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Bilateral Vestibular Loss Leads to Active Destabilization of Balance During Voluntary Head Turns in the Standing Cat

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Stapley, Paul J., Lena H. Ting, Chen Kuifu, Dirk G. Everaert, and Jane M. Macpherson. Bilateral vestibular loss leads to active destabilization of balance during voluntary head turns in the standing cat. *J Neurophysiol* 95: 3783–3797, 2006. First published March 23, 2006; doi:10.1152/jn.00034.2006. The purpose of this study was to determine the source of postural instability in labyrinthectomized cats during lateral head turns. Cats were trained to maintain the head in a forward orientation and then perform a rapid, large-amplitude head turn to left or right in yaw, while standing freely on a force platform. Head turns were biomechanically complex with the primary movement in the yaw plane accompanied by an ipsilateral ear-down roll and nose-down pitch. Cats used a strategy of pushing off by activating extensors of the contralateral forelimb while using all four limbs to produce a rotational moment of force about the vertical axis. After bilateral labyrinthectomy, the initial components of the head turn and accompanying postural responses were hypermetric, but otherwise similar to those produced before the lesion. However, near the time of peak yaw velocity, the lesioned cats produced an unexpected burst in extensors of the contralateral limbs that thrust the body to the ipsilateral side, leading to falls. This postural error was in the frontal (roll) plane, even though the primary movement was a rotation in the horizontal (yaw) plane. The response error decreased in amplitude with compensation but did not disappear. We conclude that lack of vestibular input results in active destabilization of balance during voluntary head movement. We postulate that the postural imbalance arises from the misperception that the trunk was rolling contralaterally, based on signals from neck proprioceptors in the absence of vestibular inputs.

INTRODUCTION

The precise role of vestibular afferent input in posture and balance control is unclear, especially for voluntary movement. We previously showed that cats with bilateral vestibular loss respond normally to unexpected disturbances of standing balance in the horizontal plane (Inglis and Macpherson 1995; Macpherson and Inglis 1993). Yet, these cats are quite unstable when performing voluntary head turns during stance and locomotion, often losing their balance entirely (Thomson et al. 1991). Similarly, humans with bilateral vestibular loss show normal responses to translation during stance (Horak 1990; Horak et al. 1990). However, even well-compensated patients with bilateral vestibular loss exhibit ataxic gait when asked to turn their head while walking forward, and often adopt a

strategy of “fixing” the head to the trunk (Herdman 1994). Humans and animals lacking vestibular input demonstrate broad-based stance and ataxic gait and often show difficulty balancing in a variety of simple tasks (Lacour and Borel 1993; Marchand et al. 1988). Exactly why the loss of vestibular input has such a profound effect on standing balance under some conditions and not others is not known.

It is widely regarded that the postural control system integrates vestibular, somatosensory (cutaneous and proprioceptive), and visual sensory inputs for balance, and weights each input according to the context and prior experience of the task (Horak and Macpherson 1996). Together these inputs provide a complete picture of body orientation and dynamics within a particular environmental context, on which the responses required for maintaining balance during different postural control tasks are generated. It is believed that multiple sources of sensory input are required to resolve ambiguities regarding the position and motion of the body within the gravitational force field. We do not know how the loss of one input system, such as vestibular, affects the computation of body posture and movement.

One explanation may be that a loss of vestibular input leads to a miscalculation of trunk-in-space while the head is moving. *Trunk-in-space* refers to the orientation and motion of the trunk relative to Earth-based coordinates (such as line of gravity). It is widely accepted that inputs from vestibular receptors (semicircular canals and otoliths) combine with neck afferent inputs (muscle spindles, joint receptors) by vestibulospinal and cervicospinal reflex pathways, respectively, to maintain stance independent of head orientation (Pompeiano 1984; Roberts 1978; Wilson and Peterson 1981). It has also been suggested, from psychophysical studies in humans, that vestibular and neck afferent inputs are used for perception of the position and motion of trunk-in-space (Mergner et al. 1997). That is, trunk-in-space may be computed by the combination of the head-in-space signal (vestibular) and the head-on-trunk signal (neck proprioceptive). If so, the absence of either vestibular or neck proprioceptive information would lead to an erroneous estimate of trunk-in-space.

This study was designed to investigate the effect of absence of vestibular afferent input on balance control in the standing cat, during voluntary head movements to the left and right. Specifically, we sought to determine the underlying cause for

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the dramatic loss of balance during head turns immediately after bilateral labyrinthectomy. We began with the premise that, during head turns in the intact cat, vestibular and neck afferent inputs combine, to inform the nervous system that the trunk remains stable in space, that is, vestibular and neck signals effectively cancel one another. After labyrinthectomy, motion of the head-in-space is no longer sensed by vestibular afferents yet motion of the head-on-trunk continues to be sensed by neck afferents. Thus the unopposed neck afferent signal results in a large apparent error signal during movements of the head. This error signal represents a misperception of trunk-in-space, thus leading to an erroneous postural response and imbalance. We hypothesized that, if this schema is correct, then labyrinthectomized cats should produce inappropriate postural adjustments during active head turns. Our results show that balance was actively destabilized after inappropriate electromyographic (EMG) activity that was initiated after the head began turning. This finding supports the idea that vestibular input is necessary for accurately computing motion of the trunk-in-space while the head is moving relative to the trunk.

METHODS

Subjects and implantation

Experiments were conducted with the approval of the local Institutional Animal Care and Use Committee and conformed to the guidelines established by the National Institutes for Health regarding the care and treatment of animals. A total of six adult cats, all females (Br, Kn, So, St, Ti, and Ve), ranging in weight from 3.4 to 4.9 kg, were used in this study. After a training period, animals were implanted with pairs of multistranded stainless steel wires with Teflon insulation into 16 selected fore- and hindlimb, axial, and neck muscles for the measurement of EMG activity. After control experiments, three of the six cats (St, Ti, and Ve) underwent a complete bilateral labyrinthectomy (see details of the procedure below). For both procedures, animals were prepared for surgery in aseptic conditions and under general anesthesia.

Experimental protocol

Cats stood unrestrained on four force plates mounted on a movable platform, with their weight equally distributed between left and right sides. Training procedures were previously described in detail (Macpherson et al. 1987). Each animal stood at a constant fore-hindpaw distance during experiments, which corresponded to its preferred stance distance as determined during free stance on the laboratory floor. Each animal was trained using food reward and positive reinforcement to maintain quiet stance with its head oriented forward and level. They were then trained to make rapid voluntary head movements followed by a hold period, to their left and right sides (schematic in Fig. 1A). An experimenter stood in front of the animal and threw an object (such as a Frisbee or cat toy) to the animal's right or left side, to elicit large, rapid head movements. We found it necessary, especially for the postlesion condition, to use large objects of interest to the subjects, to maintain their attention and elicit the numbers of head movements required within and across days. Labyrinthectomized cats have great difficulty fixating and tracking small objects such as the lights that are typically used for visual-tracking experiments.

Each 5-s trial consisted of ≥ 1 s of quiet stance during which the animal's head was oriented forward, followed by a voluntary movement of the head in the direction of the cue, and the maintenance of a leftward or a rightward final head position. A food reward followed each correctly executed trial. Trials were eliminated if the head moved

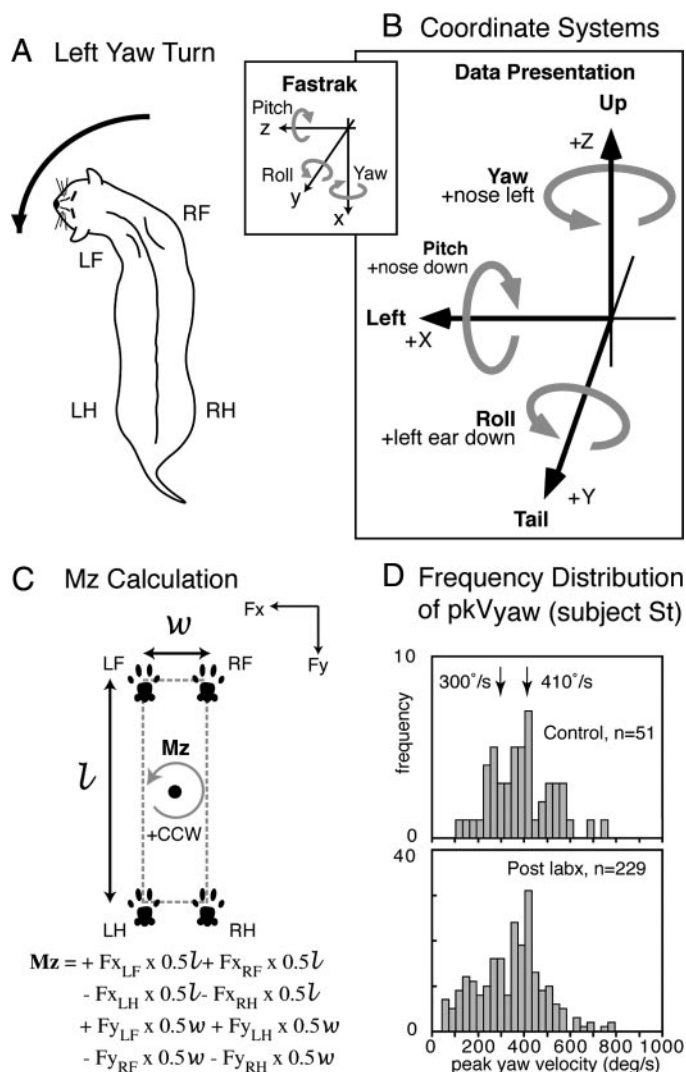


FIG. 1. *A*: top-down schematic view of a leftward head turn in yaw. *B*: coordinate systems. Data Presentation system is the primary reference frame, including body kinematics and ground reaction forces. Head angle data are shown according to the following convention: pitch was defined as the rotation of the head around the mediolateral (X) axis (positive = nose down), roll about the anteroposterior (Y) axis (positive = left ear down), and yaw about the vertical (Z) axis (positive = nose left). Fastrak reference frame shows the coordinate system of the Fastrak device during data collection. Note that the x -axis is opposite to and collinear with the z -axis of the Data Presentation frame; the z -axis is collinear with the x -axis of the Data Presentation frame; the y -axes are identical. *C*: formula for calculating total body moment of force about the z -axis, M_z , from force components F_x and F_y of the left and right forelimbs (LF, RF) and hindlimbs (LH, RH). l = center-center distance between front and rear force plates; w = center-center distance between left and right force plates; CCW, counterclockwise. *D*: frequency distribution of peak yaw velocity (absolute values) from control trials (above) and postlesion trials (below) of left and right turns for cat St. Of the 51 control trials, 17 (1/3) fell below 300°/s and 17 (1/3) fell above 410°/s. Averages were generated from trials falling within each third of the control data. Same cutoffs (300 and 410°/s) were used for the postlesion data, even though the frequency distribution was not identical to that of the control.

before the cue or if a limb was lifted off the force plate before or during the movement. During control sessions, ≤ 20 trials for each direction of head movement were collected (total 40 trials per session); ≤ 10 sessions of normal head movements were recorded from each animal.

