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Bilateral Vestibular Loss in Cats Leads to Active Destabilization of Balance During Pitch and Roll Rotations of the Support Surface

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Macpherson JM, Everaert DG, Stapley PJ, Ting LH. Bilateral vestibular loss in cats leads to active destabilization of balance during pitch and roll rotations of the support surface. *J. Neurophysiol* 97: 4357–4367, 2007. First published April 11, 2007; doi:10.1152/jn.01338.2006. Although the balance difficulties accompanying vestibular loss are well known, the underlying cause remains unclear. We examined the role of vestibular inputs in the automatic postural response (APR) to pitch and roll rotations of the support surface in freely standing cats before and in the first week after bilateral labyrinthectomy. Support surface rotations accelerate the body center of mass toward the downhill side. The normal APR consists of inhibition in the extensors of the uphill limbs and excitation in the downhill limbs to decelerate the body and maintain the alignment of the limbs with respect to earth-vertical. After vestibular lesion, cats were unstable during rotation perturbations and actively pushed themselves downhill rather than uphill, using a postural response that was *opposite* to that seen in the control trials. The extensors of the uphill rather than downhill limbs were activated, whereas those of the downhill limbs were inhibited rather than being excited. We propose that vestibular inputs provide an important reference to earth-vertical, which is critical to computing the appropriate postural response during active orientation to the vertical. In the absence of this vestibular information, subjects orient to the support surface using proprioceptive inputs, which drives the body downhill resulting in instability and falling. This is consistent with current models of sensory integration for computation of body posture and orientation.

INTRODUCTION

One of the significant and debilitating features of vestibular loss is difficulty with balance under certain conditions but not others (Brandt 1991). For example, subjects with vestibular loss are able to maintain balance during translation of the support surface, but not during rotation (Allum and Pfaltz 1985; Horak et al. 1990). Likewise, vestibular-absent cats show normal latencies and muscle activation patterns in the postural response to translation in multiple directions in the horizontal plane (Inglis and Macpherson 1995), but, as we describe in this report, they have difficulty with rotation. Although the situations in which instability is experienced are well known, the underlying cause remains a mystery. In this report, we describe the underlying cause of postural difficulties during rotation of the support surface in cats with complete bilateral vestibular loss resulting from surgical labyrinthectomy. Our subjects were tested immediately after lesion, prior to the compensation phase in which changes are known to occur in the brain (Igarashi 1984; Kitahara et al. 1998). Studying the acute effects of lesion simplifies the inferential interpretation of the normal role of vestibular signals. In contrast, people with vestibular loss who participate in postural studies are usually well past the compensation phase.

The results of this study in the cat likely apply to humans as well. Even though cats are habitual quadrupeds, the neural mechanisms and biomechanical principles of postural control are similar to those of the bipedal human (Dunbar et al. 1986; Horak and Macpherson 1996; Ting and Macpherson 2004). Surface rotation produces similar imbalance in cats and people and analogous automatic postural responses. In both species, rotation of the support surface accelerates the body center of mass (CoM) toward the downhill side. In roll about the anteroposterior axis for example, the downhill limb is initially unloaded and any tonically active muscles are shut down while the uphill limb is loaded. The upward platform motion passively flexes the uphill limbs; this stretches the extensors and may elicit a short-latency burst of electromyographic (EMG) activity, depending on platform kinetics (acceleration and velocity). The appropriate postural response requires activation of the extensors in the downhill limb(s) and inactivation of extensors in the uphill limb(s), to brake the CoM motion and minimize body tilt. In pitch about the frontal axis, the human foot performs the combined tasks of the forelimbs and hindlimbs of the cat. A toes-down (human) or head-down (cat) rotation moves the CoM forward, unloading the ankle extensors (human) and forelimb extensors (cat) and stretching ankle flexors (human) and hindlimb extensors (cat). The automatic postural response activates the ankle extensors (human) and forelimb extensors (cat) to produce a downward force against the surface under the toes (human) and forelimbs (cat) and correct the forward tilt of the body (Diener et al. 1984; Nashner 1976; Ting and Macpherson 2004). Even though the specifics of body configuration and muscle activation pattern may differ across species, the effect of the surface rotation on balance and the strategy for recovery are similar: the body tilts downhill and forces are produced on the downhill side by appropriate muscle activation, to reduce body sway and restore upright stance.

There are several possible underlying causes for which vestibular-absent people and cats have difficulty with balance on tilting surfaces. The automatic postural response (APR) may be reduced in amplitude or even absent and therefore insufficient to stop the sway induced by the perturbation (Allum and Pfaltz 1985; Carpenter et al. 2001; Diener and

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Dichgans 1988). Alternatively, the APR may be delayed or, finally, it may be wrong.

In this report, we demonstrate that rotation of cats with bilateral vestibular loss evokes an APR at the normal latency but with the wrong pattern, opposite to the response observed prior to lesion. We argue that the erroneous response stems from an incorrect estimate of the CoM motion relative to the gravity vector due to the absence of an accurate reference to earth vertical. Preliminary results were published in abstract form (Everaert et al. 2005).

METHODS

The subjects for this study, three adult female cats (*Ti,* 3.6 kg; *St,* 4 kg; and *Ve,* 3.4 kg), were trained using positive reinforcement to stand, unrestrained, on four force plates mounted on a rotating platform. The cats were trained to stand quietly with their weight evenly distributed between left and right sides. They were required to remain standing during and after rotation but were not rewarded for any particular response to the platform motion.

After several months of training, subjects were placed under general anesthesia and implanted for EMG recording with pairs of Teflon-coated stainless steel wire electrodes in 16 muscles of fore- and hindlimbs, neck, and trunk using aseptic technique (for implant details see (Macpherson 1988). The muscles of interest for the current study were proximal and distal extensors of the limbs that are typically recruited during the rotation APR (Ting and Macpherson 2004). EMG electrodes were accessed through a pair of connectors cemented to the skull. Animals were allowed to recover fully before collecting control data.

During testing, once a subject was standing quietly with its weight evenly distributed between right and left sides, the platform was rotated without warning with a ramp-and-hold profile of 6° amplitude and 40°/s peak velocity. These parameters are comparable to those used in human studies of platform rotation [e.g., 3° at 36°/s (Allum and Pfaltz 1985); 7.5° at 50°/s (Carpenter et al. 2001); and 3° at 50°/s (Nardone et al. 1990)] and, most importantly, the velocity was high enough to reliably evoke an APR in our subjects (see Diener et al. 1984).

Control data were collected over several days, and on each day at least five sequential trials were recorded for each of the four directions (see Fig. 1*A* for coordinate systems), head-down (+pitch), head-up (−pitch), left-down (+roll) and left-up (−roll). Total trial duration was 3 s including 0.28 s of background period prior to the onset of platform movement. Platform angular position and velocity, three components of linear force from each of the four force plates, and raw EMG activity were recorded at 1,200 Hz using an Amlab system (Amlab Technologies, Lewisham, NSW, Australia). Body kinematics were recorded at 120 Hz using a Vicon system (Vicon, Lake Forest, CA) with reflective markers of 7 mm diam placed bilaterally on the forelimbs (metacarpo-phalangeal (MCP), wrist, elbow, and shoulder (gleno-humeral) joints and the tip of the scapular spine), and hindlimbs (metatarso-phalangeal (MTP), ankle, knee, and hip joints and the iliac crest of the pelvis).

Head position (6 df) was recorded at 120 Hz using a Minibird (Ascension Technology, Milton, VT) with the transmitter mounted ~20 cm above the head of the cat, and the receiver mounted on one of the EMG connectors. Using a custom-built device, the position and orientation of the transmitter were measured relative to the Earth-referenced system of the Vicon device prior to the series of experiments. The angular offsets were then programmed back into the Minibird system at the beginning of each data collection session, to align the Minibird coordinate system with earth vertical and platform horizontal (Fig. 1*A,* Minibird). Although aligned, the Minibird native

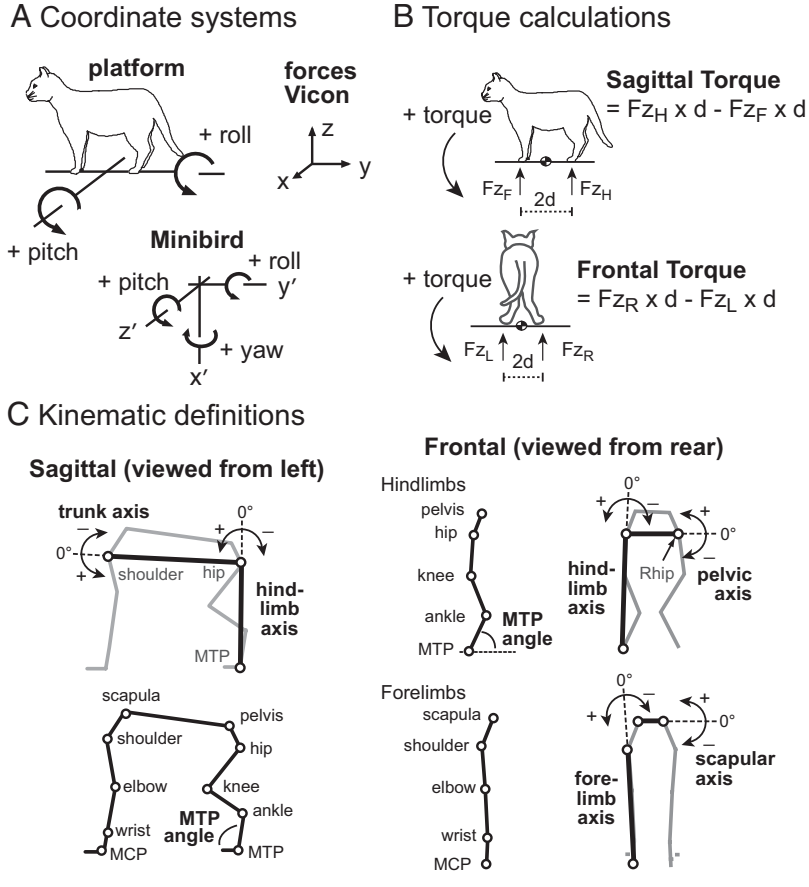

FIG. 1. *A*: coordinate reference frames. Platform rotation was specified as positive pitch in the head-down direction and positive roll, left side-down. Axes of rotation were collinear with the surface of the force plates and intersected in the midline, half-way between the centers of the front and back force plates. The force and Vicon reference frames were aligned with the platform, with the *x* axis along the pitch axis, *y* axis along the roll axis, and *z* axis parallel to the gravity vector. The Minibird coordinate system was aligned with the other systems but was rotated 90° about the *y* axis. The measured angles were the Euler angles with pitch about the *z′* axis and roll about the *y′* axis. *B*: formulas for computing sagittal and frontal plane torque about the axis of platform rotation. Fz_H, summed vertical force under hindlimbs; Fz_F, under forelimbs; Fz_L, under left fore- and hindlimb; Fz_R, under right fore- and hindlimb; 2d, distance between fore- and hindpaws for sagittal plane, between left and right paws for frontal plane. *C*: body axis and joint angle definitions are shown on the stick figures for sagittal and frontal planes. Arrows indicate the directions of positive and negative angle change of body axes.

coordinate system was rotated 90° about the *y* axis relative to the Vicon and force systems. In its native system, the Minibird angular coordinates matched those of the platform pitch and roll. The Minibird linear position data (*x, y, z* in mm) were expressed in the same system as the Vicon data for the purposes of illustration (i.e., z'-axis data were labeled as *x*-axis data). The position and orientation of the Minibird receiver relative to each cat's head was measured in stereotaxic coordinates post mortem.

After completion of control data collection, animals were labyinthectomized bilaterally under full anesthesia using aseptic techniques. The vestibule was accessed by drilling through the temporal bone, and the sensory tissue was destroyed. With this method, activity in the afferent fibers is retained but no longer modulated by head acceleration in space. The details of the surgery can be found in our previous publication in which the same subjects were studied in a voluntary head-movement task (Stapley et al. 2006).

After lesion, all three cats showed similar deficits in their free-ranging behaviors on the floor: broad-based stance, ataxic gait, head instability, and inability to track moving objects visually. Before lesion, all three cats showed strong eye and even head nystagmus during rotation in the dark in both vertical and horizontal planes, followed by brisk postrotary nystagmus. After lesion, none of the cats exhibited any nystagmus during or immediately after rotation in the dark. All three cats were unable to right themselves during vertical falls. Two of the animals (*Ti, St*) stood independently on the platform from the first day after lesion whereas *Ve,* although able to stand on the floor, refused to stand on the rotating platform without experimenter contact until the second week. This was deemed a behavioral issue rather than a difference in severity of lesion between *Ve* and the other two cats. In fact, from day 3 onward *Ve* stood independently and performed the voluntary head-turn task on a different platform used for that particular study (Stapley et al. 2006).

All cats were unstable while standing on the platform and frequently required steadying support by an experimenter, especially at the end of platform rotation. The criteria for even weight distribution during the background quiet stance period was relaxed, to accommodate the swaying of the lesioned animals. The data in this report were collected from *Ti* and *St* during the first week postlesion, from trials in which the subjects remained standing without assistance (days 3, 4, 6, and 7 for *Ti;* days 4–6 for *St*). Data are included from *Ve* in the third week after lesion (days 14 and 15) because they showed the same global postural deficit as the other cats.

Data were imported into MATLAB (Mathworks, Natick, MA) files and processed off-line. The time of platform onset was determined from the platform angular velocity traces. Digital filtering used a sixth-order Butterworth algorithm. Raw EMGs were high-pass filtered (35 Hz) to remove movement artifact and the mean was subtracted. Then EMGs were full-wave rectified and low-pass filtered (35 Hz). Forces were low-pass filtered at 100 Hz and kinematic data at 7 Hz. The kinematic variables of interest in this study are the limb and trunk axis angles relative to earth horizontal and the MTP joint angle, illustrated in Fig. 1*C*. The limb axis is defined as the line between the MTP and hip joints for the hindlimb and MCP and shoulder joints for the forelimb. The sagittal plane trunk axis is the line between shoulder and hip joints. Two frontal plane trunk axes were computed: the line between the left and right hip joints (termed the pelvic axis) and the line between left and right scapular tips (termed the scapular axis). Sagittal and frontal plane torques about the platform axis of rotation were calculated based on the vertical forces under hind- and forelimbs and force-plate distances (Fig. 1*B*). These torque variables provide a good global measure of the effect of the perturbation and the active response of the animals.

