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

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ABSTRACT

Previous studies have shown significant effects of increased postural anxiety in healthy young individuals when standing quietly or performing voluntary postural tasks. However, little is known about the influence of anxiety on reactive postural control. The present study examined how increased postural anxiety 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. Electromyographical data from 12 different postural leg, hip and trunk muscles was collected simultaneously. Full body kinematic data was also used to determine total body centre of mass (COM) and segment displacements.

Four distinct changes were observed with increased postural anxiety: a) increased amplitude in balance correcting responses (120-220 ms) in all leg, trunk and arm muscles b) decreased onset latency of deltoid responses, c) reduced magnitude of COM displacement and d) reduced angular displacement of leg, pelvis and trunk. These observations suggest that changes in dynamic postural responses with increased anxiety are mediated by alterations in neuro-muscular control mechanisms, and thus may contribute significantly to the patho-physiology of balance deficits associated with aging or neurological disease.
INTRODUCTION

Postural anxiety has been related to balance and gait impairments (Tinetti et al. 1994; Vellas et al. 1997; Meyers et al. 1996; Hill et al. 1996; Maki et al. 1991) and increased risk of falls (Cummings et al. 2000). Postural anxiety is not only a consequence of poor balance but can also contribute to changes in balance control. For example, studies have shown that postural anxiety can influence changes in the way in which balance is controlled during quiet standing (Carpenter et al. 1999a, 2001a; Adkin et al. 2000; Maki and McIlroy 1996; Nakahara et al. 2000), anticipatory control of voluntary movements (Adkin et al. 2002) and walking (Brown et al. 2002).

Postural reactions to unexpected perturbations are unique from other balance tasks in that they consist of stereotypical patterns of triggered postural responses in leg, trunk and arm muscles, which have onset latencies of approximately 100 ms. The onset latency of triggered postural responses is earlier than that expected for voluntarily initiated movements (Nashner and Cordo 1981). Furthermore, the relatively long latency of the triggered responses and their appearance in muscles that are both stretched and unloaded by the postural perturbation (Diener et al. 1983; Allum et al. 1994; Carpenter et al. 1999b), distinguishes triggered responses from that of spinal stretch reflexes. Current evidence has suggested that triggered responses are most likely mediated by supra-spinal pathways that pass through regions in the brainstem or higher cortical areas (Macpherson et al. 1999). Therefore, changes in the amplitude or timing of triggered postural responses are often used as an indicator of changes in the gain or processing requirements of sensory information used to trigger or modulate postural responses.
Although postural anxiety has been suspected to have an influence on postural reactions to unexpected perturbations (Bloem et al. 2001; Maki and Whitelaw 1993; Okada et al. 2001), there have been no studies to date that have manipulated anxiety in order to determine how it may influence stretch reflexes, triggered postural responses and voluntary reactions in response to externally generated postural perturbations. Therefore, the goal of the present study was to determine how increased anxiety influences the muscular and biomechanical responses of healthy young adults to unexpected rotations of the support surface. Information gained from this study has two important applications. Firstly, the knowledge may be used to gain a better understanding of the connections between neural areas controlling emotional behaviour and those involved in balance control. Secondly, the study will provide important insight into the possible mechanisms through which fear and anxiety may contribute to balance deficits in individuals with increased fear of falling, such as the elderly (Niino et al. 2000), patients with Parkinson’s disease (Bloem et al. 2001), vestibular loss or proprioceptive deficit (Yardley and Hallam 1996).

**MATERIALS AND METHODS**

**Subjects**

Ten university students (6 male; mean age +/- 1SD = 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 orthopaedic disorder as verified by self report. Prior to the
experiment, anthropometric measures were recorded, including height (mean +/- 1SD =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. All subjects were completely naïve to the experimental design and balance perturbations used in the study.

