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RESEARCH ARTICLE

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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 (BVL) in humans on postural responses to multidirectional 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 multidirectional rotational perturbations of the support surface. Fourteen normal healthy adults and five BVL patients stood on a dual axis rotating platform which rotated 7.5° at 50°/s through eight different directions of pitch and roll combinations separated by 45°. 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 muscles. Only SOL muscle activity demonstrated a 38- to 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 and 220 ms was, with one exception, severely reduced in BVL patients for eyes open and eyes closed conditions. SOL 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 and 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 and 260 ms and increased torque between 280 and 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 and 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 from 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 spatiotemporal 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.

Keywords Balance control · Vestibulo-spinal system · Proprioceptive reflexes · Vestibular loss

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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 1982; 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 (forward or backward) 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; Horak et al. 1990; Allum et al. 1994; Allum and Honegger 1998; 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 quadrupedal animals (Macpherson 1988a, b, 1994; Rushmer et al. 1988) and humans (Moore et al. 1988; Maki et al. 1994; Henry et al. 1998a, b; Carpenter et al. 1999), as well as perturbations delivered to the trunk and pelvis (Gilles et al. 1999; Rietdyk 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 multidirectional 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 (Nashner et al. 1982; Horak et al. 1990; Allum et al. 1994). 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 labyrinthectomized cats during sudden unexpected multidirectional translations. Since the biomechanical constraints imposed by surface perturbations are different for quadrupeds 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 sen-

sory input for adequate directional information underlying balance commands (Allum and Honegger 1998). Although such a switching mechanism has been proposed (Nashner et al. 1982; Horak et al. 1994), 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 multidirectional rotations of the support surface. The first goal of the present study was to determine whether the current understanding of the effects of bilateral peripheral vestibular loss (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.

Materials and methods

This study examined the effect of multidirectional rotations of the support surface on muscular and biomechanical responses in normal healthy young adults and subjects with BVL acquired idiopathically as adults at least 2 years prior to these experiments. Fourteen normal controls (seven male, seven female; mean age=22.71 years, SD 2.40 years; height=1.73 m, SD 0.08 m; weight=69.5 kg, SD 11.7 kg) and five BVL patients (four male, one female; mean age=39.4 years, SD 6.18 years; height=1.72 m, SD 0.07 m; weight=74.2 kg, 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 orthopedic injuries as verified by extensive questioning. Normal vestibular function was further verified using Romberg and Unterberger stance tests. BVL was characterized by no response (slow phase velocity less than 2°/s) to bithermal caloric irrigation (100 cc water for 30 s) of each ear and by horizontal vestibulo-ocular reflex responses to whole body rotations of 80°/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 d.c. 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 eight different directions in the pitch and roll planes. A clockwise increasing notation, as viewed from above, was used to specify rotation direction. The 0° rotation direction represented a pure 'toes-down' tilt of the platform, conversely, 180° direction represented a pure 'toes-up' rotation. Pure roll movements were assigned angles of 90° to the right and 270° to the left. Combinations of pitch and roll rotations were used to provide four other directions, each separated by 45°. Platform rotations had a constant amplitude of 7.5° and angular velocity of 50°/s. One series of 44 randomly presented directional stimuli was always performed first with eyes open. Following a 5- to 10-min rest period to minimize any confounding effects due to order and/or fatigue, a second series of 44 random stimuli was 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 eight perturbation directions were presented randomly five or six times.

Each perturbation was preceded by a random 5- to 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 online 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- to 20-s interstimulus delay was initiated automatically once the platform had returned to its original prestimulus 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 electromyographic (EMG) recordings

All biomechanical and 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, 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°/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 occurring across different signals, all signals were digitally low-pass filtered offline at 25 Hz using a zero phase-shift tenth-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 (QUADS), and paraspinal (PARAS) muscles. Electrodes were mounted on the PARAS at the L1–2 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 bandpass analog filtered between 60 and 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 (five or six 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 predetermined 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 ms) responses (Carpenter et al. 1999). Response latencies for balance correcting responses were determined semiautomatically based on the following criterion: later than 90 ms, burst longer than 40 ms, and a continuous amplitude of at least 2 SD 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 Figs. 2, 3, 8, 9). Average trunk angular velocity was calculated over 60 ms during time intervals between 160 and 220, 240 and 300, and 470 and 530 ms. Torque changes were calculated between 160 and 260, and 280 and 380 ms. All EMG areas, average trunk velocities, and ankle torque changes were analyzed in a 2×8 (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 coordination 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.