The head angular position data were transformed relative to stereotaxic zero based on the post mortem measures of the Minibird receiver position for each cat such that, for example, at 0° roll, the mid-sagittal plane of the head was parallel to the gravity vector. Angular velocity of the head-in-space was computed by differentiating (using a weighted difference function) the transformed head pitch and roll angular positions. Because the Minibird receiver was located at a distance from the skull-C_1 joint, the measured linear position is due to both head rotation about the skull-C_1 joint as well as linear motion of the skull-C_1 joint. Therefore the coordinate origin of the head linear position data were transformed mathematically from the position of the Minibird receiver to the position of the skull-C1 joint based on the post mortem measures. Because the placement of the Minibird receiver differed across cats, we wanted a uniform representation of head linear motion which could be compared across subjects. The skull-C_1 joint is the center of rotation for small pitch motions of the head (Peterson and Richmond 1988) and therefore linear motion at this joint is independent of the angular rotation about the joint, at least in the pitch plane. The native Minibird coordinate system was used in the transform of the origin of the position data because of the noncommutative nature of the Euler angles. The linear position data of the skull-C_1 joint were differentiated to estimate head linear velocity.

RESULTS

In summary, the lesioned animals had difficulty maintaining balance during platform rotation and frequently required intervention by an experimenter. The imbalance arose from an improper postural response that was opposite to the control response, with the result that the cats actively pushed themselves toward the downhill side.

The effect of platform rotation before and after lesion can be seen globally in the example stick figures drawn at a series of time points over the course of a trial (Fig. 2). Prior to lesion, cats tilted slightly toward the downhill side during both pitch

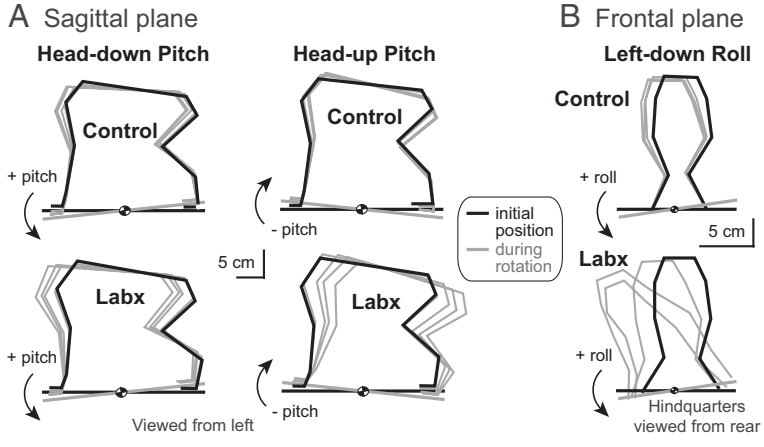

FIG. 2. Typical kinematic profiles during pitch and roll rotation. *A*: sagittal plane pitch rotations (left fore- and hindlimb segments, *subject Ti*). *B*: frontal plane roll rotation (left and right hindlimb segments, subject St). Left-up roll (not shown) was similar but opposite to left-down. Stick figures show mean positions of body segments before (black lines) and at 140, 300, 560, and 975 ms after onset of platform rotation (gray lines). Data are averaged across all trials before (control) and in the 1st week after bilateral labyrinthectomy (Labx), within each subject. Note the large increase in downhill rotation of the body after lesion, especially for roll. The horizontal axis was scaled up for the frontal plane figures (see scale bars) for easier viewing.

and roll rotation and remained slightly tilted relative to earth-vertical during the hold phase of the perturbation. After vestibular loss, cats showed considerable increase in body sway as they actively pushed themselves downhill and frequently lost balance, particularly during roll rotations.

Automatic postural response (APR)

The automatic postural response to rotation consists of a series of events: first, after a characteristic delay from the onset of platform motion, the nervous system activates (or inhibits) specific groups of muscles throughout the body. After a delay due in part to excitation-contraction coupling time, the change in EMG activity results in a change in force applied at the ground by each leg; the net effect of the force is expressed in this study as torque generated about the CoM. This torque, arising from the evoked EMG activity, affects body sway with a delay related to the inertia of the body and can be observed in various kinematic variables. Figure 3 (*left*) illustrates this sequence using shaded rectangles to indicate the time period of the APR events and arrows to illustrate the time delays from EMG activation to torque generation and finally to body motion (limb axis angle). Teasing out the quantitative effects of the APR on forces and kinematics is difficult because the perturbation itself induces passive changes in these variables that combine with the active response of the APR. However, a pre- and postlesion comparison of EMG, force, and kinematic variables can provide important insights into the mechanism of the APR because the perturbation is identical and the initial stance is similar for each subject under the two conditions. Specific responses to rotation are detailed in the following description.

The responses to rotation of the intact cats prior to lesion were similar to those described in our previous study (Ting and Macpherson 2004). The initial effect of the perturbation *prior* to the APR was a short-latency (<40 ms) activation of extensors in the uphill limbs (Fig. 3, *A,* LGAS in head-down pitch; *B,* SOL in left-down roll) and inhibition of extensors in the downhill limbs (Fig. 3*A,* LGAS in head-up pitch; 3B, SOL in left-up roll). The short-latency activation was not usually as pronounced after lesion as compared with before (Fig. 3 thick gray traces vs. thin black traces, respectively). This early response was not part of the APR. Instead it was destabilizing and added to the torque evoked by the perturbation which rotated the body to the downhill side as seen in the early, downhill displacement of the limb axis angles (Fig. 3).

Subsequently, the APR was evoked at around 70 ms and in the control case was characterized by inhibition in the uphill limb extensors (thin black traces: Fig. 3, *A,* LGAS head-down; *B,* SOL left-down) and excitation in the downhill (thin black traces: Fig. 3, *A,* LGAS head-up; *B,* SOL left-up). The control APR contributed to the reversal and decrease of the torque back toward the baseline (see thin black traces in the shaded region of torque plots in Fig. 3). Finally, the reduction in torque helped to slow down body sway (represented by the right hindlimb axis angles in Fig. 3).

The APR following labyrinthectomy was opposite to that of the control condition, i.e., *excitation* in the uphill limb extensors and *inhibition* in the downhill (compare control and Labx EMG traces from each muscle within the region bounded by the gray rectangles in Fig. 3). This inappropriate APR was followed by an *increase* in downhill torque (note the divergence of the control and Labx torque traces within the gray