**Apparatus**

Surface rotations were delivered using a single axis, rotating platform that was bolted firmly to the front edge of a hydraulic lift (Pentalift, Guelph). The rotating platform was designed so that the participants could stand in a constant position, while the axis of rotation delivered by the platform motor could be manually turned (up to 180 deg) relative to the position of the participant to achieve multi-directional perturbations. This method is distinctly different than that used by Moore et al. (1988) in which the participant 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. When the hydraulic lift was in the lowest position, the top surface of the rotating platform was 60 cm above the ground. Handrails (165 cm long, 125 cm high) 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. Adjustable heel guides were used to align the ankle with the pitch axis of rotation and maintain 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 a safety harness worn by the participants throughout the experiment. 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.

Procedure
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. A 5 minute rest was given before the platform was moved to the second surface height condition (either low or high) to remove any possible effects due to fatigue.

Participants were required to focus on a target, placed at eye level on the wall approximately 6 m in front of them, and assume 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 had a constant amplitude of 7.5 deg and 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 randomly presented 6 times. Directions were 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 experimenter altered the direction of the platform’s axis of rotation manually. Before moving the platform axis to its desired location, the platform was moved randomly in different directions and over different time periods to eliminate any timing or auditory information that would otherwise allow the perturbation direction to be anticipated. 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. The platform axis was not in the field of view and therefore, subjects did not have any visual information that would help them to predict the new orientation of the platform.

Data Collection

Recordings of all biomechanical and electromyographical (EMG) data commenced 2 s prior to the onset of the perturbation and were collected for 5 s. EMG recordings were sampled at 1024 Hz. EMG was recorded from disposable surface electrodes, placed 2 cm
apart along the muscle bellies of 12 different muscles: left soleus, left tibialis anterior and bilaterally on rectus femoris, biceps femoris, gluteus medius, paraspinals and middle deltoid (acromial head). Electrode leads were attached to a preamplifier unit that 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 one at the 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 complete questionnaires probing their general 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. 2002). General balance confidence was estimated on a percentage scale at each surface height, with 0 representing ‘no confidence’ and 100 representing ‘complete confidence’. Task specific balance efficacy was assessed using a percentage scale to estimate the subject’s own abilities to (1) avoid a fall, (2) maintain concentration, (3) overcome worry, and (4) reduce 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.
After the completion of a series of postural perturbations and quiet stance trials at each surface height, participants were seated and asked to complete self-rated questionnaires that assessed their perceived anxiety and perceived stability during the stance trial at the previous surface height. 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.

The reliability of the anxiety and balance confidence scales used in the present study has been verified independently. Intra-class correlation co-efficients were calculated from the scores of 30 subjects that performed static and dynamic balance tasks in low and high threat conditions over three consecutive test sessions within the same week. Results confirmed a high reliability for the questionnaires for the assessment of balance confidence (r=0.86), task specific balance efficacy (r=0.78-0.85), perceived stability (r=0.88) and perceived anxiety (r=0.70-0.84) for dynamic balance tests performed in low and high threat conditions (L Grin, unpublished observations).
Data Analysis

Zero latency for each trial was determined as the first inflection 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 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).

Onset latencies for stretch reflexes and balance correcting responses were calculated for each trial and muscle. For each subject, the six individual trials for each muscle and direction 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. Latencies were first 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.
Position data was digitally filtered at 25 Hz using a zero-phase shift, dual pass Butterworth filter. Total body COM displacement was calculated in the anterior-posterior (A-P) and medial-lateral (M-L) 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. 1998). 2D angular displacements of the lower leg were calculated from ankle and knee markers of the left leg. 3D angular displacements of the pelvis and trunk were calculated using euler angles (Söderkvist and Wedin 1993) based on rigid bodies defined by bilateral markers on the ASIS and iliac crest (pelvis) and shoulders and zyphoid process (trunk). Direction vectors for COM were calculated using the resultant vector of pitch and roll displacements for individual trials. All EMG areas, latencies, segment angles and COM results were averaged across perturbation direction. Subject averages were averaged together to yield group averages for low and high threat conditions.

**Statistical Analysis**

EMG areas, latencies and kinematic results were examined using a 2 x 6 (threat by direction) within-subject analysis of variance (ANOVA). Significant main and interaction effects were analyzed using pre-planned comparisons using t-tests with Bonferroni corrections (alpha=0.05). Scores for general balance confidence, task specific balance efficacy, perceived anxiety and perceived stability were examined using repeated measures one way ANOVA, with a level of significance of 0.05.
RESULTS

Perceived Anxiety and Balance Confidence

Postural threat had a significant influence on participant’s general balance confidence, confidence to specifically avoid a fall, perceived anxiety and perceived stability. Balance confidence significantly decreased an average of 18% \((F(1,9)=27.21, p<0.001)\), and confidence to avoid a fall decreased an average of 16% \((F(1,9)=13.52, p<0.01)\) when standing in the high threat compared to the low threat condition. Following performance of the balance task, the participants’ perceptions of anxiety were increased by an average of 30% in the high threat compared to low threat condition \((F(1,9)=10.55, p<0.02)\). In addition, participants’ perceived stability significantly decreased an average of 14% when standing in the high threat compared to low threat condition \((F(1,9)=11.12, p<0.02)\).

Total Body Centre of Mass

The total body COM was displaced in the same direction as the platform perturbation. Therefore, for backward left perturbations, the total body COM was displaced backwards and to the left (Figure 1). Similarly, forward left perturbations caused the total body COM to be displaced forwards and to the left.

The magnitude of COM displacement was influenced by increased postural threat. As shown in figures 1 and 2, the COM displacement in the vertical direction had a significantly larger peak displacement for backward perturbations, and smaller peak displacement for forward perturbations, in the high threat compared to the low threat condition \((F(5,45)=2.58, p=0.03)\). There was a trend for smaller peak COM
Influence of postural anxiety on postural reactions

Displacements in the A-P direction in high threat compared to low threat conditions (F(1,9)=3.26, p=0.10). Small reductions observed in peak M-L COM displacement for perturbations with a roll component in high threat compared to low threat conditions (figure 1 and 2), were not found to be statistically significant (height F(1,9)=1.09, p=0.32; height by direction F(5,45)=1.45, p=0.22). Although, the magnitudes of peak COM displacement were altered with increased postural threat, there were no changes in the resultant directional vectors for the displacement of the COM, either in the sagittal or horizontal planes.

Changes in COM displacement were supported by observed changes in segment displacements. Backward left perturbations caused the left lower leg segment to be rotated backwards, reaching a peak angular displacement at 250 ms after the onset of platform rotation. The pelvis and trunk segments pitched forward in the opposite direction to leg displacements (figure 1). Similar but opposite segment displacements were observed for forward perturbations, with the lower leg rotating forward, and pelvis and trunk segments displaced backwards.

In general, high threat conditions were associated with decreased amplitude of segment movements in legs, pelvis and trunk. However, changes associated with postural threat were relatively small, and therefore, statistical comparisons were not significant. Backward rotation of the lower legs was reduced in amplitude in the high threat compared to the low threat condition. The peak forward pitch angle of the pelvis and
Influence of postural anxiety on postural reactions

trunk were also reduced in the high threat compared to low threat condition (Figure 1). Similar observations were observed in the forward left directions.

Amplitude and directional sensitivity changes in leg and trunk muscles

Backward perturbations (toe-up) elicited an early stretch reflex in the soleus muscle at an average latency (and standard error) of 46.4 +/-3.05 ms (figure 3) followed by large balance correcting responses in tibialis anterior (to control backward rotation of the lower leg), rectus femoris (to maintain knee extension) and paraspinals (to control trunk flexion). In contrast, forward perturbations (toe-down) elicited medium latency 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 4). Large balance correcting responses (120-220 ms) were elicited in the soleus muscle (to control forward leg rotation) and distinct bursts of activity were observed in left rectus femoris and biceps femoris muscles (to provide stability at the knee and hip joint).

In perturbations with a roll component, the legs are initially displaced in the same direction as platform movement, while the upper body counter-rotates in the opposite direction. Therefore, for backward and forward left perturbations the legs are rotated to the left, while the upper body is rotated to the right. Large balance correcting activity is observed in hip and trunk muscles which are initially unloaded by the perturbation. Therefore, for left perturbations, distinct bursts of activity are observed in the left gluteus medius and right paraspinal muscles (figures 3 and 4).
Comparisons between low and high threat conditions, did not reveal any differences in background muscle activity (measured 500 ms prior to perturbation onset) for any muscle, except biceps femoris (threat x direction interaction F(5,39)=2.61, p<0.04). Likewise, there were no differences in the latency of the stretch reflexes or balance correcting responses for any leg or trunk muscle. The onset 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, differences between threat conditions were observed in the amplitude of balance correcting responses (120-220 ms) for all postural muscles. For backward left perturbations, significantly larger response amplitudes were observed in the primary balance correcting muscles such as tibialis anterior, biceps femoris, rectus femoris and paraspinals (figure 3). For forward left perturbations there was greater muscle activity in soleus, biceps femoris, rectus femoris and paraspinal muscles, a muscle that was minimally activated in the low threat condition for this direction (figure 4).

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 figure 5 depict the magnitude and directional sensitivity of EMG areas calculated over the balance correcting period between 120-220 ms. ANOVA results revealed a significant
main effect of threat on amplitude of balance correcting responses for all muscles analyzed. As observed in figure 5, participants had larger responses between 120-220 ms in the high compared to low threat condition for soleus (F(1,9)=5.06, p<0.05), tibialis anterior (F(1,9)=13.59, p<0.01), rectus femoris (F(1,9)=5.43, p<0.04), biceps femoris (F=(1,9)=8.42, p<0.02), gluteus medius (F(1,9)=24.07, p<0.001) and paraspinals (F(1,9)=5.14, p<0.05). All of these muscles demonstrated normal directional sensitivity in the high threat condition, with response amplitudes for particular directions similar to those observed in the low threat condition.

Statistical results did not reveal significant differences in EMG amplitude for any muscle during the later periods between 240-340 or 350-700 ms.

Shoulder Muscle Responses

Backward left perturbations elicited distinct bursts of muscle activity in both left and right deltoid activities with onset latencies similar to that seen for balance correcting responses in more distal leg and trunk muscles (see Table 1). These responses were observed without the presence of any prior stretch or unloading responses, and returned to resting levels of activation after 250 ms (figure 6A). Forward left perturbations also elicited distinct balance correcting activity in 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 6A).
In the high threat condition, both the onset latency and amplitude of balance correcting activity in deltoids was significantly different from responses seen in the low threat condition. ANOVA results revealed a significant effect of threat ($F(1,6)=9.77, p<0.02$) for onset latencies in left deltoid muscle (Table 1). Onset latencies in left deltoid were earlier (on average by 18.8 ms) in the high threat condition for all perturbation directions. For example, backward left perturbations had mean onset latencies (and st. error of mean) of 85.7 +/- 5.9 ms in the high threat compared to 103.7 +/- 5.0 ms in the low threat condition. Likewise, forward left perturbations, had mean onset latencies (and st. error of mean) of 90.8 +/- 4.1 ms in the high threat compared to 106.8 +/- 5.6 ms in the low threat condition (figure 6A).

Balance correcting amplitudes in deltoids were also larger during the high compared to low threat condition (figure 6A and B). As shown in polar plots in figure 6B, significantly larger balance correcting responses were present in deltoid muscles ($F(1,9)=4.85, p<0.05$) 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 (figure 6B).

**DISCUSSION**

The purpose of the study was to determine how changes in postural anxiety could influence the reactive mechanisms associated with triggered postural responses to an
Influence of postural anxiety on postural reactions

unexpected balance perturbation. Our results have demonstrated four distinct changes in the balance response that are influenced by increased postural anxiety a) increased amplitude in balance correcting responses (120-220 ms) in all leg, trunk and arm muscles b) decreased onset latency of deltoid responses, c) reduced magnitude of COM displacement and d) reduced angular displacement of leg, pelvis and trunk.

Previous studies on postural anxiety have used either static balance (Carpenter et al. 1999a, 2001a; Adkin et al. 2000; Maki et al. 1991, Maki and McIlroy 1996), or dynamic postural tasks in which the internally generated (Adkin et al. 2002) or external postural perturbations (Brown and Frank 1997; Okada et al. 2001; Wada et al. 2002) were predictable in direction. In such circumstances, the opportunity exists to use anticipatory postural adjustments in muscle tone or postural leaning to improve stability or performance on the postural task. For example, participants have been shown to lean away from the direction of the perceived threat (i.e. edge of a high surface), shifting the mean position of the COM backward when standing in a high compared to low threat condition (Carpenter et al. 1999a, 2001a; Adkin et al. 2000, 2002). Likewise, background activity increased in tibialis anterior and decreased in triceps surae muscles when participants stood in more threatening conditions (Brown and Frank 1997, Carpenter et al. 2001a). Such changes are not unique to studies that use environmental change to alter postural threat. Maki and McIlroy (1996) observed forward leaning and increased background activity of tibialis anterior in anxious subjects during quiet stance. Pre-stimulus changes in background activity or muscle length have the potential to have confounding effects on subsequent muscular responses. For example, pre-leaning 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). Likewise, 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).

In contrast to previous studies, the present experiment utilized external postural perturbations that were delivered in multiple directions relative to the position of the perceived postural threat. In such situations, using anticipatory changes in pre-stimulus posture may not be advantageous, since an anticipatory change used to protect against perturbations in one direction will have adverse effects for other directed perturbations. Furthermore, stringent control of lower leg angle prior to the onset of each perturbation trial was used to ensure that subjects did not lean prior to the perturbation. As a result of these methodological considerations, we did not observe any changes in background muscular activity preceding the onset of postural perturbations between the high and low height conditions. Furthermore, there were no significant changes in the timing or amplitudes of short latency stretch reflexes that would suggest differences in tone or pre-stimulus posture between the high and low threat conditions.

Even with pre-stimulus posture controlled, some findings from the present study were consistent with that of previous studies. The reduced amplitude of peak A-P COM
Influence of postural anxiety on postural reactions

Displacement in high threat conditions (figures 1 and 2) corresponds with reduced COM displacements observed in subjects that were unexpectedly pushed from behind in high compared to low threat conditions (Brown and Frank 1997). Preserved normal onset latencies in leg muscle responses with increased anxiety (figures 3 and 4) was also observed in fearful subjects reacting to unexpected surface translations (Okada et al. 2001; Bolmont et al. 2002). Furthermore, Okada et al. (2001) observed increased co-contraction of tibialis anterior and gastrocnemius muscles during the first 200 ms of the postural response in older adults with fear of falling which corresponds to increased amplitudes of tibialis anterior and solus muscle activity observed between 120-220 ms with increased postural anxiety (figure 5).

In addition to the above findings, the current study has uncovered a number of new aspects of balance control, which provide important insights into how anxiety can influence dynamic postural control. The use of surface rotations provides an important advantage over translations, as it allows a separation of early stretch responses and subsequent balance correcting responses (Diener et al. 1983). With this paradigm we have been able to show that changes associated with increased postural anxiety influence mechanisms controlling long latency reflexes between 120-220 ms without altering either short spinal reflexes or later voluntary postural responses, which have latencies >200 ms. Increased amplitudes of balance correcting responses were observed, not only in lower leg muscles, but also in muscles of the upper leg, trunk and arms (figures 3-6). Combined with the fact that the directional sensitivity of muscular responses are generally preserved with increased postural anxiety (figures 5 and 6), our findings
Influence of postural anxiety on postural reactions

suggest that increased anxiety influences changes in the gain of postural responses without altering the basic postural synergy.

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 when postural anxiety increased. McIlroy and Maki (1995) also reported earlier and larger activation of arm muscles in young controls during larger, and possibly more threatening perturbations. Evidence has shown that some voluntary arm movements can be triggered earlier (with onsets as early as 90 ms) when accompanied by an acoustic startle stimulus (Valls Sole et al. 1999; Carlsen et al. 2003). One distinction between the present study and previously observed startle responses is that startle responses are typically of similar amplitude and duration, shifted in time with earlier onset and offsets, whereas in the present study, the deltoid responses in the high threat condition were triggered earlier, and were larger and longer in duration compared to low threat condition. Thus, further studies are required to understand the nature of early arm responses in fearful conditions.

The biomechanical changes observed in the current study were consistent with those expected with observed gain increases in balance correcting responses in distal and proximal postural muscles. For example, for backward left perturbations, greater activation of tibialis anterior would be required to reduce the peak backward displacement of the lower leg angle, while increased gain of paraspinals and biceps femoris activity would contribute to reducing the peak forward displacement of the trunk and pelvis respectively. Furthermore, the decreased range of angular displacements in the
lower leg, pelvis and trunk would contribute to the tighter control of the COM movements in the sagittal plane observed in the high compared to low threat condition (figure 2). Adoption of postural strategies to maintain a tighter control of the COM and decrease angular displacements of the limbs during conditions of increased anxiety have been observed previously during quiet standing (Carpenter et al. 2001), rising to the toes (Adkin et al. 2002), walking (Brown et al. 2002) and unexpected perturbations to the trunk (Brown and Frank 1997). More research is required to determine the extent to which a more tightly controlled COM during dynamic balance tasks may adversely affect balance performance and lead to falls. It has been hypothesized that co-contraction and stiffening responses, particularly in the trunk, may contribute to balance deficits in older adults (Allum et al. 2002) and patients with Parkinson’s disease (Carpenter et al. 2004).

Possible neural links between fear and postural control – central influences

Although, we did not have any direct measure of physiological arousal in the present study, the presently observed changes in balance confidence and perceived anxiety have been shown previously to parallel changes in physiological measures, such as increased blood pressure and increased skin conductance, in participants standing under similar postural threat conditions (Carpenter et al. 1999c; Adkin et al. 2002). This converging evidence provides convincing support that our manipulation of height has an influence on higher brain centers associated with anxiety or emotional behaviour.

Previous studies have demonstrated that changes in trait anxiety or mood states can influence postural control (Wada et al. 2001; Bolmont et al. 2002). Thus the mechanisms
that control balance or posture may receive information from areas of the brain that are associated with emotion/mood processing, such as the amygdala or parabrachial nucleus (PBN). One possible neural link between balance and anxiety/fear behaviours is via the vestibular system. Balaban (2002) has proposed three potential neural networks that could potentially modulate balance reactions via the vestibular system: vestibulo-parabrachial nucleus network, coeruleo-vestibular network and raphe-nuclear-vestibular network. Research has shown that direct neural projections are found between the PBN and vestibular nuclei (Balaban, 1996). Based on these connections, Balaban (2001) proposed that the vestibulo-parabrachial nucleus network may allow outputs from vestibular nuclei to be modulated with respect to the safety or danger associated with incoming inertial-gravitational information. Thus it is possible that the PBN could modulate the gain of the output from the vestibular nuclei, via these pathways, and consequently could alter the amplitude of postural changes controlled by the vestibular system.

There is also anatomical evidence for modulatory roles of the noradrenergic pathways on vestibular nuclei. Schuerger and Balaban (1999) have shown in rabbits and monkeys that vestibular neurons receive noradrenaline from locus ceruleus and subcereleus, particularly to areas of the superior and lateral vestibular nucleus which control postural and eye movements. Since locus ceruleus has been shown to become active in times of increased vigilance or arousal, it would have the potential to modulate vestibular function during periods of increased arousal and anxiety (Balaban and Thayer, 2001).
Vestibular neurons also receive serotonergic inputs from raphe-nuclei. Animal studies have shown connections between raphe nuclei and vestibular nuclei, and that serotonin release is associated with the degree of arousal (Rueter et al. 1997). Serotonergic activation is believed to facilitate motor output and inhibit sensory processing during periods of fear or anxiety expression. Li Volsi et al. (1998) demonstrated that local application of serotonin in the lateral and inferior vestibular nucleus increases EMG activity of limb muscles via the lateral vestibular spinal tract. This evidence would suggest that actions of serotonin in the vestibular nucleus would increase the sensitivity of the lateral vestibular-spinal tract with increased arousal (Balaban and Thayer, 2001).