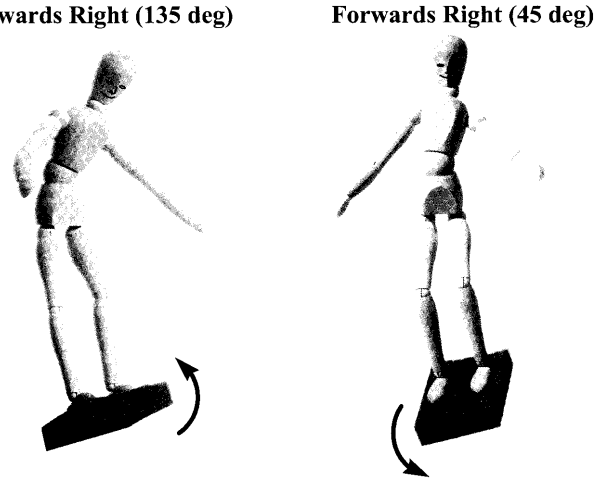

Fig. 1 Graphical schematic representation of stimulus-induced movements of the head, trunk, and leg segments in response to unexpected support surface rotations directed backward to the right (135°) and forwards to the right (45°)

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°) and forward to the right (45°). 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 multilink fashion (Fig. 1 *left*). The uphill leg (left) is driven upward by the elevated side of the platform while the lower leg falls simultaneously backward 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 forward at 50 ms. The initial roll is rapid, but the pitch is only rapid after 100 ms (see Fig. 7). The uphill leg buckles during the

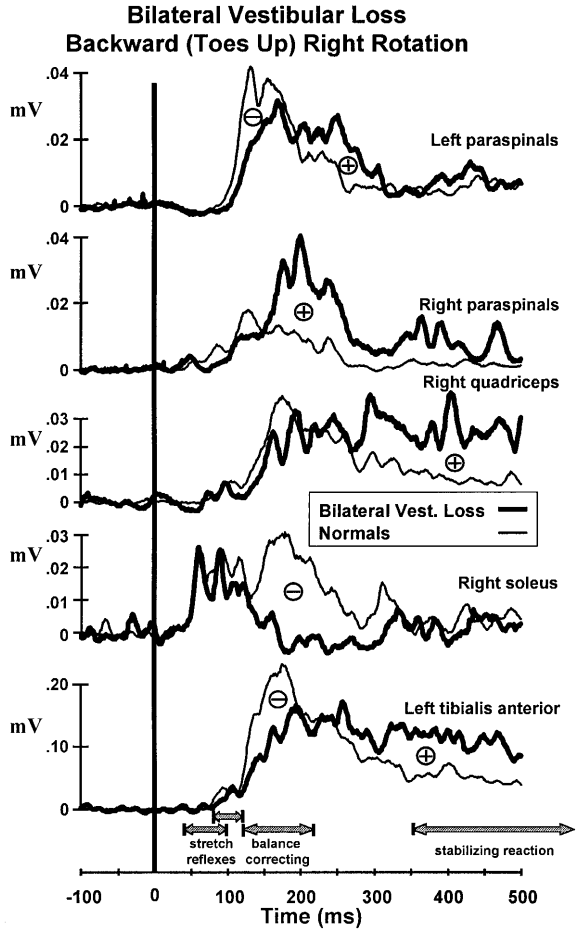

Fig. 2 Average electromyographic (EMG) traces from 14 normal subjects (*thin lines*) and five bilateral vestibular loss patients (*thick lines*), measured during eyes closed trials in response to unexpected surface rotations directed backward to the right (135°). The *black vertical line* at 0 ms represents the onset of ankle rotation. *Grey 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. Note: for vestibular loss patients: (1) normal onset of stretch, unloading, and balance correcting (120–220 ms) responses, (2) decreased amplitude of balance correcting responses in left tibialis anterior, right soleus, and left paraspinal, (3) increased amplitude of right paraspinal balance correcting activity, and (4) excessive stabilizing activity (350–700 ms) in right paraspinal muscle

rapid trunk roll, flexing at the knee and ankle joint (see Fig. 1 *left*). As illustrated in Fig. 2, the initial dorsiflexion of the ankles causes a prominent stretch reflex in SOL muscles of normal subjects at a latency of 54 ms. Shortly thereafter, small amplitude stretch reflexes in the right 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 TA, right QUADS, and left PARAS demonstrated dominant balance correcting responses to counter rotation of the ankle, knee, and hip, respectively (Fig. 2). During the stabilizing period elevated levels of muscle activity in right TA, SOL, and QUADS 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 backward to the right. As observed in Fig. 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 QUADS, 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 (Fig. 2). However, differences in the magnitude of the balance correcting response were observed with BVL subjects. For example, BVL subjects generated only negligible balance correcting activity in SOL after the initial stretch reflex compared to normals (Fig. 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 Fig. 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 (Fig. 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 (Fig. 3).

