Influence of Postural Anxiety on Postural Reactions to Multi-Directional Surface Rotations

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Carpenter, M. G., J. S. Frank, A. L. Adkin, A. Paton, and J.H.J. Allum. Influence of postural anxiety on postural reactions to multidirectional surface rotations. J Neurophysiol 92: 3255–3265, 2004. First published August 4, 2004; doi:10.1152/jn.01139.2003. 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 yr, range: 22–27 yr) were required to recover from unexpected rotations of the support surface (7.5° 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. Electromyographic data from 12 different postural leg, hip, and trunk muscles was collected simultaneously. Full body kinematic data were also used to determine total body center of mass (COM) and segment displacements. Four distinct changes were observed with increased postural anxiety: increased amplitude in balance-correcting responses (120–220 ms) in all leg, trunk, and arm muscles; decreased onset latency of deltoid responses; reduced magnitude of COM displacement; and 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 neuromuscular control mechanisms and thus may contribute significantly to the pathophysiology of balance deficits associated with aging or neurological disease.

INTRODUCTION

Postural anxiety has been related to balance and gait impairments (Hill et al. 1996; Maki et al. 1991; Meyers et al. 1996; Tinetti et al. 1994; Vellas et al. 1997) and increased risk of falls (Cumming 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 (Adkin et al. 2000; Carpenter et al. 1999d, 2001b; 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 ~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 (Allum et al. 1994; Carpenter et al. 1999a; Diener et al. 1983) 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 brain stem 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 Whitehall 1993; Okada et al. 2001), there have been no studies to date that have manipulated anxiety to determine how it may influence stretch reflexes and 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. First the knowledge may be used to gain a better understanding of the connections between neural areas controlling emotional behavior and those involved in balance control. Second, 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) and patients with Parkinson’s disease (Bloem et al. 2001), vestibular loss, or propioceptive deficit (Yardley and Hallam 1996).

METHODS

Subjects

Ten university students [6 male; age = 25.5 ± 5.3 (SD) yr] 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 orthopedic disorder as verified by self report. Prior to the experiment, anthropometric measures were recorded, including
height (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 naive 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 (≤180°) relative to the position of the participant to achieve multi-directional perturbations. This method is distinctly different from 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 center 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 tightly 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-min 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 ~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 that 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° and velocity of 50°/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 six different directions randomly presented six times. Directions were separated by 45° 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 electromyographic (EMG) data commenced 2 s prior to the onset of the perturbation and were collected for 5 s. EMG recordings were sampled at 1,024 Hz. EMGs were 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 encumbrance was placed on the participant. Kinematic data were recorded at 64 Hz using the OPTOTRAK (Northern Digital Canada, 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, and temple and one at the center of zyphoid). Three additional ireds were placed at the front corners and center 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 avoid a fall, maintain concentration, overcome worry, and 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 that 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 three different elements of anxiety: somatic, worry, and concentration. Participants were required to score each item using a nine-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 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 coefficients 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 300 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 predetermined time intervals associated with early stretch (40–100 ms), medium-latency responses (80–120 ms), balancing 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 >50 ms) over a threshold of 2 SD 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 were 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 that 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). Two-dimensional angular displacements of the lower leg were calculated from ankle and knee markers of the left leg. Three-dimensional 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 $2 \times 6$ (threat by direction) within-subject ANOVA. Significant main and interaction effects were analyzed using preplanned comparisons using t-test 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 with the low-threat condition. After performance of the balance task, the participants’ perceptions of anxiety were increased by an average of 30% in the high-threat compared with 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 with low-threat condition [$F(1,9) = 11.12, P < 0.02$].

**Total body COM**

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 backward and to the left (Fig. 1). Similarly, forward left perturbations caused the total body COM to be displaced forward and to the left.

The magnitude of COM displacement was influenced by increased postural threat. As shown in Figs. 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 with the low-threat condition [$F(5,45) = 2.58, P = 0.03$]. There was a trend for smaller peak COM displacements in the A-P direction in high-threat compared with 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 with low-threat conditions (Figs. 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 backward, 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 (Fig. 1). Similar but opposite segment displacements were observed for forward perturbations, with the lower leg rotating forward, and pelvis and trunk segments displaced backward.

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 with the low-threat condition. The peak forward pitch angle of the pelvis and trunk were also reduced in the high-threat compared with low-threat condition (Fig. 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 of 46.4 ± 3.05 (SE) ms (Fig. 3) followed by large balance-correcting re-
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 latencies of 88.0 ± 2.33 and 85.3 ± 2.87 ms, respectively (Fig. 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 (Figs. 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 × 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
type of muscle. The onset and amplitude of initial stretch
reflexes in soleus in backward left perturbations was similar in
the high- compared with low-threat condition (onset latency =
$42.3 \pm 2.09$ ms). Likewise, for forward left conditions, there
were no significant differences in amplitude or onset of initial
stretch reflexes in tibialis anterior ($83.7 \pm 2.67$ ms) or rectus
femoris ($81.6 \pm 2.91$ 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 femo-
ris, and paraspinals (Fig. 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 direc-
tion (Fig. 4).

