ELSEVIER

Neuropsychologia 44 (2006) 1878–1890

NEUROPSYCHOLOGIA

www.elsevier.com/locate/neuropsychologia

Asymmetrical perception of body rotation after unilateral injury to human vestibular cortex

John W. Philbeck [a,*], Marlene Behrmann [b], Tim Biega [c], Lucien Levy [c]

[a] *Department of Psychology, George Washington University, 2125 G. Street, NW, Washington, DC 20052, USA*
[b] *Carnegie Mellon University, Department of Psychology, Pittsburgh, PA, USA*
[c] *George Washington University Medical Center, Department of Radiology, Washington, DC, USA*

Received 8 June 2005; received in revised form 3 February 2006; accepted 15 February 2006
Available online 31 March 2006

Abstract

Vestibular information plays a key role in many perceptual and cognitive functions, but surprisingly little is known about how vestibular signals are processed at the cortical level in humans. To address this issue, we tested the ability of two patients, with damage to key components of the vestibular network in either the left or right hemisphere, to perceive passive whole-body rotations (25–125°) about the yaw axis. In both patients, the posterior insula, hippocampus, putamen, and thalamus were extensively damaged. The patients' responses were compared with those of nine age- and sex-matched neurologically intact participants. The body rotations were conducted without vision and the peak angular velocities ranged from 40° to 90° per second. Perceived rotation was assessed by open-loop manual pointing. The right hemisphere patient exhibited poor sensitivity for body rotations toward the contralesional (left) hemispace and generally underestimated the rotations. By contrast, his judgments of rotations toward the ipsilesional (right) hemispace greatly overestimated the physical rotation by 50–70° for all tested magnitudes. The left hemisphere patient's responses were more appropriately scaled for both rotation directions, falling in the low-normal range. These findings suggest that there is some degree of hemispheric specialization in the cortical processing of dynamic head rotations in the yaw plane. In this view, right hemisphere structures play a dominant role, processing rotations in both directions, while left hemisphere structures process rotations only toward the contralesional hemispace.

Keywords: Self-motion; Updating; Path integration; Manual pointing; Spatial cognition

The vestibular system plays a central role in a wide variety of perceptual and cognitive functions, including spatial orientation, postural stability, motion perception, and oculomotor control (Brandt & Dieterich, 1999; Dieterich et al., 2003; Suzuki et al., 2001). Despite the importance of vestibular signals for normal perception and action, however, surprisingly little is known about how these signals are processed and represented at the cortical level. Although there is suggestive evidence that perception of angular head motion in opposite directions may exhibit some degree of hemispheric specialization, the extent to which this might be true is currently unclear. The study presented here describes two patients (JM and TG) who suffered damage to key cortical and subcortical components of the vestibular network in the right and left hemispheres, respectively, and, therefore, promises to provide insight into the perceptual representation of vestibular information in cortex.

Vestibular signals travel from the VIIIth cranial nerve and vestibular nuclei to several nuclei in the thalamus, via the rostral midbrain; then from the thalamus, vestibular signals project directly or indirectly to several cortical structures (Berthoz, 1996; Brandt & Dieterich, 1999). In the monkey, these cortical regions include area 2v in the intraparietal sulcus; area 7, in the inferior parietal lobe; the ventral intraparietal region (VIP); the medial superior temporal and visual posterior Sylvian regions of the temporal lobe (MST and VPS); area 3aV, a sensorimotor region in the central sulcus; a region in the cingulate cortex; and a core region densely interconnected with nearly all of the foregoing regions, known as the parieto-insular vestibular cortex (PIVC) (Guldin & Grüsser, 1998). The PIVC is located near the posterior end of the insula in the depths of the lateral sulcus. In humans, the cortical vestibular structures are known with less certainty, but evidence from a variety of sources (principally

* Corresponding author. Tel.: +1 202 994 6313; fax: +1 202 994 1602.
E-mail address: philbeck@gwu.edu (J.W. Philbeck).

0028-3932/$ – see front matter © 2006 Elsevier Ltd. All rights reserved.
doi:10.1016/j.neuropsychologia.2006.02.004

functional neuroimaging) suggests that a similar organization holds true, with regions around the temporoparietal junction, central sulcus, and intraparietal sulcus being the likely human homologues of areas PIVC, 3aV, and 2v, respectively (Bottini et al., 1994, 2001; Brandt & Dieterich, 1999; see also the pioneering work of Friberg, Olsen, Roland, Paulson, & Lassen, 1985). In addition to these areas, the posterior insula, putamen, anterior cingulate cortex, and the hippocampus are sometimes engaged by vestibular stimulation (Bottini et al., 2001; Vitte et al., 1996). Given that vestibular stimulation often generates concurrent signals in other sensory modalities (Petit & Beauchamp, 2003), a current topic of research concerns whether activation in these latter areas during vestibular stimulation is indeed due to the vestibular component (Bense, Stephan, Yousry, Brandt, & Dieterich, 2001; Bucher et al., 1998; Emri et al., 2003; Fasold et al., 2002; Lobel, Kleine, Bihan, Leroy-Willig, & Berthoz, 1998; Suzuki et al., 2001).

Many of the foregoing functional neuroimaging studies have used caloric vestibular stimulation (CVS) to activate the vestibular system, a technique that involves introducing water or gas into the external auditory canal; warm and cold stimulation is thought to activate and inhibit, respectively, vestibular nerve signals arising from one or more of the semicircular canals of the stimulated ear. Unilateral warm and cold CVS in supine observers is associated with a preponderance of ipsilateral and contralateral activity, respectively, in cortical vestibular regions, although there is a substantial amount of bilateral activity (e.g., Dieterich et al., 2003). These results suggest that cortical vestibular processing exhibits some, albeit incomplete, degree of functional hemispheric specialization (Bottini et al., 2001; see also Kahane, Hoffman, Minotti, & Berthoz, 2003; Penfield & Jasper, 1954). The full extent and significance of this lateralization is currently unknown. One outstanding question concerns whether head motions in opposite directions might be encoded in lateralized cortical regions. The asymmetrical pattern of brain activity seen after unilateral CVS hints that this might be true to some extent, but the perceptual correlates of this brain activity have not been systematically assessed.

A potential source of insight into these issues comes from patients who have sustained a unilateral brain injury to some part of the vestibular network. One might expect that if there is indeed some hemispheric specialization in encoding head rotations in different directions, damage to one of these functionally specialized structures would selectively impair the perception of head rotation for some directions but not others, perhaps as part of a more general asymmetry in perceived body orientation based on vestibular signals. Brain lesions in or near certain vestibular regions are known to elicit directional biases (typically toward the ipsilesional side) in static orientation tasks; examples include subjective judgments of visual vertical, body vertical, and a host of judgments that are typically impacted by hemispatial neglect (Bisdorff, Wolsley, Anastasopoulos, Bronstein, & Gresty, 1996; Brandt, Dieterich, & Danek, 1994; Halligan, Fink, Marshall, & Vallar, 2003; Karnath, Johannsen, Broetz, & Küker, 2005).

To date, little is known about the impact of unilateral brain injury specifically on the perception of *dynamic* angular head motion. This is the issue that we address in the current paper. Several studies have tested patients with right hemisphere lesions in or around the temporoparietal junction (Farrell & Robertson, 2000; Philbeck, Behrmann, & Loomis, 2001; Tropper, Melvill Jones, Bloomberg, & Fadlallah, 1991). Although these patients tend to underestimate, there is no clear pattern of asymmetry in terms of perceiving clockwise versus counterclockwise rotations. Similarly, when saccadic eye movements are used to indicate the magnitude of body rotations, the final eye positions of patients with lesions restricted to the right temporoparietal junction show no evidence of asymmetry (Israël, Rivaud, Gaymard, Berthoz, & Pierrot-Deseilligny, 1995). Importantly, the lesions in these studies were relatively localized and may have permitted a significant amount of residual vestibular processing to proceed in undamaged components of the vestibular network. The impact of more extensive unilateral vestibular cortex injuries on the perception of dynamic angular body motion is as yet unknown. Examining the effects of extensive disruption of vestibular processing in one hemisphere promises to provide insight into the functional role of the entire vestibular network.

Given the multiplicity of cortical regions implicated in vestibular processing, extensive lesions within the vestibular network may well result in a range of behavioral deficits that depend on exactly which components of the network have been affected. A complete characterization of these deficits, and the brain regions they implicate, would therefore require testing a large number of patients with a variety of lesions. Our approach in this paper is to focus on two individuals to illustrate two possible consequences of extensive lesions in multiple components of the vestibular network, with an emphasis on evaluating possible asymmetries in the perception of dynamic angular head motion. To our knowledge, JM, a patient with right hemisphere injury, is the first to be described in whom there is a strong asymmetry in perceiving leftward vs. rightward whole-body rotations. We tested a second patient, TG, who had a left hemisphere injury and was relatively well-matched to JM in terms of the location and extent of his lesioned tissue. TG demonstrated little or no asymmetry in perceiving body rotations. For comparison, we also tested nine neurologically intact participants. We assessed perceived body rotation using an open-loop pointing paradigm: participants viewed an earth-fixed target on a table directly in front of them, then covered the eyes and underwent a passive whole-body rotation (25–125° left or right of straight ahead). After the rotation, participants manipulated a pointer without vision to indicate the remembered location of the target. These responses were not visually guided and were non-ballistic, so they were unlikely to recruit special-purpose visuomotor spatial representations which might be functionally distinct from conscious perceptual representations (Milner & Goodale, 1995). In addition to these experimental trials, we also verified that participants could localize targets visually and point to the remembered locations after a short delay (perceptuomotor performance trials). This allowed us to assess whether there were any biases in directing attention or motoric actions toward locations on either side of the body midline when no body rotations were involved (Behrmann, Ghiselli-Crippa, & Di Matteo, 2001–2002; Karnath

& Perenin, 1998). Vestibular stimulation can cause a temporary remission of biases associated with hemispatial neglect in some brain-injured patients, suggesting that a shift in the perceived body midline may have occurred (Bisiach & Vallar, 2001; Rode et al., 1992; Rode & Perenin, 1994). If body rotation induces shifts of the perceived body midline, this could generate systematic differences in responses for leftward versus rightward rotations, even in the absence of an asymmetry in perceiving the rotations. To check this, on some trials we passively rotated participants and asked them to point straight ahead without vision after the rotation (midline shift trials).

1. Methods

1.1. Participants

Two patients with large unilateral cortical and subcortical injuries and nine neurologically intact control subjects gave their informed consent to participate prior to inclusion in the study. The study was approved by the Carnegie Mellon University ethics committee (Pittsburgh, USA) and was performed in accordance with the ethical standards of the 1964 Declaration of Helsinki.

1.1.1. Case histories

1.1.1.1. Patient JM. Seven years prior to testing, JM, a right-handed male with a master's degree in engineering, suffered an extensive right middle cerebral artery infarction that left him with a large right frontotemporal lesion (see Lesion analyses, below). He exhibited a left homonymous hemianopia following the infarct, but this had resolved by the time of testing. At testing, JM was 55 years old and his corrected visual acuity in each eye was 6/12. He exhibited mild left-sided hemiparesis and typically wore a leg brace to aid in locomotion. Although he could walk unassisted, he usually walked with a cane. He denied any current dizziness or history of falls and/or disorientation. Previous testing had shown JM to have mild to moderate left-sided hemispatial neglect (Behrmann & Plaut, 2001). To assess this at the time of testing, we administered the Behavioral Inattention Test (Wilson, Cockburn, & Halligan, 1987), which includes line bisection, line cancellation, letter cancellation, copying, and figure drawing tasks. The cut-off score for this test is 129 out of 146 possible points. JM's score was in the normal range (139) at the time of testing.

1.1.1.2. Patient TG. TG, a left-handed male, was found to have an anaplastic astrocytoma 1.25 years prior to testing.[1] Approximately 8 months prior to testing, he underwent a surgical resection of the tumor, leaving him with a large left frontotemporal lesion (see Lesion Analyses, below, and Table 1). A shunt was inserted shortly thereafter due to complications arising from this procedure. At the time of testing, TG was 47 years old and medically stable, though he was non-ambulatory due to right hemiparesis. He exhibited mild to moderate expressive aphasia and a left homonymous hemianopia. His visual acuity was not evaluated clinically, but based upon our testing, his vision was more than adequate for localizing nearby targets when allowed to move his head, as was the case in this study. His educational background was not recorded, but in our testing he successfully demonstrated comprehension of complex procedural instructions and gave no indication of any severe intellectual impairment. He exhibited mild right-sided neglect (Behavioral Inattention Test score of 114) at the time of testing.

Table 1
Lesion analyses for patients JM and TG

Brain region	Percentage damaged			
	Patient JM		Patient TG	
	Right	Left	Right	Left
Vestibular regions				
Temporoparietal junction	12.5	0	0	29[a]
Intraparietal sulcus	5	0	0	29[a]
Superior temporal cortex	17.5	0	0	41.5[b]
Anterior cingulate cortex	17.5	0	0	16.5[a]
"Neck" sensorimotor region, central sulcus	7.5	0	0	4
"Hand" sensorimotor region, central sulcus	25	0	0	4
Posterior insula	90	0	0	92.5
Hippocampus	65	0	0	75
Putamen	100	0	0	90
Thalamus	87.5	0	0	90
Other affected regions				
Frontal cortex	60	0	0	15[a]

Note: Percentage of damaged tissue in each region represents the mean rating of two independent raters. Unless otherwise indicated, interrater judgments differed by less than 10% points.
[a] Interrater judgments differed by 17–42% points.
[b] Interrater judgments differed by 67% points.

1.1.2. Lesion analyses

Fig. 1 shows structural neuroimaging scans of JM's brain taken seven years after his infarct, obtained by magnetic resonance imaging (MRI) using high resolution, three-dimensional gradient echo pulsing sequences. Fig. 2 shows MRI scans of TG's brain taken seven months after surgery to remove an astrocytoma. Both sets of scans were evaluated by two raters working independently (the third and fourth authors). The raters judged the percentage of damaged tissue in a variety of regions assumed to be part of the vestibular network. The two scorers' ratings were generally highly correlated (average $R^2 = 0.80$). The results of this analysis are presented in Table 1. This lesion analysis confirmed that JM suffered a large right frontotemporal lesion that impacted many components of the right hemisphere vestibular network. Notably, the right thalamus, the thalamic output fibers in the internal capsule, and the posterior insula were all extensively damaged. A small amount of the right posteromedial thalamus was spared, along with a very minimal amount of the genu and posterolateral portions of the internal capsule. Lesions of approximately the same volume were present in many of the same vestibular structures in TG, with only a small amount of sparing of left medial thalamic nuclei. TG's frontal lesion was smaller in volume than JM's.

1.1.3. Neurologically intact control participants

We tested four males with no history of neurological disorder as control subjects. Their mean age was 46.5 years (range 35–57). To provide a more robust estimate of normal performance, we also included data from five additional neurologically intact males collected in a previous study (Philbeck et al., 2001). This previous study included all the trial types used in the current experiment, in the same order and using the same methods; after undergoing the trials described below, these subjects participated in an additional block of trials investigating somewhat different issues than those presented here. Thus, before exposure to the additional trials, participants in the previous study received the same treatment conditions as the other subjects in the current experiment. All nine individuals in the aggregated group of control participants were college graduates and three had obtained a Ph.D. degree. The mean age of the nine men was 54.7 years; there were three subjects in the 22–42 year age range, four in the 70–73 year age range, and two subjects quite close in age to JM (52–57 years). Overall, the

[1] Some evidence suggests that functional hemispheric lateralization of cortical vestibular processing may be more closely linked with handedness than is lateralization of language processing (Dieterich et al., 2003). As yet, it is unclear what might be the consequences of this linkage for representations of dynamic angular head motion in right- versus left-handed individuals. Testing in a larger group of patients is required to assess the representativeness of both JM and TG's; nevertheless, the rarity of studies involving left-handed patients with large left hemisphere lesions makes TGs case particularly valuable, especially in light of the apparent linkage between handedness and vestibular processing.

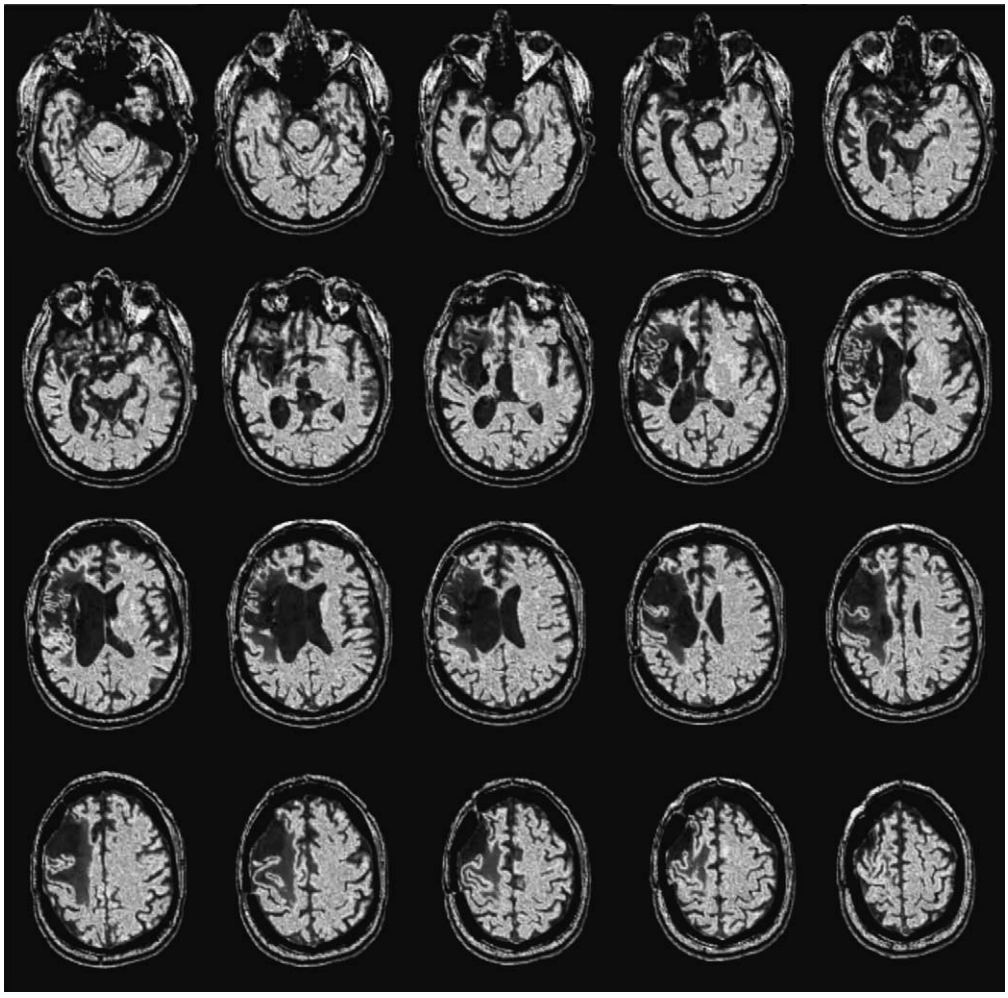

Fig. 1. Neuroimaging data (axial T1-weighted magnetic resonance imaging) for Patient JM. The right hemisphere appears on the left side of the images and the anterior of the brain is on the top.

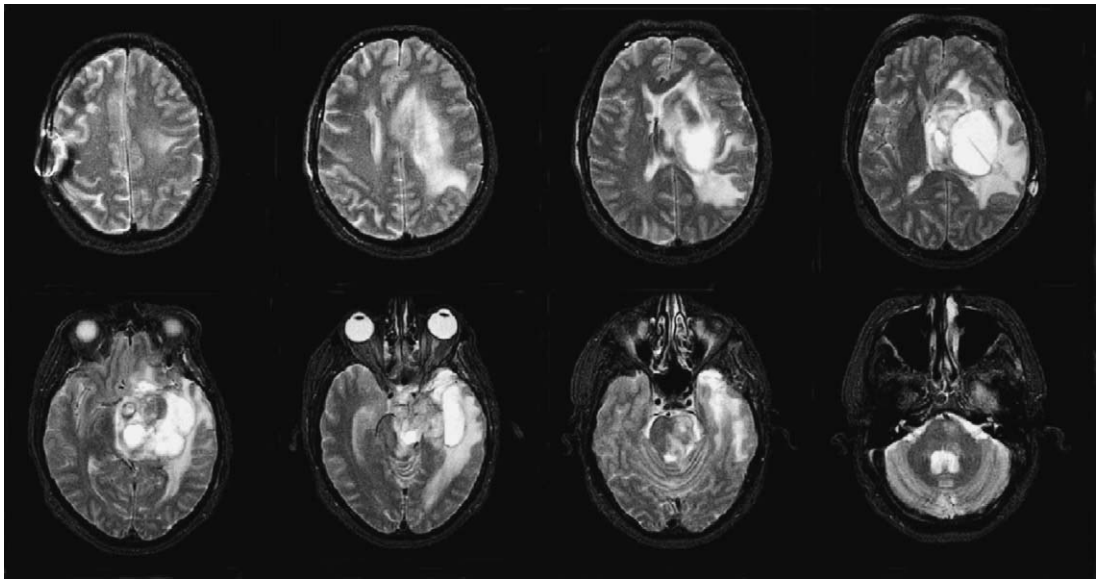

Fig. 2. Neuroimaging data (axial T2-weighted magnetic resonance imaging) for Patient TG. Image orientation is same as in Fig. 1.

group was roughly balanced in terms of including subjects older and younger than JM, with two being especially good age matches.

1.2. Apparatus

The experiment was conducted in a well-lit, indoor room. Each participant sat in a swivel chair with his feet on the chair's footrest. The footrest prevented leg muscle proprioception from providing information about the chair rotation, thereby providing a more narrow focus on vestibular information. Our methodology did not rule out possible contributions of somatosensory information or from eye movements by way of the vestibular-ocular reflex (VOR), so the sensory information signaling body rotations should be considered a composite of these sources. We will return to this issue in the General Discussion. A pointing device was affixed to the chair 23 cm directly in front of the participant. It was mounted in a horizontal plane at approximately waist level, and rotated with the chair. The pointer itself consisted of a thin rod, which extended 16 cm from its axis of rotation. Just below the pointing rod was a polar scale, from which pointing responses could be recorded to the nearest degree.

The chair was centered in the middle of a table, 1.52 m^2 and 0.76 m high, which had a circular hole (0.76 m in diameter) cut in the center (see Fig. 3). The chair was accessible via a removable panel in the table. The table itself was inscribed with markers that were used to align the chair when administering whole-body rotations. In addition, four clear-glass flashlight bulbs (12 V, 80 mA) were mounted on the table at a radius of 76 cm from the chair's rotation axis and at eccentricities of 25° and 65° left and right of straight ahead. Although we did not formally measure the luminance of the bulbs, each bulb had approximately the same apparent brightness as a standard hand-held flashlight and no participant demonstrated any difficulty in detecting when a particular bulb was illuminated. Because the overhead room lights were illuminated throughout the study, all of the flashlight bulbs were visible even when extinguished, and each subtended approximately 0.20° × 0.20° of visual angle. On certain trials, however, one of the bulbs was flashed at about 7 Hz for 2 s to specify it as a target location. A fifth possible target location, designated as "the origin", lay straight ahead from the participant's starting position and was visible at the perimeter of the table as a joint in the table surface.

1.3. Procedure

On each trial, the task was to use the dominant hand to manipulate a pointer to indicate the direction of a specified target. Participants were alerted to the fact that the rotation axis of the chair-fixed pointer was offset from that of the chair itself. This was demonstrated by indicating a point on the table 90° to the left relative to a coordinate frame centered on the chair's rotation axis, and showing that an accurate pointing response to that location would require that the pointer be set more than 90° to the left. Thus, ideally, participants would ignore the chair's rotation axis when responding and align the pointer with an imaginary line connecting the pointer rotation axis with the physical target location. Participants began each trial with the blindfold raised and their trunk and head oriented toward the "origin" landmark. In trials involving body rotations, participants also wore hearing protectors (overall noise reduction rating, 20 dB) to minimize auditory information that might be informative about their orientation in the room. After every pointing response, the experimenter turned the pointer toward the participant's abdomen (i.e., 180°). This ensured that all responses were equated in terms of requiring at least some motor behavior to set the pointer.

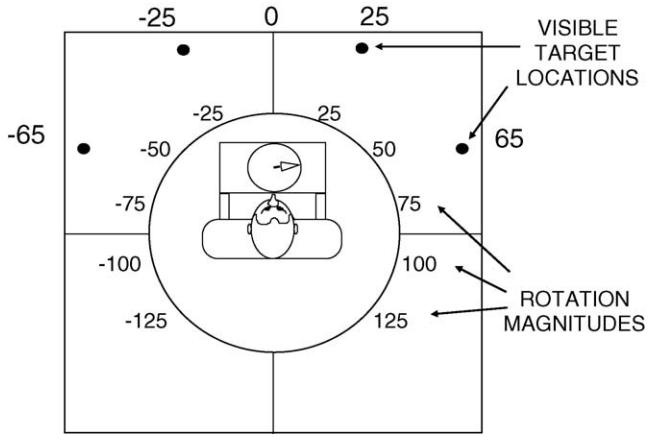

Fig. 3. Schematic overhead view of the experimental apparatus. The observer sits in a swivel chair; a pointing device is mounted on the chair and rotates with it. The chair is surrounded by a table. Four target lamps are placed on the table surface at 25° and 65° on either side of the observer's initial orientation.

There were three trial types (see below). Except for midline shift trials, an abbreviated set of trials was conducted with unrestricted vision immediately before beginning data collection. These practice trials allowed us to verify that the participants understood the tasks before moving on to perform them without vision. Other than the feedback provided by vision during these practice trials, no error feedback was given. The three trial types were blocked and presented in the following block order. The running order was fully randomized within each block.

1.3.1. Perceptuomotor performance trials

In these trials, the four electric lamps at 25° and 65° left and right of straight ahead served as four possible target locations, with the origin landmark being a fifth possible location. On each trial, the experimenter specified one of these five locations as the target, either verbally, in the case of the origin, or by flashing one of the lamps. After approximately 5 s, the experimenter gave a verbal signal to point, at which time participants made their pointing response. Each of the five locations was presented three times apiece in random order, first with vision during the response to verify that participants understood the task, and then in a second block in which participants donned a blindfold before pointing. In the second block, after vision was occluded by the blindfold, a 5-s delay was imposed before the pointing response; this delay was longer than the duration of the largest body rotation (125°). These trials allowed us to assess participants' ability to localize individual targets and use the pointing device to point to their remembered locations after a short delay.

A slightly different methodology was used for two control participants, who saw targets at 75° left and right of straight ahead instead of 65° and pointed immediately after covering their eyes. To take the difference in target locations into account, all responses were converted to signed errors relative to the physical target eccentricity. We analyzed the data in the perceptuomotor performance trials both with and without these two subjects included, and the results were highly similar. We will therefore ignore these minor methodological differences hereafter and report the results of the full group.

1.3.2. Midline shift trials

Participants began these trials by viewing the origin landmark. After approximately 5 s, they donned the blindfold and the experimenter administered a passive whole-body rotation of either 75° or 125° to the right or left (three times per condition in random order). Participants then used the pointer to indicate the direction of "straight ahead," still without vision. When pointing, participants were instructed to imagine a line extending from the trunk straight ahead, and to set the pointer to be collinear with that imaginary line. Thus, the ideal pointing response after the body rotation in these trials was always 0° in the reference frame of the chair-fixed pointing device. These trials allowed us to assess the degree to which passive rotations may have affected the participants' localization of their own egocentric body midline.

1.3.3. Experimental trials

Participants viewed the origin landmark for several seconds and donned a blindfold. The experimenter then administered a passive whole-body rotation. The participant's head was unrestrained, but in practice, participants held their head stationary with respect to their body during the body rotations and uncontrolled head motion was minimal. There were 10 possible rotation magnitudes (25–125° in 25° increments on the left and right sides). Each magnitude was presented six times in random order. After the rotation, participants used the pointing device to indicate the location of the earth-fixed "origin" landmark seen prior to the rotation. To administer passive whole-body rotations, the experimenter manually rotated the chair until markers on the back of it were aligned with other

markers on the table. He attempted to generate bell-shaped velocity profiles without jerky stops and starts. He had administered well over 1000 rotations using this apparatus when conducting a previous experiment (Philbeck et al., 2001) and had become adept at producing stereotyped velocity profiles. Over- or undershoots of the nominal rotation magnitudes were recorded to the nearest degree, and trials on which the experimenter erred more than 3° in producing the nominal rotation were discarded and re-run later in the experiment. The actual rotations, rather than the nominal, were used when calculating response errors. In our previous experiment (Philbeck et al., 2001), we recorded several typical chair rotations for each rotation magnitude via an infrared video tracking system (Optotrak, Waterloo). Off-line analysis of these data showed that the peak angular velocities ranged between about 40° and 90° per second, increasing with the rotation magnitude. The average movement durations ranged between 875 and 2131 ms, increasing with rotation magnitude. An abbreviated set of practice trials was conducted with unrestricted vision prior to beginning collection of the experimental data.

1.4. Data analysis

Prior to analysis, the raw pointing responses were transformed to the corresponding values that would be produced if the rotation axis of the pointer were centered on that of the chair. This was intended to facilitate comparison of the pointing responses with the nominal target eccentricities and body rotation magnitudes (thus, after the transformation, an accurate indication of the starting location after a body rotation of 90° would be −90°). In preliminary data analyses, we used circular statistics to characterize central tendencies and dispersions (Fisher, 1993), but these measures were virtually identical to the means and standard deviations (S.D.) of the raw responses owing to the relatively restricted distributions of the data, so we did not proceed further with circular analyses.

Our primary focus is on differences in JM's performance after whole-body rotations toward the left and right. We will report two-tailed, paired-sample *t*-tests ($\alpha = .05$) comparing JM's data for leftward versus rightward responses in the various tasks, and report similar analyses for TG. We will use analysis of variance (ANOVA) to characterize the performance of the control participants, manipulating the independent variables within subjects using a repeated measures design. To compare the patient data with those of the control participants, we will indicate JM's and TG's performance in terms of *z*-scores based on the means and standard deviations of the control data in each condition. We will take *z*-scores above 2 and below −2 to be evidence of significant deviation from normal performance. As we will see, JM's perception of dynamic whole-body rotation was sufficiently unusual that our interpretation of his performance would not change substantially if a considerably more conservative cut-off were chosen. The multiplicity of different trial types means that a relatively large number of statistical tests will be performed. Most of these tests are descriptive and are not intended to test our primary hypotheses. Instead of correcting for multiple comparisons, we will present exact *p*-values and mean squared errors to give an indication of effect sizes.

2. Results

2.1. Perceptuomotor performance trials

2.1.1. Control group

We first used ANOVA to compare visually guided versus open-loop responses in the control group. Before analysis, responses were converted to signed errors, with negative and positive values indicating errors to the left and right, respectively, of the nominal target. These error scores were then averaged across repetition. Stimulus side (left/right), availability of vision (eyes open/eyes closed), and stimulus eccentricity (25°/65°) were varied within subjects. None of these variables yielded main effects (side: $F[1,8] = 0.233$, M.S.E. = 16.23, $p = 0.64$; vision: $F[1,8] = 0.315$, M.S.E. = 5.2, $p = 0.59$; direction: $F[1,8] = 1.46$, M.S.E. = 10.39; $p = 0.26$). For our purposes, the lack of differentiation by stimulus side is especially noteworthy. There were no other reliable main effects or interactions, except for a small side × direction interaction ($F[1,8] = 5.8$, M.S.E. = 122.68, $p = 0.043$). The largest difference between the marginal means in this interaction was only 3.56° (comparing responses to targets 25° left and right of straight ahead), so the practical significance of this interaction is likely to be negligible. The data for both visually guided and open-loop responses are plotted in Fig. 4, which also shows that the mean signed error in pointing straight ahead was less than 1° in both cases.

2.1.2. Patient JM

On average, JM's pointing responses in the visually guided and open-loop perceptuomotor performance trials fell within 3.5° of the nominal target. His performance ranged between −2.86 and 0.92 standard deviation units of the control data (mean = −0.66). Although his responses to some particular tar-

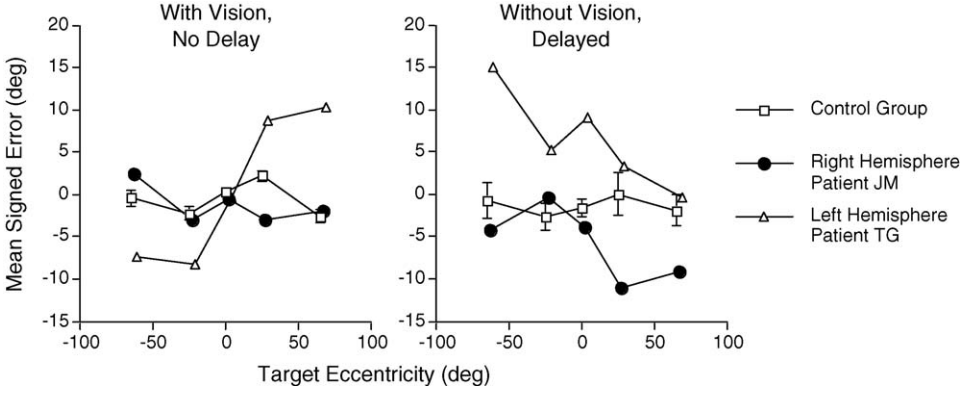

Fig. 4. Mean signed pointing error in the perceptuomotor performance trials for JM, TG, and the nine control subjects. Responses are shown as a function of the five possible target eccentricities. Negative values of target eccentricity signify targets appearing on the left of straight ahead, and negative response values signify responses to the left of the specified target. No body rotations were imposed before responding. Error bars for the control subjects denote ±1 standard error of the mean. Horizontal lines indicate accurate responding, and data points for JM, TG, and the control group have been shifted laterally slightly to aid visibility. (Left panel) Pointing responses made under visual control. (Right panel) Pointing responses made without vision after a 5 s delay.

gets were more error-prone than those of the control group (see Fig. 4), his pointing in the perceptuomotor performance trials was globally within normal limits. A tendency to point somewhat to the left of targets was evident, however; a two-tailed *t*-test showed that this bias was more pronounced for right- than left-sided targets ($p < 0.001$). To assess these biases relative to the control group, we calculated a mean difference score for each participant (mean "left" error minus mean "right" error) in the eyes closed condition. The mean control group difference score was −0.77° (S.D. = 9.90°); JM's difference score of 7.83° fell within 0.87 standard deviation units of the control group mean, indicating that the difference between his left-and right-sided responses fell within the normal range. Biases in left- versus right-sided responses in individual subjects are taken into account in Section 2.4.

2.1.3. Patient TG

TG's pointing responses fell within 3.5° of the nominal target on average in the visually guided and open-loop perceptuomotor performance trials, although his performance ranged from −2.75 to 5.52 standard deviation units from the Control group (mean = 1.15). In visually guided trials, he tended to create larger-than-normal responses for targets on both sides; by contrast, in open-loop trials, he exhibited a tendency to point to the right of targets. A two-tailed *t*-test showed that this bias in open-loop responding was more pronounced for left- than right-sided targets ($p < 0.001$), opposite to the pattern seen in JM. In terms of the difference between mean "left" errors and mean "right" errors, however, TG's difference score of 8.63 fell within the normal range (0.95 standard deviation units above the control group mean).

2.1.4. Response consistency (within-subject random error)

Table 2 shows the average response consistency for JM, TG, and the control group, as measured by the within-subject standard deviations (S.D.s) across repetitions in each condition. As might be expected, pointing without vision tended to be more variable than visually guided responses for all participants. The consistency of JM's and TG's responses was generally within normal limits. To assess systematic differences in response consistency in left- versus right-sided responses, we calculated difference scores for each participant, comparing the mean within-subject S.D.s for left and right responses in the eyes closed condition. JM's and TG's difference scores fell within 1.24 and 1.72 S.D. units, respectively, of the control group mean.

2.2. Midline shift trials

2.2.1. Control group

We performed an ANOVA on the control data, with body rotation direction (rightward/leftward) and rotation magnitude (75°/125°) included as within-subject factors. This analysis showed no main effects or interactions (all Fs [1,8] < 1.05, all ps > 0.33); the mean signed error for the controls was −0.65°. Taken together, these results indicate that the control group did not experience a shift in the perceived body midline after whole-body rotations.

Table 2
Response consistency, perceptuomotor performance trials

Viewing condition	Target eccentricity (°)			
	−65	−25	25	65
With vision				
Control mean (S.E.)	2.17 (0.52)	1.23 (0.34)	1.07 (0.18)	1.46 (0.37)
JM mean	1.15	0.87	0.87	1.00
TG mean	2.52	2.89	1.15	5.51
JM *z*-score	−0.65	−0.36	−0.37	−0.41
TG *z*-score	0.22	1.62	0.16	**3.64**
Without vision + delay				
Control mean (S.E.)	4.80 (1.09)	4.06 (0.69)	3.37 (0.22)	3.47 (0.39)
JM mean	3.66	2.44	3.63	4.71
TG mean	0.89	4.12	4.26	4.63
JM *z*-score	−0.35	−0.78	0.40	1.06
TG *z*-score	−1.19	0.03	1.36	1.00

Notes: Mean scores give the average within-subject response consistency for each condition, as measured by within-subject standard deviations (°); values in parentheses give the between-subject variability, as measured by standard errors ($n = 9$), associated with the control group means. Negative values of target eccentricity denote targets presented left of straight ahead. Boldface type highlights *z*-scores that exceed +2.

2.2.2. Patient JM

JM's performance ranged between −1.6 and −2.19 S.D.s of the control data (mean = −1.82), and his mean pointing error was −8.46°. A two-tailed *t*-test confirmed that his responses did not differ depending on the rotation direction ($p = 0.77$). We again calculated difference scores for each participant, comparing mean responses after leftward versus rightward turns. JM's difference score of −0.25° fell −0.16 S.D. units from the control group mean. Thus, the small amount of bias in JM's straight ahead judgments was not differentiated by the direction of the body rotation, suggesting that the body rotations did not induce direction-specific midline shifts.

2.2.3. Patient TG

TG's responses were generally biased to the right for body rotations in both directions. A two-tailed *t*-test showed that this bias was somewhat more pronounced for leftward than rightward rotations (+8.92° versus +5.33°, respectively; $p < 0.01$). However, TG's leftward versus rightward rotation difference score was −3.58°, placing his performance within the normal range (1.64 S.D. units below the control group mean).

2.2.4. Response consistency (within-subject random error)

JM's and TG's response consistency, as measured by within-subject S.D.s, were both within normal limits (see Table 3).

2.3. Experimental trials

2.3.1. Control group

In these trials, signed errors were calculated such that positive and negative values indicated pointing responses that were

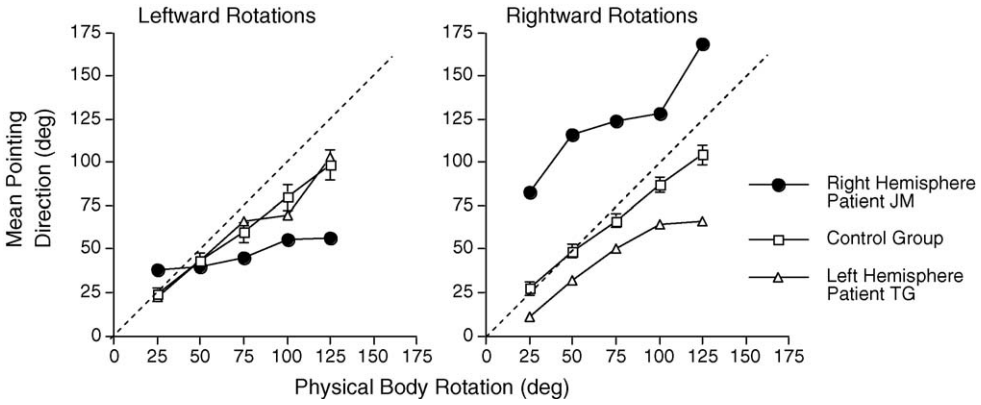

Fig. 5. Mean pointing responses in the experimental trials for JM, TG, and the nine control subjects. Responses are shown as a function of the five possible body rotation magnitudes toward the left and right of the subjects' heading prior to the rotation. Error bars for the control subjects denote ±1 standard error of the mean, and diagonal lines indicate accurate responding.

larger or smaller than required, respectively. For our purposes, the most critical finding is that in the control group, there was no main effect of side ($F[1,8] = 2.86$, M.S.E. = 707.00, $p = 0.13$). There was an effect of rotation magnitude ($F[4,32] = 18.24$, M.S.E. = 1690.68, $p = 0.0001$), with signed errors tending to increase with rotation magnitude; this effect is apparent in Fig. 5 as a tendency for responses to increasingly underestimate the physical rotation magnitude. This pattern is consistent with previous findings in neurologically intact participants (Blouin, Gauthier, & Vercher, 1995; Yardley, Gardner, Lavie, & Gresty, 1999, see also Guedry, Stockwell, & Gilson, 1971). There was no side × magnitude interaction ($F[4,32] = 0.301$, M.S.E. = 9.06, $p = 0.88$).

Table 3
Descriptive statistics, midline shift trials

Statistic	Rotation magnitude (°)			
	−125	−75	75	125
Signed error				
Control mean (S.E.)[a]	−1.01 (1.28)	−0.41 (1.31)	−0.65 (1.46)	−0.54 (1.73)
JM mean	−7.67	−9.00	−8.33	−8.83
TG mean	8.17	9.97	5.50	5.17
JM *z*-score	−1.73	**−2.19**	−1.75	−1.60
TG *z*-score	**2.38**	**2.57**	1.40	1.10
Response consistency				
Control mean (S.E.)[a]	2.21 (0.46)	1.56 (0.26)	2.67 (0.48)	2.36 (0.38)
JM mean	1.53	1.73	0.58	1.26
TG mean	2.36	0.58	0	1.04
JM *z*-score	−0.5	0.22	−1.46	−0.97
TG *z*-score	0.11	−1.26	−1.86	−1.17

Note: Mean signed errors and response consistency (measured by within-subject standard deviations) are given in degrees. Boldface type highlights *z*-scores that exceed ±2. Negative and positive values of rotation magnitude denote rotations toward the left and right, respectively. Negative and positive values of signed error denote pointing errors to the left and right, respectively, of the nominal target.

[a] Values in parentheses denote the between-subject variability, as measured by standard errors ($n = 9$) for each condition.

2.3.2. Patient JM

JM's performance was strikingly different from the control group's in the experimental trials. The pattern is most obvious when the data are plotted as mean responses rather than signed error scores (see Fig. 5). For body rotations toward the left, JM's responses were poorly scaled, and exhibited a constant offset of approximately 30°. For rightward body rotations, the overall response gain was normal, but the responses exhibited a large constant offset of 50–70°. To provide an overall assessment of JM's left–right differences relative to the control group, we calculated mean difference scores for each participant, comparing responses in leftward versus rightward body rotation trials; JM's mean difference score of −74.6° fell 7.13 S.D. units away from the control group mean of −5.6°. Not surprisingly, a two-tailed *t*-test comparing JM's responses for left versus right body rotations was highly significant ($p < 0.0001$).

2.3.3. Patient TG

TG generally underestimated body rotations in both directions, and although his responses showed a hint of asymmetry, their pattern differed from JM's. Specifically, the pattern of large offsets after leftward versus rightward body rotations, so salient in JM's data, was entirely absent in TG. TG's mean signed error for left versus right rotations was −14.1° and −30.2°, respectively, and his mean left–right difference score was 16.12°. This value fell just outside the normal range (+2.18 S.D. units). His responses for left versus right body rotations differed significantly ($p < 0.01$). Inspection of TG's data (Fig. 5) indicates that the overall left–right asymmetry was largely driven by response differences in the 125° rotation conditions.

2.3.4. Response consistency (within-subject random error)

Table 4 shows the pattern of response consistency across rotation side and magnitude, as measured by the within-subject S.D. JM's responses exhibited normal consistency for body rotations to the left; by contrast, rightward body rotations produced a more erratic pattern, with individual rotation magnitudes eliciting either normal or abnormally high variability. TG's response consistency was within normal limits for most of the tested body

Table 4
Response consistency, experimental trials

Rotation direction	Rotation magnitude (°)				
	125	100	75	50	25
Rightward					
Control mean (S.E.)	14.11 (2.34)	11.64 (2.38)	10.92 (1.43)	13.67 (2.19)	7.16 (1.40)
JM mean	17.99	16.56	12.82	10.46	13.96
TG mean	30.55	14.54	17.18	17.10	7.97
JM *z*-score	0.55	0.69	0.44	−0.49	1.61
TG *z*-score	**2.34**	0.41	1.45	0.52	0.19
Leftward					
Control mean (S.E.)	14.94 (3.05)	10.88 (1.80)	10.39 (1.42)	7.12 (1.20)	5.06 (0.89)
JM mean	15.26	32.03	23.53	6.59	29.88
TG mean	11.41	17.93	15.25	6.90	5.50
JM *z*-score	0.03	**3.93**	**3.07**	−0.15	**9.34**
TG *z*-score	−0.39	1.31	1.14	−0.06	0.17

Notes: Mean scores give the average within-subject response consistency for each condition, as measured by within-subject standard deviations (°); values in parentheses give the between-subject variability, as measured by standard errors ($n = 9$), associated with the control group means. Boldface type highlights *z*-scores that exceed ±2.

rotations, with the notable exception of rightward body rotations of 125°, which produced particularly variable responses.

To characterize more fully the pattern of responses with increasing body rotation magnitude, we used the least squares criterion to fit straight lines through the mean response data plotted against the physical rotation magnitude. The parameters of these functions are given in Table 5. Although the fits to the control group data were quite good, with average squared correlations of 0.84, the fits to JM's data tended to be somewhat poorer, averaging 0.40. This analysis showed that JM's responses after leftward body rotations exhibited abnormalities in both slope and intercept, while responses to rightward rotations showed a more normal slope with an abnormally large intercept. TG's slope for leftward body rotations (0.75) was higher than his slope for rightward body rotations (0.57), though both were within normal limits. TG's intercepts and squared correlations for both rotation directions were within normal limits.

2.4. Recalibrated experimental data

It is clear that the small biases in JM's pointing in the perceptuomotor performance and midline shift trials cannot account for the dramatic left–right differences in his experimental data. Nevertheless, we performed an analysis to recalibrate each participant's experimental data and effectively take individual perceptuomotor and midline shift biases into account. We first assumed that when participants indicate perceived straight ahead by open-loop manual pointing, the responses are subject to the same biases both before and after body rotations (e.g., due to physical constraints imposed by the hand posture). To estimate biases in pointing straight ahead not specifically due to body rotation, we averaged each participant's indications of straight ahead obtained under delayed, open-loop conditions (no body rotation). We then subtracted this value from individual responses in midline shift trials to effectively remove straight-ahead pointing biases not due to body rotation. The resulting data give a rough indication of how much the perceived body midline is biased during body rotations of 75° and 125° in either direction, after taking into account biases unrelated to body rotation. Based on these data, we used linear interpolation to estimate the amount of midline shift bias accrued during each of the other rotation magnitudes used in experimental trials. We then subtracted the resulting biases at each rotation magnitude from the pointing responses in experimental trials. The perceptuomotor performance trials, by virtue of being performed open-loop and after a delay, provide a means of estimating additional biases due to motor and mnemonic factors. To remove these biases, we fit straight lines through the function relating pointing responses with the physical target eccentricity in perceptuomotor performance trials (eyes closed condition), using the least squares criterion. We used the parameters of these lines to transform the experimental data and effectively remove biases due to motor and mnemonic factors. One-tailed *t*-tests showed that this recalibration significantly reduced the absolute error in JM's and TG's responses (both $ps < 0.01$), although similar *t*-tests on the control group data showed that the recalibration did not change absolute error reliably in this group. Analyses of JM's transformed data did not differ substantially from the untransformed experimental data. TG's left versus right difference score using the recalibrated experimental data still fell just outside the normal range (2.31 S.D. units above the control group mean), but a two-tailed *t*-test relating TG's recalibrated responses for leftward versus rightward rotations was no longer significant ($p = 0.18$). Table 5 shows the parameters of straight lines fit through JM's and TG's recalibrated data, along with the corresponding average parameters for the control group.

3. Discussion

Our results demonstrate some of the functional consequences of large unilateral lesions in key components of the cortical vestibular network, particularly with regard to sensing dynamic

Table 5
Parameters of linear functions relating indicated to physical body rotation (experimental trials)

Calibration type	Leftward rotations			Rightward rotations		
	Slope	Intercept	R^2	Slope	Intercept	R^2
No recalibration						
Control mean (S.E.)	0.74 (0.06)	5.50 (3.63)	0.81 (0.03)	0.77 (0.05)	8.90 (4.12)	0.86 (0.03)
JM	0.21	31.15	0.23	0.73	68.61	0.56
TG	0.75	4.58	0.66	0.57	2.21	0.72
JM *z*-score	**−2.76**	**2.35**	**−6.13**	−0.25	**4.84**	**−3.12**
TG *z*-score	0.06	−0.09	−1.60	−1.46	−0.54	−1.40
Perceptuomotor + midline shift recalibration						
Control mean (S.E.)	0.78 (0.08)	3.07 (3.75)	0.82 (0.03)	0.79 (0.06)	7.55 (3.69)	0.86 (0.03)
JM	0.21	46.93	0.19	0.74	60.60	0.54
TG	0.92	−7.75	0.68	0.62	7.38	0.71
JM *z*-score	**−2.39**	**3.90**	**−6.91**	−0.27	**4.80**	**−3.09**
TG *z*-score	0.58	−0.96	−1.55	−0.96	−0.02	−1.47

Notes: Boldface type highlights *z*-scores that exceed ±2. Values in parentheses give the between-subject variability, as measured by standard errors ($n = 9$), associated with the control group means.

angular head motion. JM, a right hemisphere patient, significantly underestimated whole-body rotations toward the contralesional side and significantly overestimated rotations toward the ipsilesional side. His responses after rightward body rotations were also considerably more variable than those of the control group. Although he exhibited some slight pointing biases in trials that did not involve body rotations, these biases were not sufficient to explain the large asymmetry in his manual indications of body rotation, which were observable over seven years after his infarct. TG, a left hemisphere patient, showed a relatively small amount of asymmetry in estimating body rotations, with contralesional rotations being underestimated somewhat more than ipsilesional rotations, but this asymmetry largely disappeared when pointing biases unrelated to body rotation were taken into account. There was no reliable tendency toward asymmetry in the control group.

The large volume of JM's and TG's lesions preclude a fine-grained analysis of which brain regions might participate most directly in the perception of body rotations. However, it is clear that two components thought to be especially important for vestibular processing, the thalamus and the posterior insula, were among the most extensively damaged in both patients. The ventral posterolateral nucleus of the thalamus provides the primary pathways by which vestibular information enters the cortex from subcortical structures (Akbarian, Grüsser, & Guldin, 1992; Hawrylyshyn, Rubin, Tasker, Organ, & Fredrickson, 1978); this nucleus was unilaterally destroyed in both JM and TG. The posterior insula has been implicated as a core structure in the cortical vestibular network, and is densely interconnected with many other components of the network (Bucher et al., 1998; Dieterich et al., 2003; Guldin & Grüsser, 1998; Suzuki et al., 2001). The combination of extensive lesions in both the thalamus and posterior insula, then, is likely to have deprived the ipsilesional hemispheres in both patients of their primary sources of subcortical vestibular inputs and disrupted processing in their core vestibular structures. This type of disconnection would affect processing throughout the larger vestibular network, including the hippocampus and frontal lobe (Fasold et al., 2002; Vitte et al., 1996). The degree of functionality in other components of our patients' vestibular network is not known, and it is possible that some vestibular information could reach these structures via extra-thalamic pathways (e.g., through the cerebellum; Berthoz, 1996; Kotchabhakdi & Walberg, 1978). Even if there is some residual vestibular processing in JM's and TG's ipsilesional hemispheres, however, it is quite likely that processing in the entire vestibular network has been severely compromised on the lesioned side. Thus, in JM's case, it is reasonable to expect his perceptual asymmetries to be overwhelmingly determined by the strong asymmetry in the functionality of the two cerebral hemispheres, such that his responses were primarily determined by vestibular processing in his intact left hemisphere. Following the same logic, TG's responses primarily reflect vestibular processing in his intact right hemisphere. More extensive testing is required to confirm these ideas; however, there is so little research investigating the consequences of large unilateral lesions on the perception of dynamic angular head motion that these data nevertheless provide important insights by illustrating two possible outcomes of this type of brain injury. Interestingly, smaller unilateral lesions in specific cortical components of the vestibular network are not associated with asymmetrical perception of dynamic head motion (e. g., Israël et al., 1995). It may be that asymmetry only emerges when processing is disrupted in multiple components of the network.

Until a larger sample of patients has been tested, we must remain cautious about the extent to which our two patients may be informative about functional organization of vestibular processing in the population at large. In particular, even though TG's aphasia suggests that he was left hemisphere dominant for language, it is difficult to know whether his left-handedness may be evidence of an altered brain organization relative to the majority of right-handed individuals. Recent evidence indicates that the organization of vestibular processing may be somewhat more closely associated with handedness than is the organization of language processing (Dieterich et al., 2003) but the full extent of

this linkage remains poorly understood, especially concerning the possible association between handedness and the representation of dynamic head motion.

Nevertheless, assuming that the functional cortical organization in our patients is not grossly abnormal, one interpretation that accords well with existing data is that vestibular cortical regions in the right hemisphere may play a dominant role for encoding body rotations about the yaw axis. Judging from TG's data, the right hemisphere is capable of supporting nearly normal perception of body rotations in both directions when left hemisphere cortical vestibular processing is compromised. JM's data indicate that the left hemisphere plays a more specialized role, primarily encoding rotations toward the contralateral hemispace. JM's pattern of asymmetry, furthermore, suggests that the left hemisphere receives inhibitory inputs from the right hemisphere. Damage to the dominant right hemisphere structures for encoding leftward rotations results in under-perception of leftward body rotations. The intact left hemisphere structures register rightward body rotations in a normal fashion (as indicated by the normal slope of the function relating the physical body rotation to JM's responses—see Table 5). However, the normal inhibitory input from the right hemisphere vestibular cortex has been removed, due to the large lesion, and this results in disinhibition of the left vestibular cortex and over-perception of rightward body rotations. Functional neuroimaging after cold CVS shows evidence of strong deactivations in cortical regions ipsilateral to the stimulated ear, suggesting that some kind of inhibitory processes may be at work (Bottini et al., 2001). This asymmetrical pattern is quite similar to the impact of right versus left hemisphere cortical injuries on the manifestation of hemispatial neglect (Corbetta, Miezin, Shulman, & Petersen, 1993; Kim et al., 1999; Mennemeier, Vezey, Chatterjee, Rapcsak, & Heilman, 1997; Mesulam, 1981). This similarity lends some credence to the possibility that JM and TG are not idiosyncratic cases. Interestingly, JM did not show any standard symptoms of neglect at the time of testing. The processes underlying hemispatial neglect are not likely to be the basis of JM's perturbed pointing, however, because patients do not typically show asymmetrical perception of whole-body rotations even when they manifest neglect symptoms (Philbeck et al., 2001). The fact that TG's responses in body rotation trials were relatively symmetrical despite his neglect symptoms further supports this view.

Although the relation between CVS and illusions of body motion is complex, warm and cold CVS in supine observers tend to produce illusory body motion toward the ipsilateral and contralateral sides, respectively (Lidvall, 1961); warm CVS is associated with a preponderance of activation in ipsilateral cortical vestibular structures, while cold CVS is associated with a preponderance of contralateral activation (e.g., Dieterich et al., 2003; Fasold et al., 2002; Suzuki et al., 2001; Vitte et al., 1996). It may be tempting to conclude that perceived unidirectional body rotation is a direct functional correlate of cortical vestibular activation in these studies, but if this is true, the neuroimaging results would suggest that the left hemisphere predominantly encodes body rotations toward the *ipsilateral* side of the body. If JM's case is representative, however, our results argue for a predominantly *contralateral* representation of vestibular information in the left hemisphere (perhaps in concert with the right hemisphere, which may encode rotations toward both the ipsi- and contralateral side of the body). Interactions between hemispheres likely play a significant role in this apparent discrepancy, although these interactions remain poorly understood. As it is typically utilized in functional neuroimaging studies, CVS generates signal changes in the vestibular nerve of a single ear, whereas natural head rotation affects signals in both ears (for a review, see Highstein, 1996). Thus, neuroimaging studies tend to reflect vestibular processing that has been deprived of its normal bilateral peripheral input. In addition, cold CVS is associated with strong *deactivations* in some ipsilateral regions (most notably those related to vision and eye movements), as well as the aforementioned preponderance of contralateral activations and bilateral activations (Bottini et al., 2001). The functional significance of all these factors is currently unclear. Taken together, these findings highlight the need for a more comprehensive understanding of the functional correlates of activity in vestibular cortex and indicate that activation should not necessarily be taken at face value as indicating ongoing perceptual processes.

For both leftward and rightward body rotations, JM's responses exhibited a large constant offset. This is observable in the abnormally high intercept values in Table 5. The cause of this offset is unknown, but one possibility is that non-vestibular (e.g., somatosensory) cues played a role in registering the change in acceleration at the onset and offset of rotations in both directions. These cues may have generated a default value of perceived rotation, with the dynamic angular motion signals being evaluated relative to that value. Eye movements related to the vestibular-ocular reflex (VOR) might also have contributed to self-motion sensing for rotations in both directions. Certain brain injuries, at both the cortical and subcortical levels, can result in asymmetrical VOR (for reviews, see Berthoz, 1996; Brandt, 1999). We did not measure VOR in JM, so the possibility remains that asymmetries in VOR-related eye movements may have influenced his judgments. We think it unlikely, however, that eye movement impairments would make more than a minor contribution toward JM's asymmetries in perceiving whole-body rotations.

Even without a fine-grained localization of function, this study provides valuable insight into the possible consequences of cortical injury on vestibular processing. In particular, very little is known about the impact of unilateral cortical damage on the ability to sense dynamic body rotations. To our knowledge, this study is the first to show large asymmetries in the perception of angular head motion after unilateral injuries in vestibular cortex. Our results complement the findings of neuroimaging studies and emphasize the need for further investigation of the functional correlates of activity in vestibular cortex.

Acknowledgements

This work was supported by NIH Grant F32 MH11791, NIH Grant RO1 NS052137, and James S. McDonnell Foundation Grant 97-17 to JWP, as well as NIH Grant RO1 MH54246 to MB. Correspondence concerning this article should be addressed to J.W. Philbeck, Department of Psychology, George Washington University, 2125 G. Street, N.W., Washington, DC 20052 (e-

mail: philbeck@gwu.edu). The authors would like to thank Dr. Maxim Hammer for assistance in clinical interpretation and Dr. Jeff Gourley for assistance in manuscript preparation.

References

Akbarian, S., Grüsser, O.-J., & Guldin, W. O. (1992). Thalamic connections of the vestibular cortical fields in the squirrel monkey (*saimiri sciureus*). *Journal of Comparative Neurology*, *326*, 423–441.

Behrmann, M., Ghiselli-Crippa, T., & Di Matteo, I. (2001–2002). Impaired initiation but not execution of contralateral saccades in patients with hemispatial neglect. *Behavioral Neurology*, *13*, 39–60.

Behrmann, M., & Plaut, D. C. (2001). The interaction of spatial reference frames and hierarchical object representations: Evidence from figure copying in hemispatial neglect. *Cognitive, Affective, and Behavioral Neuroscience*, *1*, 307–329.

Bense, S., Stephan, T., Yousry, T. A., Brandt, T., & Dieterich, M. (2001). Multisensory cortical signal increases and decreases during vestibular galvanic stimulation (fMRI). *Journal of Neurophysiology*, *85*, 886–899.

Berthoz, A. (1996). How does the cerebral cortex process and utilize vestibular signals? In R. W. Baloh & G. M. Halmagyi (Eds.), *Disorders of the vestibular system* (pp. 113–125). Oxford: Oxford University Press.

Bisdorff, A. R., Wolsley, C. J., Anastasopoulos, D., Bronstein, A. M., & Gresty, M. A. (1996). The perception of body verticality (subjective postural vertical) in peripheral and central vestibular disorders. *Brain*, *119*, 1523–1534.

Bisiach, E., & Vallar, G. (2001). Unilateral neglect in humans. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology: Vol. 1* (2nd ed.). North-Holland, Amsterdam: Elsevier Science.

Blouin, J., Gauthier, G. M., & Vercher, J.-L. (1995). Failure to update the egocentric representation of the visual space through labyrinthine signal. *Brain and Cognition*, *29*, 1–22.

Bottini, G., Karnath, H.-O., Vallar, G., Sterzi, R., Frith, C. D., Frackowiak, R. S. J., et al. (2001). Cerebral representations for egocentric space: Functional-anatomical evidence from caloric vestibular stimulation and neck vibration. *Brain*, *124*, 1182–1196.

Bottini, G., Sterzi, R., Paulesu, E., Vallar, G., Cappa, S. F., Erminio, F., et al. (1994). Identification of the central vestibular projections in man: A positron emission tomography activation study. *Experimental Brain Research*, *99*(1), 164–169.

Brandt, T. (1999). *Vertigo: its multisensory syndromes* (2nd ed). London: Springer-Verlag.

Brandt, T., & Dieterich, M. (1999). The vestibular cortex. Its locations, functions, and disorders. *Annals of the New York Academy of Sciences*, *871*, 293–312.

Brandt, T., Dieterich, M., & Danek, A. (1994). Vestibular cortex lesions affect the perception of verticality. *Annals of Neurology*, *35*, 403–412.

Bucher, S. F., Dieterich, M., Wiesmann, M., Weiss, A., Zink, R., Yousry, T. A., et al. (1998). Cerebral functional magnetic resonance imaging of vestibular, auditory, and nociceptive areas during galvanic stimulation. *Annals of Neurology*, *44*, 120–125.

Corbetta, M., Miezin, F. M., Shulman, G. L., & Petersen, S. E. (1993). A PET study of visuospatial attention. *Journal of Neuroscience*, *13*(3), 1202–1226.

Dieterich, M., Bense, S., Lutz, S., Drzezga, A., Stephan, T., Bartenstein, P., et al. (2003). Dominance for vestibular cortical function in the non-dominant hemisphere. *Cerebral Cortex*, *13*(9), 994–1007.

Emri, M., Kisely, M., Lengyel, Z., Balkay, L., Márián, T., Mikó, L., et al. (2003). Cortical projection of peripheral vestibular signaling. *Journal of Neurophysiology*, *89*, 2639–2646.

Farrell, M. J., & Robertson, I. H. (2000). The automatic updating of egocentric spatial relationships and its impairment due to right posterior cortical lesions. *Neuropsychologia*, *38*, 585–595.

Fasold, O., von Brevern, M., Kuhberg, M., Ploner, C. J., Villringer, A., Lempert, T., et al. (2002). Human vestibular cortex as identified with caloric stimulation in functional magnetic resonance imaging. *NeuroImage*, *17*, 1384–1393.

Fisher, N. I. (1993). *Statistical analysis of circular data*. Cambridge: Cambridge Universitiy Press.

Friberg, L., Olsen, T., Roland, P., Paulson, O., & Lassen, N. (1985). Focal increase of blood flow in the cerebral cortex of man during vestibular stimulation. *Brain*, *108*(3), 609–623.

Guedry, F. E., Stockwell, C. W., & Gilson, R. D. (1971). Comparison of subjective responses to semicircular canal stimulation produced by rotation about different axes. *Acta Oto-laryngologica*, *72*, 101–106.

Guldin, W. O., & Grüsser, O.-J. (1998). Is there a vestibular cortex? *Trends in Neurosciences*, *21*, 254–259.

Halligan, P. W., Fink, G. R., Marshall, J. C., & Vallar, G. (2003). Spatial cognition: Evidence from visual neglect. *Trends in Cognitive Sciences*, *7*, 125–133.

Hawrylyshyn, P. A., Rubin, A. M., Tasker, R. R., Organ, W., & Fredrickson, J. M. (1978). Vestibulo-thalamic projections in man–a sixth primary sensory pathway. *Journal of Neurophysiology*, *41*, 394–401.

Highstein, S. M. (1996). How does the vestibular part of the inner ear work? In R. W. Baloh & M. Halmagyi (Eds.), *Disorders of the vestibular system* (pp. 3–11). New York: Oxford University Press.

Israël, I., Rivaud, S., Gaymard, B., Berthoz, A., & Pierrot-Deseilligny, C. (1995). Cortical control of vestibular-guided saccades in man. *Brain*, *118*(5), 1169–1183.

Kahane, P., Hoffman, D., Minotti, L., & Berthoz, A. (2003). Reappraisal of the human vestibular cortex by cortical electrical stimulation study. *Annals of Neurology*, *54*, 615–624.

Karnath, H.-O. (1994). Subjective body orientation in neglect and the interactive contribution of neck muscle proprioception and vestibular stimulation. *Brain*, *117*, 1001–1012.

Karnath, H.-O., Johannsen, L., Broetz, D., & Küker, W. (2005). Posterior thalamic hemorrhage induces "pusher syndrome". *Neurology*, *64*, 1014–1019.

Karnath, H.-O., & Perenin, M.-T. (1998). Tactile exploration of peripersonal space in patients with neglect. *NeuroReport*, *9*, 2273–2277.

Kim, Y.-H., Gitelman, D. R., Nobre, A. C., Parrish, T. B., LaBar, K. S., & