothing.

Apparatus

As shown in figure 1, surface rotations were delivered using a single axis rotating platform which was bolted firmly to the front edge of a PENTALIFT hydraulic lift (minimum deck height = 20 cm, maximum=160 cm). The motor for the rotating platform could be swivelled 180° around a vertical axis, relative to its fixed base. A footplate with heelguides was attached to the top surface of the rotating platform that could also be swivelled 180° around a vertical axis. Therefore, with this system, the top of the rotating platform (on which the participants stood) could be maintained in a constant position, while the axis of rotation delivered by the platform motor could be manually turned relative to the position of the participant to achieve

platform rotations in multiple directions. This method is distinctly different than that used by Moore et al. (1988) in which the participant was turned relative to the axis of platform rotation. The benefit of the present apparatus was that the participant was completely unaware of the direction of upcoming rotation.

The distance of the axis of rotation to the front edge of the hydraulic lift was 38 cm. The top surface of the rotating platform was 40 cm above the deck of the hydraulic lift. Therefore, when the hydraulic lift was in the lowest position, the top surface of the rotating platform was 60 cm above the ground. Handrails were located 38 cm on the left and right side of centre of the rotating platform and bolted to the deck of the hydraulic lift so they could be raised along with the moving platform. There were no handrails located either in the front or back of the platform. Handrails were 165 cm long and 125 cm high above the top surface of the rotating platform (figure 1).

Procedure

Participants initially were fitted securely with a climbing harness that would support body weight from straps under the legs and around the shoulders and chest. Participants were seated while the hydraulic lift was raised to the first surface height condition, representing either a low postural threat (surface height from top of rotating surface to ground = 60 cm) or high postural threat (surface height from top of rotating surface to ground = 160 cm) condition. Order of initial surface height presentation was counter-balanced between subjects to remove any confounding effects due to learning. Seated participants completed questionnaires probing their balance confidence and task specific balance efficacy related to their ability to recover from balance perturbations at the height at which they were presently seated (Adkin et al. 2001b). Subjects then stood on the platform and the heelguides were adjusted to align the ankle joints with the pitch axis of the platform. The heelguides maintained a constant stance width and foot angle within and between participants. The feet were lightly strapped across the bridge of the foot to the surface of the platform and a climbing rope, attached to the ceiling, was fixed to the back of the safety harness. The supporting rope had enough slack so as to not provide any cutaneous information during normal movements on the platform, while still able to provide support in case of a fall. Two spotters were arranged with one on the hydraulic lift

behind the participant, and the other on the floor to the side of the hydraulic lift to lend support in case of a fall.

Participants were required to focus on a target on the wall approximately 6 m ahead and assumed a normal upright standing position, with knees straight and arms hanging comfortably at their sides. While standing in their 'preferred stance' position, a temporary ink marking was made on the side of their left lower leg which was aligned with a sight mounted on a fixed rigid rod located lateral to the left leg. Prior to each perturbation, the sight was used to verify that the subject was standing in their preferred stance position before the next perturbation was initiated. Subjects were presented with 37 randomly directed platform rotations in one series. All platform rotations were at a constant amplitude (7.5 deg) and had a velocity of 50 deg/s. The first trial of each series was an adaptation trial, which was excluded from further analysis to reduce habituation effects (Keshner et al. 1987). The following 36 perturbations consisted of 6 different directions, each randomly presented 6 times. Directions were achieved by unlocking the turning mechanisms on the motor and top plate of the rotating platform, and manually turning the pitch axis of rotation relative to the participant. Directions were each separated by 45 deg and will be referred to using clockwise notation, as if viewed from above. The perturbation directions were forward (toes-down – 0°), backward (toes-up - 180°) and four combinations of pitch and roll including forward right (45°), backward right (135°), backward left (225°) and forward left (315°). Between each trial, the orientation of the platform was manually altered by the experimenter without revealing the identity of the subsequent trial to the subject. Subjects were permitted to grasp the handrails in between each trial while the orientation of the platform was changed, however they were required to continuously look ahead at the target and were not aware of the new orientation of the platform.

At the end of each series of 37 trials, the feet were unstrapped, and the participant performed a 1 minute trial of quiet stance with eyes open and arms hanging at the sides. The participant was then seated and completed questionnaires on perceived anxiety and perceived stability with respect to their previous performance at the present surface height. A 5 minute rest was given to remove any possible effects due to fatigue, after which the platform was moved to the second surface height condition (either low or high).

While seated at the new surface height, questionnaires regarding the subject's balance confidence and task specific balance efficacy were repeated. Subjects stood and were required to assume the same normal 'preferred stance' as in the previous height condition, by ensuring the marking on the lower leg was aligned with the sighting rod on the platform frame. A second series of 37 randomly presented platform rotations was performed (36 trials plus 1 adaptation trial) followed by a one minute quiet standing trial. Participants were then seated and completed the perceived anxiety and perceived stability questionnaires, with respect to their latest performance at the new surface height.

Data Collection

Recordings of all biomechanical and electromyographical (EMG) data commenced 2 seconds prior to the onset of the perturbation and lasted 5 seconds. EMG recordings were sampled at 1024 Hz. Electromyographical recordings were made from disposable surface electrodes, placed 2 cm apart along the muscle bellies of 10 different muscles: left soleus, left tibialis anterior and bilaterally on rectus femoris, biceps femoris, gluteus medius, paraspinals and medial deltoids. Electrode leads were attached to a preamplifer unit which was attached to the rail beside the participant during testing. Therefore, no additional weight or encumberance was placed upon the participant. Kinematic data was recorded at 64 Hz using the OPTOTRAK (Northern Digital Canada Inc., Waterloo) motion analysis system. Twenty-one infrared emitting diodes (ireds) were placed on anatomical landmarks (bilaterally on the ankle, knee, greater trochanter, anterior superior iliac spine, iliac crest, lower rib, shoulder, elbow, wrist, temple and centre of zyphoid). Three additional ireds were placed at the front corners and centre of the forceplate to define pitch and roll movements of the moving platform.

Prior to each series of perturbations (both at low and high threat conditions), seated participants were required to estimate both their general balance confidence as well as their task specific balance efficacy in their abilities to (1) avoid a fall, (2) maintain concentration, (3) overcome worry, and (4) reducing nervousness during the postural task of recovering from an unexpected perturbation. This is in keeping with recommendations of McAuley and Mihalko (1998), that efficacy measures must be developed which are specific to the task.

General balance confidence was estimated on a percentage scale at each surface height, with 0 representing no confidence and 100 representing complete confidence.

After the completion of a series of postural perturbations and quiet stance trials at each surface height, seated participants completed perceived anxiety and perceived stability questionnaires. Perceived anxiety was assessed using a 16 item questionnaire, contextually modified from Smith et al. (1990), which probed 3 different elements of anxiety: somatic, worry, and concentration. Participants were required to score each item using a 9 point scale ranging from (1) 'I don't feel at all' to (9) 'I feel extremely'. For example, one question pertaining to somatic related anxiety (modified context underscored) reads 'My heart was racing when standing at this height' . Items were summed for a total perceived anxiety score for each threat condition. Perceived stability was estimated on a percentage scale, with 0 representing a feeling of complete instability and 100 representing a feeling of complete stability.

Data Analysis

Zero latency for each trial was determined as the first inflexion of the platform angle measured from an angular potentiometer. EMG signals were digitally full wave rectified and low pass filtered at 100 Hz. For each trial, background activity recorded 500 ms prior to perturbation onset was averaged for each muscle and subtracted from the rest of the EMG signal. EMG areas for all left sided muscles were calculated using trapezoid integration within pre-determined time intervals associated with early stretch (40-100 ms), medium latency responses (80-120 ms), balance correcting responses (120-220 ms), secondary balance correcting responses (240-340 ms) and stabilizing reactions (350-700 ms).

In order to determine the asymmetry of paraspinal muscle activity for perturbations containing a roll component we calculated an 'asymmetry ratio'. The asymmetry ratio was calculated by dividing the EMG areas (between 120-220 ms) recorded for backward left perturbations by those recorded for backward right perturbations. Purely symmetrical responses between the two perturbation directions will have a ratio equal to 1, whereas smaller or larger ratios indicate asymmetrical activation.

Onset latencies for stretch reflexes and balance correcting responses in left sided muscles were calculated for each trial and muscle. For each subject, the six individual muscle traces for a specific direction in a single series were displayed together on a screen. EMG latencies were determined using a semi-automatic computer algorithm that selected the first point that activity rose (and remained active longer than 50 ms) over a threshold of 2 standard deviations above mean activity calculated over the 500 ms period just prior to perturbation onset. Each latency first was selected by the computer algorithm, then approved or manually corrected by the operator. The same operator selected all of the latencies to maintain consistency across trials.

Note that EMG from one subject could not be used for analysis due to equipment difficulties. Across all subjects, a motor artifact was found in the left paraspinal traces which prevented further analysis. Thus, for graphical purposes throughout the paper, the mirror image of the directional responses of the right paraspinal muscle will be used to represent the left muscle responses.

Total body COM displacement was calculated in the anterior-posterior (A-P) medial-lateral (M-L) and vertical directions using a 14 body segments model which included 2 lower legs, 2 thighs, pelvis, 4 trunk, 2 upper arm, 2 lower arm and a head segment (for details refer to Winter et al. 1997). Position data was digitally filtered at 25 Hz using a zero-phase shift, dual pass Butterworth filter, from which segment angular displacements were calculated in the pitch and roll direction for each body segment. All EMG areas, latencies and kinematic results were averaged across perturbation direction. Subject averages were averaged together to yield group averages for low and high threat conditions.

The mean position of COM was calculated for each one minute standing trial. The mean value was then subtracted from each signal and filtered at 1.2 Hz using a zero-phase shift dual pass Butterworth filter. Root mean square (RMS) and mean power frequency (MPF) were calculated from the filtered COM signal (with the bias removed) for each standing trial.

Statistical Analysis

All EMG areas, latencies and kinematic results were examined using a 2 x 6 (threat by direction) repeated measures between and within subject analysis of variance. All significant main and interaction effects were further analysed using individual t-tests with a level of signficance of 0.05. Mean position of COM, RMS and MPF values calculated over the one minute standing trials were analyzed using a repeated measures one-way analysis of variance, with a level of significance of 0.05. Similarly, scores for general balance confidence, task specific balance efficacy, perceived anxiety and perceived stability were also examined using repeated measures one way ANOVA, with a level of significance of 0.05.

RESULTS

Segment Movements

As shown in figure 2, backward left perturbations caused the ankle initially to be driven passively into dorsi-flexion. At the same time the left lower leg segment rotated backwards, reaching a peak angular displacement at 250 ms after the onset of platform rotation (figure 3). Backward lower leg rotation, pulled the knee into extension and caused the upper thigh segment to also pitch backwards. With the leg segments falling backward, the pelvis and trunk segments pitched forward in the opposite direction (figure 2 and 3). Peak angular displacement was almost two times greater in the pelvis segment than for the upper trunk segment.

In general, when the platform rolls to the left, the lower leg, thigh and pelvis are rolled to the left, while the upper trunk is rolled in the opposite direction to the right (figure 3). Similar to pitch directed displacements, the pelvic segment had the largest angular deviations compared to all other leg and trunk segments. The pelvis began to roll left at approximately 50 ms and reached a peak left angular displacement of 2.5 deg by 200 ms. In contrast, the upper trunk fell to the right, in the opposite direction to roll displacement of the thigh and pelvis. Trunk roll angular displacement occurred around 30 ms and reached a peak amplitude of 1.2 deg at approximately 200 ms.

Standing in a high threat condition had only minor influence on segment displacements for backward left perturbations (figure 3). In the high threat condition, backward rotation of the

lower legs was reduced in amplitude but had similar velocity compared to the low threat condition. The degree to which the knee was pulled into extension was slightly reduced in the high threat condition, and remained less extended throughout the trial. There were minimal differences in the angular displacement profiles of both the pelvic and trunk segments, which pitched forward with similar peak amplitude and time to peak velocity in both the low and high threat conditions. In the roll plane, there were no noticeable differences in the traces of any of the leg, knee, pelvis or trunk roll angle for backward left perturbations between the two height conditions.

Forward left perturbations elicited segment displacements which were distinctly different from backward left perturbations in the pitch plane, with similar displacements between the two perturbations observed in the roll plane (figure 2). As shown in figure 4, as the platform rotated forward and to the left, the ankle became plantarflexed and inverted. The lower leg fell forward, reaching peak forward rotation after 200 ms, then returning slightly more vertical position where it was held throughout the remainder of the trial. The thigh segment was simultaneously pitched backwards reaching a peak angular displacement with similar time to peak as seen in the lower leg (200 ms). Forward lower leg rotation coupled with backward upper leg rotation resulted in a rapid flexion of the knee joint before straightening to within 1 deg of initial standing posture by 500 ms. The pelvic segment was first displaced backward, then reversed direction and pitched forwards after 150 ms. It should be noted that the overall pitch displacement of the pelvis was small for forward left perturbation (less than 1.5 deg) compared to the larger (>4 deg) rotation observed for backward left perturbations (figure 3 and 4). The upper trunk also pitched backwards for forward left perturbations. Trunk pitch backward displacement was later than for the pelvis, and continued to rotate backwards until 350 ms when it became relatively stable.

Roll angles for forward left perturbations were similar to those observed for backward left perturbations. As shown in figure 4, the lower leg had minimal roll rotation to the left which did not begin until after 200 ms. The thigh and pelvic segments were rotated to the left in the same direction as platform roll, reaching maximum roll at approximately 200 ms where they

remained throughout the trial. In contrast, the upper trunk segment rolled in the opposite direction (right) to leg and pelvic rotation, with similar onset and time to peak amplitude.

The effects of increased postural threat had a more dramatic influence on segment control during forward left perturbations compared to backward perturbations. Whereas most differences in high versus low threat conditions were in the lower leg angle in backward left perturbations, forward left perturbations had most changes occurring in the thigh and trunk segments. There was no noticeable difference in the forward displacement of the lower leg for high versus low threat conditions. In contrast, the thigh pitched backwards with a larger peak amplitude displacement and remained further back compared to the low condition for the remainder of the trial. These changes in thigh displacement yielded a larger peak knee angular displacement in the high threat condition. Despite changes in peak amplitude, there were no observable differences in onset or time to peak displacement for knee angle. Pelvis pitch angle reached the same peak amplitude in the high compared to low condition, but maintained a more flexed position throughout later periods of the response. Trunk pitch angle was similar in high and low conditions for the first 200 ms. In both conditions the trunk initially pitched backward into extension. However, in the high condition, trunk pitch angle was reversed after 200 ms and brought into a flexed position. In the roll direction, there was less lower leg roll in the high compared to low condition. Otherwise, little observable differences in roll angle were observed between height conditions for either the thigh, pelvis or trunk segments.