Vestibular loss subjects did not differ from normals in the onset of stretch reflexes or unloading reflexes (Fig. 3). BVL subjects exhibited distinct pattern differ-

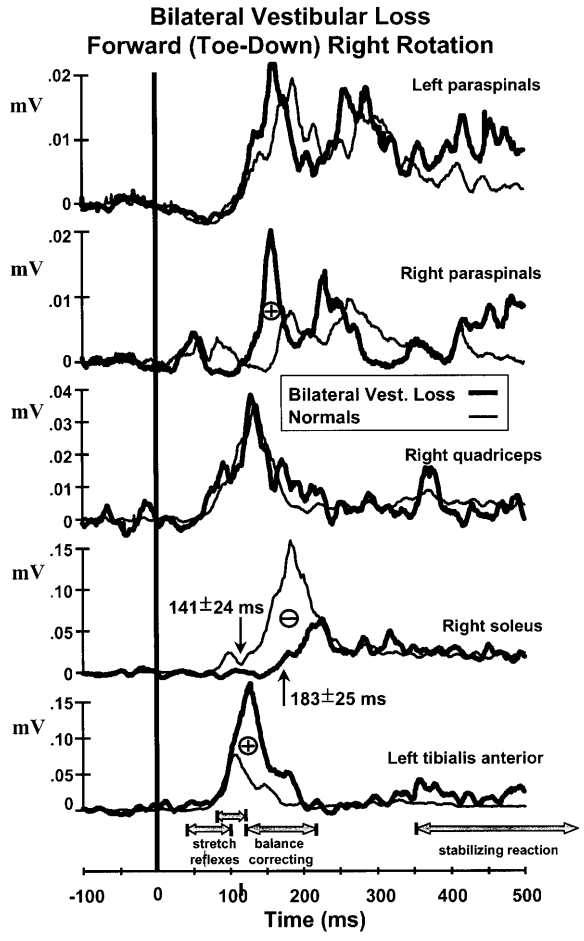

Fig. 3 Average EMG traces from 14 normal subjects (*thin line*) and five bilateral vestibular loss patients (*thick line*) measured during eyes closed trials in response to unexpected surface rotations directed forward to the right (45°). The *black vertical line* at 0 ms represents the onset of ankle 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*. *Grey 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) responses. Note: for vestibular loss patients: (1) delayed onset and decreased amplitude of balance correcting response in right soleus, (2) increased amplitude of balance correcting activity in left tibialis anterior and right paraspinals, and (3) increased stabilizing activity in left and right paraspinals. Note that the scales of muscle activity are not the same as those in Fig. 2

ences 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 SOL 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°, 140.6±23.7 ms for 45°, and 140.9±27.2 ms for 315° 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° ($P<0.001$), 183.6±25.1 ms for 45° ($P<0.013$), and 179.1±18.5 ms for 315° ($P<0.022$). Similar differences were observed in the left SOL. 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 (Fig. 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 occurring between 40 and 120 ms)

There were no significant effects of BVL on the amplitude of stretch reflexes over the period we analyzed (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 Fig. 4 and the responses in Figs. 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° and 135° (clockwise notation) with a maximum activity vector at 338° 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° with maximum activity vectors oriented close to 180° 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° 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° 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 (Fig. 4).

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

The amplitude of balance correcting activity measured between 120 and 200 ms was also significantly influenced by the direction of the perturbation with each muscle having clearly defined ranges of activation. Balance