Postural adjustments of the animals were quantified in terms of three-dimensional (3D) forces exerted by each paw against the sup-

port surface, EMG activity, head linear and angular position in space, and 3D positions of the body segments, bilaterally for all cats. Head position was recorded using a 6-df magnetic tracking device (Fastrak by Polhemus, Colchester, VT) and body kinematics were recorded using an optoelectronic system (Vicon, Lake Forest, CA). The Amlab system (Amlab Technologies, Lewisham, NSW, Australia) was used to collect force, raw EMG (1,000 sa/s), and Fastrak (100 sa/s) data and to trigger the Vicon collection (100 sa/s). The onset of data collection for each trial was timed to the video sync signal of the Vicon system.

Using a custom-built frame, the coordinate system of the Vicon was transformed before each recording session to be collinear with the gravity vector, Earth horizontal, and the long axis of the platform and thus collinear with the force recording system (Fig. 1B, Data Presentation). Passive reflective markers (7 mm in diameter) were placed at the following anatomical landmarks on both sides of the body: metacarpophalangeal (MCP); wrist, elbow, and shoulder (glenohumeral) joints; scapula tip at the top of the spine; metatarsophalangeal (MTP); ankle, knee, and hip joints; and the iliac crest of the pelvis. For sites over which the skin was particularly loose (e.g., knee, shoulder, scapula), the markers were placed using palpation of the joint while the cat was standing in position on the platform.

The Fastrak transmitter was mounted about 20 cm above the head of the cat and the receiver attached to the EMG connector, which was cemented to the skull. Using a custom-built device, the position and orientation of the transmitter were measured relative to the Earth-referenced system of the Vicon device before the series of experiments. The angular offsets were then programmed back into the Fastrak system at the beginning of each data collection session, to align the Fastrak coordinate system with Earth vertical and platform horizontal (Fig. 1B, Fastrak). Although aligned, the Fastrak coordinate system was rotated 90° about the *y*-axis relative to the Vicon and force system. The Fastrak data consisted of three linear positions (*x*, *y*, *z* in mm) and the quaternion (four components). We found the quaternion representation of angular position to be the most efficient and reliable method for on-line data acquisition. The position and orientation of the receiver relative to each cat's head were measured in stereotaxic coordinates post mortem.

Once the control data were collected, the vestibular system of each of three animals was lesioned bilaterally in one surgery using the technique described by Money and Scott (1962). An opening was made in the vestibule, and mechanical disruption was used to destroy the hair cells in the vestibule and neighboring ampullae. The horizontal and anterior canals were typically visualized and disrupted while drilling through the temporal bone. After surgery, animals were placed in a padded cage and allowed to recover from the anesthetic. Behavioral techniques used to evaluate the completeness of the lesion were described previously (Thomson et al. 1991). None of the three animals tested in our study showed signs of nystagmus or postrotatory nystagmus at any time during the postlesion monitoring period (≤ 1 mo). All animals were unable to right themselves when dropped without vision from a supine or upside-down position onto a thick foam surface (Money and Scott 1962; Watt 1976).

Data analysis

The various types of data were all imported into one file for each trial and subsequent analysis was performed using MATLAB (The MathWorks, Natick, MA). Head linear displacement data were expressed in the Vicon coordinate system based on the measured position of the electrical center of the Fastrak transmitter. Head angular displacement was expressed as Euler angles (computed from the quaternion), of pitch, roll, and yaw rotations about the Fastrak *z*-, *y*-, and *x*-axes, respectively. The angular data were then transformed relative to stereotaxic zero based on the post mortem measures of the Fastrak receiver position for each cat. For simplicity, the head angular data are shown in the figures according to the main coordinate system illustrated in Fig. 1B, Data Presentation, to conform to the force and

Vicon coordinate system. Note the only difference with the Fastrak system is the reversal in sign for the yaw rotation (nose-left turn is expressed as positive). However, it is important to note that all data transformations used a rotation matrix in the original recording coordinate system because of the noncommutative nature of rotation axes.

Data were filtered and processed off-line using custom MATLAB routines, applying a fourth-order Butterworth filter with zero phase shift: force data were filtered at 100 Hz, kinematic data at 7 Hz, and EMG data were high-pass filtered at 35 Hz, demeaned, rectified, and low-pass filtered at 30 Hz. Head angular velocity was computed from the position data. Force plate data were used to compute ground reaction force (GRF) vectors in three planes and the position of the center of pressure (CoP) in the horizontal plane (resultant of the vertical forces). In addition, the horizontal plane forces from the four limbs were used to calculate whole body moment of force (Mz) around a vertical axis passing through a point central to the four force plates (Fig. 1C).

For each trial, the onset and end of head movements were determined from the yaw angular velocity. From the 5-s acquisition period, data were analyzed for a 2.5-s period, 1 s before and 1.5 s after the onset of head movements, and averaged both to the onset and peak of angular velocity of the yaw head movement. Trials were selected for analysis based on the following criteria: 1) the initial head position was within ± 20 deg of zero in the yaw plane; 2) yaw angular velocity had a relatively uniform, monophasic initial peak (although some smaller peaks on the trailing end were allowed, as shown in Fig. 2A); and 3) after the initial peak, yaw velocity did not cross zero or increase significantly in the opposite direction. In other words, the head either remained steady at the final position or rotated in the same direction in a second movement, but never reversed in movement toward the initial position. The criteria had to be relaxed for the postlesion trials because the head movements were more irregular and ataxic and the velocity profiles were often segmented. However, movements with a reversal in yaw position were excluded.

RESULTS

In summary, head turns before and after labyrinthectomy were characterized by simultaneous rotation of the head and scapular girdle in yaw and lateral bending of the anterior trunk, accompanied by a rotational moment of force (Mz) at the ground around a vertical axis. Postural adjustments for the head turns were similar before and after lesion until near the time of peak angular velocity, after which the lesioned animals became unstable and leaned or fell, most often to the ipsilateral side with respect to the direction of head movement.

Postural strategies accompanying head movements in the intact cat

Leftward yaw movements were accompanied by left ear-down roll rotations, as illustrated in Fig. 2A for the representative single trial from subject So. Similarly, rightward yaw turns were accompanied by right ear-down roll rotations. In both left and right turns, the head pitched in a nose-down direction. Left and right head turns were reasonably symmetric but, for clarity, only leftward turns will be shown in illustrations. Some of the EMGs in the figures are from right turns and have therefore been reflected to the opposite limb, for illustration purposes.

The motion of the body over time is shown in the series of stick figures in Fig. 2B, with the same trial shown from two different viewpoints. The initial position (Fig. 2B, black sticks)

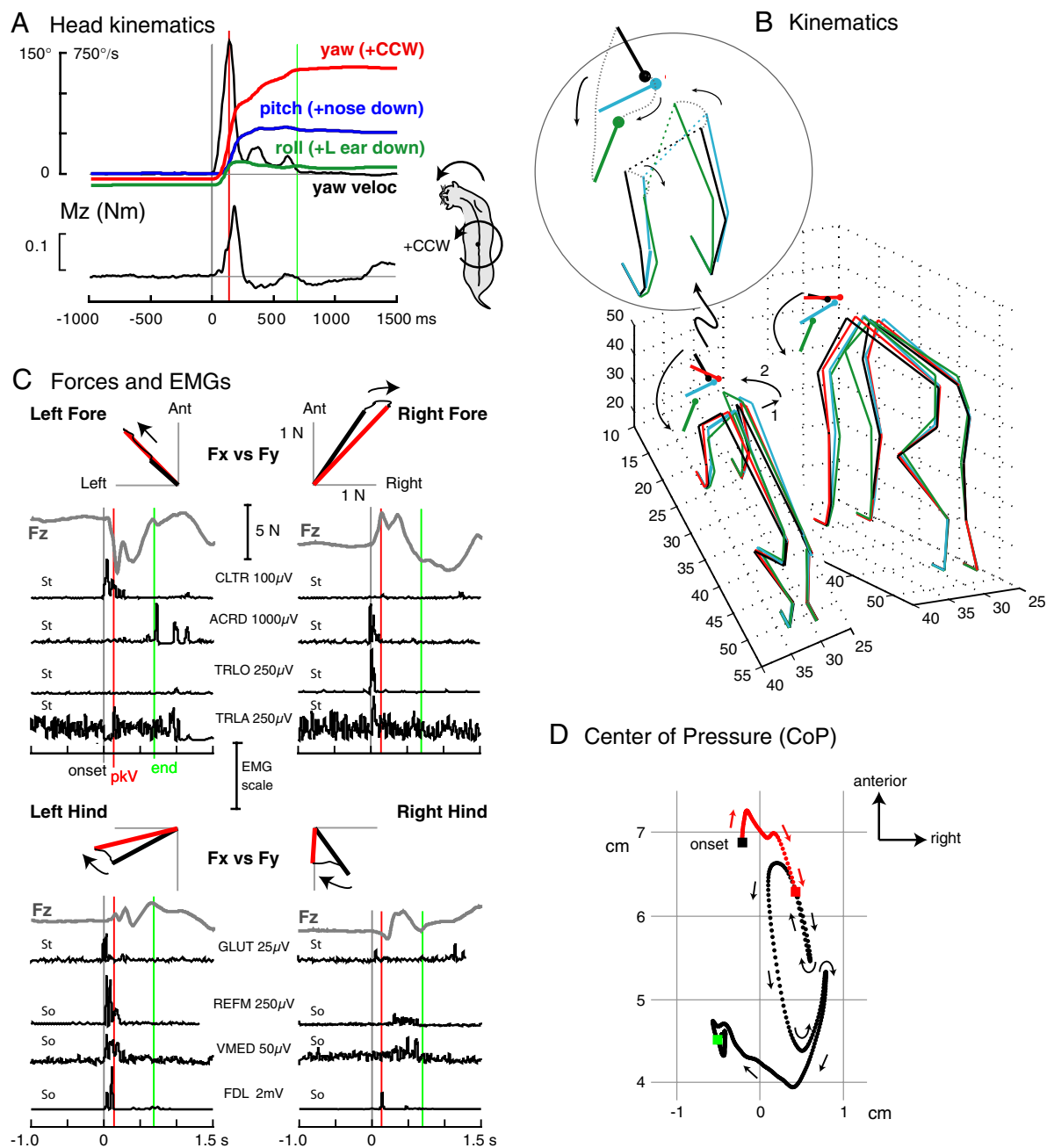


FIG. 2. Characteristics of a left head turn in an intact cat (one representative trial, cat So). **A**: head angular position in yaw (red trace), pitch (blue trace), and roll (green trace) from 1 s before to 1.5 s after the onset of the head movement (vertical gray line). Head yaw angular velocity is shown in black. Vertical red line marks the time of peak yaw angular velocity. Whole body moment of force around the vertical axis (M_z) is shown in the bottom panel. **B**: 3D kinematics of head and body motion over time from 2 different perspectives (top and side views). Black sticks show the initial position of each segment 200 ms before the onset of head movement; red sticks, at the peak of yaw angular velocity; cyan sticks, when the forequarters are maximally displaced to the contralateral (right) side; and green sticks, the final position of the body at the end of head movement. For each time point, the head is represented by a circle showing the computed position of the skull–C1 joint and a line indicating the orientation of the head in space, relative to stereotaxic zero. *Circled inset*: enlargement of the 2 forelimb sticks (from paws to shoulder joints) and the head symbol at 3 time points. Left and right shoulder markers are joined by dotted lines of the corresponding color, to highlight the rotation of the shoulder girdle. Note how the 2 shoulder joints traverse arcs in opposite directions (gray dotted lines). Skull–C1 joint moves laterally as the head rotates in yaw. Axis units in centimeters. **C**: forces and EMGs. Representative EMGs and vertical ground reaction force (F_z) are plotted for each limb over time with onset, peak yaw velocity, and end of movement indicated by the gray, red, and green lines, respectively. EMGs are a composite from 2 subjects, St and So. At the top of the traces for each limb are shown horizontal plane force vectors (F_x vs F_y) plotted at the time of onset (black) and peak angular velocity (red) of the yaw head movement. Thin lines indicate trajectories of the vector endpoints over time. Vectors are plotted from a single origin for each paw and represent the cat-generated forces that are equal and opposite to the ground reaction force (GRF). Arrows indicate the CW rotation of the vectors in the horizontal plane. **D**: trajectory of center of pressure (CoP) in the horizontal plane. Dots are plotted every 5 ms, with red before peak yaw velocity and black after. Black, red, and green squares represent the times of onset, peak yaw velocity, and end of the head movement, respectively. Arrows indicate direction of motion across various time points.

shows the head oriented forward. As the head turns to the left, the anterior trunk moves rightward (Fig. 2B, arrow 1, red, cyan sticks). The shoulder girdle then simultaneously moves forward and rotates counterclockwise (Fig. 2B, arrow 2, green sticks) as the trunk flexes laterally (trunk flexion was not captured by the kinematic recordings but was visually obvious during data collection). At the end of head movement, the anterior trunk moves back toward the left while the shoulder girdle remains rotated and the trunk laterally flexed. The hindlimbs and pelvis show little motion, except for a slight extension toward the end of the head turn. This sequence of head and trunk motion varied somewhat across subjects but the basic characteristics were robust and repeatable.

The contralateral forelimb exhibits an increase in vertical force (F_z) and the ipsilateral forelimb a decrease, simultaneous with movement onset (Fig. 2C). The hindlimbs typically show little change in vertical force until after the peak in yaw angular velocity. The net effect of changes in F_z is shown in the motion of the CoP (Fig. 2D). The CoP initially moves forward then rightward, reflecting the push-off of the contralateral forelimb at the initiation of the leftward head turn. After peak yaw velocity, the CoP moves backward as the hindlimbs increase F_z .

Three of the four limbs produce a clockwise (CW) rotation of the horizontal plane force vector (F_x vs. F_y) at the support surface (Fig. 2C, shown as cat generated forces that are equal and opposite to the GRF). The horizontal plane force components give rise to the ground reaction moment, M_z , which rotates in a counterclockwise (CCW) direction and reflects the angular acceleration of the body about a central vertical axis, as the animal performs the head movement (Fig. 2A). All but the slowest head turns are accompanied by a reaction moment, M_z , in the same direction as the head turn.

Figure 2C illustrates the basic muscle activation pattern accompanying a leftward head turn. The onset of the head movement is preceded by activation of extensors of the right forelimb [acromiodeltoideus (ACRD), long head of triceps brachii (TRLO), lateral head of triceps brachii (TRLA)], which contribute not only to support of this limb (increased F_z), but also to the rotation of the horizontal plane force vector (and thus M_z). The unloading of the left forelimb is accompanied by reduced activity in TRLA. The activation of cleidotrapezius (CLTR) in this limb may contribute to the head turn and/or the increase in horizontal plane force vector amplitude. In the ipsilateral (left) hindlimb, the abductor, gluteus medius (GLUT), and anterior muscles, rectus femoris (REFM), vastus medialis (VMED), and flexor digitorum longus (FDL), likely contribute primarily to rotation of the horizontal plane force vector because there is little change in the support force (F_z) before peak yaw velocity. None of the recorded muscles of the contralateral, right hindlimb showed a significant increase in activity, so it is not clear whether the contribution of that limb to M_z was passive or active. Because the force vector rotated in the adduction direction, the relevant muscles may not have been sampled.

Head turns from all subjects exhibited a wide range of peak amplitude and peak yaw velocity. Trials were averaged in three groups based on peak velocity. Left and right head turns for control data were combined to generate a frequency distribution of absolute values of peak yaw velocity within each subject (e.g., Fig. 1D, cat St). The distribution was divided into

thirds to generate the cutoff velocity values for each group average. These same cutoffs were used for the postlesion data within a subject, regardless of the distribution of that data set. This method was found to produce the best matching of average peak velocity before and after lesion for each of slow, medium, and fast head turns. Angular displacement of the head in roll was consistently toward the side of the head turn (i.e., left yaw and left-ear-down roll) but the peak amplitude and velocity of the roll motion were considerably smaller than in yaw, as expected. Peak roll angular velocity was linearly related to peak yaw velocity (Fig. 4, middle), but the slope varied across subjects (0.14, 0.18, 0.25, 0.27, 0.29, and 0.41 deg/s of roll per deg/s of yaw across the six subjects). M_z increased with peak yaw velocity across all subjects as illustrated in Fig. 3A (bottom traces). The initial peak of M_z increased linearly with peak yaw velocity (Fig. 4, left) with a characteristic slope for each subject. On average, CoP moved a small distance contralaterally along the x -dimension until the time of peak yaw velocity, consistent with the vertical force changes in the forelimbs (Fig. 3B). After peak velocity, the CoP reversed direction along the x -dimension and ended in a position ipsilateral to the starting point, consistent with the shift of the head and shoulder girdle to the side of the head turn. The anteroposterior motion of the CoP was more variable (Fig. 3B).

Postural strategies accompanying head movements in the labyrinthectomized cat

For the first several days to 1 wk after labyrinthectomy, cats were ataxic and exhibited the behaviors previously described in detail (Thomson et al. 1991). Initially, all three cats required light touch for balance while standing on the platform, and frequently lost balance or stepped during head turns (Table 1). Actual falls were prevented by the experimenter. Only those trials in which the cat stood without support before the head turn were accepted for further analysis. Trials classified as "supported" could not be analyzed because the ground reaction forces were contaminated by the force generated by the experimenter.

In the postlesion animal, the initial phase of the head movement was similar to that of the control trials. However, around the time of peak yaw velocity, the cat initiated a thrust, which propelled the body to the side of the head turn, often with enough force to cause falling. As the animals compensated over time for the loss of vestibular inputs, the amplitude of this thrust decreased and balance was more easily maintained.