Protective Arm Responses

The normal response for compensatory arm pitch movements is to move the arms in the same direction as trunk movement. Therefore, for backward left perturbations, which cause forward pitch rotation of the trunk, the left arm pitched forward (shoulder flexion) (see figure 5). Shoulder flexion begins around 150 ms and reaches almost 3 deg flexion by 400 ms. The arms are then held in the flexed position for the remainder of the trial, likely to counterbalance the backward shift of the COM. Likewise for forward left perturbations, which cause the trunk to extend backward, the left arm pitched backward (shoulder extension) (see figure 5). Shoulder extension begins at 100 ms and reached peak angle by 200 ms, before being brought back to starting position at 500 ms. Note that left shoulder extension movements (for forward left

perturbation) are much faster and smaller (< 1 deg) compared to shoulder flexion movements (seen in backward left perturbations). Arm movements in the roll direction were found to precede arm pitch movements by almost 100 ms. However, the first adduction of the arms, between 20 and 200 ms, was likely attributed to a passive movement relative to the trunk. For left perturbations, the trunk rolls to the right in young adults, which will cause the left arm to fall into adduction relative to the trunk. After 200 ms this passive movement of the left arm was overcome by active abduction of the arms away from the body. For backward left perturbations the time to peak arm abduction was similar to that of peak arm flexion at 400 ms. Arm abduction was less for forward left perturbations, returning only to the pre-stimulus position by 500 ms.

Distinct differences in arm pitch and roll movements were observed when subjects stood in a high threat condition. For backward left perturbations, initial arm pitch angle was oppositely directed in the high compared to the low threat condition (figure 5). As described previously, in the low condition the arms were pitched immediately forward in the same direction as trunk flexion. However, in the high condition, the arms were brought backwards into extension for over 100 ms before changing direction and pitching forward similar to the low condition. For forward left perturbations, backward arm pitch angle is faster and reaches a larger amplitude in the high compared to the low height condition. Arm roll movements occurred earlier and reached larger peak abduction amplitudes in the high compared to low threat condition. Earlier and larger arm abduction in the high height condition was observed for both backward left and forward left perturbations.

Total Body Centre of Mass

Changes observed in joint and segment profiles appear to successfully control movements of the COM for both forward and backward directed perturbations. Range of total body COM displacement did not exceed 2 cm in either the A-P, M-L or vertical directions for any perturbation direction (figure 6). The COM was displaced in the same direction as the platform perturbation. As shown in figure 7b, the resultant vectors of A-P and M-L displacement of the COM lie along the directions of the platform rotation. Therefore, for backward left perturbations, the total body COM was displaced backwards and to the left. Forward left

perturbations caused the total body COM to be displaced forwards and to the left. COM was displaced upwards in backward left perturbations, and slightly downward for forward left perturbations (figures 6 and 7a).

In the high threat condition the total body COM had similar patterns of displacement compared to the low condition. For both backward left and forward left perturbations, the peak displacement of total body COM was reduced in both the A-P direction, and to a lesser extent in the M-L direction (figure 6 and 7a). Upward vertical displacement of total body COM was greater in the high standing condition for both backward and forward directed perturbations. In the A-P direction, peak displacement was reduced for all perturbation directions, except pure toes-up rotations (180 deg) which was larger. Differential changes in A-P peak COM displacement approached statistical significance (height by direction interaction $F(5,45)=2.04$, $p<0.0915$). Similarly, peak M-L displacement of total body COM was reduced in all directions which had a roll component; however these results were not statistically different (height $F(1,5)=0.02$, $p=0.8810$; height by direction $F(5,45)=1.06$, $p<0.3935$). COM displacement in the vertical direction was significantly different (height $F(1,9)=6.06$, $p<0.0360$) between high and low threat conditions, with higher peak vertical displacements observed in the high threat condition across all perturbation directions (mean difference = 0.67 cm) (refer to figure 7). Although, the magnitudes of peak COM displacement were altered due to increased postural threat, there was no change in the resultant directional vector for the displacement of the COM (figure 7b).

Leg, Hip and Trunk Muscle Responses

Backward left perturbations caused the ankle to initially dorsiflex (figure 3) which elicited an early stretch reflex in the soleus muscle at an average latency (and standard error) of 46.4 +/- 3.05 ms (figure 8). Extension of the knee joint may be related to a stretch reflex observed in biceps femoris (figure 8). As described above, backward perturbations caused the trunk to rotate forward, while the total body COM was displaced backward (figures 3, 6). These changes elicited large balance correcting responses in paraspinals (to control trunk flexion), rectus femoris (to maintain knee extension), and tibialis anterior (to provide dorsiflexing ankle

torque to counteract the backward displacment of COM). Large gluteus medius responses on the left side were required to maintain stability of the hip joint in the roll direction (figure 8).

In contrast, forward left perturbations initially caused the ankle to plantarflex and knees to flex (figure 4).These stimulus induced link movements elicited stretch reflexes in different muscles including tibialis anterior and rectus femoris with average (and standard error) latencies of 88.0 +/- 2.33 ms and 85.3 +/- 2.87ms respectively (figure 9). The total body COM displaced forward for forward left perturbations (figure 2,4), requiring a large balance correcting response to be generated by the soleus muscle. Distinct bursts of activity during the balance correcting response period were also recorded in other postural leg and trunk muscles. Left rectus femoris and biceps femoris muscles were co-activated to provide stiffness at the knee and hip joint. Similar to backward pertubations, gluteus medius activity acted to increase stability of the hip joint in the roll direction (figure 9).

In the high threat condition, there were no differences in the latency of the stretch reflexes or balance correcting responses for any muscle. The pattern and amplitude of initial stretch reflexes in soleus in backward left perturbations was similar in the high compared to low threat condition (mean and standard error for onset latency = 42.3 +/- 2.09ms). Likewise, for forward left conditions there were no significant differences in amplitude or onset of initial stretch reflexes in tibialis anterior (83.7 +/- 2.67 ms) or rectus femoris (81.6 +/- 2.91 ms). In contrast to normal stretch reflexes, differences between threat conditions were observed in subsequent balance correcting responses for all postural muscles. For example, the amplitude of the balance correcting responses were elevated for the high condition for soleus and tibialis anterior for forward left and backward left directions respectively (figure 10). For backward left perturbations, the largest amplitude changes were observed in the primary balance correcting muscles such as biceps femoris, paraspinals, rectus femoris and tibialis anterior (figure 8). For forward left perturbations there was excessive muscle activity in soleus, biceps femoris and paraspinal muscles, a muscle that was minimally activated in the low threat condition (figure 9). Distinct bursts of muscle activity were observed during the secondary balance correcting period (240-340 ms) in tibialis anterior muscles that were not present in the low condition (figure 9 and 10). Similar increases in secondary balance correcting activity was

observed in biceps femoris with larger amplitude than for low threat conditions. During the later period of time between 350-700 ms there were no observable differences in response amplitude or pattern of response with increased postural threat (figures 8 and 9).

Changes in Amplitude and Directional Sensitivity with Increased Postural Threat

Illustrating EMG areas on a polar plot, with mean amplitudes plotted along axes that correspond to different perturbation directions, allows for an easy visualization of the magnitude and directional sensitivity of different postural muscles. The polar plots in figures 11 and 12 depict the magnitude and directional sensitivity of EMG areas calculated over the balance correcting period for all muscles recorded on the left side of the body. Statistical comparisons revealed a significant influence of threat on amplitude of balance correcting responses across all directions for all muscles analyzed. As observed in figure 11, participants had larger responses between 120-220 ms in the high compared to low threat condition for tibialis anterior ($F(1,9)=13.59$, $p<0.0050$), soleus ($F(1,9)=5.06$, $p<0.051$) gluteus medius ($F(1,9)=24.07$, $p<0.0008$) and rectus femoris ($F(1,9)=5.43$, $p<0.0447$). All of these muscles demonstrated normal directional sensitivity, with response amplitudes for particular directions similar to those observed in the low condition. As shown in figure 12, significantly larger balance correcting responses were also observed for biceps femoris ($F=(1,9)=8.42$, $p<0.0176$), paraspinals ($F(1,9)=5.14$, $p<0.0495$) during the high threat condition. In addition to increased amplitude of balance correcting response, biceps femoris and paraspinals demonstrated a change in the directional sensitivity with increased postural threat. In the low condition, balance correcting activity in biceps femoris was largest for backward perturbations with least activity for the forward direction. However, in the high height condition, biceps femoris activity was much larger in all directions, with particularly large differences seen in the forward direction compared to low threat condition (figure 12). Paraspinal balance correcting activity was normally tuned to backward directions which initially unload the muscle. For example, left paraspinal was most active for pertubations backward to the right. Minimal activity is observed in the paraspinal muscle initially stretched by the perturbation i.e. backward left directions for left paraspinal. However, in the high threat condition, paraspinal activity became more symmetrical, with equal amplitude responses seen for both backward left and backward right perturbations in the left paraspinal muscle (figure 12). Evidence for more

symmetrical activation of paraspinals in the high threat condition was supported from assymetry ratios calculated between backward left and backward right perturbation directions for the left paraspinal muscle. In the low threat condition, the median assymetry ratio was 2.94 compared to a more symetrical ratio of 1.10 seen in the high threat condition, suggesting equal activation of this muscle for both roll directions.

Shoulder Muscle Responses

Backward left perturbations elicited distinct bursts of muscle activity in both left and right medial deltoid activities with onset latencies similar to that seen for balance correcting responses in more distal leg and trunk muscles (mean and standard error of onset = 103.7 +/- 5.87 ms for left deltoid). Balance correcting responses appeared to be symmetrical, with similar amplitudes observed between left and right deltoids for the same perturbation direction (figure 5). These responses were observed without the presence of any prior stretch or unloading responses, and return to resting levels of activation after 250 ms. Forward left perturbations also elicited distinct balance correcting activity in medial deltoids. Onset latency of 106.8 +/- 5.62 ms for left deltoid for forward left perturbations was similar to that for backward left pertubations; however the overall amplitude of the response was reduced in forward compared to backward perturbations (figure 5).

In the high threat condition, both the onset latency and amplitude of balance correcting activity in medial deltoids was signficantly different from responses seen in the low threat condition. ANOVA results revealed a significant effect of threat ($F(1,6)=9.77$, $p<0.0204$) for onset latencies in left deltoid muscle. Onset latencies in left medial deltoid were earlier (average 18.8 ms) in the high threat condition for all perturbation directions. For example for forward left perturbations, mean onset latencies (and st. error of mean) were 90.8 +/- 4.09 ms in the high threat compared to 106.8 +/- 5.62 ms in the low threat condition. Latencies were slightly earlier in the deltoid muscle that was contralateral to the side of the platform roll compared to the muscle on the same side. Balance correcting amplitudes were also larger during the high compared to low threat condition (figure 5). Differences appeared to be directionally dependent, with largest differences observed between threat conditions for forward directions

and in muscles contralateral to platform roll (deltoid right for forward and backward left perturbations).

As shown in polar plots in figure 12 (right panel), significantly larger balance correcting responses were present in deltoid muscles ($F(1,9)=4.85$, $p<0.0551$) during the high threat condition. Deltoid responses were relatively symmetrical in the low height condition, with equal amplitude responses for perturbations to the left and to the right. However, in the high standing height condition, deltoid balance correcting responses were proportionately larger for forward perturbation directions which rolled away from the side of the muscle. In other words, roll perturbations caused the trunk to rotate in the opposite direction to platform roll (figure 3 and 4). Therefore, under more threatening conditions, deltoid responses seem to be tuned more to perturbation directions that will cause the trunk to roll to the same side of the deltoid muscle.

Quiet Standing

When standing for one minute in the high height, differences were observed in the mean position, as well as amplitude and frequency characteristics of COM displacement compared to standing in the low threat condition. The mean position of COM during the one minute standing trial was shifted a mean distance of 0.28 cm forward in the high threat condition. MPF of COM displacement was increased on average by 0.015 Hz in the A-P direction and 0.023 Hz in the M-L condition in the high compared to low threat condition. Average RMS of COM displacement was decreased in the high threat condition by 0.081 cm in the A-P and 0.040 cm in the M-L direction. It should be noted that these trends did not reach statistical significance for any variable tested.

Perceived Anxiety and Balance Efficacy

Postural threat had a significant influence on participant's balance confidence, balance efficacy to specifically avoid a fall, perceived anxiety and perceived stability. Participants estimated their balance confidence to be signficantly lower ($F(1,9)=27.21$, $p<0.001$), and reported lower self efficacy to avoid a fall ($F(1,9)=13.52$, $p<0.01$) when standing in the high threat compared to the low threat condition. Following their performance on the moving the platform, the

participants reported experiencing significantly higher perceptions of anxiety ($F(1,9)=10.55$, $p<0.02$) in the high threat condition. In addition, participants felt significantly less stable when standing in the high threat compared to low threat condition ($F(1,9)=11.12$, $p<0.02$).

DISCUSSION

A perceived risk of injury, as a result of postural instability, may contribute to changes in postural control through alterations in central set (Brown and Frank, 1997; Adkin et al. 2000). Changes in central set, as a result of increased postural threat, have the potential to influence postural control in two different ways. First, postural threat may alter aspects of pre-stimulus posture, or preparatory postural adjustments that precede either a reactive or voluntary postural response. Alternatively, postural threat may modulate the reactive or voluntary component of the postural response directly. For example, Adkin et al. observed changes in the performance of a voluntary rise to toes task, as well as the preceding anticipatory postural adjustment in healthy subjects standing in a condition of increased postural threat. Brown and Frank (1997) also observed changes in pre-stimulus postural parameters, as well as changes in postural reaction to an unexpected perturbation when standing in a condition of increased postural threat. In the following discussion we will examine the results of increased postural threat on postural reactions to multi-directional perturbations with respect to these two possible mechanisms of central set.

Influence of Postural Threat on Pre-stimulus Posture

Previous studies have shown that increased postural threat can influence aspects of balance control which would precede any reactions to a balance perturbation. Participants have been shown to lean away from the direction of the perceived threat (i.e. edge of a high surface) and therefore, shift the mean position of the COM backward when standing in a high compared to low threat condition. Furthermore, increased background activity in tibialis anterior, and decreased activity in triceps surae muscles has been observed in participants standing in more threatening conditions (Brown and Frank, 1997, Carpenter et al. 2001a). Such changes are not unique to studies using environmental changes to alter postural threat. Maki and McIlroy (1996) observed forward leaning and increases in background activity of tibialis anterior in anxious subjects while standing and performing a secondary cognitive task. Maki and

Whitelaw (1993) also showed a tendency to lean forward with experience and prior information.

Changes in control of postural sway during quiet stance also has been observed when participants stood in conditions of increased postural threat. Decreased amplitude and increased frequency of centre of pressure (COP) oscillations have been observed in participants standing in high compared to low threat conditions (Carpenter et al. 1999a; Adkin et al. 2000). These changes were considered indicative of a stiffening strategy used to exert tighter control over the COM under threatening conditions. This hypothesis was later supported by findings showing that ankle stiffness significantly increases and amplitude of COM decreases under conditions of increased threat (Carpenter et al. 2001a). Elderly fallers with a fear of falling have been shown to have increased amplitude and velocity of COP displacement (Maki et al. 1994b). In patients with phobic postural vertigo, Krafczyk et al. (1999) reported higher frequency sway and presumably stiffer control compared to normals. Taken as a whole, the body of evidence does support a significant influence of postural threat and fear of falling on postural control parameters during quiet standing, which may potentially influence the normal response parameters of subsequent balance correcting responses. For example, increased background activity has been shown to affect amplitude of short and medium latency stretch responses and may also influence longer latency balance correcting responses (Bloem et al. 1993; Bedingham and Tatton 1984; Allum and Mauritz, 1984). Pre-leaning also has been shown to significantly increase the stretch reflex and decrease balance correcting responses to postural perturbations (Diener et al. 1983; Allum and Pfaltz 1985; Schieppati et al. 1995; Horak and Moore 1993) and thus may interact with subsequent postural reactions (Maki and McIlroy, 1996).