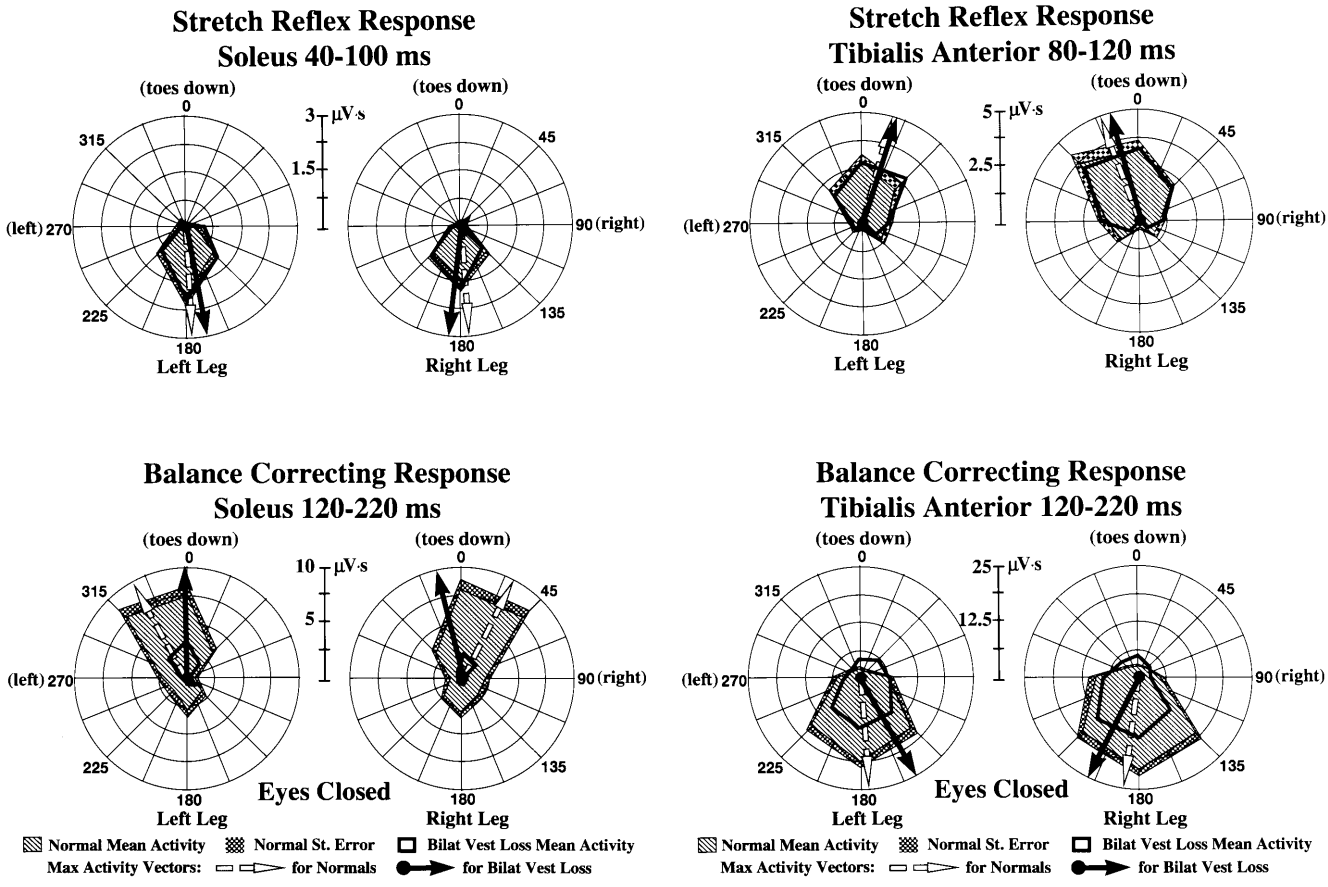

Fig. 4 Polar plots for tibialis anterior (*right panel*) and soleus (*left panel*) muscles under eyes closed conditions averaged over three distinct time intervals representative of stretch (40–100 ms and 80–120 ms) and balance correcting (120–220 ms) responses. Each *radial line* or spoke represents 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

correcting activity in SOL, TA, and QUADS muscles was oriented 180° from directions that elicited stretch reflexes in both normal and BVL subjects (Fig. 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 (Figs. 4, 5). The amplitudes for five directions and their standard deviations have been plotted on horizontal bar representations in the *upper* and *lower parts* of Fig. 5 for TA and SOL, respectively, to highlight the significant interaction between BVL and perturbation direction for these muscles. As observed in Fig. 5, for pure toe-up rotation (180°), 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 backward to the right (135°). Likewise, the same trend of reduced amplitude response in BVL subjects was observed for left TA when the perturbation was backward to the left (Fig. 4). In contrast, when perturbations are composed of pure rotations to the right (90°) and forward roll right (45°), 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° toes-down perturbations. BVL patients showed significantly larger responses between 120 and 220 ms compared to normals, in both left and right TA ($P<0.05$) for the 0° direction (Fig. 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. SOL balance correcting activity was also influenced by a significant interaction between vestibular loss and perturbation direction. As observed in Fig. 5, the magnitude of this interaction was different for right and left muscles for rightward perturbations. For the

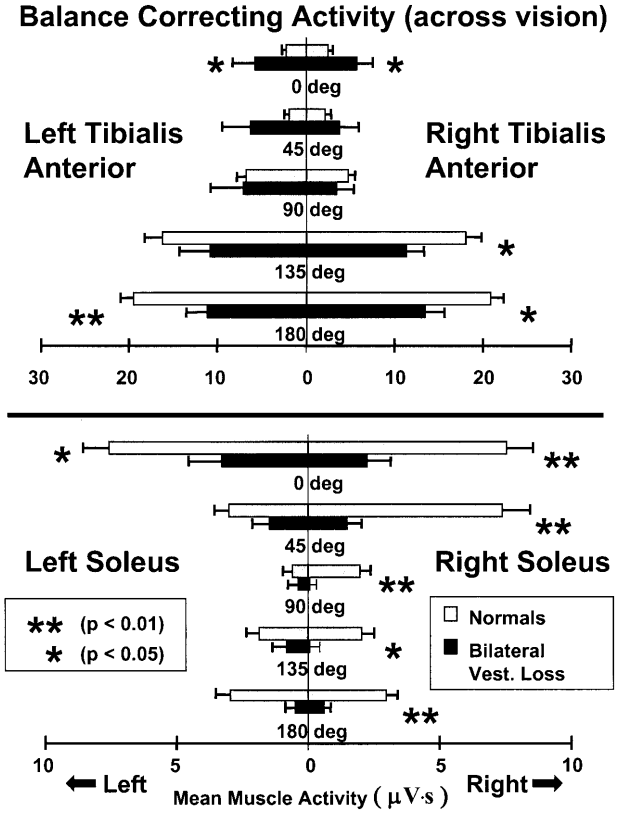

Fig. 5 Mean amplitudes and standard error bars (eyes open and eyes closed combined) for balance correcting response (120–220 ms) in *upper half* left and right tibialis anterior muscles and *lower half* left and right soleus muscles. Normal amplitudes are shown as *white bars* and bilateral vestibular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

right SOL (downhill leg), vestibular loss patients had significantly lower balance correcting activity for all directions (P<0.05). Less significant differences were observed in the left SOL muscle on the uphill leg (Figs. 4, 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 Fig. 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°), vestibular loss patients also had significantly less activity in the right SOL compared to normals (P<0.05). Notably, in normals this activity is much smaller than for toe-down perturbations. For the 0° 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 Fig. 4 with respect to the directions of normal subjects were not significant.