Our current results are consistent with a mechanism of increased vestibular gain with increased postural anxiety. Previous studies have demonstrated that reduced vestibular information, in patients with bilateral compensated and acute unilateral vestibular loss, are associated with a preservation of the onset timing, pattern and directional sensitivity of postural responses, but a decreased amplitude of balance correcting responses between 120-220 ms (Carpenter et al. 1999d; 2001b; Allum and Pfaltz 1985; Keshner et al. 1987; Allum et al. 1994; Runge et al. 1998). Thus, the reciprocal changes observed in the present study (increased amplitude of balance correcting responses with increased postural anxiety), would be consistent with changes that would be expected from an increase in vestibular gain. Other studies have also discovered changes in anxious subjects which coincide with changes expected from altered vestibular function. Yardley et al. (1995), observed increased gain of vestibular-ocular reflex in subjects with increased anxiety. Individuals with increased anxiety demonstrated changes in frequency
components of postural sway (Wada et al. 2001) and changes in aspects of the SOT test (Bolmont et al. 2002) that are associated with altered vestibular inputs. However, more detailed studies must be performed to understand the possible links of the vestibular system and emotional control centers.

Alternatively, reciprocal projections between the basal ganglia and amygdala have been considered important in attaching contextual or emotional weighting to movements, and could also play an important role in modulating dynamic balance. Interestingly, PD patients tested with a similar multi-directional protocol to the present study demonstrated increased balance correcting response amplitudes, and earlier and larger amplitudes of deltoid muscle responses (Carpenter et al. 2004), which correspond to changes observed in healthy young individuals standing under conditions of increased postural threat.

Possible neural links between fear and postural control – peripheral influences
The observed changes in balance correcting response amplitudes, without accompanying changes in stretch reflex amplitudes or later voluntary responses could also be attributed to changes in gamma motor neuron activity. Prochazka et al. (1985) showed that gamma motor neuron activity was increased in cats when performing more difficult postural tasks, such as standing on an elevated surface or walking on a beam. Although there is no direct evidence for alpha-gamma decoupling during standing in humans, microneurographic recordings from gamma efferent and 1a afferent fibres in relaxed humans (Ribot-Ciscar et al. 1986, 2000; Rossi-Durand 2002) have provided supporting evidence for increased fusimotor drive with increased arousal. Increased fusimotor drive
would serve a dual benefit in conditions of increased difficulty or postural threat. First, the associated change in 1a afferent firing rate with increased gamma activity (Rossi-Durand 2002; Ribot-Ciscar et al. 2000) would saturate the synapse due to depletion of neurotransmitter at the synapse (Hultborn et al. 1996; Pinniger et al. 2001) and thus decrease the likelihood that smaller stretch inputs could trigger potentially destabilizing stretch reflexes. Secondly, increased gamma activation would increase the sensitivity of the muscle spindles to stretch information and thus enhance the sensory information received by the cortex via ascending pathways (Llewellyn et al. 1990). At this point, independent control of alpha-gamma activation is still controversial (Gandevia and Burke 1985) and requires further investigation to determine whether this mechanism can provide any functional changes in muscles that are non-passive or posturally-stabilizing.

Summary

The study has demonstrated that amplitude and, in some cases the timing, of muscular and kinematic responses to an unexpected balance perturbation are susceptible to the influence of increased postural anxiety. These findings support the hypothesis that neural mechanisms responsible for anxiety-related behaviour may contribute significantly to the patho-physiology of balance impairments and falls in older adults and patients with balance disorders such as Parkinson’s disease, vestibular loss and proprioceptive deficits. Further research is needed to better understand the circuitry that links neural regions and pathways associated with anxiety and fear and balance control, and how these mechanisms may be influenced by age and disease. This information will be critical for clinicians and researchers to discriminate between the physiological and psychological
Influence of postural anxiety on postural reactions

origins of a balance disorder and to develop new training and intervention programs aimed at improving both the physical and cognitive components of a balance deficit.