In the present study, the same trends for changes in postural sway characteristics were observed during quiet standing as reported previously. The mean RMS of COM displacement was reduced and mean power frequency was increased in the high versus low threat condition. These changes did not reach statistical significance, but the magnitude of change observed in the present study for RMS and MPF are similar to those reported previously (Carpenter et al. 1999a; Adkin et al. 2000), suggesting that this is a problem related to small sample size.

Despite the clear indications that postural threat influences postural control during quiet stance, we observed no significant changes in the postural control preceding the onset of postural perturbations. Background activity was not significantly increased in any muscle tested in the high versus low threat condition when measured 500 ms prior to the onset of perturbation. This implies that pre-stimulus changes in background activity in previous studies may have been secondary effects related to an unchecked leaning strategy. The lack of observable changes in background activity is likely explained by the stringent control of lower leg angle prior to the onset of each perturbation trial. Previous research has shown a significant correlation between leaning and background muscle activity (Sinha and Maki, 1996; Horak and Moore, 1993; Carpenter et al. 2001a). Negligible changes in background activity and pre-leaning were further confirmed by the equal amplitude stretch reflexes in both high and low threat conditions. No observable differences in amplitude or timing of the stretch reflexes in the high threat condition also argues against any change in alpha motor drive due to increased threat.

Alternatively, pre-stimulus changes seen in previous studies may be due to the predictable nature of the protocol. All previous studies on postural threat have used either static posture (Carpenter et al. 1999a;2001a; Adkin et al. 2000), or dynamic tasks in which the direction of the voluntary movement (Adkin et al. 2001a) or postural perturbation (Brown and Frank, 1997) was predictable and in the same direction as the postural threat. In these circumstances, the opportunity exists to use anticipatory postural adjustments in muscle tone or postural leaning to improve stability or performance in upcoming trials. In contrast, the present study employed unpredictable perturbations that were delivered in different directions relative to the position of the perceived postural threat. In such situations, using anticipatory changes in pre-stimulus posture may not be advantageous. For example, it may provide protection against perturbations in one direction but will have adverse effects for perturbations in other directions. Therefore, it may be hypothesized that in cases where balance perturbations are unpredictable, changes in set due to increased postural threat may more likely manifest themselves in automatic postural responses which can be appropriately tuned to the direction (Moore et al. 1988; Henry et al. 1998; Carpenter et al. 1999c), amplitude (Diener et al. 1984; 1991), and velocity of the perturbation (Allum and Pfaltz, 1985; Allum et al. 1993).

Influence of Postural Threat on Automatic Responses

Central set has been shown to exert significant influence on the amplitude of automatic postural reactions to unexpected perturbations, whether through prior knowledge (Maki and Whitelaw 1993; Diener et al. 1991) or expectation (Keshner et al. 1987, Sveistrup and Woollacott, 1997; Chong et al. 1999; Horak et al. 1989). In the present study postural threat was shown to have a significant influence on the magnitude of automatic postural responses in leg and trunk muscles. The overall gain of the balance correcting responses were increased in all muscles recorded when standing in the high threat compared to low threat condition. In tibialis anterior, soleus, gluteus medius and rectus femoris, the muscles maintained sensitivity to particular directions, suggesting that the responses were being altered online and modulated by increased postural threat. These results do not support a global activation pattern, or startle response, which would be activated independently for the direction of perturbation (McIlroy and Maki, 1994). In biceps femoris and paraspinals, the directional sensitivity was altered in the high threat condition. Biceps femoris had equally large responses for all directions, which created the largest difference in forward perturbations (directions which had minimal biceps femoris activity in the low condition). Paraspinals had a more symmetrical activation pattern for laterally directed perturbations for high versus low threat conditions. In the low condition, left paraspinals had a large balance correcting response for directions which initially unloaded the muscle (in this case, back to the right, which causes the trunk to fall forward and left). In contrast, backward left perturbations cause an initial stretch reflex in the left paraspinals which is followed by relatively low balance correcting activity (Carpenter et al. 1999c). When standing under high threat condition, the left paraspinal had equally large amplitudes for both backward left and backward right directions that would stretch or unload the muscle respectively. Presumably similar symmetrical activation for both backward left and backward right directions would be seen in the opposite paraspinal as well, which would lead to co-contraction between bilateral paraspinals for these directions. Increased co-contraction in bilateral paraspinals and increased activation of biceps femoris, and rectus femoris, will all contribute to greater trunk stiffness in the high threat condition.

Trunk stiffness in high threat condition was confirmed with the biomechanical analysis. During backward left perturbations, forward trunk angular displacement and hip angular displacement reached a lower peak amplitude with slightly earlier time to peak amplitude in the high compared to low threat condition. Increased stiffness has been previously reported clinically in patients with pathological fear related disorders. Tijssen et al. (1995) described a temporary generalized stiffness of the body in a subset of patients with excessive startle reflexes. Stiff-man syndrome, involves involuntary stiffness of the axial muscles induced by a startling or emotional stimuli (McEnvoy, 1991). Although these are extremely rare clinical examples, there existence suggests the possibility that far less dramatic changes in trunk stiffness may be achieved using similar pathways in normals responding to a postural threat.

Protective Arm Movements

Protective arm movements provide an important defense strategy against falls (Maki and McIlroy, 1997). Unlike leg and trunk muscles which had changes in amplitude but not timing or pattern of response, arm muscles responses were significantly earlier and larger in conditions of increased postural threat. Left deltoid activity had latencies that were, on average, 18.8 ms earlier in the high compared to low threat condition across all directions (i.e at 90.8 +/- 4.09 ms in the high threat compared to 106.8 +/- 5.62 ms in the low threat condition for forward left perturbations). McIlroy and Maki (1994) also reported earlier and larger activation of biceps brachii muscles in young controls during large amplitude compared to small amplitude perturbation. It is possible that the larger perturbations represent more threatening conditions to subjects and may cause earlier arm muscle responses similar to that found in the present study under conditions of increased threat. McIlroy and Maki (1995) argued that the responses were not startle responses but triggered automatic responses because they were scaled to both perturbation size and direction. Valls Sole et al. (1999) have reported that some voluntary movements, including arm movements, can have onsets as early as 90 ms when accompanied by an acoustic startle stimulus. It is unlikely that the earlier deltoid responses seen in the present study are consistent with the early voluntary movements triggered by a startling stimulus. Valls Sole et al. (1999) reported that the responses were shifted in time, with earlier onset and offset, whereas in the present study, the deltoid responses

have an earlier onset latency but appear to have the same offset latency (thus an extended response duration) in the high threat compared to the low threat condition.

An alternative explanation is that an earlier startle reflex may have been triggered at 80-90 ms, which was blended with the normally timed balance correcting response in the same muscle. Indeed, when the traces are inspected closely, there appears to be an initial peak in some traces prior to the normally timed balance correcting response which would suggest an earlier burst of activity. This is supported by the significantly larger EMG areas for left deltoid measured during both medium latency period (between 80-120 ms) and the later balance correcting period (between 120-220 ms). However, the presence of slightly earlier and larger amplitude responses in the left compared to right deltoid muscle for left directed perturbations would suggest that these responses are oriented to the direction of perturbation and not a simple startle reflex (McIlroy and Maki, 1994).

The third possibility is that the rapid early arm movements are a preprogrammed response used to exert a early protective mechanism to reduce movements of the COM in threatening conditions. Rapidly raising or lowering the arms has been shown to have significant secondary effects on joint moments at other lower leg and hip joints (Eng et al. 1992; Hodges et al. 2000; Ishac et al. 2001). Raising the arms generates reaction moments including an extensor moment at the hip, flexor moment at the knee and plantarflexor moment at the ankle. The net result of arm raising is a forward displacement of the total body COM (Friedli et al. 1988; Eng et al. 1992; Ishac et al. 2001). The stimulus induced changes in joint angles seen in the present study for a toe up rotation, consist of hip flexion, knee extension and ankle dorsiflexion, with a backward displacement of the COM (figure 2). Therefore, the forward pitch rotation of the arms (raising relative to the trunk), which is a feature of the response to toe-up rotations, will generate internal reaction moments at each joint which oppose initial stimulus induced moments to that caused by the perturbation. Likewise, lowering the arms (pulling toward the trunk) is followed by reactive flexor moment at the hip, extensor moment at the knee and dorsiflexion moment at the ankle (Eng et al. 1992; Ishac et al. 2001). For toes down perturbations, in which the arms are moved backward (toward the body), stimulus induced body movements included hip extension, knee flexion and ankle plantarflexion, and were

accompanied by a forward displacement of the COM (figure 2). Therefore, in this case the backward arm movements will also generate reaction moments which will counteract the stimulus induced joint moments caused by the perturbation. The early and larger arm activation seen in threatening conditions may be a pre-programmed response to generate earlier and larger stabilizing joint moments.

It is interesting that for backward perturbations arm responses are directed differently in the high threat condition, being pulled back instead of flexing forward as seen in the low threat condition. One possible explanation is that the arms are initially brought in to the trunk as part of an initial startle response. Alternatively, the initial backward displacement of the arms may represent a default strategy to protect against a possible fall toward the edge. In this case, the participants may first generate arm movements in anticipation of a forward perturbation (towards the direction of threat) and then adjust the direction of arm movements once the initial perturbation has been accounted for.

More detailed analyses of arm movement strategies and involvement of other shoulder, arm and back muscles is needed to further establish the role of compensatory arm responses in normal healthy and pathological populations.

Effects on Centre of Mass Displacement

The relatively small changes in COM displacement between the high and low threat conditions despite significant changes in segments angles and EMG amplitudes supports the notion that the COM represents a key variable controlled by the central nervous system. In the high threat condition, the A-P displacement of the COM was reduced in magnitude across all perturbation directions (significant only at the 0.10 level). This reduction in A-P COM displacement could have been achieved by a number of factors such as increased amplitude of muscle activity during balance correcting period, increased co-contraction of paraspinal muscles and larger arm movements causing inter-link changes in stimulus induced joint movements.

Observation of reduced peak amplitude of COM to postural perturbations in threatening conditions in the present study was similar to that found previously by Brown and Frank

(1997). However, there was no evidence for decreased time to peak COM velocity in the present study. It is possible that changes in pre-stimulus posture between threat conditions, such as increased forward leaning, increased background activity, and presumably ankle stiffness, contributed to the differences in time to peak COM velocity observed by Brown and Frank (1997). In contrast, the more stringent control over the prestimulus posture, and a more unpredictable nature of the perturbation directions used in the present study, may explain the lack of observable changes in either background activity, postural leaning, or stiffness that would have facilitated changes in COM velocity.

The most significant change in COM displacements with increased threat was seen in the vertical direction. For all directions, COM was displaced higher in the high threat compared to low threat condition. The greater upward movement of the COM potentially could be a strategy for converting the kinetic energy which moves the COM off balance to potential energy in order to decrease the COM horizontal velocity.

Less change in M-L displacement of the COM, may have been related to the constant location of the percieved threat along the pitch plane. Further research is required to determine whether postural reactions in the roll plane will be more greatly influenced by a postural threat located to the side or back of a subject.

Clinical Implications

The results of the present study have shown that increased postural threat in otherwise healthy adults can significantly alter the response characteristics of a postural reaction to a sudden perturbation. Unless accounted for, response characteristics related to an increased fear of falling in patients with balance deficits may otherwise be falsely attributed to the physiological disorder. Particularly, when studies are limited to a small number of muscle responses, or perturbation directions, the ability to discriminate between psychological and physiological manifestations of a balance disorder may become more difficult. For example, PD patients tested with a multi-directional protocol have been shown to have increased balance correcting response amplitudes, alterations in directional sensitivity of soleus and paraspinal muscles which leads to ankle and trunk stiffness, and earlier and larger amplitudes of deltoid muscle

responses (Carpenter et al. 2001b). Some of these responses have also been observed in the present study in the same muscles of healthy young individuals standing under conditions of increased postural threat. These similarities should emphasize the need, both in clinical and experimental conditions, to carefully consider the potential of fear of falling to influence certain aspects of postural behaviour in some patient groups.

Possible Confounding Influences

There are a number of factors that may have had a confounding influence on our results that should be considered. In the present study only a single constant location of postural threat was used (edge of high surface was always located in front of the participant). The pitch oriented nature of the postural threat may have restricted our ability to elicit changes in segment movements and COM displacements in only the pitch plane (particularly for forward perturbations, which moved the COM toward the edge). However, it should be noted that roll perturbations elicit trunk pitch displacements, as well as trunk roll displacements. This is not the case for pure pitch perturbations which elicit trunk pitch but not roll angular displacements (Carpenter et al. 1999c). Therefore, even roll perturbations in the present study would have generated postural instability in the trunk which was directed towards the direction of the postural threat. It is still possible that a different location of the perceived postural threat, (i.e. positioning the edge of the high surface to the side or behind the participant) may yield distinctly different postural strategies. Further studies into the influence of the location and different types of postural threat should be performed to further unravel this issue.

A second limitation to this study is that no direct physiological measure of fear or anxiety was recorded to confirm the threatening nature of our manipulation in postural threat. Previous studies using the same surface heights have shown significant changes in blood pressure and skin conductance in participants of similar age and characteristics to those participating in the present study (Carpenter et al. 1999b; Adkin et al. 2001a). Nonetheless, participants in the present study reported significantly lower balance confidence, decreased stabililty as well as increased perceived anxiety while standing under conditions of increased postural threat. Similar changes in balance confidence and percieved anxiety have been shown previously to

parallel changes in physiological measures in participants standing under identical postural threat conditions (Carpenter et al. 1999b; Adkin et al. 2001a).