Stabilizing reactions (responses occurring between 350 and 700 ms)

Consistent with both stretch and balance correcting responses, stabilizing reactions were significantly influenced by the direction of the perturbation. As observed in Fig. 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. SOL 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] (Fig. 6 *upper part*). 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 (Fig. 6) and QUADS, as perturbation direction moved from forward right to backward right directions, BVL subjects had greater stabilizing amplitudes compared to normals (Fig. 6) and these responses were larger for eyes closed compared to eyes open conditions. However, for the pure toes-up (180°) 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 (Fig. 6 *upper right*). The net result of the changed amplitudes of stabilizing reactions and foregoing balance corrections in BVL subjects is shown in the *lower half* of Fig. 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.

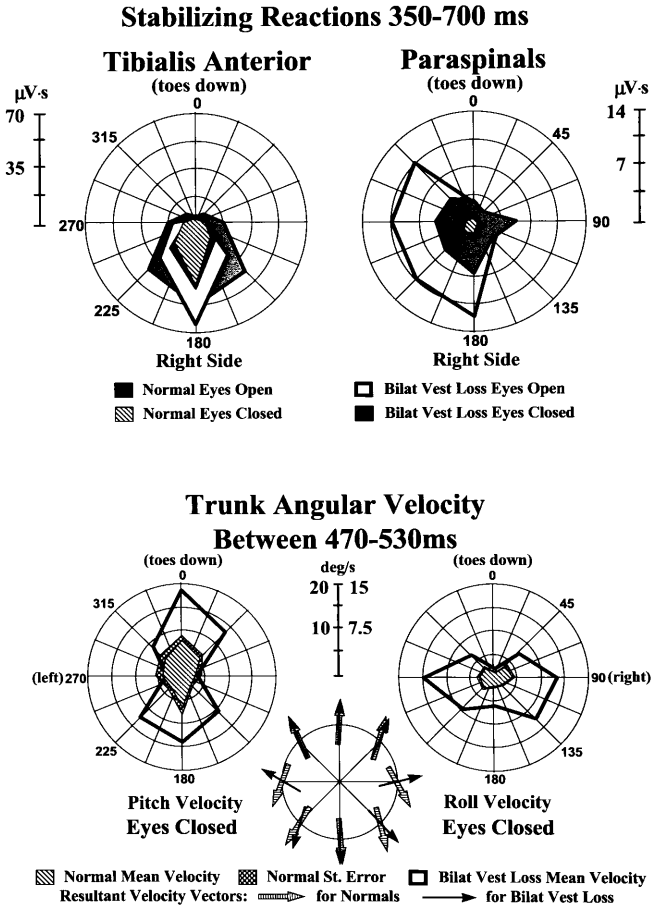

Fig. 6 *Upper half* shows polar plots of average EMG activity during the period between 350 and 700 ms for normals with eyes open (*filled, black*) and eyes closed (*filled with diagonal hatch lines*), and bilateral vestibular loss patients with eyes open (*filled white*) and eyes closed (*filled gray, black border*) measured in right tibialis anterior and right paraspinals muscles. The response amplitude represented by each of the *concentric circles* in the plot is scaled according to the *vertical scale* beside each set of plots. *Lower half* shows mean absolute values for average trunk angular velocity calculated between 470 and 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*). 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