Figure 5 illustrates these features in a side-by-side comparison of two single trials from cat St, one on the 4th day after lesion and the other from the control data set. Trials were matched by peak yaw velocity. The trajectory of head movement during the leftward turn was similar across the two trials (Fig. 5A), but two characteristic features stand out in the postlesion case: 1) a rhythmic tremor is obvious in the roll angular position signal; and 2) the head turn shows a large overshoot and return in the yaw signal as well as M_z , characteristic of vestibular hypermetria. The contralateral (right) forelimb extensors, TRLA and TRLO, show an initial burst related to the thrust of the limb, which contributes to the initiation of the head turn. The slightly larger amplitude in the

A Averages for Left Head Turn Across 3 Velocity Ranges

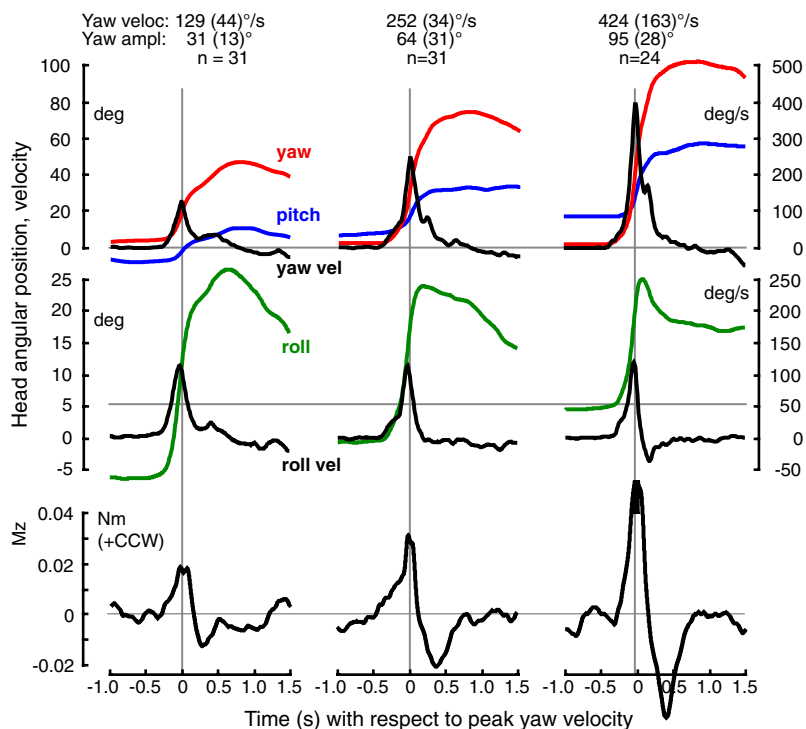
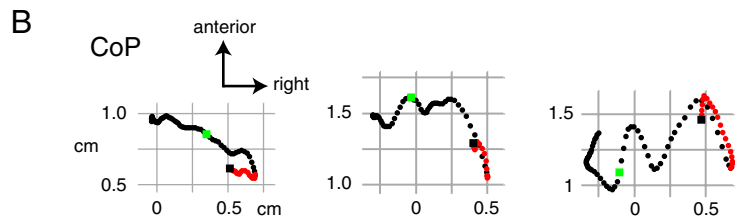


FIG. 3. Left head turn data averaged across trials by velocity, for cat Ti. Trials were grouped according to peak yaw velocity and averaged for slow, medium, and fast head turns [mean (SD) velocity and amplitude for the 3 groups is shown at the top of each column of figures]. Velocity cutoffs were based on combined left and right turns and so these averages for left turns only do not have equal numbers of trials. *A*: time traces of head angular position and velocity, and moment about the z-axis. Note that Mz increases with peak yaw velocity (conventions as in Fig. 2A). *B*: average horizontal plane CoP displacements for each velocity (conventions as in Fig. 2D).



postlesion trial is consistent with the hypermetria. The most marked effect of vestibular loss is seen near the time of peak yaw velocity (Fig. 5A, gray bars) when there is an additional burst of EMG activity in the postlesion trial but not the control. This abnormal EMG is closely followed by a second peak in the force traces that is not present in the control trial.

Like the EMGs, the initial period of force trajectories in horizontal and frontal planes is similar in control and postlesion trials (Fig. 5B). The initial direction of the horizontal plane force vectors is more laterally directed in the postlesion trial, suggesting a strategy of bracing to stabilize stance. Nevertheless, the vectors rotate in similar manner to the control trial up

to the time of peak yaw velocity (Fig. 5B, top, gray arrows). Near the time of peak yaw velocity, the postlesion trial exhibits a large lateral and downward thrust from both fore- and hindlimb on the contralateral (right) side. The black arrows in the top left of Fig. 5B show the direction of thrust for the right limbs in the horizontal plane. Figure 5B, bottom left shows the net frontal plane vector from both fore- and hindlimb on the right side of the cat. Note that the initial rightward and downward force trajectory (red dots) shows a return toward the origin by the end of peak yaw velocity. This first force pattern is similar to the control and is part of the normal initiation of the head turn. The trajectory then abruptly changes direction

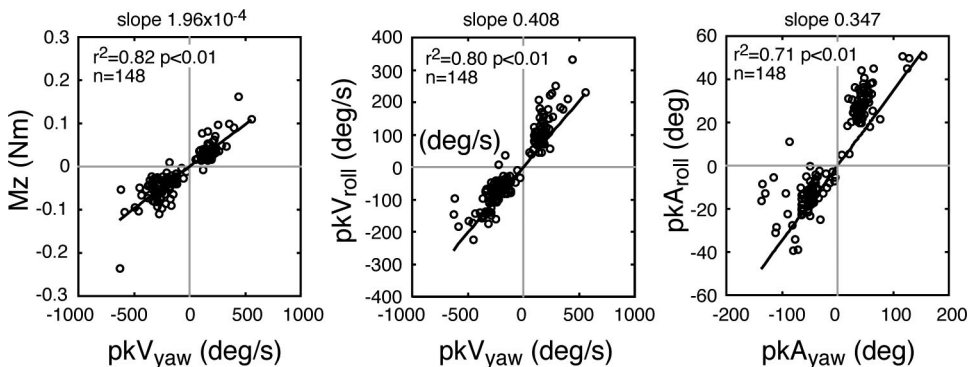


FIG. 4. Linear regressions among dynamic variables, for individual trials of both left and right turns, from cat Ve. *Left*: peak yaw velocity vs. peak Mz. *Middle*: peak yaw velocity vs. peak roll velocity. *Right*: peak yaw amplitude vs. peak roll amplitude. By definition, left turns have positive amplitude and velocity values, and right turns, negative values.

TABLE 1. Numbers of head-turn trials classified by behavior after labyrinthectomy

Subject	Days Postlesion	Supported	Balanced	Falls	Step/Lift
St	1-3	34	42	13	16
	4-7	1	121	5	9
	11-19	0	143	1	11
	43	0	58	0	0
Ve	4-8	0	229	1	18
	11-31	0	242	0	2
	37-43	0	84	0	0
Ti	1-3	2	79	14	5
	4-8	0	133	0	3
	9-38	0	203	0	2

Values are means \pm SD. For supported trials, the experimenter provided light touch before and during head turn. For the remainder of the trials, cats were freely standing before head turn and ended the trial in one of three conditions: 1) balanced = remained standing, feet in place; 2) falls = falling during head turn, which required a catch by the experimenter; and 3) step-lift = head turn induced stepping off force plate by one or more paws, or lifting and replacing of one or more paws.

and points downward and outward a second time (see black dots, black arrow). The outcome of this second thrust down and to the right is a fall to the left, as illustrated in the kinematic trajectories. This response was consistent across all three labyrinthectomized cats (Fig. 7).

The stick figures of Fig. 5C show the similarities in the initial phase of the head movement in the control and postlesion trials. In both, the anterior trunk moves slightly to the right (Fig. 5C, arrow 1) as the head begins rotating to the left. Then, the shoulder girdle rotates CCW in yaw and returns to the left as the head movement is completing (Fig. 5C, arrow 2). However, as a result of the abnormal rightward thrust in the postlesion trial, the trunk then accelerates to the left and the cat falls to that side (Fig. 5C, bottom stick figures; note the right forepaw has stepped to the left while the right hind lifts off the force plate).

Figure 6 compares control and postlesion data for cat St across three velocity ranges, averaged with respect to peak yaw velocity. Trials from the 1st wk only were included in these postlesion data, to eliminate any significant effects of compensation. On average, the control and postlesion data were well matched for yaw velocity and amplitude profiles (Fig. 6A, top traces). Peak Mz was larger in the postlesion case across all velocities, and followed by a reversal, especially at the highest velocity. This was reflected in the increase in slope in the relationship between peak yaw velocity and peak Mz for all three lesioned cats (Fig. 9). The frontal plane force trajectories of the right side illustrate the abnormal thrust that follows peak yaw velocity in the postlesion averages (Fig. 6B). The downward and rightward force after peak yaw velocity increased with velocity of the head turn.

Figure 6C shows a composite from all three cats of averaged EMGs, comparing pre- and postlesion activity across the three velocity ranges. Extensors of the contralateral fore- and hindlimbs show the large activation just before peak yaw velocity that characterizes the postlesion data (Fig. 6C; see, e.g., gray bars in traces of velocity range 3). Also of interest is the reciprocal inhibition in some extensors of the ipsilateral fore- and hindlimb (Fig. 6C, gray bars in TRLA, GLUT). The late extensor activation in the ipsilateral limbs is a reaction to

increased loading of those limbs as the body is thrust to the ipsilateral side. The EMG, kinetic, and kinematic data suggest that the lesioned animal actively destabilizes its balance after the head movement is under way, by applying force against the ground to drive the body toward the ipsilateral side.

One might suppose that overbalancing to the ipsilateral side after vestibular lesion merely arises from hypermetria during the latter part of the head turn, when the body normally follows the head as in the control condition. The postlesion hypermetria is exemplified by the increase in peak Mz, which indicates that the net acceleration of the body around a central vertical axis was higher than that in the control case, for a given peak velocity of head turn. Was the excessive rotational acceleration sufficient to cause instability and falling? To explore this possibility, head turns were studied in two cats, which demonstrated similar hypermetria during head turns. These cats had somatosensory loss but normal vestibular function. Somatosensory loss was induced by pyridoxine intoxication, which causes loss of peripheral afferent fibers in the diameter range of $\geq 7-9 \mu\text{m}$, affecting primarily group I muscle and large cutaneous afferents of the limbs (Stapley et al. 2002). Both subjects (Br and Kn) showed hypermetria as evidenced by the higher-peak Mz in the postlesion head turn compared with control (Fig. 7). Nevertheless, neither somatosensory loss animal showed any evidence of abnormal thrust in the frontal plane force trajectories from the contralateral limbs (Fig. 7, far right column). Instead, the force trajectories are disorganized and irregular compared with control.

Change in voluntary head turns with time vestibular compensation

Head turns were recorded up to about 40 days after labyrinthectomy. As previously reported by Thomson et al. (1991) animals showed the greatest ataxia and instability during the first 3 days after labyrinthectomy. Some improvement was observed by the end of the first week. By the end of the month, the animals were able to run in the lab and even negotiate cornering without falling. The head turn data were divided into three to four periods and averaged by velocity to examine the extent of recovery. Only trials in which the animals were able to stand unaided on the platform were included. Cats Ti and St were able to stand independently for a portion of trials from the first day. Cat Ve, although able to stand independently on the floor, could not stand on the platform without light support until day 3 and would not produce head turns until day 4. Figure 8 shows plots of frontal plane force trajectories for the contralateral limbs before lesion and during recovery for all three subjects for the highest velocity average. During the acute phase (days 1-3) animals were highly ataxic and at high risk for falls as mentioned above (Table 1). Frontal plane force generated at the time of peak head velocity was very large and resulted in falling or stepping to the ipsilateral side with respect to the head turn. Over time, all animals showed considerable improvement in balance and were able to remain standing, feet in place, after head turns. Even though the amplitude of the frontal plane force decreased over time, the abnormal thrust downward and contralateral remained and the profile of the force vector trajectory never returned to the control pattern. Thus vestibular compensation was accompanied by a reduction in amplitude of the active destabilization but no modification of

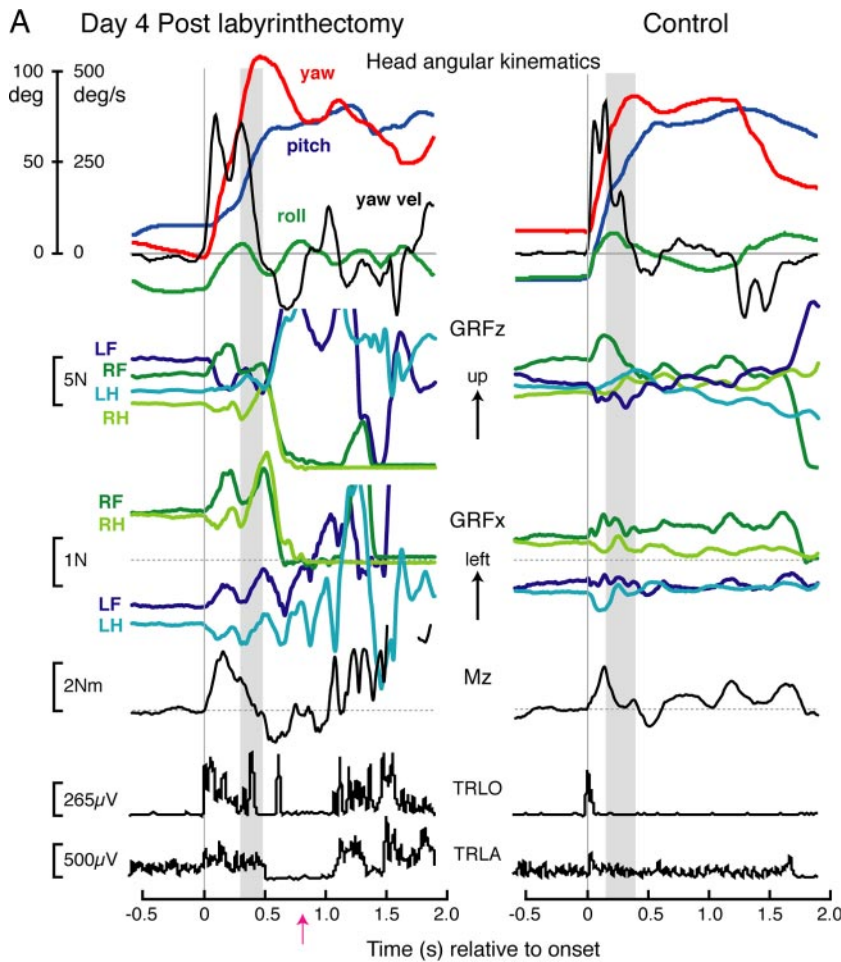
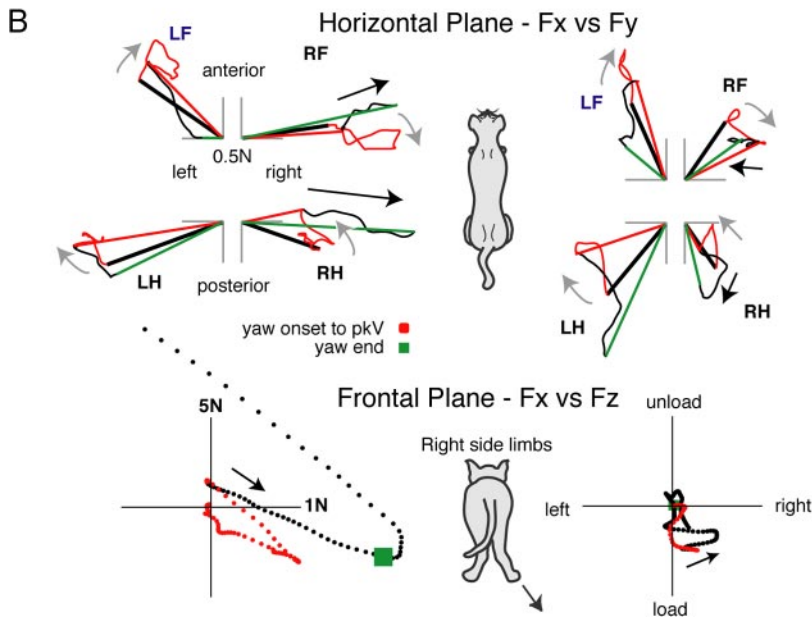


FIG. 5. Representative trials of a left head turn in cat St, before and 4 days after labyrinthectomy. *A*: head angular kinematics, ground reaction force components (GRFz, GRFx), moment (Mz), and right forelimb extensor EMGs (TRLO, TRLA) plotted against time relative to onset of head movement at *time 0* (vertical gray line). Trials were matched by similar peak yaw velocity and segmentation of the yaw velocity curve. Note the similarity between lesion and control trials from onset of head movement to peak velocity. Vertical gray bars highlight the period from peak yaw velocity to the end of head movement. Note during this period in the postlesion trial, the increase in vertical and lateral forces of the right limbs (green traces) and the extra burst of activity in the forelimb extensors compared with the control trial, suggesting an active push-off by the right side limbs. Pink arrow beneath the time axis indicates the moment at which the right limbs lose contact with the force plates as the animal falls to the ipsilateral (left) side. *B, top*: horizontal plane vector trajectories of cat-generated force (conventions as in Fig. 2C). *Bottom*: cat-generated force in the frontal plane. Dots represent the change in net force from background (Fx vs. Fz) for the right side fore- and hindlimbs combined. Red dots are values from onset to peak velocity of the head movement; black dots, from peak velocity to stable posture after the end of head movement (control) or to the time the right limbs lost contact with the force plates (postlesion). Time between each dot is 5 ms. Forces at the end of head movement are represented by the green lines in the horizontal plane plots and the green square in the frontal plane plots. Gray arrows indicate the rotation of the force vectors in the horizontal plane from onset to peak yaw velocity, which generate the moment about the z-axis. Black arrows highlight the differences between postlesion and control trials for the period after peak yaw velocity. Note the large rightward thrust in the postlesion trial. This thrust is also seen in the frontal plane (*bottom plots*) where the combined force of the right fore- and hindlimbs is downward and to the right, beginning at the time of peak yaw velocity. LF, RF left, right forelimb; LH, RH left, right hindlimb.



direction of the erroneous response. The relationship between peak yaw velocity and peak Mz showed a dramatic increase in slope during the initial period after lesion, compared with control (Fig. 9). The slope decreased over time but did not return to control levels, suggesting that the hypermetria was reduced but not completely compensated.

DISCUSSION

Cats that underwent a complete bilateral labyrinthectomy showed frequent instability and loss of balance during voluntary head turns. The initial components of the head turn and accompanying postural responses were hypermetric, but simi-

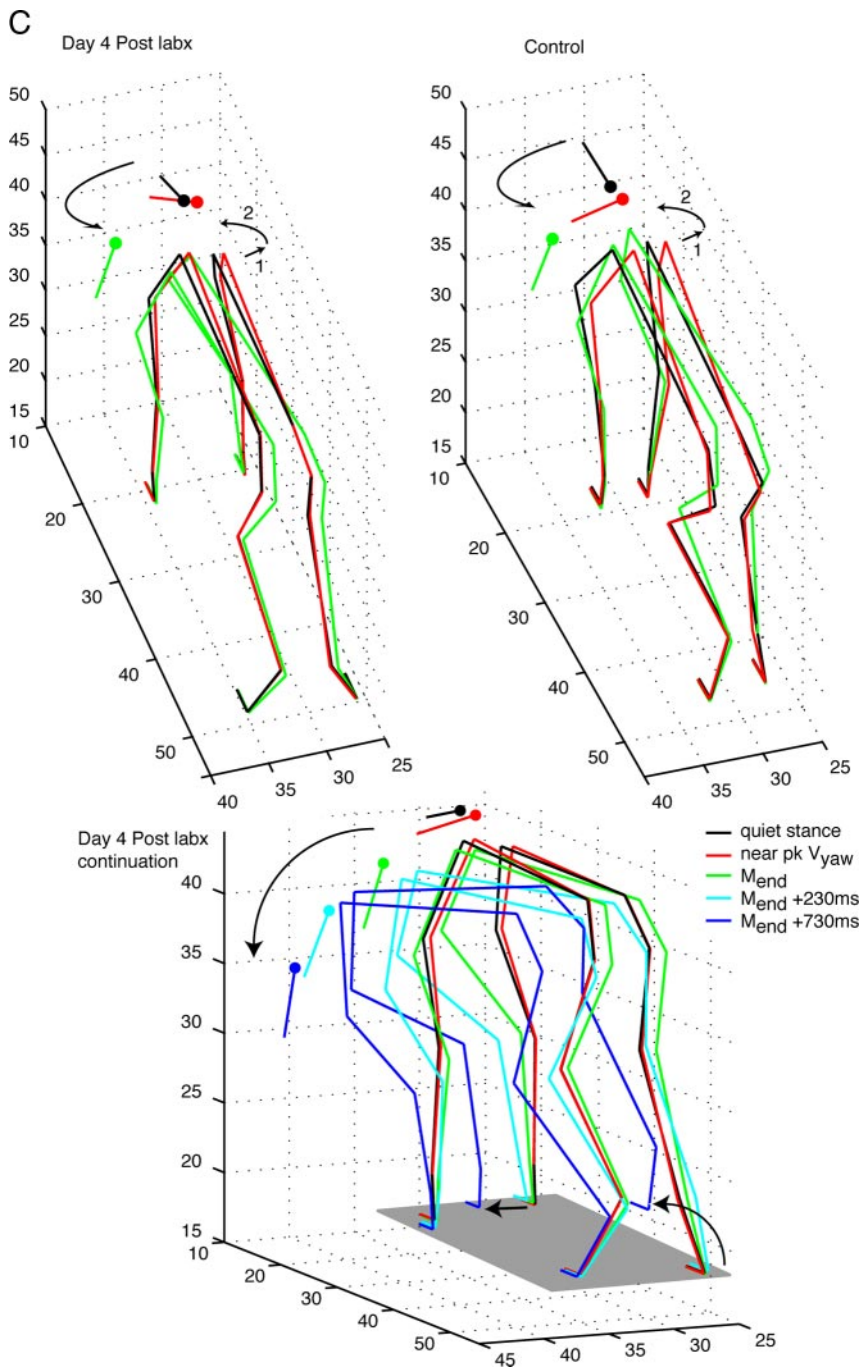


FIG. 5. (continued). C: stick figures illustrating body motion during the same trials shown in A and B. Top 2 panels highlight the initial similarity in body motion before and after lesion, as the forequarters move first to the right (red sticks), then back to the left as the head turns left. Bottom panel: continuation of the postlesion trial in which the animal falls to the ipsilateral side, losing contact with the right side force plates and stepping to the left (see arrows). Axis units in centimeters.

lar to those produced before the lesion was made. Unlike in control trials, however, the lesioned cat produced bursts in extensor muscles of the contralateral fore- and hindlimbs near the time of peak yaw velocity, which thrust the body to the ipsilateral side, leading to falls. We conclude that lack of vestibular input results in an active destabilization of balance during voluntary head movement. Furthermore, we postulate that this is explained by the erroneous perception by the nervous system that the trunk was falling to the contralateral side as the head turned in yaw, leading to a “corrective” postural response, which was, in fact, destabilizing. The inability of vision to compensate for the vestibular loss is likely attributable to blurring of the visual image during

the rapid head movement, as a result of an absent vestibuloocular reflex.

That the lesioned animal actively generates a fall is clear from the sequence of events during the voluntary head turn. The falling motion of the body was consistently preceded by a downward and outward force generated by the limbs contralateral to the side of the head turn. Furthermore, this force was preceded by activation of contralateral extensor EMGs, which was not observed in the control head turns. The abnormal force was a linear thrust primarily in the frontal plane, in contradistinction to the rotational force, M_z , which accompanied the head turn in both control and lesioned trials. Finally, the abnormal EMG burst began after movement initiation and near

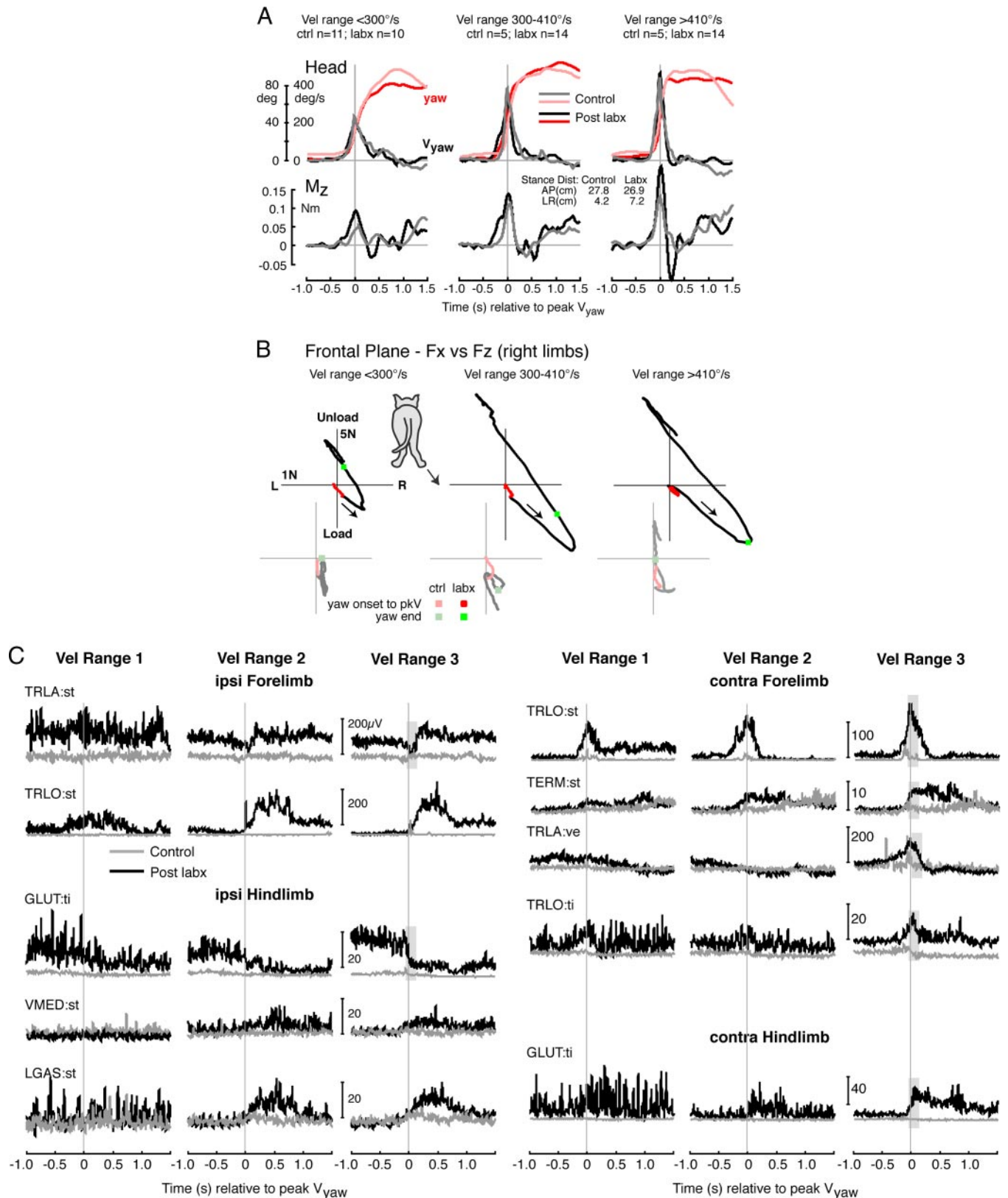


FIG. 6. Left head turn data averaged across trials by velocity, for control and the 1st week postlabyrinthectomy (post labx) trials (cat St). Data were averaged on the time of peak yaw velocity. *A*: head kinematics for control (gray and pink traces) and post labx (black and red traces) averages were well matched for each of the 3 mean peak velocities. Nevertheless, the peak moment about the z-axis (Mz) was larger after lesion. *B*: averaged change in frontal plane force. Conventions as in Fig. 5*B*. Note in the post labx data the increase in the downward and rightward force with velocity, after the time of peak yaw velocity (black trajectories). *C*: EMG activity from the ipsi and contra fore- and hindlimbs from control (gray traces) and post labx (black traces) data averaged on peak yaw velocity (vertical gray line), across 3 velocity ranges. Muscle names are as in Fig. 2; LGAS, lateral gastrocnemius. EMGs from each of the 3 animals (ti, ve, st) are represented and the source indicated alongside the muscle name. Gray bars highlight post labx activity around the time of peak yaw velocity, consistent with a thrust of the contralateral limbs leading to active destabilization of the body.

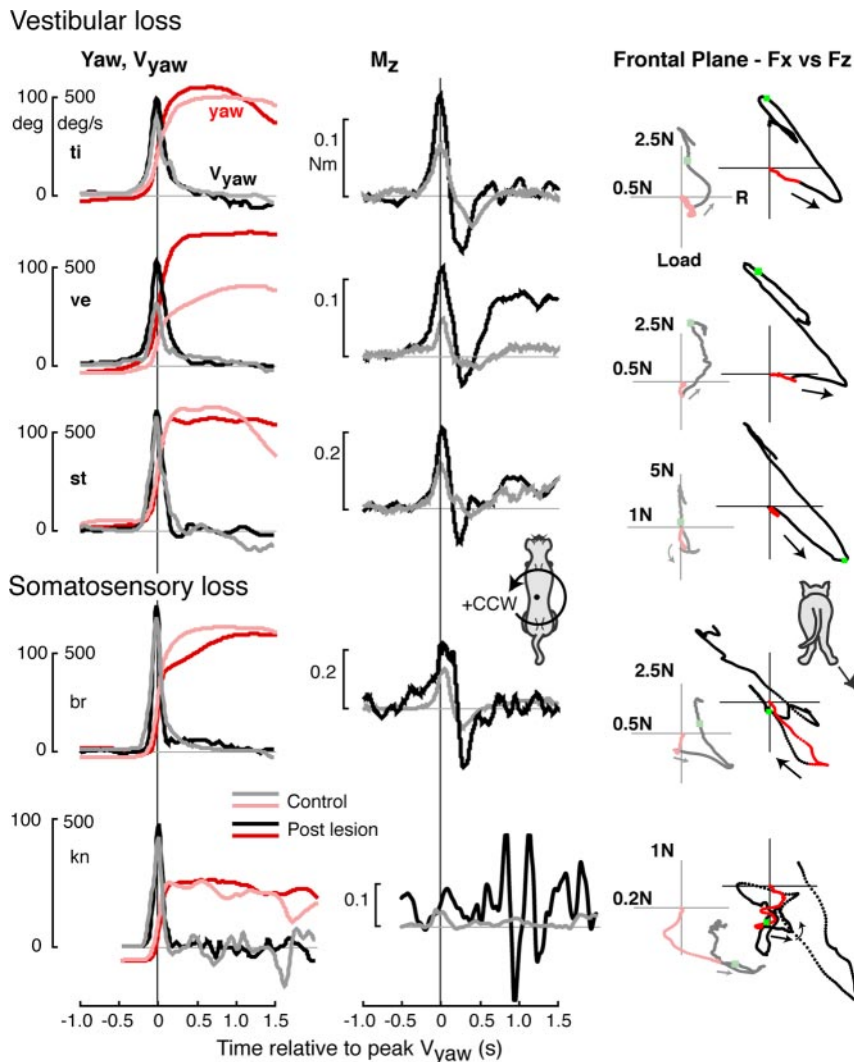


FIG. 7. Head kinematics, M_z , and change in frontal plane force averaged with respect to peak yaw velocity for the highest-velocity group within each cat. Pre- and postlesion data are shown for the 3 vestibular cats (above), and 2 somatosensory-loss cats (below). Note the hypermetria in the M_z traces for all cats. Only the vestibular-loss animals displayed the downward and rightward force thrust beginning around the time of peak yaw velocity. Force trajectories for the somatosensory-loss animals were more disorganized and erratic. Arrows indicate the direction of the frontal plane force vector trajectory immediately after peak yaw velocity.

the time of peak yaw velocity, suggesting that the imbalance was linked to some feature of the head movement.

The question arises, then, as to the origin of the active destabilization characteristic of the labyrinthectomized animal. Because the abnormal EMG bursts started after the head was in motion, it is likely they were initiated by sensory feedback from the ongoing head movement itself. The relevant sensory signals would likely be those occurring ≥ 40 ms before the onset of the abnormal EMG bursts, to allow time for the postural system to process the inputs and generate the response. The fall typically occurred in the frontal plane, leading us to suggest that the critical afferent signal that triggered the fall was linked to motion of the head in the frontal (roll) plane. As shown in Figs. 2, 3, and 5, a voluntary head turn is accompanied by a stereotypical roll rotation of the head in the direction of ipsilateral ear down. During the head turn, the intact cat receives both vestibular signals encoding velocity of head roll in space and neck proprioceptive signals of head roll with respect to the trunk. When the head-on-trunk is subtracted from head-in-space, the result is zero, which indicates that the trunk is not moving and therefore no postural correction is necessary. In contrast, the lesioned cat lacks the signal of head roll in space; thus we suggest that the neck proprioceptive input of head-on-trunk, in the absence of an accompanying head-in-

space input, is interpreted as the body rolling under a stable head. In other words, the lesioned animal perceives that their trunk is falling in the frontal or roll plane, rather than the head rolling on a stable trunk. Figure 10 illustrates how a left ear-down roll of the head, which accompanies a left turn, could be misinterpreted as a rightward fall of the body in the absence of vestibular input. Such a misperception would trigger an erroneous postural response to thrust the body to the ipsilateral or left side, consistent with our observations.

The absence of otolith inputs may also contribute to an illusory body motion to the contralateral side. The head turns in our study were characterized by a significant translation component, ipsilateral and backward (e.g., Figs. 2B and 5C), which would normally stimulate the otolith organs. After labyrinthectomy, a leftward head turn could be misinterpreted as a rightward linear translation of the body in space, thus reinforcing the illusion in the roll plane.

Concerning the yaw rotation, there is no evidence that the lesioned cat misperceived the rotation of the body about the vertical axis. Perhaps the combination of the motor command (efference copy), visual feedback, and limb proprioceptive inputs relating to M_z were sufficient to override the lack of a yaw vestibular signal and indicate the successful completion of the primary goal of a gaze shift. The nose-down pitch rotation

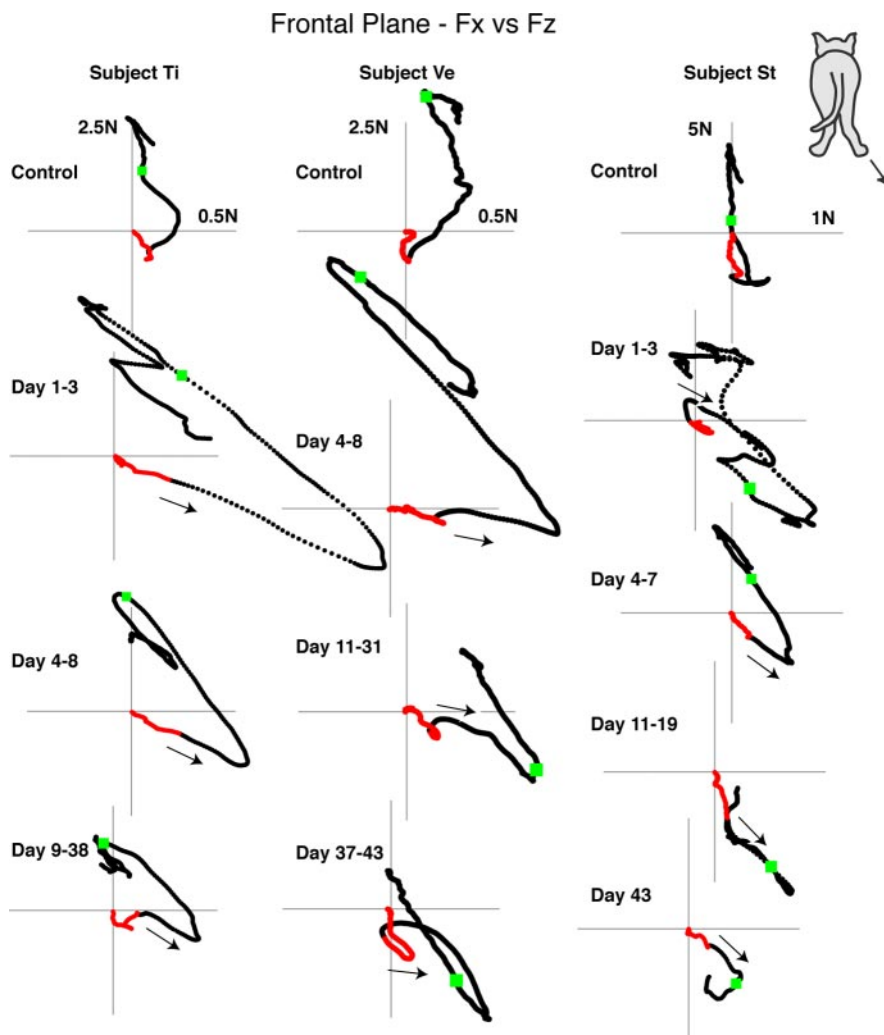


FIG. 8. Change in frontal plane forces before lesion and during the vestibular compensation period. Frontal plane force trajectories were averaged for the periods indicated for each cat after lesion, for the highest-velocity trials and with respect to peak yaw velocity. Arrows indicate direction of the frontal plane force trajectory after peak yaw velocity. Note the change in orientation of the trajectories for control compared with postlesion data. Note further that the basic shape of the trajectories does not change over days postlesion, whereas the extent, or amplitude, becomes smaller.

that accompanied the head turn may have generated a misperception that the trunk was rotating tail down in pitch. If so, the effects were too subtle to be detected in our analysis, probably because of the inherent stability of the cat in pitch arising from the long base of support.

Our proposal lends support to the classical hypothesis that a combination of vestibular and neck afferent information contributes directly to trunk stability in space (Lindsay et al. 1976; Roberts 1978; Von Holst and Mittelstaedt 1950). Various authors have proposed that to maintain balance as the head turns, vestibulospinal and cervicospinal reflexes sum together to conserve a stable trunk position (Mergner et al. 1983; Pompeiano 1984; Roberts 1978; Wilson and Peterson 1981). In the decerebrate preparation, Lindsay et al. (1976) demonstrated that vestibular and neck reflexes are cancelled out when the head is rotated on a stable trunk. The reflex studies are supported by the finding that some neurons in the vestibular nuclei are modulated by opposing directions of vestibular and neck stimuli for yaw rotations (Anastasopoulos and Mergner 1982) and pitch and roll rotations (Boyle and Pompeiano 1980; Kasper et al. 1988a,b); activity in these neurons is cancelled out in a frequency-dependent manner, during head rotation on a stationary body (i.e., vestibular and neck stimulation combined). Furthermore, some neurons responded to head rotation in a manner consistent with the encoding of head position in

space (Kasper et al. 1988b). More recent studies in awake, behaving animals (reviewed in Cullen and Roy 2004) have shown that the responses of vestibular neurons depend on the current behavior and may reflect signals not only from vestibular afferents but also from proprioceptive afferents and efference copy signals of the motor command during active movements.