A third limitation of the present study was the restriction in available recovery strategies to be used by the participants. By lightly strapping the feet to the support surface, we have restricted the participants ability to take a compensatory step, a strategy which has been shown to be an important protective response to unexpected perturbations (Maki and McIlroy, 1996). However, it was deemed necessary in the present study to restrict motion of the feet during perturbations for two different reasons 1) to ensure safety due to the small support surface upon which the subjects were standing 2) to maintain constant foot placement and body position between and within subjects. We acknowledge that by denying participant's ability to use a stepping responses we may have increased the likelihood to compensate with arm responses or other balance strategies. However, similar arm responses in young adults have been observed even when stepping responses were available (McIlroy and Maki, 1994)

Conclusions

Increased postural threat (and presumably fear of falling) has a significant impact on the normal postural reaction to unexpected perturbations to balance. Using multi-directional perturbations removes the subjects ability to predict the direction of perturbation and with control of leaning may eliminate alterations to pre-stimulus posture that may interact with subsequent balance correcting responses. In addition, multi-directional perturbations allows for insight into changes in directional sensitivity of muscle responses and segment movements to differently directed perturbations, which will contain an element of falling in the direction of the threat. Increased postural threat was associated with increased amplitude of muscle responses, and alterations of directional sensitivity in hip and trunk muscles which may contribute to increased trunk stiffness. Lack of changes in early stretch reflex amplitudes and background activity suggests these changes are not due to leaning or alterations in spinal neuronal drive. Arm movements provided an important protective strategy which were larger in threatening conditions. These findings may have important implications for using dynamic posturography to screen or discriminate between patients which may be prone to increased fear of falling.

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REFERENCES

Adkin AL, Frank JS, Carpenter MG, Peysar GW (2001a) Fear of falling modifies anticipatory postural control. Experimental Brain Research (In press)

Adkin AL, Frank JS, Carpenter MG, Peysar GW (2000) Postural control is scaled to level of postural threat. Gait Posture 12:87-93

Adkin AL, Frank JS, Jog MS (2001b) Fear of falling and postural control in Parkinson's disease. Movement Disorders (submitted)

Allum JHJ, Pfaltz CR (1985) Visual and vestibular contributions to pitch sway stabilization in the ankle muscles of normals and patients with bilateral peripheral vestibular deficits. Exp Brain Res 58: 82-94

Allum JHJ, Mauritz KH (1984) Compensation for intrinsic muscle stiffness by short-latency reflexes in human triceps surae muscles. J Neurophysiol 52:797-818

Allum JH, Honegger F, Schicks H (1993) Vestibular and proprioceptive modulation of postural synergies in normal subjects. J Vestib Res 3:59-85

Arfken CL, Lach HW, Birge SJ, Miller JP (1994) The prevalence and correlates of fear of falling in elderly persons living in the community. American Journal of Public Health 84:565-570

Bedingham W, Tatton WG (1984) Dependence of EMG responses evoked by imposed wrist displacements on pre-existing activity in the stretched muscles. Can J Neurol Sci 11: 272-280

Bloem BR, Grimbergen YAM, Cramer M, Willemsen MD, Zwinderman AH (2001) Prospective assessment of falls in Parkinson's disease. J Neurol (in press)

Brown LA, Frank JS (1997) Postural compensations to the potential consequences of instability: kinematics. Gait and Posture 6: 89-97

Carpenter MG, Allum JH, Honegger F (1999c) Directional sensitivity of stretch reflexes and balance corrections for normal subjects in the roll and pitch planes. Exp Brain Res129: 93-113

Carpenter MG, Allum JHJ, Honegger F, Adkin AL, Bloem BR. (2001b) Postural abnormalities to multidirectional stance perturbations in Parkinson's disease. Brain (submitted)

Carpenter MG, Allum JHJ, Honegger F. (2001c) Vestibular influences on human postural control in combinations of pitch and roll planes reveal differences in spatiotemporal processing. Experimental Brain Research (In press)

Carpenter MG, Frank JS, Brawley LR, Adkin AL. (1999b) Influence of threat on postural control in young and older adults. International Symposium on Gait Disorders: Book of Abstracts: 108-109

Carpenter MG, Frank JS, Silcher CP, Peysar GW. (2001a) The influence of postural threat on the control of upright stance. Exp Brain Res 138: 210-218

Carpenter MG, Frank JS, Silcher CP. (1999a) Surface height effects on postural control: a hypothesis for a stiffness strategy for stance. J Vestib Res 9: 277-286

Chong RKY, Horak FB, Woollacott MH (1999) Time-dependent influence of sensorimotor set on automatic responses in perturbed stance. Exp Brain Res 124: 513-519

Cumming RG, Salkeld G, Thomas M, Szonyi G. (2000) Prospective study of the impact of fear of falling on activities of daily living, SF-36 scores, and nursing home admission. J Gerontol A Biol Sci Med Sci 55: M299-305

Cummings SR, Nevitt MC. (1994) Non-skeletal determinants of fractures: The potential importance of the mechanics of falls. Osteoporosis International 1: S67-70

Diener HC, Bootz F, Dichgans J, Bruzek W (1983) Variability of postural "reflexes" in humans. Exp Brain Res 52: 423-428

Diener HC, Dichgans J, Bootz F, Bacher M (1984) Early stabilization of human posture after a sudden disturbance: influence of rate and amplitude of displacement. Exp Brain Res 56:126-134

Diener HC, Horak F, Stelmach G, Guschlbauer B, Dichgans J (1991) Direction and amplitude precuing has no effect on automatic posture responses. Exp Brain Res 84:219-223

Downton JH, Andrews K. (1990) Postural disturbance and psychological symptoms amongst elderly people living at home. International Journal of Geriatric Psychiatry 5: 93-98

Eng JJ, Winter DA, MacKinnon CD, Patla AE (1992) Interaction of reaction moments and centre of mass displacement for postural control during voluntary arm movements. Neursci Res Commun 11: 73-80

Friedli WG, Cohen L, Hallett M, Stanhope S, Simon SR (1988) Postural adjustments associated with rapid voluntary arm movements. II. Biomechanical analysis. J Neurol Neurosurg Psychiatry 51: 232-243

Henry SM, Fung J, Horak FB (1998) EMG responses to maintain stance during multidirection surface translations. J Neurophysiol 80:1939-1950

Hill KD, Schwarz JA, Kalogeropoulos AJ, Gibson SJ (1996) Fear of falling revisited. Arch Phys Med Rehabil 77: 1025-1029

Hodges PW, Cresswell AG, Daggfeldt K, Thorstensson A (2000) Three dimensional preparatory trunk motion precedes asymmetrical upper limb movement. Gait Posture 11: 92-101

Holliday PJ, Fernie GR, Gryfe CI, Griggs GT (1990) "Video recording of spontaneous falls of the elderly," slips, stumbles, and falls: Pedestrian footwear and surfaces, ASTM STP 1103, B.E. Gray, Ed., American Society for Testing and Materials, B.E. Gray (Ed.) Philadelphia: 7-16

Horak FB, Diener HC, Nashner LM (1989) Influence of central set on human postural responses. Journal of Neurophysiology 62: 841-852

Horak FB, Moore SP (1993) The effect of prior leaning on human postural responses. Gait Posture 1:203-210

Ishac MG, Patla AE, Winter DA (2001) The effect of postural constraint on anticipatory control during voluntary arm movement. In: Duysens J, Smits-Engelsman BCM, Kingma H, editors. Control of Posture and Gait. Maastricht : Symposium of the International Society for Postural and Gait Research: 365

Keshner EA, Allum JH, Pfaltz CR (1987) Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit. Exp Brain Res 69: 77-92

Krafczyk S, Schlamp V, Dieterich M, Haberhauer P, Brandt T (1999) Increased body sway at 3.5-8 Hz in patients with phobic postural vertigo. Neurosci Lett 259: 149-152

Lepicard EM, Venault P, Perez-Diaz F, Joubert C, Berthoz A, Chapouthier G (2000) Balance control and posture differences in the anxious BALB/cByJ mice compared to the non anxious C57BL/6J mice. Behav Brain Res 117:185-195

Maki BE, Holliday PJ, Topper AK (1994b) A prospective study of postural balance and risk of falling in an ambulatory and independent elderly population. Journal of Gerontology: Medical Sciences 49: M72-M84

Maki BE, McIlroy WE. (1997) The role of limb movements in maintaining upright stance: the 'Change in Support' strategy. Phys Ther 77: 488-507

Maki BE, McIlroy WE, Perry SD (1994a) Compensatory responses to multi-directional perturbations. In: Taguchi K, Igarashi M, Mori S (eds) Vestibular and Neural Front, Elsevier Science, Amsterdam, pp 437-440

Maki BE, McIlroy WE (1996) Influence of arousal and attention on the control of postural sway. Journal of Vestibular Research 6:53-59

Maki BE, McIlroy WE (1998) Control of compensatory stepping reactions: Age related impairment and the potential for remedial intervention. Physiotherapy Theory and Practice 15: 69-90

McAuley E, Mihalko SL (1998) Measuring exercise related self efficacy. In: Duda JL (ed.). Advances in Sport and Exercise Psychology Measurement: 371-390.

McEvoy KM. Stiff-man syndrome. (1991) Mayo Clin Proc 66: 300-304

McIlroy WE, Maki BE (1995) Early activation of arm muscles follows external perturbation of upright stance. Neurosci Lett 184: 177-80

Moore SP, Rushmer DS, Windus SL, Nashner LM (1988) Human automatic postural responses: responses to horizontal perturbations of stance in multiple directions. Exp Brain Res 73:648-658

Myers AM, Powell LE, Maki BE, Holliday PJ, Brawley LR, Sherk W (1996) Psychological indicators of balance confidence: relationship to actual and perceived abilities. Journal of Gerontology 51A: M37-M43

Niino N, Tsuzuku S, Anko F, Shimokata H. (2000) Frequencies and circumstances of falls in the National Instititute for Longevity Sciences, Longitudinal Study of Aging (NILS-LSA). Journal of Epidemiology 10:S90-S94

Schieppati M, Nardone A, Siliotto R, Grasso M (1995) Early and late stretch responses of human foot muscles induced by perturbation of stance. Exp Brain Res 105: 411-422

Sinha T, Maki BE (1996) Effect of forward lean on postural ankle dynamics. IEEE Trans Rehabil Eng 4:348-359

Smith RE, Smoll FL, Schutz RW (1990) Measurement and correlates of sports specific cognitive and somatic trait anxiety: The Sport Anxiety Scale. Anxiety Research 2: 263-280.

Tijssen MA, van Dijk JG, Roos RA, Padberg GW (1995) Startle disease: growing rigid with fear. Ned Tijdschr Geneeskd 139:1940-1943

Sveistrup H, Woollacott MH (1997) Practice modifies the developing automatic postural response. Exp Brain Res 114: 33-43

Tinetti ME, Mendes de Leon CF, Doucette JT, Baker DI (1994) Fear of falling and fall-related efficacy in relationship to functioning among community-living elders. Journal of Gerontology 49: M140-7

Valls-Sole J, Rothwell JC, Goulart F, Cossu G, Munoz E (1999) Patterned ballistic movements triggered by a startle in healthy humans. J Physiol 516: 931-938

Vellas BJ, Wayne SJ, Romero LJ, Baumgartner RN, Garry PJ (1997) Fear of falling and restriction of mobility in elderly fallers. Age Ageing 26:189-93

Walker JE, Howland J (1991) Falls and fear of falling among elderly persons living in the community: occupational therapy interventions. Am J Occup Ther 45:119-22

Winter DA, Patla AE. (1997) Signal Processing and Linear Systems for the Movement Sciences. Waterloo: Waterloo Biomechanics

Yardley L, Hallam RS (1996) Psychosocial aspects of balance and gait disorders. In: Bronstein AM, Brandt TH, Woollacott M, ed. Clinical Disorders of Balance Posture and Gait. New York: Oxford University Press: 251-267

Figure 1 Illustration of apparatus used to deliver multi-directional postural perturbations under different levels of postural threat achieved by adjusting surface height. The present photograph depicts the high threat condition (surface height 160 cm above ground).

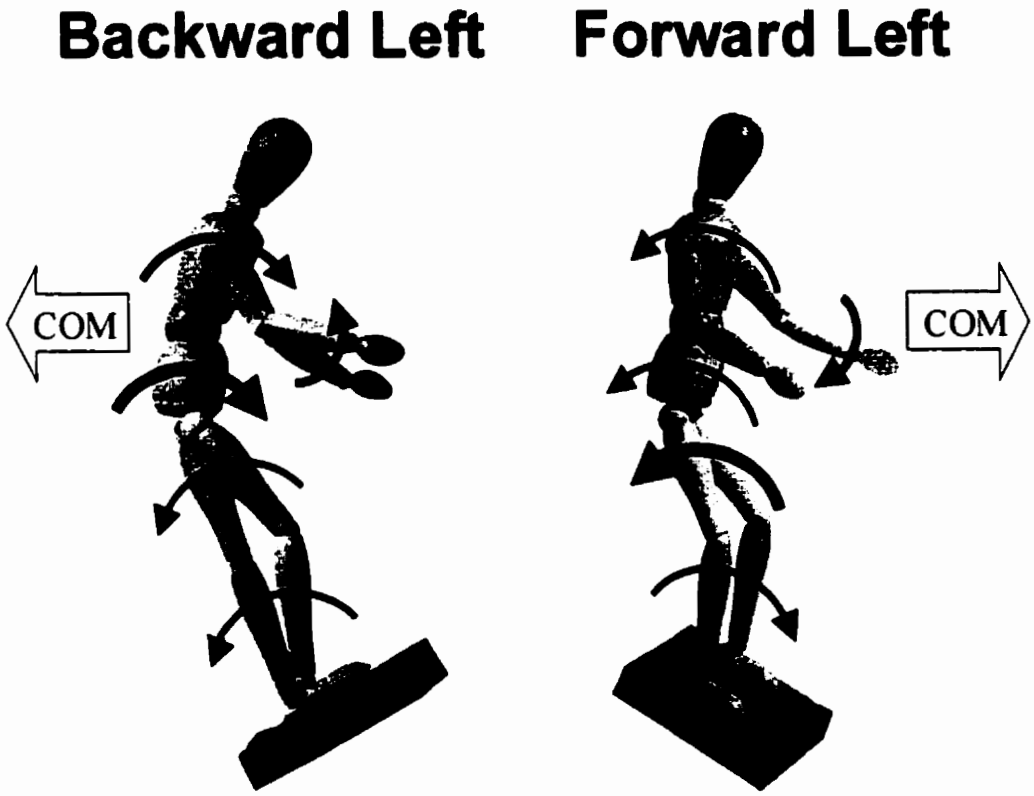

Figure 2 Illustration of initial body segment movements induced by platform perturbations directed backward to the left (225 deg) and forward to the left (315 deg). Arrows represent the direction of absolute initial stimulus induced segment rotations of the lower leg, thigh, pelvis, upper trunk and upper arm segments.

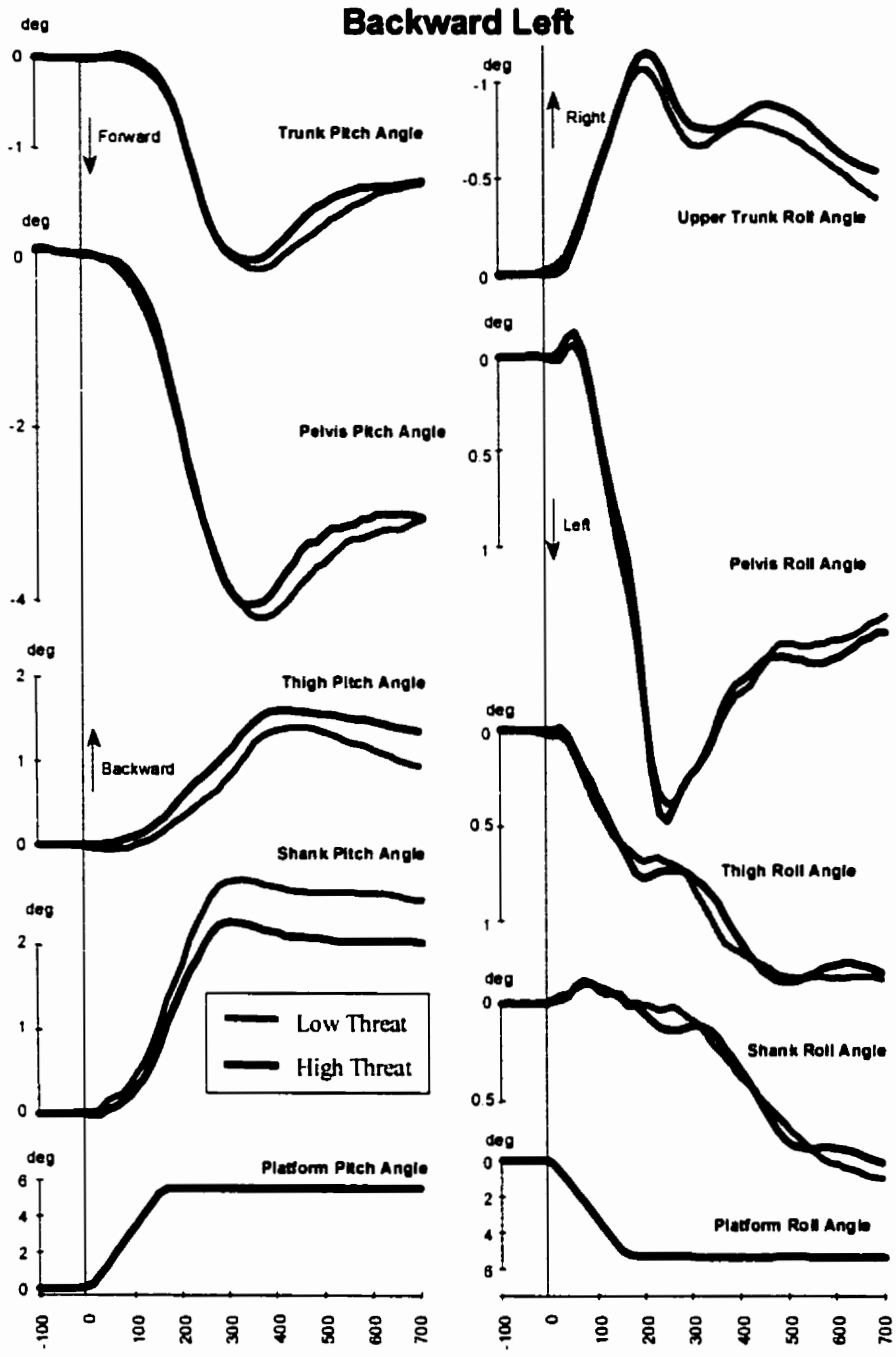

Figure 3 Segment angular displacements to backward left (225 deg) perturbations (7.5 deg at 50 deg/s). Traces on left panel represent angular displacements in the pitch plane, whereas traces on right are in the roll plane. Each of the traces shows the average for 10 participants to 6 randomized repititions of the stimulus direction. The thick black vertical line at 0 ms represents the onset of support surface rotation. A positive deflection of the traces represents absolute backward angular displacement, while a negative deflection represents absolute forward angular displacement.

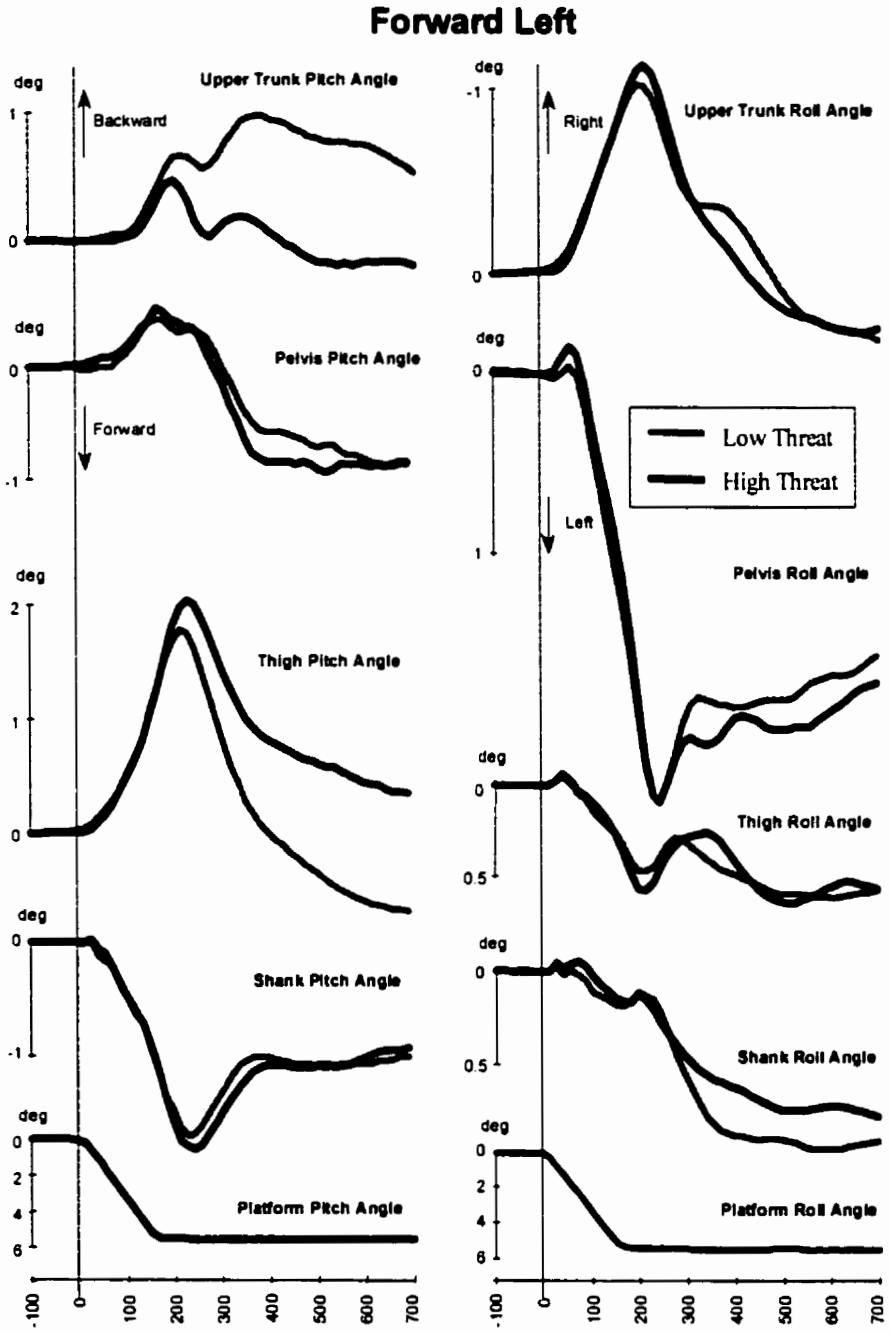

Figure 4 Segment angular displacements to forward left (225 deg) perturbations (7.5 deg at 50 deg/s). Details of the responses have been provided in the legend of figure 3.

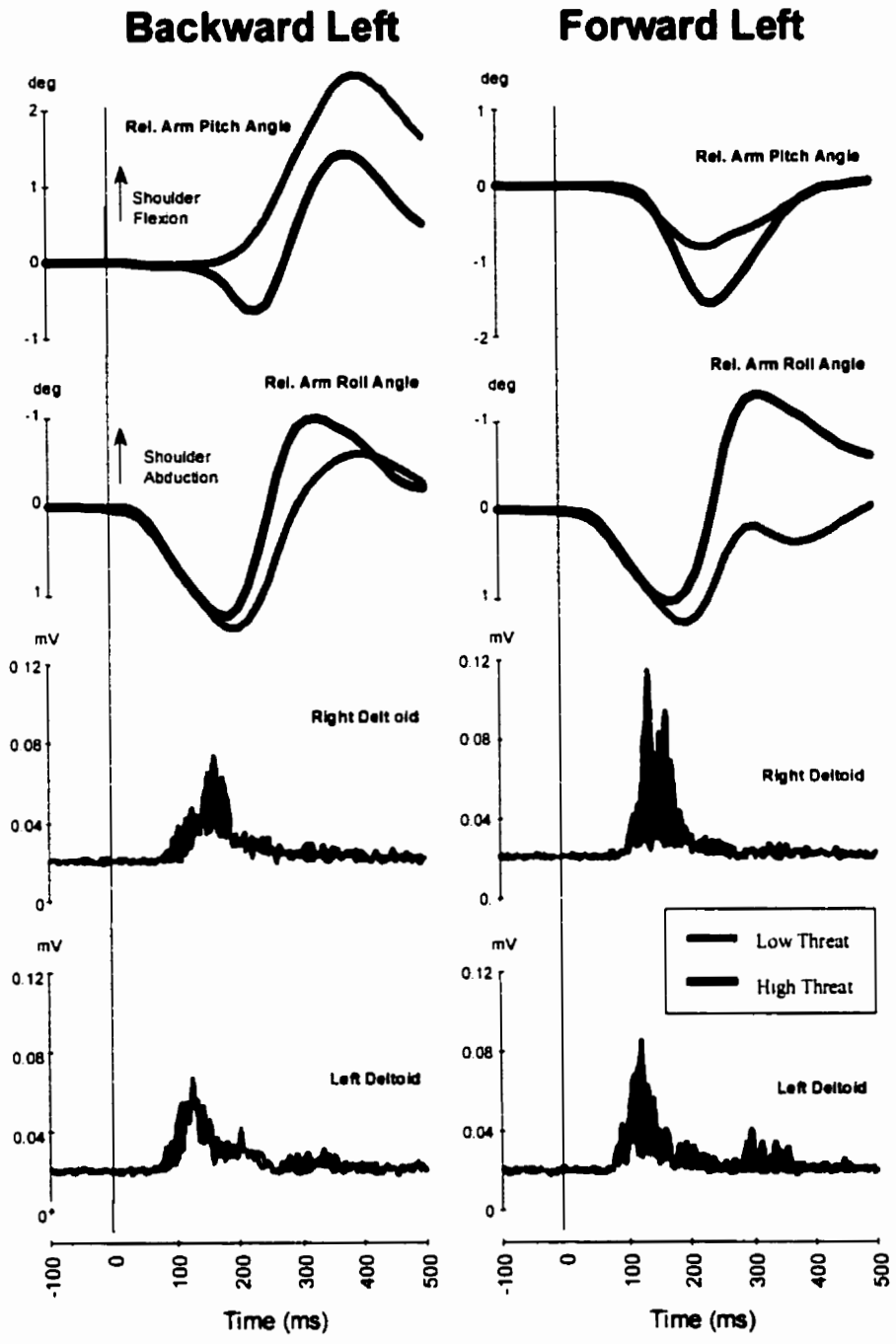

Figure 5 Arm pitch and roll angular displacements relative to the trunk are shown in the upper panel for backward left (left panel) and forward left (right panel) perturbations. A positive deflection of the traces represents either shoulder flexion (pitch) and shoulder abduction (roll) relative to the trunk. A negative deflection of the traces represents either shoulder extension (pitch) or shoulder adduction (roll) relative to the trunk. Bottom panel contains average muscle responses in right and left medial deltoid muscles. Black shaded area represents periods that activity is greater in the high threat compared to low threat condition. Details of the responses have been provided in the legend of figure 3.

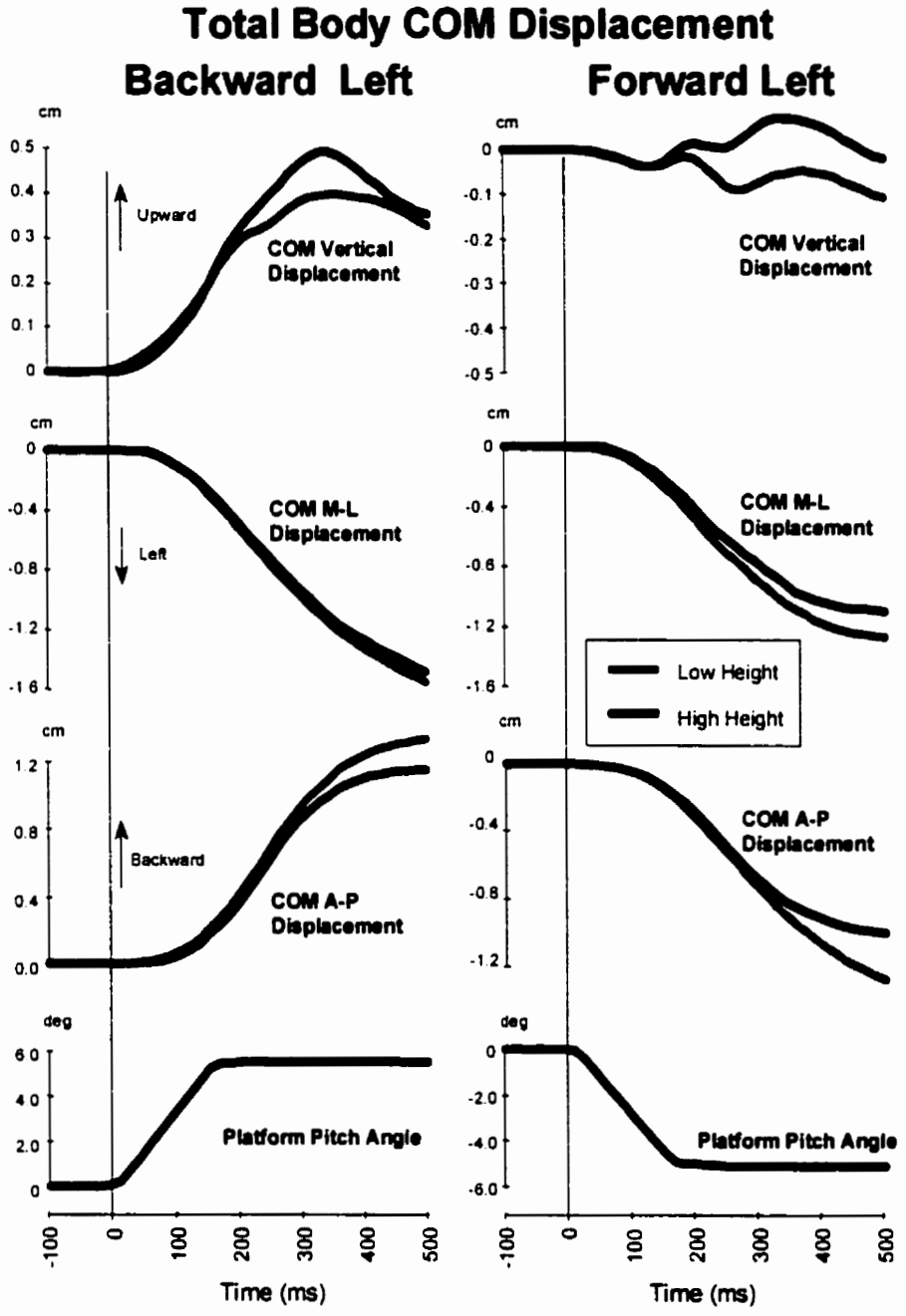

Figure 6 Average linear displacement for the total body COM for backward left (225 deg) and forward left (315 deg) perturbations. Details of the responses have been provided in the legend of figure 3.