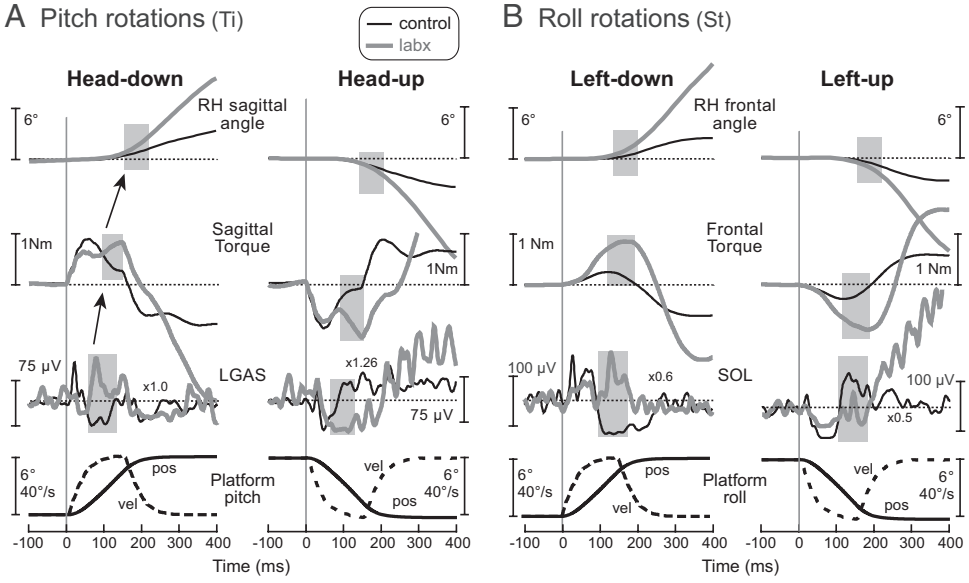

FIG. 3. Automatic postural response and succeeding events during platform rotation in pitch (*A*) and roll (*B*). Traces show the mean response within subjects across all trials during the control period (thin black lines) and in the 1st week after labyrinthectomy (Labx - thick gray lines). Gray rectangles overlying electromyographs (EMGs) indicate the period of the automatic postural response. Note the opposite response in the EMG data after lesion compared with control. Gray rectangles overlying the torque data show the effect on the torque of EMG activity during the postural response, assessed after a delay of 30 ms from onset of EMG to accommodate excitation-contraction coupling. Note the divergence in direction of control and Labx torque traces during this period. Gray rectangles overlying the right hindlimb (RH) axis angle highlight the onset of body sway in the direction of platform rotation. The control EMG and torque responses helped slow down the body sway induced by the perturbation. Following lesion, the EMG and torque responses accelerated body sway toward the downhill side. Data were taken from *subjects Ti* (*A*) and *St* (*B*). Vertical lines mark the onset of platform movement as determined from platform velocity data. For EMGs, the display gain of the control trace was multiplied by the indicated factor, to have equal mean background levels for control and Labx traces and allow better visual comparison of responses. LGAS, right lateral gastrocnemius (ankle extensor/knee flexor); SOL, right soleus (ankle extensor); pos, angular position; vel, angular velocity

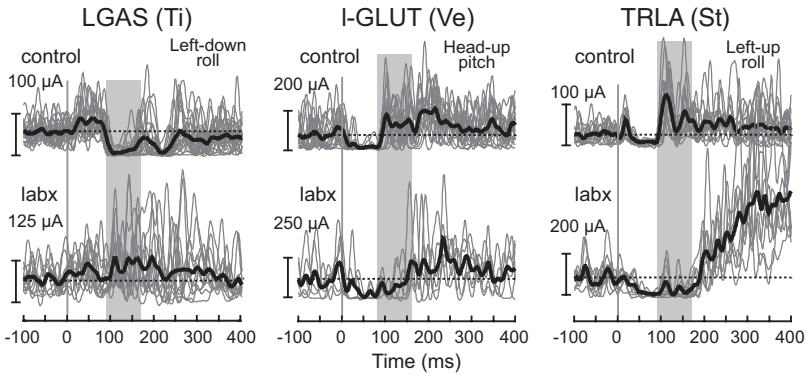

FIG. 4. Consistency in EMG response to rotation. One example set of EMGs from each subject shows the mean response (black traces) and all individual trials (gray traces) during the control and postlesion (Labx) periods. Gray rectangles highlight the region of the automatic postural response. Vertical lines indicate the onset of platform motion. LGAS, right lateral gastrocnemius; RLA, right triceps brachii, lateral head (elbow extensor); l-GLUT, left gluteus medius (hip extensor/abductor).

rectangles of Fig. 3). The increase in torque was then followed by a rapid increase in body sway toward the downhill side, greater than the sway seen in the control trials (Fig. 3, gray traces).

The opposite nature of the APR before compared with after lesion is consistent and repeatable as seen at the single trial level in both EMGs and torques. Figure 4 shows one example from each cat of all trials of EMG recorded (gray traces) from a single muscle before (control) and after (Labx) lesion as well as the mean activity (black traces). The region of the APR is highlighted by the gray rectangles. Right lateral gastrocnemius (LGAS), a distal hindlimb extensor, was inhibited in the control trials and excited after lesion during left-down roll in *Ti* (Fig. 4, *left*). Left gluteus medius (l-GLUT), a proximal hindlimb extensor, was activated in the control trials and inhibited after lesion for head-up pitch in *Ve* (Fig. 4, *middle*). Similarly, right triceps brachii (TRLA), a proximal forelimb extensor, was activated in the control and inhibited after lesion during left-up roll in St (Fig. 4, *right*).

Table 1 shows the mean and SD of the APR-induced torque response in all three cats before and after lesion. The sign of the torque response was opposite for pre-compared with postlesion measures in every trial, indicating the robustness of the inappropriate postural response induced by vestibular loss. Torque data are presented from trials in the first week following lesion for *Ti* and *St,* showing that the postural reversal occurred prior to any significant compensation. Supporting data from week 3 in *cat Ve* are also shown because the postural reversal persisted. Change in torque was measured for each trial from 100 ms after the onset of platform movement until the time of peak platform velocity to avoid the confound of the effect of platform deceleration. The decrease in platform velocity, or deceleration, acts as a second perturbation, causing a torque that is opposite to the torque induced by the initial platform acceleration (see Fig. 3). This deceleration is actually stabilizing to the animal because it opposes the downhill motion of the body. EMG responses to this phase of the perturbation were not analyzed. Although the limb axis rotation shown in Fig. 3 was initiated near the time of platform peak velocity, this motion was associated with the initial component of the perturbation. The long delays reflect the inertia of the body.

Limb and trunk kinematics

During rotation, the limb axes initially remained vertical while the trunk axis rotated with the platform, in both control and postlesion trials. This is evident in Fig. 5 in which the trunk axis and MTP plots deviate from zero shortly after the platform begins to move while the limb axis plots remain at zero and deviate only after a delay. The limb axis angles reflect body sway and the delay in motion is due to the mass and inertia of the body.

In the control data of pitch perturbations (Fig. 5*A,* thin black traces), the trunk rotated close to the full 6° of platform tilt,

TABLE 1. *Change in torque (Nm) evoked by EMG activation during APR*

	Head-Down Platform Pitch		Head-Up Platform Pitch	
Subject	Control	Labx	Control	Labx
Ti	−0.55 ± 0.12 (23)	+0.28 ± 0.12 (7)	+0.27 ± 0.10 (24)	−0.55 ± 0.30 (7)
St	−0.19 ± 0.07 (14)	+0.30 ± 0.11 (12)	+0.19 ± 0.07 (15)	−0.49 ± 0.19 (11)
Ve	−0.20 ± 0.08 (11)	+0.12 ± 0.07 (17)	+0.15 ± 0.10 (14)	−0.14 ± 0.12 (10)
	Left-Down Platform Roll		Left-Up Platform Roll	
Ti	−0.03 ± 0.02 (27)	+0.07 ± 0.09 (15)	+0.05 ± 0.02 (18)	−0.10 ± 0.06 (12)
St	−0.11 ± 0.07 (15)	+0.07 ± 0.04 (14)	+0.05 ± 0.05 (13)	−0.09 ± 0.06 (12)
Ve	−0.08 ± 0.03 (15)	+0.02 ± 0.03 (15)	+0.06 ± 0.03 (16)	−0.05 ± 0.03 (10)