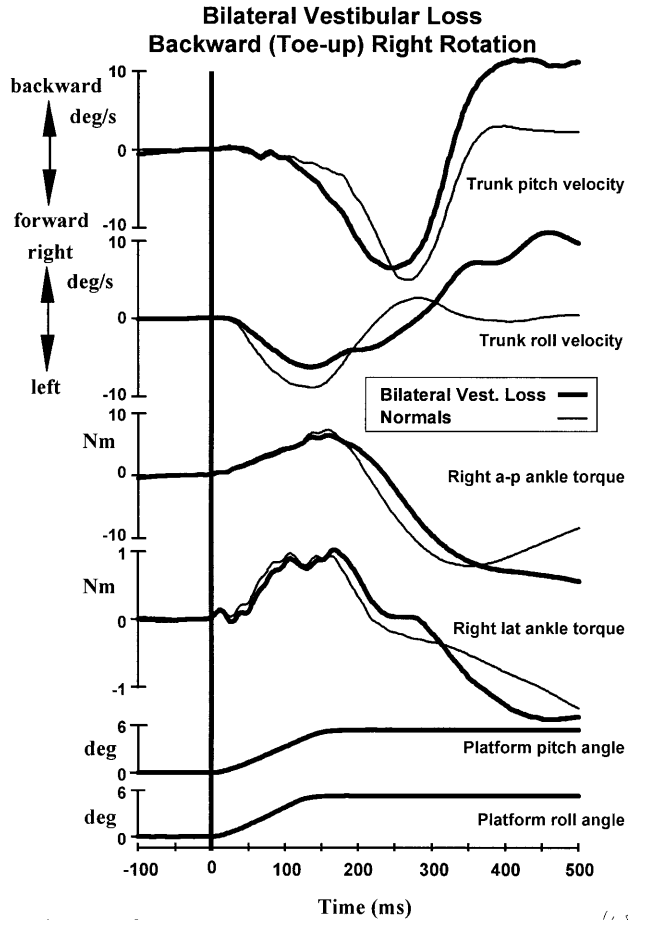

Fig. 7 Average biomechanical traces for normals (*thin lines*) and bilateral vestibular loss patients (*thick lines*), measured during eyes closed trials in response to unexpected surface rotations directed backward to the right (135°). Refer to Fig. 3 for details. *a-p* Anterior/posterior

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 (Fig. 7). In forward perturbations, normal ankle torque is initially plantarflexing, followed by rapid dorsiflexion beginning at 120 ms and peaking at ca 375 ms (Fig. 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 Fig. 9, where the ankle torque change between two different time periods is plotted for each perturbation direction. During the early period between 160 and 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 (Fig. 9 *upper right*). A significant interaction between group and direction was also observed between 280 and 380 ms for A-P [$F(7,119)=4.25$, $P<0.0003$] and M-L [$F(7,119)=2.13$,

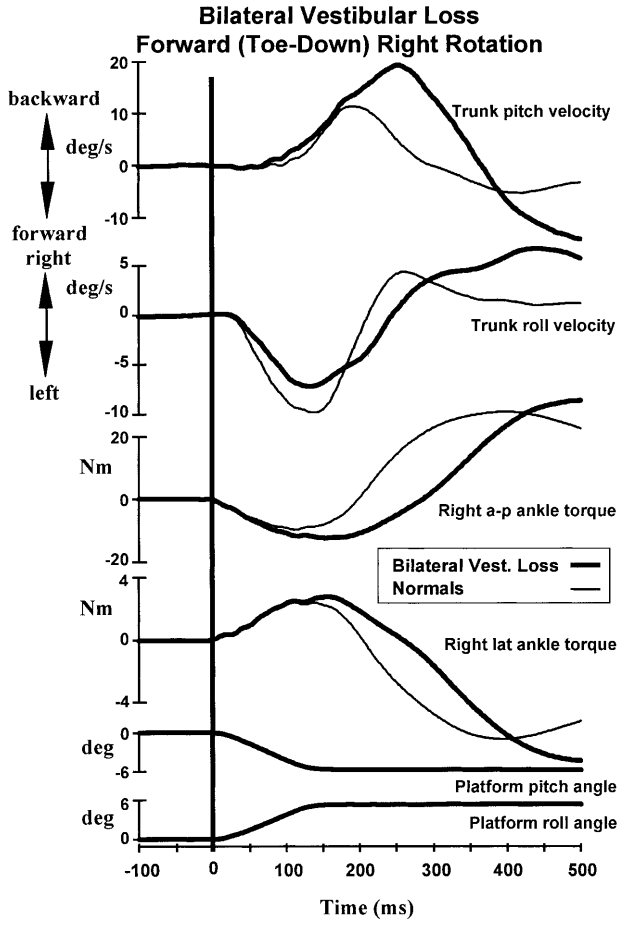

Fig. 8 Average biomechanical traces for normals (*thin line*) and bilateral vestibular loss patients (*thick line*), measured during eyes closed trials in response to unexpected surface rotations directed forward to the right (135°). Refer to Fig. 3 for details

$P<0.0457$] 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 (Fig. 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 Fig. 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 (Figs. 1, 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 (Figs. 7, 8). Platform rotation to the right caused trunk roll to the left with peak velocities reaching 10°/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 (Figs. 7, 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 (Figs. 6, 7, 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 (Fig. 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 Figs. 6, 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 a large overcorrecting ‘stabilizing’ response in BVL patients (Fig. 6) also in the direction of the initial platform motion, however without a falling tendency. As shown in Fig. 9, differences between BVL and normals extended to all perturbation directions. During the period between 160 and 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 (Fig. 9 *lower left*). During the later period between 240 and 300 ms, BVL patients had significantly larger [$F(1,17)=8.82$, $P<0.0086$] pitch velocities across all perturbation directions (Fig. 9 *lower right*). Significant group by direction interaction effects were observed during the later period between 470 and 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 (Fig. 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 and 220 ms were oriented opposite to the direction of platform movement for both normal and BVL subjects (Fig. 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 Fig. 9. However, during this latter period, the