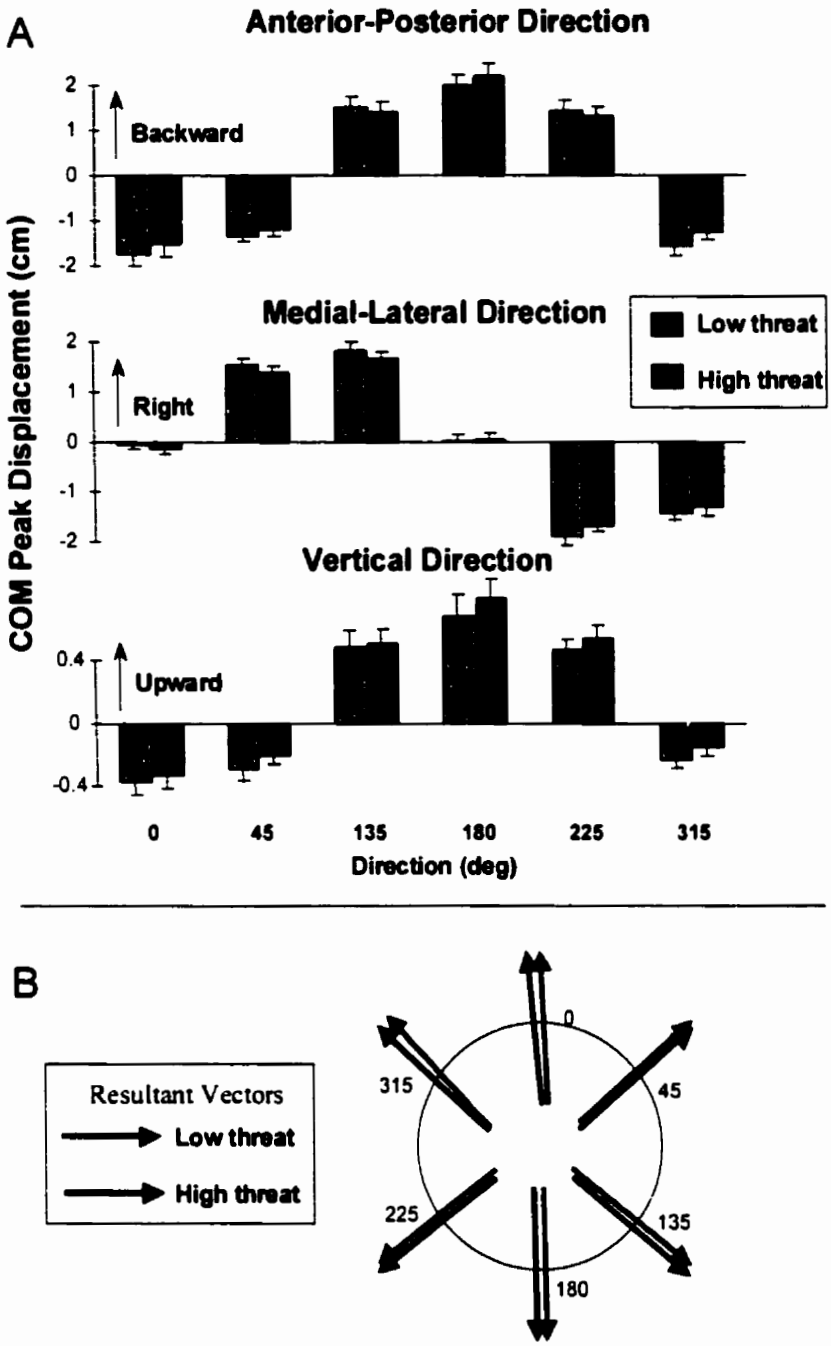

Figure 7 a) Mean and standard error values for peak displacement of COM in the A-P (upper panel), M-L (middle panel) and vertical (lower panel) planes for different perturbation directions. b) Resultant vectors calculated from peak A-P and M-L COM displacements are shown as directional arrows for each perturbation direction.

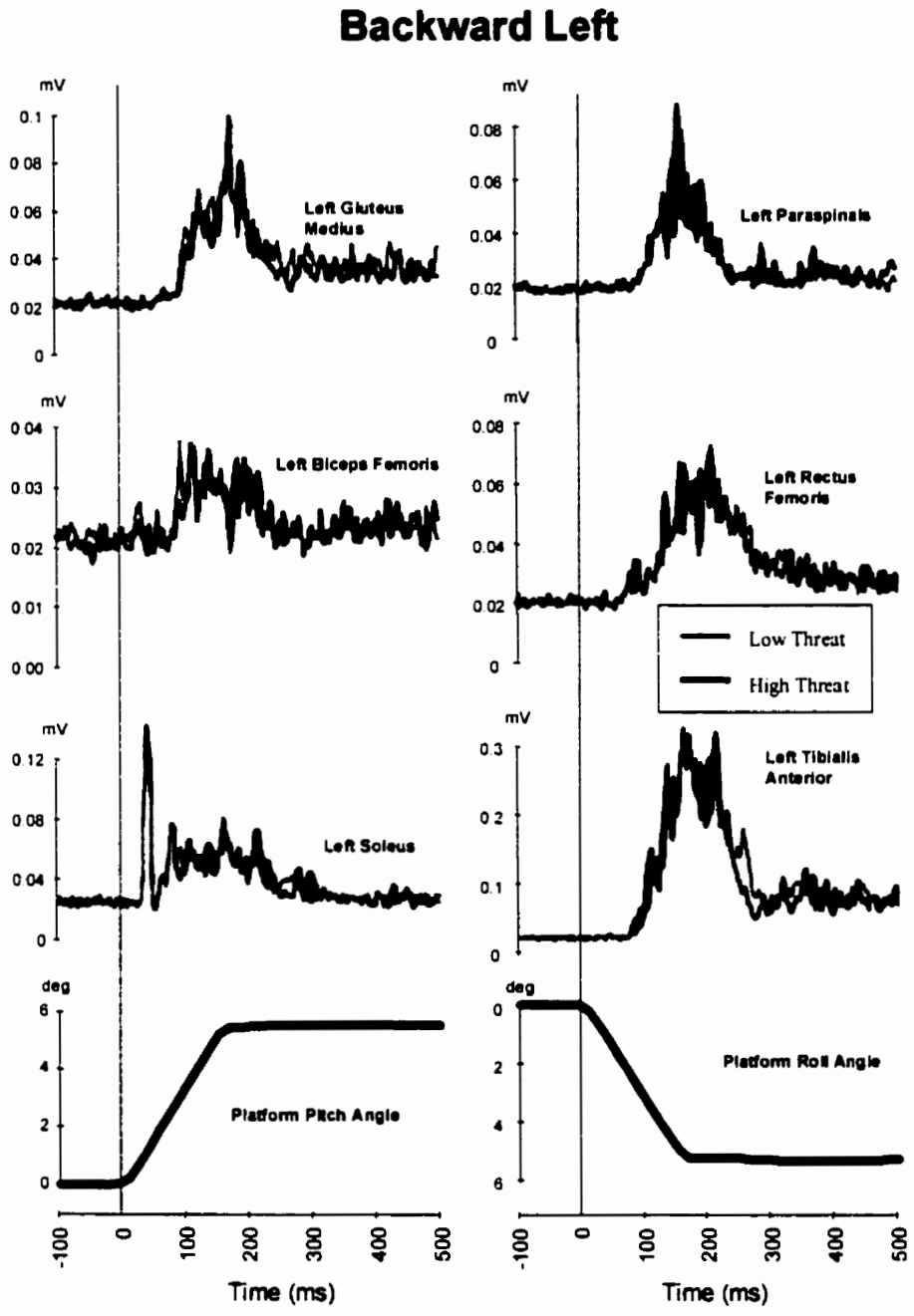

Figure 8 Mean muscle responses for left sided postural leg, hip and trunk muscles for backward left (225 deg) perturbations. Black shaded area represents periods that activity is greater in the high threat compared to low threat condition. Details of the responses have been provided in the legend of figure 3.

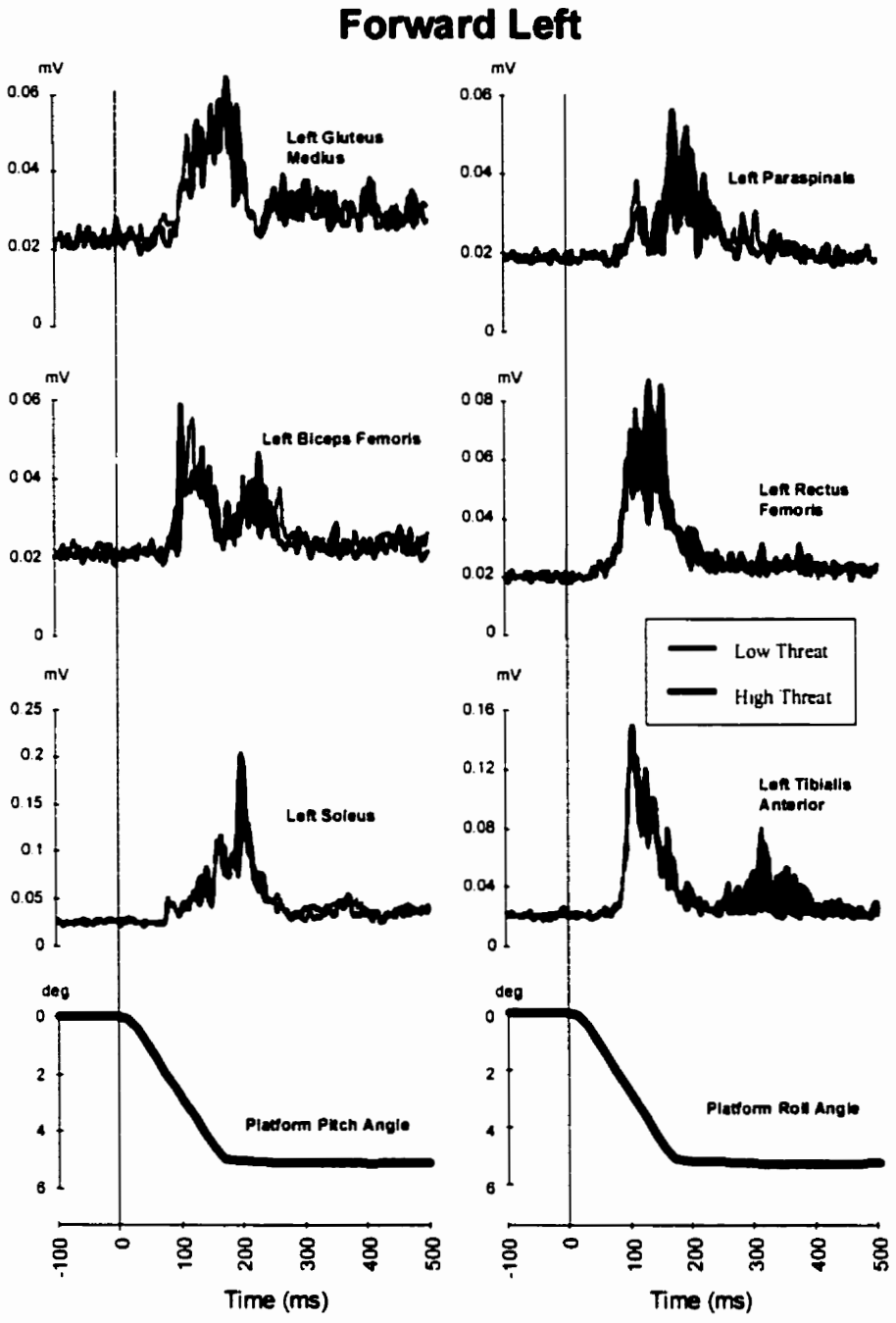

Figure 9 Mean muscle responses for left sided postural leg, hip and trunk muscles for forward left (315 deg) perturbations. Black shaded area represents periods that activity is greater in the high threat compared to low threat condition. Details of the responses have been provided in the legend of figure 3.

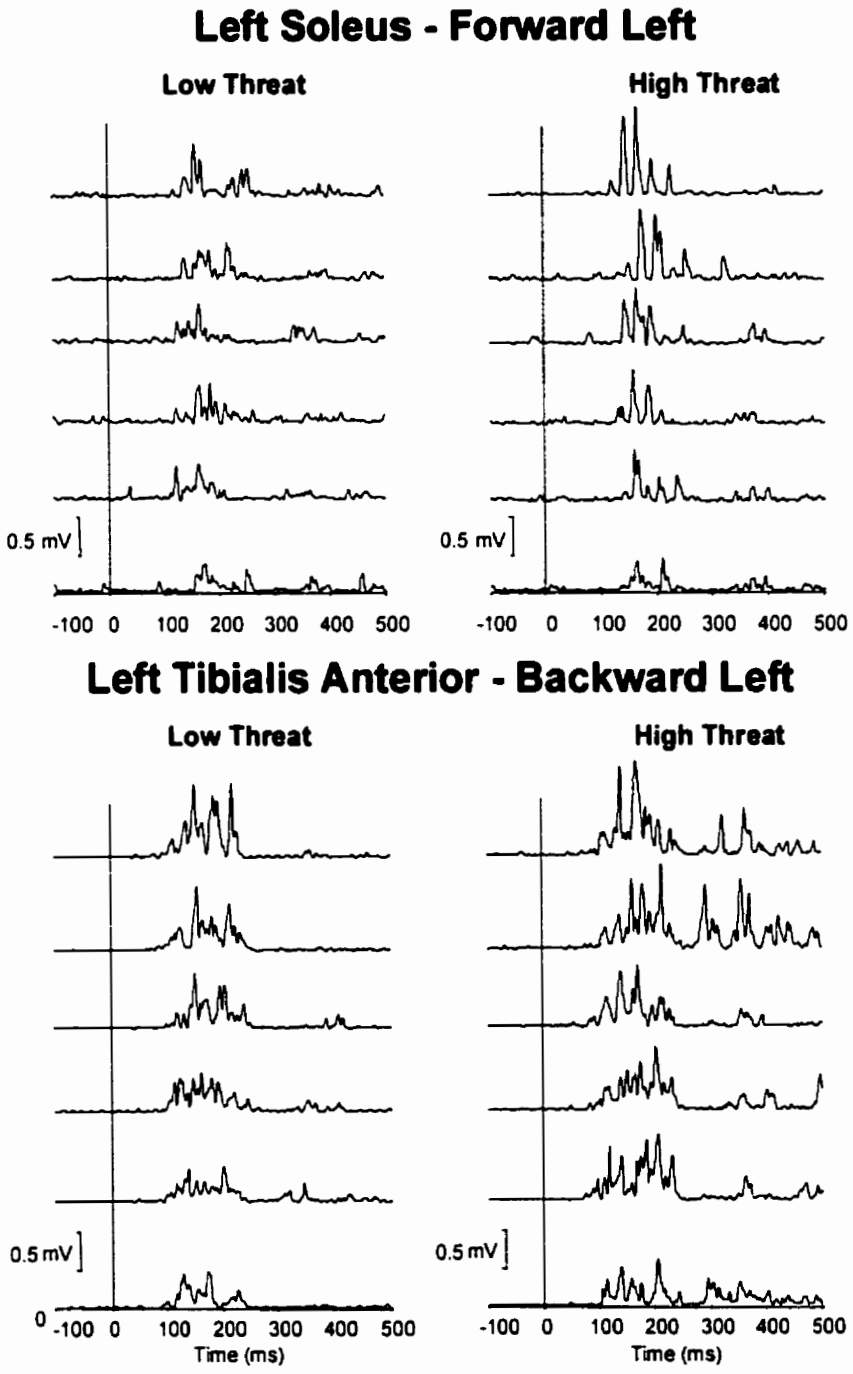

Figure 10 Profiles demonstrating the balance correcting responses from left soleus and left tibialis muscles measured from a single representative participant during both low threat and high threat conditions.