Values are means ± SD (n = trials in parentheses) recorded before (control) and after bilateral labyrinthectomy (Labx). Labx data are from the first week post-lesion for *Ti* and *St* and the third week for *Ve.* EMG, electromyograph; APR, automatic postural response

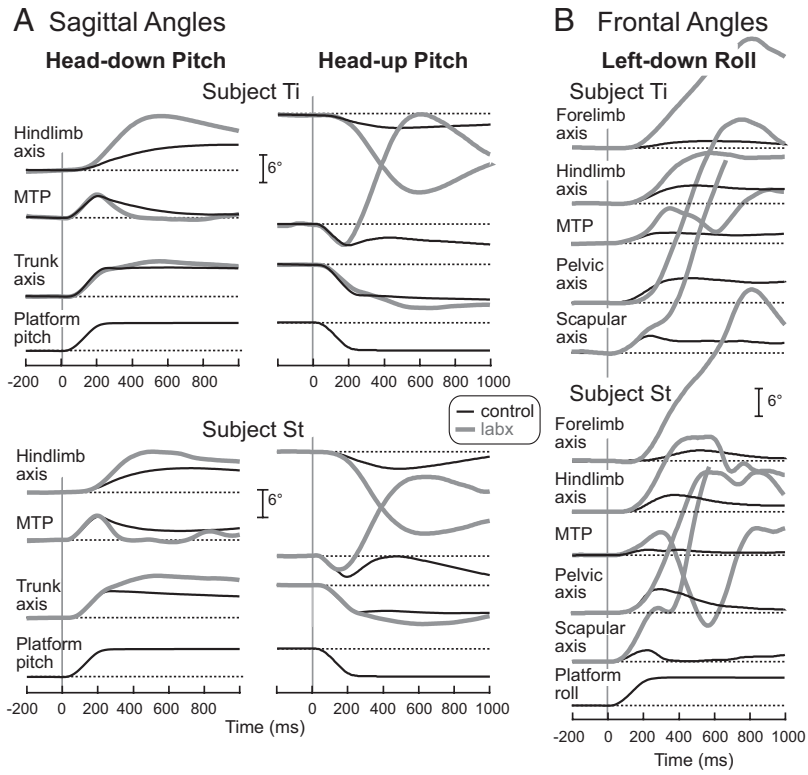

FIG. 5. Kinematics. Time traces show the average angular excursion of trunk and limb axes, left mtp joint, and platform in the sagittal plane during pitch rotations (*A*) and in the frontal plane during left-down roll (*B*) both before (thin black lines) and after (thick gray lines) lesion. *Top*: data from *subject Ti; bottom*: data from *subject St*. In pitch, both fore- and hindlimb axes showed similar changes, so only the hindlimb data are shown. In roll, frontal plane excursions of both pelvic and scapular axes are shown because they differed in amplitude. Similarly, both fore and hindlimb axes on the left side are shown to illustrate the consistently larger excursion of the hind quarters during roll rotations. Vertical lines show onset (at *time 0*) of platform rotation, determined from platform velocity data. Body axis and joint angle definitions are illustrated in Fig. 1*C*.

whereas in roll perturbations (Fig. 5*B,* thin black traces), trunk rotation was smaller than that of the platform with the pelvic axis tilting more than the scapular axis. For all perturbations, the limb axes began to rotate downhill near the end of the support-surface rotation and remained tilted during the static hold period with the limbs oriented at an angle in between support surface perpendicular and earth vertical. The limb axis angular displacement was considerably smaller in roll than in pitch.

After vestibular lesion, the sequence of trunk axis rotation followed by limb axis persisted, but rotation of the trunk and especially the limb axes was greater than in the controls (Fig. 5, thick gray traces). These perturbations were clearly challenging to the lesioned animals, and they sometimes had to lift a paw or step off the force plate to maintain balance (5/26 trials for *Ti* and 9/25 trials for *St* in the 1st week, and 2/50 trials for *Ve* in the 3rd week following lesion).

The gain of the postural response with respect to the surface rotation can be defined as the ratio of the limb axis angle to the platform angle and is shown in Fig. 6 as a function of time during both the ramp and hold phases of rotation. Both limb axis and platform angles were measured in earth-based coordinates. In the intact cat, limb axis gains increased during rotation and reached a peak, usually <1 for pitch (Fig. 6*A,* control) and 0.5 for roll (Fig. 6*B,* control), shortly after the beginning of the hold phase of rotation. The gain then decreased and stabilized on average at a value between 0 and 1 but larger for pitch than for roll. After lesion, the limb axis gains greatly exceeded 1 at the peak and generally remained higher than in controls during the hold phase (Fig. 6, Labx). A gain of 1 signifies that the limb axis tilted in the same direction and to the same degree as the platform rotation and therefore remained in its original orientation relative to the platform (see figurines in Fig. 6). A gain of 0 signifies that the limb axis remained oriented to earth-vertical as the platform rotated. Limb axis orientation is considered to be an important control variable during stance in the cat (Lacquaniti and Maioli 1994) and is equivalent to body sway when the axis length does not change.

Head kinematics

The data show that cats without vestibular inputs had impaired postural responses to pitch and roll rotation of the support surface. To explore how vestibular information contributes to the automatic postural response to rotation, we examined angular and linear velocity of the head in space (Fig. 7) during the initial phase of pitch and roll rotations, prior to the onset of the automatic postural response, which was around 70 ms after platform onset. In this early period, movement of the head is passively induced by inertial forces from platform motion and gravity and is therefore expected to be quite similar before and after lesion. An understanding of how the head is perturbed early-on during the various directions of rotation can lead to insights about the information that the vestibular system is sending to the postural centers and, therefore the possible role of vestibular inputs in the initial phase of the automatic postural response to rotation.

About 50 ms after the onset of platform pitch rotation, the head began to rotate opposite to the direction of the platform (Fig. 7*A, columns 1–2*); i.e., with a head-down rotation of the platform (+pitch), the head initially rotated in the nose-up, or –pitch direction. The inertia of the head and compliance of the neck joint (probably skull-C_1) may account for the opposite direction of head compared with platform rotation. Linear motion of the head was downward and forward during head-down pitch, and upward and back-

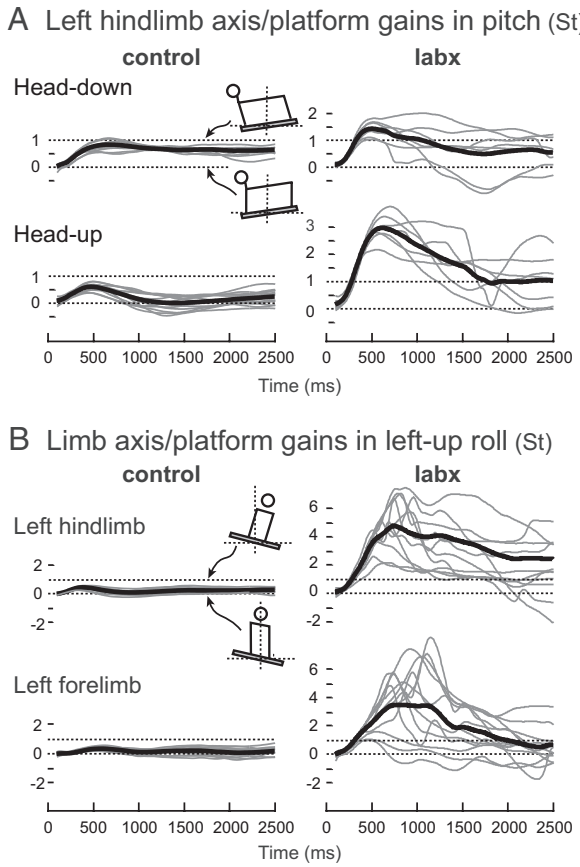

FIG. 6. Limb axis gains in pitch (*A*) and roll (*B*) for subject St. The ratio of limb axis angle to platform angle is plotted for individual trials (gray traces) and the average (black traces) before (control) and after vestibular lesion (Labx). Data from the left hindlimb shown in *A* are representative for all 4 limbs. Head-up and -down responses are shown separately because there was some asymmetry in the 2 directions. The 2 directions of roll were symmetric, so only left-up data are shown. Both forelimb and hindlimb data are shown because hindlimb gains were slightly higher for roll in all subjects. Figurines represent the idealized limb and trunk axis orientations for gains of 0 and 1. Arrows point to the relevant gain value for each figurine. With a gain of 0, the limbs remain parallel to the gravity vector; with a gain of 1, the limbs rotate with the platform, remaining perpendicular to the support surface. Onset of platform rotation occurred at *time 0*.

ward during head-up pitch (Fig. 7*B, columns 1–2*). During stance, the head position was far anterior to the axis of platform pitch rotation. Therefore as the forequarters dropped down under the force of gravity in head-down pitch, the head also dropped, translating downward and forward on an arc. The dashed lines in Fig. 7*B* (*columns 1–2*) show the theoretical linear velocity of a sphere located at the position of the head and fixed to the platform by a rigid rod. The actual vertical velocity of the head (*z* vel) lagged that of the sphere but eventually achieved the same peak velocity. In contrast, the horizontal velocity of the head (*y* vel) not only lagged but never reached the theoretical peak velocity of the sphere because the limb axes remained vertical during the initial phase of platform rotation and the head therefore subtended a much tighter arc than that of our theoretical sphere.

During platform roll rotation there was no consistent angular or linear motion of the head during the period prior to the automatic postural response, i.e., the first 70 ms (Fig. 7, *A* and *B, columns 3–4*). In contrast, a sphere rigidly fixed to the platform at the position of the head would undergo significant lateral motion (Fig. 7*B,* dashed lines in columns 3 and 4). As with platform pitch, the limb axes remained vertical during the initial phase of platform roll, thus limiting the lateral motion of the head (*x* axis). Both actual and theoretical vertical motion (*z* axis) was negligible because the head was approximately centered over the axis of roll rotation. The lack of angular motion of the head in roll is more difficult to explain, given that the pelvic and scapular axes displayed significant rotation early in the perturbation.

How is it that the early, passive trunk rotation was not transmitted to the head? The passive rotation of the pelvis would certainly have been transmitted through the lumbo-sacral to the thoracic spinal column because the lumbo-sacral vertebral joints cannot rotate around the long axis relative to each other and so the entire pelvis and lumbo-sacral region would have rotated as a unit (Macpherson and Ye 1998). In contrast, the thoracic joints between T_4 and T_{11} have a large range of motion in torsion. It is likely that the pelvic rotation was dampened out due to compliance in the deep rotator muscles somewhere between T_4 and T_{11} because no rotation was observed at the head. Unlike the pelvis, the scapulae have muscular but no bony attachments with the trunk. Instead, the trunk is suspended from the two scapulae along three lines, the spinous processes of the thoracic vertebrae and the lateral processes and ribs on each side, providing a mechanism for relative motion of the forelimbs and trunk without rotation of the upper thoracic spine (Macpherson and Ye 1998). Therefore small rotations of the scapular axis do not likely cause rotation of the spinal column. Instead, the rotation of the scapular axis in roll most likely reflects the drop of the downhill forelimb and sliding of the scapula downward over the trunk.

A similar profile of early head movement was seen postlesion (Fig. 8), reinforcing the passive nature of the early (<70 ms) effects on the head.

The movements of the head in space evoked by pitch and roll rotations of the surface do not correlate in a simple or direct way with the movements of the body induced by the same rotations. Therefore the pattern of canal and otolith signals presumed to be evoked by the observed head movements cannot be correlated in a simple way to the pattern of muscle activation comprising the APR. And yet the removal of vestibular inputs led to a consistent error in the APR to both pitch and roll rotations characterized by the exact opposite response compared with the control case.

DISCUSSION

This study presents the novel result that vestibular-absent subjects generate an erroneous automatic postural response to rotation that is opposite to the control response, and this is the cause of their difficulties in remaining balanced. This finding complements our previous study showing that vestibular-absent subjects actively destabilize themselves during voluntary head movements (Stapley et al. 2006). We will argue that this erroneous response to platform rotation arises as lesioned cats try to align their limb axes to the support surface rather than to earth vertical because information about earth vertical is no longer reliable or available to them. The problem with aligning to the surface is that the