Illustrating EMG areas on a polar plot, with mean ampli-
tudes plotted along axes that correspond to different perturba-
tion directions, allows for an easy visualization of the magni-
tude and directional sensitivity of different postural muscles.
The polar plots in Fig. 5 depict the magnitude and directional
sensitivity of EMG areas calculated over the balance-correct-
ing period between $120$ and $220$ ms. ANOVA results revealed
a significant main effect of threat on amplitude of balance-
correcting responses for all muscles analyzed. As observed in
Fig. 5, participants had larger responses between $120$ and $220$
ms in the high- compared with 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 and 340 or 350 and 700 ms.

**Shoulder muscle responses**

Backward left perturbations elicited distinct bursts of muscle activity in both left and right deltoid muscles 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 (Fig. 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 perturbations; however, the overall amplitude of the response was reduced in forward compared with backward perturbations (Fig. 6A).

In the high-threat condition, both the onset latency and amplitude of balance-correcting activity in deltoids was signif-
FIG. 5. Polar plots for population average electromyographic (EMG) areas for balance-correcting response period between 120 and 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 gray filled area) represent low postural threat condition. Each radial line represents 1 of 6 different directions (0, 45, 135, 180, 225, and 315°) in clockwise notation. Please note that no direct measurements were recorded for 90 and 270°. Therefore dashed lines may not reflect true response amplitudes for these directions. Accurate measures for tibialis anterior, soleus, qluteus medius, and paraspinal muscles for 90 and 270° can be found in previous publications (Bloem et al. 2001; Carpenter et al. 1999a). For each direction, mean values are plotted along each radial axis with magnitude represented by the distance to the center.

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.
been shown to significantly increase the stretch reflex and decrease balance-correcting responses to postural perturbations (Allum and Pfaltz 1985; Diener et al. 1983; Horak and Moore 1993; Schieppati et al. 1995) 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 (Allum and Mauritz 1984; Bedingham and Tatton 1984; Bloem et al. 1993).

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 prestimulus posture may not be advantageous because 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 prestimulus posture between the high- and low-threat conditions.

Even with prestimulus posture controlled, some findings from the present study were consistent with that of previous studies. The reduced amplitude of peak A-P COM displacement in high threat conditions (Figs. 1 and 2) corresponds with reduced COM displacements observed in subjects that were unexpectedly pushed from behind in high- compared with low-threat conditions (Brown and Frank 1997). Preserved normal onset latencies in leg muscle responses with increased anxiety (Figs. 3 and 4) was also observed in fearful subjects reacting to unexpected surface translations (Bolmont et al. 2002; Okada et al. 2001). 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; this corresponds to increased amplitudes of tibialis anterior and soleus muscle activity observed between 120 and 220 ms with increased postural anxiety (Fig. 5).

In addition to the preceding findings, the current study has uncovered a number of new aspects of balance control that 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 and 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 (Figs. 3–6). Combined with the fact that the directional sensitivity of muscular responses are generally preserved with increased postural anxiety (Figs. 5 and 6), our findings 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 (Carlsen et al.
2003; Valls Sole et al. 1999). One distinction between the
present study and previously observed startle responses is that
startle responses are typically of similar amplitude and dura-
tion, 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 with 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 in-
creases 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 with low-threat condition (Fig. 2). Adoption of postural
strategies to maintain a tighter control of the COM and de-
crease angular displacements of the limbs during conditions of
increased anxiety have been observed previously during quiet
standing (Carpenter et al. 2001b), rising to the toes (Adkin et
al. 2002), walking (Brown et al. 2002), and unexpected per-
turbations 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, par-
icularly 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 per-
ceived 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 (Adkin et al. 2002; Carpenter
et al. 1999c). This converging evidence provides convincing
support that our manipulation of height has an influence on
higher brain centers associated with anxiety or emotional
behavior.

Previous studies have demonstrated that changes in trait
anxiety or mood states can influence postural control (Bolmont
et al. 2002; Wada et al. 2001). 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 behav-
iors is via the vestibular system. Balaban (2002) has proposed
three potential neural networks that could potentially modulate
balance reactions via the vestibular system: vestibulo-parabra-
chial 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
(2002) proposed that the vestibulo-parabrachial nucleus net-
work 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 coeruleus
and subceroeleus, particularly to areas of the superior and
lateral vestibular nucleus which control postural and eye move-
ments. Since locus coeruleus 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 serotonergic release is
associated with the degree of arousal (Rueer et al. 1997). Serotone-
ergic 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 ac-
tions of serotonin in the vestibular nuclei 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. Pre-
vious studies have demonstrated that reduced vestibular infor-
mation, in patients with bilateral compensated and acute uni-
lateral 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 and 220 ms (Allum and Pfaltz 1985;
Allum et al. 1994; Carpenter et al. 1999b, 2001a; Keshner et al.
1987; 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 that 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 demon-
strated 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 gan-
glia 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, Parkinson’s disease (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.

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 fibers 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 provide a dual benefit in conditions of increased difficulty or postural threat. First, the associated change in 1a afferent firing rate with increased gamma activity (Ribot-Ciscar et al. 2000; Rossi-Durand 2002) 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. Second, 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 nonpassive 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 behavior may contribute significantly to the pathophysiology 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 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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