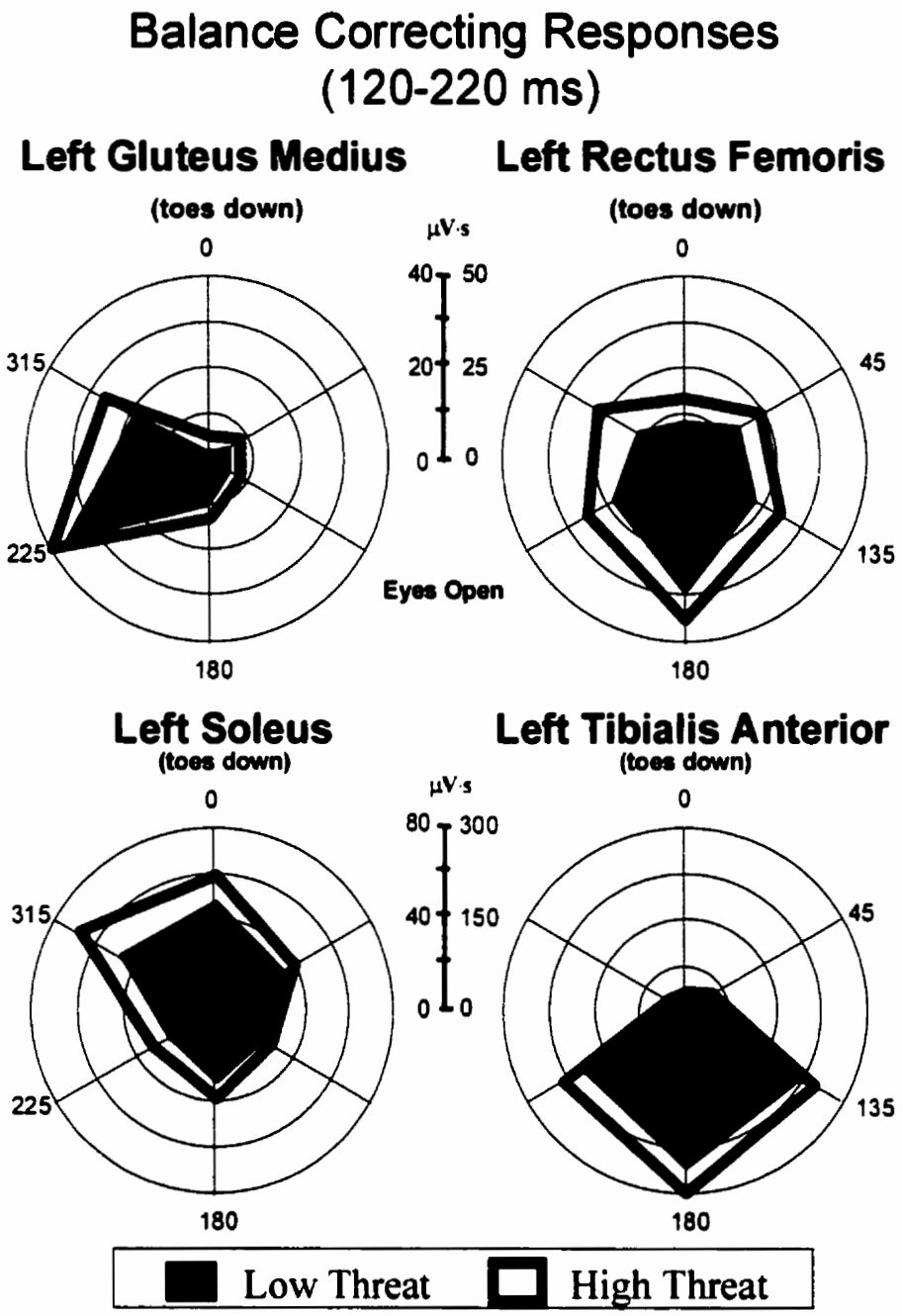

Figure 11 Polar plots for average EMG areas for balance correcting response period (120-220 ms) for left sided soleus, tibialis anterio, gluteus medius and rectus femoris muscles. Thick black lines represents high postural threat condition; grey filled area represent low postural threat condition. Each radial line represents one of six different directions (0, 45, 135, 180, 225, 315 deg) in clockwise notation. For each direction mean values are plotted along each radial axis with magnitude represented by the distance to the centre.

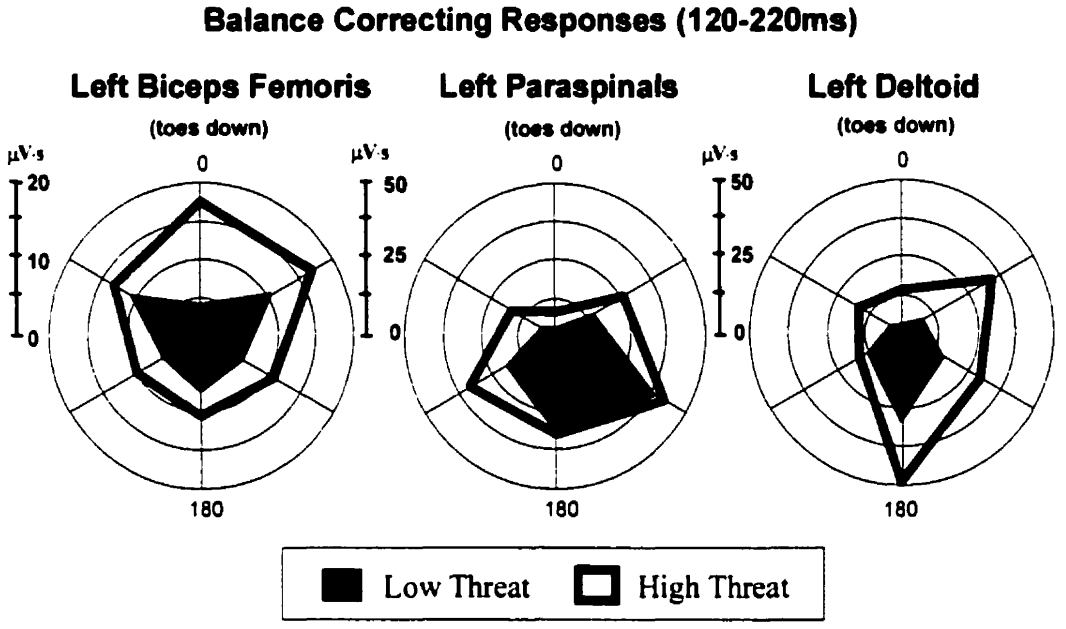

Figure 12 Polar plots for average EMG areas for balance correcting response period (120-220 ms) for left sided biceps femoris, paraspinals and deltoid muscles. Thick black lines represents high postural threat condition; grey filled area represent low postural threat condition. Details of the responses have been provided in the legend of figure 11.

CHAPTER 6

CONCLUSION

The focus of the present thesis was to examine normal, pathological and psychological factors that influence postural reactions from two new perspectives. The first goal was to determine what previously known aspects of postural reactions, established using pitch plane perturbations, can be extended to perturbations in multiple directions, which may more accurately mimic events experienced in everyday life. The second goal was to determine what new information can be extracted from multi-directional perturbations that are not available when using only pitch plane perturbations. In the following chapter, these two goals will be addressed with respect to three different aspects of postural reactions 1) possible triggering mechanisms 2) modulation of triggered postural responses and 3) trunk control.

Triggering Mechanisms

The origin of the primary triggering mechanism responsible for detecting and initiating the cascade of muscular responses required to recover from an unexpected perturbation is a contentious, and to date, unresolved issue in postural control literature. One view supports the theory of a distal to proximal activation of postural muscles, which is triggered primarily from proprioceptive input from the ankle joint (Nashner et al. 1982, Horak and Nashner, 1986). This theory has been established from observations of an initial early activation of triceps surae muscles, followed chronologically by activation in upper leg and trunk muscles. Others have reported observations of early muscle activity in more proximal muscles, such as deltoids (McIlroy and Maki, 1995) and neck muscles (Keshner et al. 1988), which are simultaneous or prior to activity observed in triceps surae muscles. These observations do not support a distal to proximal activation pattern that is triggered by early ankle proprioceptive input. Instead, they have provided evidence for a second theory which suggests that the primary trigger for postural reactions originates in various proprioceptive receptors in proximal sites such as the knees, hips, and trunk (Forsberg and Hirschfeld, 1994; Allum et al. 1993, Bloem et al. 2000; Horstmann and Dietz, 1990; Do et al. 1988; Di Fabio, 1995).

Using single directional perturbations may not provide a complete picture of the mechanisms involved in triggering postural reactions. For example, certain joints may not be displaced by perturbations within a particular direction, but may be greatly influenced by other types or direction of perturbation. For example, the knee joint remains locked into extension for pure toe-up perturbations, but has large flexion movements to both toes down and roll perturbations (Carpenter et al. 1999). Similarly, certain muscles may provide important triggering information which are more sensitive to lateral displacement of the joints through combinations of stretch and unloading of bilateral muscles. Therefore, by using multi-directional perturbations, significant new information has come to light which may reshape present hypotheses concerning the primary triggering mechanisms for postural reactions. For example, off-pitch perturbations have revealed early stretch and unloading reflexes in paraspinals (Carpenter et al. 1999; 2001a) and gluteus medius muscles (Carpenter et al. 2001b;2002), which precede stretch responses in triceps surae and tibialis anterior muscles. This finding suggests that directionally sensitive triggering information is, in the very least, available for the CNS to trigger a postural response with the same, if not earlier, latencies compared to that from ankle proprioceptive inputs.

The presence of very early head accelerations (20 ms after perturbation onset), with directionally sensitive changes in vertical and rotational accelerations suggests that vestibular inputs could also provide the triggering signal (Carpenter et al. 1999). However, the normal onset latency for balance correcting responses in leg and trunk muscles was preserved in cases of bilateral (Carpenter et al. 2001a) and acute unilateral (Carpenter et al. 1999b) vestibular loss. The exception to this finding was an increased latency in soleus muscles for toes down perturbations, which supports, at least, a partial contribution of the vestibular system to triggering postural reactions in directions in which head accelerations indicate a downward fall (Carpenter et al. 2001a).

Research using a patient with total leg proprioceptive loss due to a dorsal root ganglionopathy has provided further convincing evidence for a directionally sensitive triggering mechanism which presides above the ankle joint. The patient had absent proprioception in both the ankle and knee joint, with severe impairment but not total loss of proprioception at the level of the

hip and trunk in addition to impaired cutaneous sensation in the feet and lower legs. In controls and patients with selective loss of triceps surae reflexes due to lower leg diabetic neuropathy, onset latencies for balance correcting responses were normal, however few automatic postural responses appeared to be triggered or modulated by lower leg proprioception (Bloem et al. 2000). In contrast, the patient with total leg proprioceptive loss had significant delays in the onset of balance correcting responses in both soleus and tibialis anterior to perturbations in both pure pitch plane perturbations and perturbations in both the pitch and roll planes (Bloem et al. 2001). Surprisingly, balance correcting responses in upper leg, hip and trunk muscles were not delayed in onset, suggesting that lower leg postural responses must be primarily triggered by proprioceptive input at the level of the knee or higher.

The multi-directional perturbation paradigm has provided evidence for a primary trigger signal which originates in either the knee or hip joint, but is augmented by ascending proprioceptive information from the ankle joint and descending vestibular information (Allum, personal communication). This new hypothesis calls for more extensive research to examine the role of other trunk and hip muscles to provide relevant triggering information and subsequent balance correcting activity for multi-directional perturbations to posture.

Modulation of Automatic Postural Responses

Previous studies using pitch plane perturbations have shown that amplitude of automatic postural responses can be modified by cutaneous information (Perry et al. 2001), stretch related (Bloem et al. 2000; Inglis and Horak, 1994) and load dependent (Horstmann and Dietz, 1990) proprioceptive information, vestibular information (Allum and Honegger, 1998; Runge et al. 1998; Horak et al. 1990; Allum et al. 1994; Nashner et al. 1982), as well from higher centres such as the basal ganglia (Allum et al.1988; Dietz et al. 1988; Schieppati and Nardone 1991; Beckley et al.1993) and cerebellum (Timmann and Horak, 1997).

An issue that has not been well addressed is how the central nervous system (CNS) and different sensory systems contribute to the appropriate modulation of postural responses for different perturbation directions. Rushmer and Moore (1983) argued that postural perturbations were modulated to direction of perturbation upon observations of altered response profiles

within a single trial of unexpectedly reversing perturbation direction from backwards to forwards. Recently, these observations have been extended to include unpredictable changes in direction in both the pitch and roll plane. These studies have revealed muscle specific ranges of activation and directional sensitivity of response amplitudes of both early stretch reflexes and subsequent balance correcting responses (Carpenter et al. 1999; Henry et al. 1998; Moore et al. 1988; Maki et al. 1994).

More recent work has begun to examine how postural deficits due to vestibular loss (Carpenter et al. 2001a), proprioceptive loss (Bloem et al. 2001) and Parkinson's disease (PD) (Carpenter et al. 2001b) may influence the normal activation pattern and directional sensitivity of postural response amplitudes to multi-directional perturbations. For example, vestibular loss has been characterized by a decreased modulation of balance correcting response (120-220 ms) amplitude in tibialis anterior and soleus muscles, followed by excessive activation of these muscles and paraspinals during the later stabilizing period (between 350-700 ms). In contrast, patients with Parkinson's disease have extra responses during the so called 'medium latency' period (80-120 ms) in distal and proximal muscles followed by excessive balance correcting responses in both leg, hip and trunk muscles. In total, this represents an overactive response pattern. In both cases, despite opposite changes in amplitude modulation, the directional sensitivity of the balance correcting responses in patients with vestibular loss and Parkinson's disease are generally well preserved compared to normals. The exception is in soleus and paraspinal muscles in PD patients which have greater activation in pitch and roll directions respectively leading to co-contraction with normally directed responses in antagonist muscles. Total leg proprioceptive loss also had relatively normal directional sensitivity of balance correcting responses in distal muscles, but had more prominent shift of activity to roll directed perturbations in hip and trunk muscles. These findings in general would suggest that the original directional sensitivity of the balance correcting response in most leg, hip and trunk muscles is coded in the original primary proximal triggering signal and later modulated by lower leg proprioceptive inputs, vestibular inputs and higher centres including the basal ganglia. Recent evidence has also shown that directionally sensitive postural responses can also be modulated by changes in central set such as prior knowledge (Horak et al. 1989; Maki and Whitelaw 1993; Diener et al. 1991), experience (Keshner et al. 1987; Sveistrup and

Woollacott, 1997; Chong et al. 1999) and other factors such as a perceived increase in postural threat (Carpenter et al. 2001c; Brown and Frank, 1997). Evidence has been shown that cortico-spinal inputs are related to changes in postural responses (Keck et al. 1998), suggesting a possible descending cortico-spinal pathway is available for modulatory influence of central set on the amplitude of postural responses.

Trunk Control

The trunk consists of nearly 1/2 of the total body's COM which is perched nearly 2/3 of the total body height above the ground (Winter et al. 1991) Therefore, trunk control represents a significant challenge for the CNS during static balance conditions, and particularly during locomotion and in cases in which balance is perturbed. Early trunk motion to unexpected perturbations in the pitch plane has been shown in normal controls (Allum et al. 1993) and constitutes a major impairment in a number of balance disorders. Horak et al. (1990) have argued that vestibular loss patients are unable to control large trunk movements when standing on a narrow surface, a deficit they claim is due to an inability to generate the appropriate hip strategy response. In contrast Allum et al. (1998) and Runge et al. (1998) have shown that vestibular loss patients are capable of generating appropriate hip torques to control pitch oriented trunk motion, but are hampered by a deficit in the timing metrics. These difficulties are different than those observed in PD, which have been shown to exert a stiffer trunk control to unexpected postural perturbations, as evidenced by larger and early trunk accelerations (Allum et al. 1988).

These previous findings have remained relatively robust when examined using multi-directional perturbations. Normal subjects were shown to have trunk pitch velocities (onset latency 50-60 ms) for pitch directed perturbations, as well as perturbations in a pure roll direction (Carpenter et al. 1999). In vestibular loss patients, initial trunk pitch movements were relatively normal until 150-200 ms, but were followed by excessively large trunk corrections due to overactivity in the stabilizing period in paraspinals and tibialis anterior muscles. Trunk pitch displacement in PD was reduced for all backward rotations which caused the trunk to pitch forward. This response was likely due to increased trunk stiffness achieved through increased background activity in hip and trunk muscles, and altered activation of paraspinal

muscles leading to co-contraction between left and right muscles. New evidence has been found for altered trunk control in the pitch plane in patients with total leg proprioceptive loss (Bloem et al. 2001) and to a lesser extent in healthy elderly (Allum et al. 2001a). In all of these subjects, trunk velocity was decreased in the pitch plane, which was likely attributed to increased trunk stiffness. Carpenter et al. (2001c) provided evidence to suggest that increased trunk stiffness in the pitch plane, whether it is observed in patients with PD, total proprioceptive loss or in aging populations, may be attributed to increased fear of falling. In young normal subjects, decreased trunk displacement was observed for backward directed perturbations when standing in a high postural threat condition compared to a low threat condition. These changes in trunk displacement are associated with increased amplitudes of balance correcting responses in hip and trunk muscles and a change in directional sensitivity of paraspinal muscles leading to co-contraction and presumably increased trunk stiffness.

The major benefit of using multi-directional perturbations is the ability to observe how the trunk is controlled in both pitch and roll planes. Winter et al. (1996) demonstrated that the COP during quiet stance is independently controlled in A-P and M-L directions. Pitch and roll movements of the trunk may also be independently controlled. Observations of trunk roll displacements to off-pitch perturbations have initial onsets which are 20-30 ms prior to any pitch displacements of the trunk (Carpenter et al. 1999). Furthermore, kinematic analysis has demonstrated that the upper trunk moves in the opposite direction to that of the pelvis and lumbar region which are rolled in the same direction of the platform roll (Carpenter et al. 2001c). Therefore, it is likely that a different control system is required to compensate for pitch and roll displacements of the trunk respectively. The ability to control the spatio-temporal requirements between these two independent systems may be useful in discriminating between patients with different balance deficits.

Patients with bilateral vestibular loss have early stimulus induced trunk roll which is similar to that of healthy controls. However, after 150 ms the trunk experiences excessive movement in the opposite direction, overshooting the normal response and continuing to rotate in the same direction of platform roll, which still increases beyond 500 ms (Carpenter et al. 2001a). In contrast, PD patients have no initial movement of the trunk during the first 150 ms after

perturbation onset, due presumably to increased trunk stiffness. This was followed by trunk roll movement in the same direction as platform roll, as the subject falls like a log. It is interesting that early experiments by Martin (1965) reported similarly directed trunk roll movements in seated subjects suffering from both vestibular loss and post-encephaletic parkinsonism which he attributed to an absence of postural reflexes in these patients. However, it must be noted that Martin's observations were based on video analysis that would not allow him to observe the early passive component of trunk roll (prior to 150 ms) and did not record electromyographical data that would have provided insight into the electrophysiological aspects of these disorders. In fact, it is possible that what Martin observed was overactivity and not an absence of postural reflexes, with excessive stabilizing activity in vestibular loss subjects, and increased co-contraction and trunk stiffness in PD patients both contributing to an eventual roll of the trunk in the same direction as platform roll. More drastic changes in trunk instability in the roll plane were observed in the patient with total leg proprioceptive loss. In this case, trunk roll fell immediately in the same direction as platform roll and opposite to that of young normals, suggesting even greater elevations in background activity, co-contraction and trunk stiffness in this patient (Bloem et al. 2001). However, some of the trunk stiffness seen in both PD patients and the total leg proprioceptive loss patients may be associated with normal effects of age, as increased background activity and improper direction of trunk roll was also observed in elderly individuals compared to younger controls (although differences were not nearly as prominent as that seen in the patients – see Allum et al. 2001a). Trunk stiffness has been experimentally confirmed as a possible contributor to the abnormal roll characteristics observed in PD patients, total proprioceptive loss patients and to a lesser extent in elderly. Gruneberg et al. (2001) have shown that young normal subjects will fall in the same direction to platform roll when their trunks have been artificially 'stiffened' by wearing a corset which impeded any pitch or roll movements around the hip. Increased postural threat appeared to have no impact on control of trunk movements in the roll direction, however this may have been linked to the location of the perceived postural threat which was always positioned in the pitch plane.

In summary, large differences have been observed between patients with peripheral and central balance deficits in their ability to control stimulus induced trunk movement in the pitch and

roll planes due to unexpected multi-directional perturbations. Recent evidence has shown that the inclusion of trunk pitch velocity has greatly improved the discriminatory ability of dynamic posturography (Allum et al. 2001b). The results of the present thesis and that of other research examining multi-directional perturbations may suggest that even greater discriminatory ability may be achieved if both pitch and roll characteristics of trunk control are used as identifying factors.

REFERENCES

Allum JHJ, Bloem BR, Carpenter MG, Honegger F (2001b) Differential diagnosis of proprioceptive and vestibular deficits using dynamic support-surface posturography. Gait and Posture (In Press).

Allum JHJ, Carpenter MG, Honegger F, Adkin AL, Bloem BR (2001a) Age-dependent variations in the directional sensivity of balance corrections. In: Duysens J, Smits-Engelsman BCM, Kingma H, editors. Control of Posture and Gait. Maastricht: Symposium of the International Society for Postural and Gait Research, 912-915

Allum JHJ, Honegger F (1998) Interactions between vestibular and proprioceptive inputs triggering and modulating human balance-correcting responses differ across muscles. Exp Brain Res. 121: 478-494

Allum JHJ, Honegger F, Schicks H (1994) The influence of a bilateral peripheral vestibular deficit on postural synergies. J Vestib Res 4:49-70

Allum JHJ, Honegger F, Schicks H J (1993) Vestibular and proprioceptive modulation of postural synergies in normal subjects. J Vestib Res 3(1): 59-85

Allum JHJ, Honegger F, Schicks H J (1993) Vestibular and proprioceptive modulation of postural synergies in normal subjects. J Vestib Res 3(1): 59-85

Allum JHJ, Keshner EA, Honegger F, Wuetrich R (1988) Disturbance of posture in patients with Parkinson's disease. In: Amblard B, Berthoz A, Clarac F, editors. Posture and gait: development, adaptation and modulation. Amsterdam: Elsevier, 245-57

Beckley DJ, Bloem BR, Remler MP (1993) Impaired scaling of long latency postural reflexes in patients with Parkinson's disease. Electroenceph Clin Neurophysiol 89: 22-28

Bloem BR, Allum JHJ, Carpenter MG, Honegger F (2000) Is lower leg proprioception essential for triggering human balance corrections? Exp Brain Res 130: 375-391

Bloem BR, Allum JHJ, Carpenter MG, Verschuuren JJGM, Honegger F (2001) Triggering of balance corrections and compensatory strategies in a patient with total leg proprioceptive loss. Exp Brain Res (submitted)

Brown LA, Frank JS (1997) Postural compensations to the potential consequences of instability: kinematics. Gait and Posture 6:89-97

Carpenter MG, Allum JHJ, Honegger F (1999) Influence of acute unilateral vestibular loss on postural responses in multiple directions. Gait & Posture 9 (Suppl.1): S2

Carpenter MG, Allum JHJ, Honegger F (2001a) Vestibular influences on human postural control in combinations of pitch and roll planes reveal differences in spatiotemporal processing. ExpBrain Res (in press)

Carpenter MG, Allum JHJ, Honegger F, Adkin AL, Bloem BR (2001b) Postural abnormalities to multidirectional stance perturbations in Parkinson's disease. Brain (submitted)

Carpenter MG, Allum JHJ, Honegger F. (1999) Directional sensitivity of stretch reflexes and balance corrections for normal subjects in the roll and pitch planes. Exp Brain Res 129: 93-113

Carpenter MG, Frank JS, Allum JHJ, Adkin AL, Patton A (2001c) Influence of postural threat on postural reactions to multidirectional surface rotations Gait and Posture (submitted)

Chong RKY, Horak FB, Woollacott MH (1999) Time-dependent influence of sensorimotor set on automatic responses in perturbed stance. Exp Brain Res 124: 513-519

Diener HC, Horak F, Stelmach G, Guschbauer B, Dichgans J (1991) Direction and amplitude precuing has no effect on automatic posture responses. Exp Brain Res 84: 219-223

Dietz V, Berger W, Horstmann GA (1988) Posture in Parkinson's disease: impairment of reflexes and programming. Ann Neurol 24: 660-669

Di Fabio RP (1995) Sensitivity and specificity of platform posturography for identifying patients with vestibular dysfunction. Phys Ther. 75:290-305

Do MC, Brenière Y, Bouisset S (1988) Compensatory reactions in forward fall: are they initiated by stretch receptors? Electroenceph Clin Neurophysiol 69: 448-452

Forssberg H, Hirschfeld H (1994) Postural adjustments in sitting humans following external perturbations: muscle activity and kinematics. Exp Brain Res 97: 515-527

Grüneberg C, Allum JHJ, Honegger F, Bloem BR. The influence of artificially increased trunk stiffness on automatic balance correcting responses. In: Duysens J, Smits-Engelsman BCM, Kingma H, editors. Control of Posture and Gait. Maastricht : Symposium of the International Society for Postural and Gait Research, 2001: 359-64

Horak FB, Diener HC, Nashner LM (1989) Influence of central set on human postural responses. J Neurophysiol 62: 841-853

Horak FB, Nashner LM (1986) Central programming of postural movements: Adaptation to altered support-surface configurations. J Neurophysiol 55: 1369-1381

Horak FB, Nashner LM, Diener HC (1990) Postural strategies associated with somatosensory and vestibular loss. Exp Brain Res 82: 167-177

Horstmann GA, Dietz V (1990) A basic posture control mechanism: the stabilization of the centre of gravity. Electroenceph Clin Neurophysiol 76: 165-176

Inglis JT, Horak FB, Shupert CL, Jones-Rycewicz C (1994) The importance of somatosensory information in triggering and scaling automatic postural responses in humans. Exp Brain Res 101: 159-164

Keck ME, Pijnappels M, Schubert M, Colombo G, Curt A, Dietz V (1998) Stumbling reactions in man: influence of corticospinal input. Electroenceph Clin Neurophysiol 109: 215-223

Keshner EA, Allum JH, Pfaltz CR (1987) Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit. Exp Brain Res 69: 77-92

Keshner EA, Woollacott MH, Debu B (1988) Neck, trunk and limb muscle responses during postural perturbations in humans. Exp Brain Res 71(3): 455-466

Maki BE, Whitelaw RS (1993) Influence of expectation and arousal on center-of-pressure responses to transient postural perturbations. J Vestib Res 3: 25-39

Martin JP (1965) Tilting reactions and disorders of the basal ganglia. Brain 88: 855-874.

McIlroy WE, Maki BE (1995) Early activation of arm muscles follows external perturbation of upright stance. Neurosci Lett 184:177-80

Moore SP, Rushmer DS, Windus SL, Nashner LM (1988) Human automatic postural responses: responses to horizontal perturbations of stance in multiple directions. Exp Brain Res 73: 648-658

Nashner LM, Biack FO, Wall C III (1982) Adaptation to altered support surface and visual conditions during stance: patients with vestibular deficits. J Neuroscience 2: 536-544

Perry SD, McIlroy WE, Maki BE. (2000) The role of plantar cutaneous mechanoreceptors in the control of compensatory stepping reactions evoked by unpredictable, multi-directional perturbation. Brain Res 877: 401-406

Runge CF, Shupert CL, Horak FB, Zajac FE (1998) Role of vestibular information in initiation of rapid postural responses. Exp Brain Res 122:403-412

Rushmer DS, Russell CJ, Macpherson J, Phillips JO, Dunbar DC (1983) Automatic postural responses in the cat: Responses to headward and tailward translation. Exp Brain Res 50: 45-61

Schieppati M, Nardone A (1991) Free and supported stance in Parkinson's disease. Brain 114: 1227-1244

Sveistrup H, Woollacott MH (1997) Practice modifies the developing automatic postural response. Exp Brain Res 114: 33-43

Timmann D, Horak FB (1997) Prediction and set-dependent scaling of early postural responses in cerebellar patients. Brain 120: 327-337

Winter DA, Prince F, Frank JS, Powell C, Zabjek KF (1996) Unified theory regarding A/P and M/L balance in quiet stance. J Neurophysiol 75:2334-43

Winter DA. (1991) The biomechanics and motor control of human gait: Normal, elderly and pathological. 2nd ed. Waterloo, Canada: University of Waterloo Press