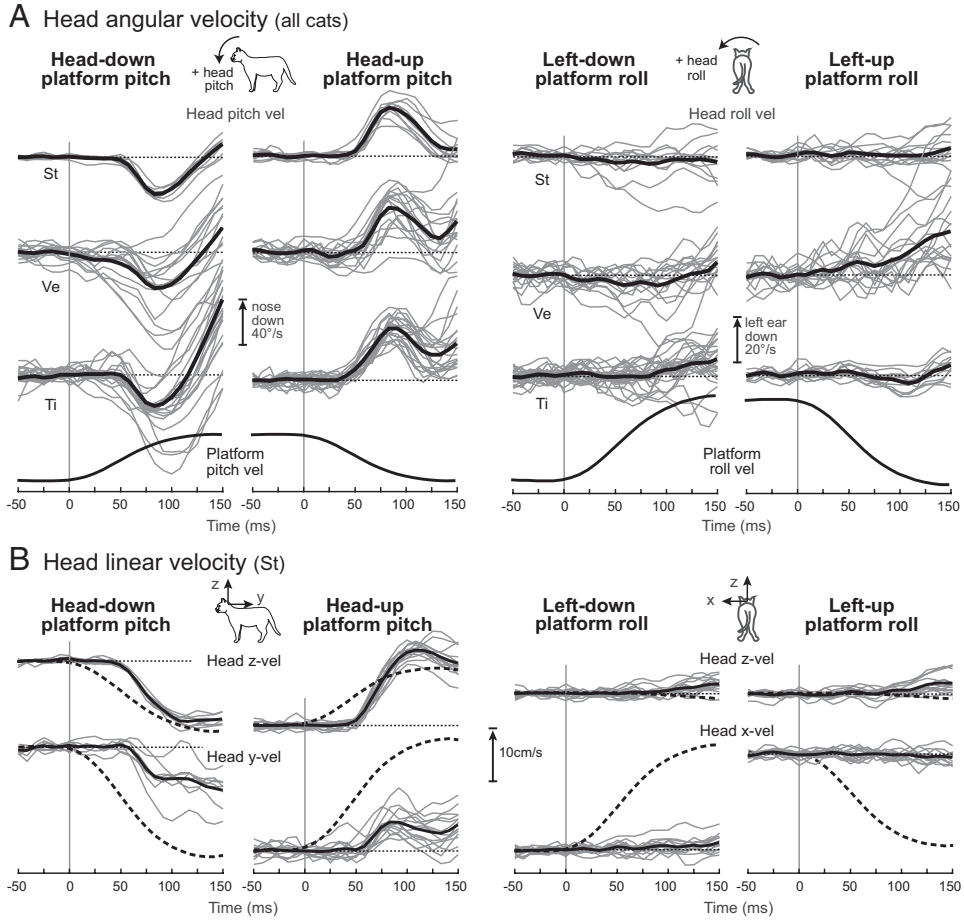

FIG. 7. Motion of the head during the early phase of platform rotation in the control condition. *A*: for each subject are shown all individual trials (gray traces) and average (black trace) of head angular velocity in pitch during pitch rotations and in roll during roll rotations of the platform. Platform angular velocity is also shown for the appropriate plane of rotation. The scale bars indicate the positive direction of head rotation as defined in the figurines at the *top*. Note that in pitch, the head rotated opposite to the platform and after a delay of ~50 ms from onset of platform motion. This pitch motion was likely passively induced by the downward acceleration of the body. During roll rotation, there was no consistent rotation of the head prior to the onset of the postural response which occurred at ~70 ms. The head was positioned directly above the axis of rotation for roll. To give a sense of the vertical displacement of the body during roll, the maximum vertical excursion of the support surface at the position of the paws was approximately ±4 mm. *B*: head linear velocity, shown from *subject St,* was similar across all 3 cats. Conventions as in *A*. The dashed lines show the theoretical linear velocity of a sphere located at the position of the head and fixed to the platform by a rigid vertical bar. Head linear motion was delayed relative to the onset of platform motion due to inertia of the body. The horizontal plane excursion of the head during pitch perturbations (Head y-vel) was much less than that of the rigid body and virtually negligible during roll rotations (Head x-vel). Vel, velocity.

resulting APR produces a force that accelerates the body downhill, in the same direction as the platform rotation, and contributes to the falling motion induced by the perturbation. In contrast, when the intact subject aligns to earth vertical, the APR generates force that opposes the downhill motion of the body and restores balance.

How do we know that the postural response in the lesioned cats is reversed and not just delayed? After all, the response postlesion of lateral gastrocnemius to head-up rotation in Fig. 3 showed a prolonged inhibition of tonic activity followed by a very late excitation, ~200 ms, whereas before lesion, the early inhibition was followed by a robust excitation at the normal APR latency. The key to this point is that the reverse pattern of response also occurred. When the control APR response was an inhibition, the postlesion response was an active excitation during the normal APR time period (e.g., Fig. 3, responses to head-down pitch and left-down roll; Fig. 4, response of LGAS to left-down roll). Therefore we conclude that the APR responses in the vestibular-absent cat reflect *active* excitations and inhibitions that were coordinated and consistent in propelling the animal to the downhill side. The consistency of the erroneous EMG response was reflected in the torque responses (Table 1).

Our conclusions are rather different from those of a recent study of people with bilateral vestibular loss (BVL) tested with pitch and roll rotations during stance (Carpenter et al. 2001). These authors concluded that the APR and subsequent torques were severely reduced in amplitude in the BVL group compared with controls. However, on examination of the published EMG traces (their Figs. 2 and 3), it is evident that several

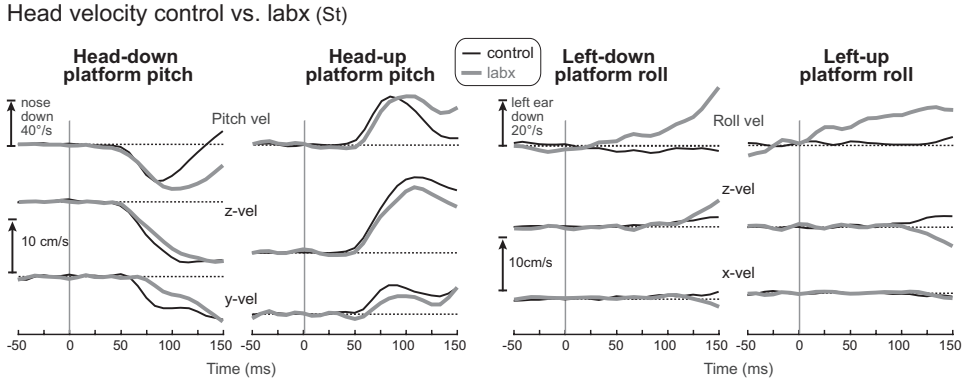

FIG. 8. Motion of the head of *subject St* during the early phase of platform rotation. Data are averaged for control trials (thin black traces) and for trials from the 1st week after lesion (thick gray traces). Note the similarity before and after lesion of the initial, passive movement of the head during the 1st 70 ms after platform onset. The largest difference was seen for head roll velocity in the left-up platform roll condition. Note, however, that the postlesion average was not stable in the background period, having a left-ear down drift that continued after the onset of platform movement. Conventions as in Fig. 7.

muscles showed reversals between the control and BVL groups during the APR time period. For example, during a toes-up/right-down rotation, the right paraspinals were shut down in the control group but excited in the BVL group. A similar inappropriate excitation was shown in this muscle group and in tibialis anterior for a toe-down/right-down rotation. Likewise, soleus was excited in the control group but inhibited in the BVL group for both these platform rotations. These observations from the published figures, although not remarked upon by the authors, are strongly suggestive that not only cats but also people lacking vestibular inputs produce erroneous, destabilizing responses to platform rotation.

It is clear that the automatic postural response to rotation in the vestibular-absent cat is coordinated and organized but acts in the wrong direction, compounding the destabilizing forces that propel the CoM downhill. It is puzzling why the lesioned cats were not able to compute direction correctly because our recent study comparing rotation and translation showed that the direction of CoM motion could, in theory, be determined from the combination of vertical and horizontal ground reaction forces, i.e., the change in force vector direction (Ting and Macpherson 2004). In general, surface rotations evoke large and immediate changes in vertical force (center of pressure) followed by small, slightly delayed changes in horizontal plane (shear) forces; surface translations evoke just the opposite: large and immediate changes in shear force followed by delayed changes in vertical force. In theory then, the direction of CoM motion for both rotations and translation could be discriminated by cutaneous receptors in the paw pads encoding change in direction of the ground reaction force vector, similar to the detection of change in slip and grip forces of the hand (Johansson and Cole 1992). Even though somatosensory inputs encoding direction of the perturbation may be available to the labyrinthectomized subject, such inputs may be unreliable under some conditions, or even ambiguous. The shear force evoked by a perturbation depends, in part, on the coefficient of friction of the surface (e.g., bare vs. icy sidewalk), so the change in force vector may not always be a reliable indicator of the direction of surface motion. Furthermore, the lack of information regarding the orientation of the surface to earth vertical, or gravity, may lead to ambiguities in interpretation of ground reaction force inputs.

We must therefore conclude that the vestibular system is critical for determining the appropriate direction of the postural response. How is vestibular information used to determine direction and, in the absence of vestibular input, what sensory signal is driving the postural control system to move the body downhill rather than uphill? To answer these questions, we must examine how the various sensory channels signal direction of a perturbation.