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Influence of postural anxiety on postural reactions


Influence of postural anxiety on postural reactions


Influence of postural anxiety on postural reactions

Table 1. *Onset latencies for left deltoid EMG responses to multi-directional rotational perturbations for low and high threat conditions*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Forward left (315°)</th>
<th>Forward (0°)</th>
<th>Forward right (45°)</th>
<th>Back left (225°)</th>
<th>Backward (180°)</th>
<th>Back right (135°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Threat</td>
<td>106.79 ± 5.62</td>
<td>95.64 ± 4.08</td>
<td>116.65 ± 9.81</td>
<td>103.67 ± 5.87</td>
<td>118.29 ± 4.73</td>
<td>117.36 ± 5.95</td>
</tr>
<tr>
<td>High Threat</td>
<td>90.83 ± 4.09</td>
<td>89.14 ± 3.33</td>
<td>92.21 ± 3.56</td>
<td>85.66 ± 4.97</td>
<td>102.76 ± 4.84</td>
<td>91.50 ± 4.53</td>
</tr>
</tbody>
</table>

Values are means ± SE, n=9. See methods for calculations.
FIGURE LEGENDS

Figure 1 – Average traces for linear displacement for the total body COM (left panel) and angular displacements of the lower leg, pelvis and trunk (right panel) measured from a single subject (mean of 6 randomized trials) for backward left (225 deg) perturbations. The thick black vertical line at 0 ms represents the onset of support surface rotation. A positive deflection of the traces represents backward or upward displacements, while a negative deflection represents forward or left displacements.

Figure 2 – Polar plots for population average of peak absolute centre of mass displacement are displayed for the anterior-posterior (left panel), medial-lateral (middle panel) and vertical (right panel) directions. Thick black lines (with white-filled area) represent the high postural threat condition; thin black lines (with grey filled area) represent the low postural threat condition. Each radial line represents one of six different directions (0, 45, 135, 180, 225, 315 deg) in clockwise notation. Please note that no direct measurements were recorded for 90 and 270 deg. Therefore, dashed lines may not reflect true response amplitudes for these directions. For each direction mean values are plotted along each radial axis with magnitude represented by the distance to the centre.

Figure 3 – Population averages for muscle responses from 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. Each of the traces shows the average for 9 participants to 6 randomized repetitions of the stimulus
Influence of postural anxiety on postural reactions

direction. The thick black vertical line at 0 ms represents the onset of support surface rotation.

Figure 4 – Population averages for muscle responses from 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 2.

Figure 5 - Polar plots for population average EMG areas for balance correcting response period between 120-220 ms are displayed for antagonist muscle pairs. Thick black lines (with white-filled area) represent the high postural threat condition, whereas thin black lines (with 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. Please note that no direct measurements were recorded for 90 and 270 deg. Therefore, dashed lines may not reflect true response amplitudes for these directions. Accurate measures for tibialis anterior, soleus, gluteus medius and paraspinal muscles for 90 and 270 deg can be found in previous publications (Carpenter et al. 1999, Bloem et al 2001). For each direction mean values are plotted along each radial axis with magnitude represented by the distance to the centre.

Figure 6 A) Average left deltoid muscle responses from a single subject (mean of 6 randomized trials) for forward left (315 deg) and backward left (225 deg) perturbations. Black shaded area represents periods that activity is greater in the high threat compared to
low threat condition. The thick black vertical line at 0 ms represents the onset of support surface rotation. B) Polar plot for population average of EMG areas for balance correcting response period (120-220 ms) for left deltoid muscle. Thick black lines (with white-filled area) represent the high postural threat condition, whereas thin black lines (with grey filled area) represent the low postural threat condition. Please note that no direct measurements were recorded for 90 and 270 deg. Therefore, dashed lines may not reflect true response amplitudes for these directions. For each direction mean values are plotted along each radial axis with magnitude represented by the distance to the centre.
Influence of postural anxiety on postural reactions

Figure 1
Influence of postural anxiety on postural reactions

Absolute Centre of Mass Displacement

Anterior-Posterior (toes down)

Medial-Lateral (toes down)

Vertical (toes down)

Displacement (cm)

Low Threat

High Threat

Figure 2
Influence of postural anxiety on postural reactions

Figure 4
Influence of postural anxiety on postural reactions

Figure 6

A  

Left Deltoid

Forward Left (315°)

Backward Left (225°)

mV

Low Threat
High Threat

Time (s)

B  

Left Deltoid

(toes down)

μV

Low Threat
High Threat

θ

0 45 90 135 180