Mergner and colleagues proposed a model for the perception of trunk-in-space using proprioceptive and vestibular afferents (Mergner and Rosemeier 1998). This model was originally based on data from perception studies in humans subjected to passive movements, but has more recently been expanded (Peterka 2002) to balance control in the pitch plane (Mergner et al. 2003). In this schema, trunk-in-space is derived from two directions of sensory "chaining" (or integration): 1) a top-down (i.e., head to trunk/center of mass) combination of vestibular inputs, and neck and trunk proprioceptive inputs (Mergner et al. 1997) and 2) a bottom-up (i.e., feet to trunk) proprioceptive chaining from limb proprioceptors to trunk (Mergner and Rosemeier 1998). Both directions of sensory integration are purported to be necessary, to resolve sensory ambiguities regarding self-motion versus support surface motion. Our study provides experimental support for the top-down concept by inferring an erroneous perception of trunk-in-space when one of the receptor types in this chain (vestibular) is not providing accurate information. Our data also suggest that the

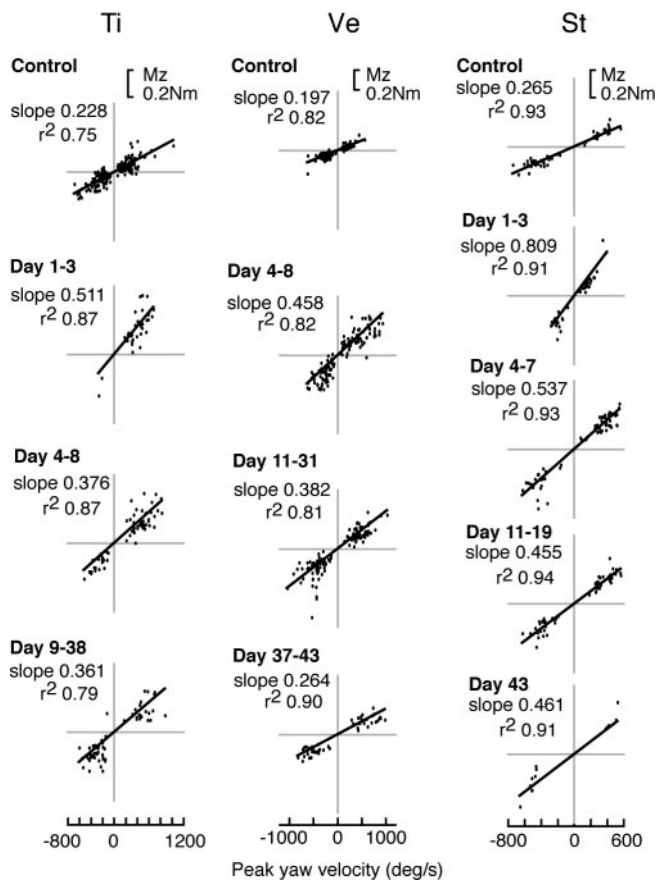


FIG. 9. Relationship between peak yaw velocity and peak Mz before lesion and during the vestibular compensation period. Units of slopes are $\text{Nm} \cdot \text{deg}^{-1} \cdot \text{s}^{-1} \times 10^{-4}$.

bottom-up proprioceptive chaining, which remains intact in the labyrinthectomized cat, is likely overridden by the top-down system during a voluntary head turn. In other words, even though the limb proprioceptive input should accurately report that the support surface is stable, and foot cutaneous input should provide veridical information about acceleration of the body relative to the support surface (Ting and Macpherson 2004), these signals are not sufficient to overcome the apparent perception that the body is falling. As compensation proceeds, a greater reliance on somatosensation may underlie the lesioned animal's ability to reduce the amplitude of the inappropriate postural imbalance.

An alternative, but less likely, explanation for the loss of balance during head turns is overbalancing that results from hypermetria. Hypermetria is an abnormal scaling (increase) of motor behavior that follows various lesions, and has previously been reported to occur after bilateral vestibular loss, during gaze shifts in monkeys (Dichgans et al. 1973) and humans (Kasai and Zee 1978), and during support surface translations in the cat (Inglis and Macpherson 1992, 1995). Inglis and Macpherson (1995) provided a discussion of the underlying basis of postural hypermetria with bilateral vestibular loss. In the present study, hypermetria was manifest by overshoot of the head angular position in yaw, and higher peak rotational moment of force, Mz, for a given peak yaw velocity of the head (i.e., increase in slope in the relationship between peak V_{yaw} and peak Mz). Despite the hypermetria demonstrated by these

animals, however, it is unlikely to be the cause of the destabilization for the following reasons. Hypermetria occurred in the horizontal (yaw) plane (rotation about the vertical axis), which was the primary plane of the voluntary movement. The hypermetric Mz is interpreted as an excessive acceleration of the body around the central vertical axis and was followed by a rapid reversal in Mz, to decelerate and stop the movement. In contrast, the postural instability occurred in the frontal (roll) plane. The destabilizing thrust was exerted laterally and downward at the contralateral limbs, resulting in a motion of the body to the ipsilateral side. It is unlikely that the abnormal force in the frontal plane arose from an excessive rotational force in the orthogonal horizontal plane. Moreover, this abnormal behavior was characterized by EMG bursts and force peaks that were distinct from the initial components of the head turn and absent in the control, as best seen in single trials (e.g., Fig. 5A) rather than averages where the variation across trials tends to blend the first and second peaks, especially in the forces.

Other important evidence that the destabilization did not result from hypermetria came from the two animals with peripheral somatosensory loss induced by high-dose pyridoxine (vitamin B6). We previously showed that somatosensory loss induces ataxia and hypermetric responses to support surface translation manifest by frequent overshoots and delayed reversals of the position of the center of mass (Stapley et al. 2002). In the present study, the two animals with somatosensory loss also showed hypermetria during voluntary head turns, but they did not show the lateral destabilizing thrust characteristic of the vestibular-loss animals. Therefore we conclude that active destabilization of balance during voluntary head turns is a specific result of the loss of accurate vestibular information regarding the acceleration of the head-in-space.

Over time, the labyrinthectomized animals compensated by reducing the amplitude of both the primary moment of force, Mz, and the subsequent destabilizing force in the frontal plane.

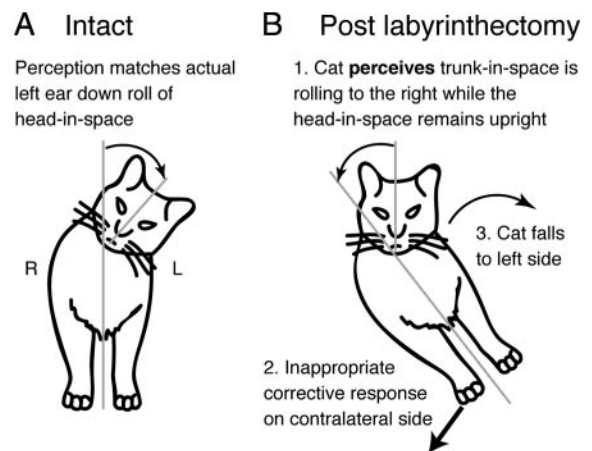


FIG. 10. Schema illustrating the proposed misinterpretation of trunk position in space after bilateral labyrinthectomy. A: in the intact cat, vestibular inputs encode the angular velocity of the head-in-space as it rolls to the left while proprioceptive input from neck muscles encodes the leftward movement of the head with respect to the trunk, leading to the correct perception that the head is moving left ear down on a stable trunk. B: in the labyrinthectomized animal, vestibular inputs send the erroneous signal that the head is not moving in space while proprioceptive inputs from neck muscles indicate a leftward movement of the head with respect to the trunk, just as in A. Combination of vestibular and neck information leads to the erroneous perception that the trunk is rolling right side down, while the head remains upright. (Figure adapted from Melvill Jones 2000.)

By the end of the first week animals could consistently produce head movements without falling. Even though the amplitude of the destabilizing force decreased, this response persisted, right up until the animals were killed. We may conclude that the compensation process allowed the animals to turn down movement amplitude but did not modify the errors in computation of trunk-in-space by the postural control system. Vestibular compensation of postural and motor deficits after labyrinthectomy is known to involve the restoration of normal, symmetrical levels of spontaneous discharge in the deafferented vestibular nuclei (Galiana et al. 1984; Gernandt and Thulin 1952; Markham et al. 1977; Precht et al. 1966; Xerri et al. 1983). Restoration of this discharge would lead to increased drive of cerebellar Purkinje cells. Thus it is likely that after bilateral labyrinthectomy, the toning down of the hypermetria seen in the present study could have resulted from a restoration of inhibitory tone from the cerebellum, perhaps through tonic descending drive in vestibulospinal and/or reticulospinal pathways. This mechanism could be viewed as a nonspecific reduction in gain of the motor output, turning down both desired as well as inappropriate actions. In part, the restoration of tonic activity in the vestibular nuclei results from increased synaptic drive from somatosensory spinal afferents (Jensen 1979). One might speculate that an increased somatosensory drive would mediate an increase in reliance on somatosensory inputs for balance control. This might manifest as an increase in weighting of the bottom-up chaining of proprioceptive inputs (Mergner and Rosemeier 1998) for accurately computing trunk-in-space. That this does not seem to be the case is evident by the persistence of the inappropriate destabilizing thrust during voluntary head turns. It is unknown whether the animals might have achieved total suppression of the abnormal response with a longer recovery period.

To conclude, the results of this study provide the first evidence that animals with vestibular loss actively generate a destabilizing force that, in the early phase after lesion, can lead to falls. These results suggest that absence of vestibular inputs leads to misinterpretation of the position and motion of the trunk-in-space during active head movements. Therefore vestibular information is critically important for calculating position of the trunk-in-space for the balance control system when active head turns are made. This mechanism may underlie the postural instability vestibular patients experience while turning their heads during stance or locomotion (Herdman 1994).

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