The signals that help determine the direction of platform rotation must be detected early enough to allow processing of the inputs, computation of the direction of perturbation, activation of the appropriate groups of motoneurons, and conduction of action potentials to the muscles, all prior to the APR latency of ~70 ms. Within the first 50 ms or so (allowing ≥20 ms for the last stages of central processing and muscle activation), the only significant angle changes are at the MTP and MCP joints while motion of the body lags behind because of inertia (Ting and Macpherson 2004). Therefore the nervous system must estimate, based on the available sensory inputs, the direction in which the CoM is *likely* to move. The visual system is too slow to contribute to the rapid postural response: the mean retinal slip response in the nucleus of the optic tract of the cat is 129 ms, about twice the latency of the automatic postural response (Distler and Hoffmann 1996). The perception of visual motion is impaired in vestibular deficiency (Grunbauer et al. 1998), so accurate detection of relative motion of the body and the environment may be faulty. Therefore we are left with vestibular and somatosensory inputs.

But early, dynamic vestibular inputs do not appear to give a clear indication of the direction of perturbation. During platform rotation in pitch, the cat's head was rotated passively by inertial forces in a direction *opposite* to body tilt, and during platform roll, the head did not move at all during the initial phase of the perturbation. It is not clear how a combination of vestibular and neck proprioceptive inputs could be used to compute unambiguously the direction of the impending motion of the CoM. In another study, re-positioning the axis of platform rotation reversed the direction of head vertical acceleration during rapid rotation of seated subjects and yet, the direction of the automatic postural response remained constant and appropriate for maintaining balance (Forssberg and Hirschfeld 1994). If vestibular inputs early in the perturbation do not inform about perturbation direction, then how else might vestibular inputs contribute?

We propose that the vestibular system provides critical information regarding the orientation of the gravity vector relative to the head that can then be used in combination with proprioceptive information to determine orientation of the limb and trunk axes with respect to gravity. It is this orientation of the body to its estimate of earth vertical which is used as the reference for interpreting the early, rapid proprioceptive inputs related to platform displacement and then computing the appropriately directed APR. In other words, the gravity reference is combined with proprioceptive information about body configuration to determine the motion of the CoM relative to that reference, during the initial phase of a perturbation. This schema is derived from studies and models of human perception (Mergner and Rosemeier 1998) and postural control (Mergner et al. 2003). A requirement of this concept is the ability to resolve the ambiguity of head tilt versus linear translation in the vestibular otolith signals. Recent behavioral and single unit recording studies of vestibuloocular reflexes show the existence of mechanisms that can parse out the head tilt component of a linear acceleration signal using a combination of canal and otolith inputs (Angelaki et al. 1999, 2004; Merfeld et al. 1999). Therefore no matter how the head is rotated and/or translated, the nervous system can determine the orientation of the head with respect to gravity, even if the head movement evoked by a perturbation is not related in a simple way to the body movement that is evoked.

In the absence of a vestibular signal of earth vertical, there still remains the visual reference frame for body orientation that comes from structures in the surrounding environment and is commonly collinear with earth vertical (Horak and Macpherson 1996). However, the ability to use such a visual reference is compromised in vestibular-absent subjects because of illusory sensations of self-motion arising from oscillopsia and difficulties in visual fixation (Brandt 1991).

The third reference frame for posture is the proprioceptive vertical based on the support surface: subjects may align their limbs (cat) or limbs and trunk (people) to the vector that is perpendicular to the surface. This kinematic reference would be derived from the relative lengths of the muscles about the limb joints including the articulations with the trunk. Alignment to the surface is observed in intact subjects for small perturbations (Peterka 2002). Electrophysiological studies in the cat suggest that limb axis orientation with respect to the support surface may be encoded by spinocerebellar tract cells that integrate proprioceptive signals originating throughout the limb (Bosco and Poppele 1997).

The APR in the labyrinthectomized cat actively rotated the body downhill, suggesting a strategy of aligning the limb axes perpendicular to the surface. Without the reference to gravity, the nervous system knows only that the body is tipping relative to the surface but does not know how the surface is moving in space. Therefore the lesioned cats aligned to the reference provided by proprioceptive information, a reference that tilted with the platform, causing them to actively push themselves downhill. Similarly, vestibular-absent subjects exposed to continuous surface rotation at a variety of amplitudes and frequencies persisted in aligning to the surface or proprioceptive vertical across the range of stimulus parameters and were not able to remaining standing at the higher amplitudes (Peterka 2002). The high stimulus-response gains of these subjects suggests that, similar to our cats, they may have produced erroneous EMG responses opposite in phase to those of control subjects, but EMGs were not recorded in the study. During sudden platform rotation, the proprioceptive vertical rotates relative to gravity whereas during translation it remains aligned with gravity. This may explain why the postural responses to rotation are opposite to control, whereas the responses to translation are appropriately tuned for direction (Horak et al. 1990; Inglis and Macpherson 1995).

Proprioceptive and cutaneous reference frames that are linked to stationary earth-based objects can provide powerful stabilizing influences. For example, vestibular-absent subjects standing on a continuously rotating surface were able to reduce body sway significantly just using light touch on an earth-fixed object (Creath et al. 2002).

The reliance of the lesioned cats on the proprioceptive reference to the platform appeared to influence not only the direction of the automatic postural response to dynamic rotation, but also the subsequent hold phase in which the platform was maintained at a static tilt angle of 6°. Such tilts may reflect a compromise or summation of the drive to align to the proprioceptive vertical relative to the surface and the drive to align to their best estimate of visual vertical, which was collinear with gravity. After vestibular lesion, the cats showed limb axis tilts in the downhill direction during the hold phase that were often higher than control and considerably more variable (e.g., Fig. 6 gain plots). Intact subjects surprisingly displayed a small tilt during the hold phase that was generally larger for pitch than for roll tilts. The most likely explanation is that experienced subjects frequently under-respond in anticipation of the end of the trial when the platform slowly returns to the origin and the body is passively restored to upright (Macpherson 1994). Given enough time on the tilted surface, presumably the intact cats would have returned to the earth vertical orientation which is characteristic of quiet stance on a statically tilted surface (Lacquaniti et al. 1990).

Our results suggest that the various sensory inputs are not used in a simple feedback structure but instead must be combined in an internal representation of body posture (Gurfinkel and Levick 1991), such as that proposed by Mergner and colleagues in which multiple sources of sensory inputs are combined through coordinate transformations for perception of body position and motion in space (Mergner and Rosemeier 1998; Mergner et al. 2003). More recent models have been proposed that include both the body and the environment to resolve sensory conflicts (Kuo 2005; Merfeld and Zupan 2002). We propose that for sudden disturbances of balance, vestibular information is critical in providing a gravity-reference that can be used to interpret the proprioceptive signals. This allows the nervous system to determine motion of the CoM with respect to gravity rather than to body or support surface coordinates as given by the proprioceptive system.

In conclusion the current study provides novel evidence in vestibular-absent subjects of a reversal in the automatic postural response to large, rapid rotation of the support surface in both pitch and roll. This abnormal response magnifies body sway, leading to active destabilization of balance. We propose that the erroneous response results from the use of a proprioceptive reference frame for balance because of the absence of an available vertical reference normally provided by the vestibular system.

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