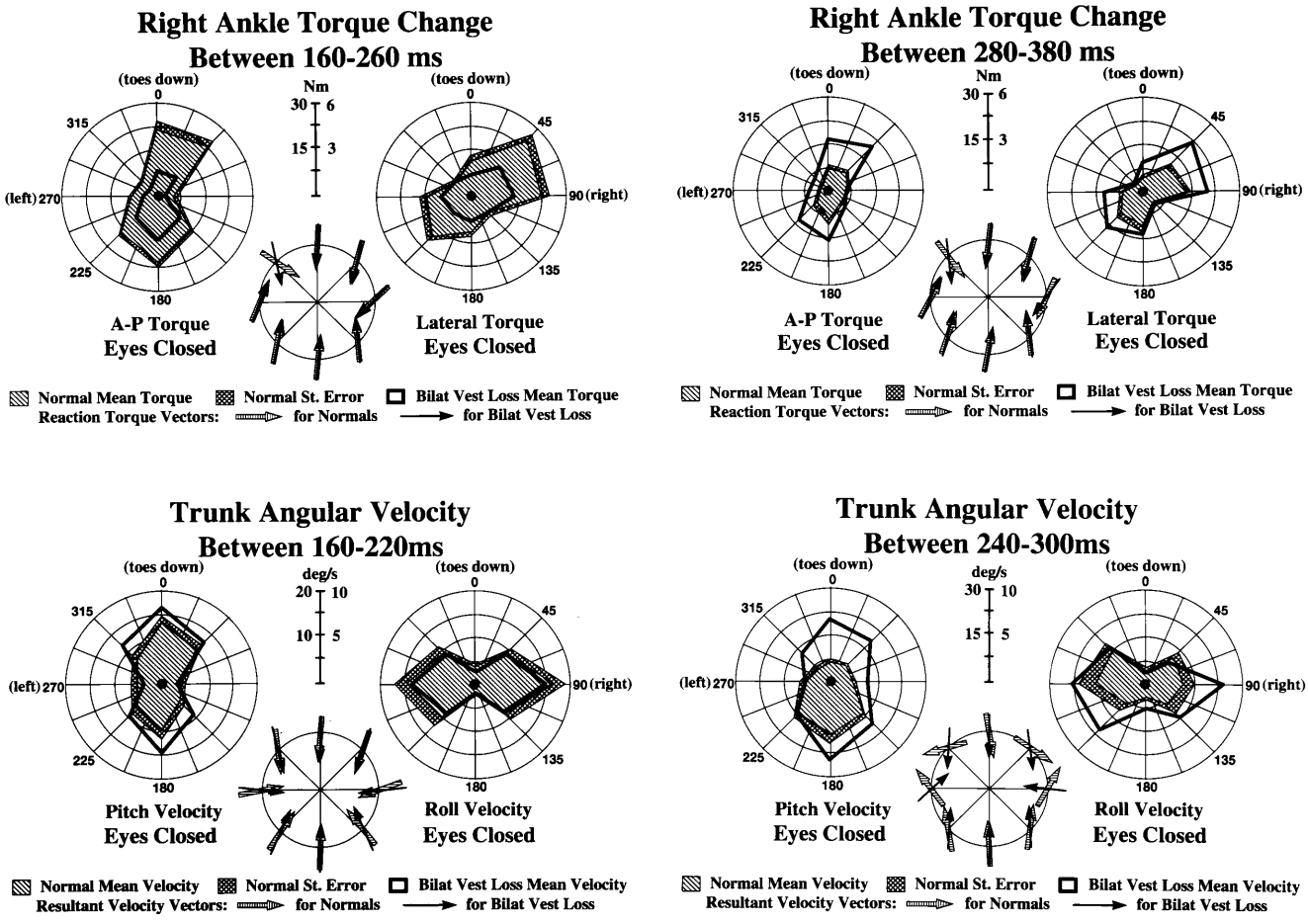

Fig. 9 *Upper half* shows mean absolute values of right ankle torque change calculated on the *left* between 160 and 260 ms and on the *right* between 280 and 380 ms. The larger polar plots to the *left* and *right* of each panel represent A-P torque and medial/lateral torque, respectively. The *lower 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* shows mean absolute values for average trunk angular velocity calculated on the *left* between 160 and 220 ms and on the *right* between 240 and 300 ms. Refer to Fig. 6 for details

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 (Fig. 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 uniplanar 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 semicircular canals, it was hypothesized that a multidirectional posturographic paradigm would provide new insights about the focus of vestibular and proprioceptive contri-

butions to balance control using the patient model of BVL. Furthermore, the different dynamics of some central vestibular neurons for roll and pitch perturbations (Angelaki and Dickman 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 semicircular 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 centers 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 BVL, the behavioral 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 (Horak et al. 1990; Allum et al. 1994; Runge et al. 1998), 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 and 220 ms in TA, SOL, and QUADS 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 PARAS 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 persists 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 unidirectional 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 (Figs. 2, 3, 5). As observed in Figs. 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 SOL, 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 (Figs. 4, 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 unidirectional perturbations, BVL patients demonstrated excessive muscular activity during the stabilizing period between 350 and 700 ms in TA, QUADS, and PARAS, for pitch and off-pitch perturbations (Figs. 2, 3, 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 and 530 ms, when normal subjects experience small residual trunk motion (Fig. 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 labyrinthectomized cats following multidirectional 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 (Figs. 3, 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 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 PARAS and left vastus lateralis (QUADS) 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 Fig. 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 mus-

cles to be oriented at approximately 60° and 300°, respectively, whereas in the present study, maximal balance correcting activity in right TA and SOL was more pitch oriented at 186° and 35°, respectively (see Fig. 4), very similar to the direction of balance correcting torque for the same foot (Fig. 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° to balance correcting activity in the same muscle (compare *upper* and *lower* plots in *left side* of Fig. 4). In contrast, PARAS balance correcting activity is oriented 90° 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 Forssberg 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 BVL (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 SOL 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 (Melvill Jones and Watt 1971; Greenwood and Hopkins 1976; 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 and 120 ms, which is confined to lower limb extensors in triceps surae muscles in falling humans (Greenwood and Hopkins 1976), baboons (Lacour et al. 1978, 1983), and cats (Watt 1976). The amplitude of the second burst has been shown to be modified by BVL (Lacour et al. 1978) as well as experience. The response amplitude decreases with multiple exposure (Lacour et al. 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 semicircular 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. submitted for publication). 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. submitted for publication) 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 contralateral *"uphill"* leg (generated by several muscles including TA) and extension of the ipsilateral *"downhill"* stance-bearing leg to platform rotation (assisted by SOL activity) are driven by vestibulospinal 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 Melvill 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 facilitatory input from the later-

al 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 behavior 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 Fig. 5, the most dramatic effects of decreased balance correcting activity in SOL due to BVL occurs for muscles that are contralateral 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 toward our second goal which was to determine what additional information, if any, might be yielded from a multidirectional 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 Fig 9, lateral torque was significantly smaller in BVL for roll directions between 160 and 260 ms, and significantly larger in roll directions between 280 and 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 (Fig. 9). Based on the assumption that the CNS controls pitch and roll torques separately (Winter et al. 1996; Matjacic et al. 2001), our observations would indicate that the coordination 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 (Figs. 6, 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 and 220 ms, followed by significantly larger trunk roll velocities during the periods between 240 and 300 ms, and 470 and 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 prominently due to excessive trunk muscle activity during the later stabilizing period, as we have shown in the present study (Figs. 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. 2001a).

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 and Honegger 1992; Allum et al. 1997). In contrast, both Allum et al. (Allum and Honegger 1992; Allum et al. 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 (Figs. 2, 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 PARAS muscles. We assume that in BVL subjects, the excessive activity in PARAS 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 sta-

bilizing period, between 470 and 530 ms, normal subjects experience small residual trunk motion following backward support-surface rotations with a roll component. BVL patients, however, exhibit excessive backward pitch and roll velocities (Fig. 7) related to hip roll torque generation as a result of further excessive PARAS activity needed in uphill muscle during later stabilizing periods (Figs. 2, 6) to avoid falling. Noticeably this activity is larger when visual inputs are present (Fig. 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 periods of 240–300 ms and 470–530 ms because PARAS muscle responses with backward roll directions of maximum activity are enhanced due to a lack of inhibitory vestibulospinal 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 SOL, 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 and 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 (Fig. 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 multidirectional acting vestibulo-ocular reflex to maintain multidimensional 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 (Fig. 6). Such an observation may suggest a greater role of trunk muscle proprioceptors in establishing appropriate head-trunk coordination 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 BVL influences normal postural reactions. Multidirectional perturbations were used to identify observable differences in muscle activation profiles, and particularly differences in trunk and ankle torque control with BVL 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 center 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 the 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, 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, Ledin T (1999) Recovery of vestibulo-ocular function in subjects with acute peripheral vestibular loss. J Vestib Res 9:135–144

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, 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

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

Allum JHJ, Held-Ziolkowska M, Adkin AL, Carpenter MG, Honegger F (2001a) Trunk sway measures of postural stability during clinical balance tests. Gait 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 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. Electroencephalogr 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, Allum JH, Carpenter MG, Honegger (2000) Is lower leg proprioception essential for triggering human automatic postural responses? Exp Brain Res. 30:375–391

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) 118:449–454

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 40:443–447

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

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, 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 (abstract). Soc Neurosci 22:1632

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

Inglis JT, Macpherson JM. (1995) Bilateral labyrinthectomy in the cat: effects on the postural response to translation. J Neurophysiol 73:1181–1191

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

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

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

Maki BE, McIlroy WE (1998) Control of compensatory stepping reactions: age-related impairment and the potential for remedial intervention. Physiother Theory 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

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

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 150:403–407

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

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

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 eighth 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 activity in humans during brief falls. Exp Brain Res 46:324–330

Wilson VJ, Melvill Jones G (1979) Mammalian vestibular physiology. Plenum, 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

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 75:2334–2343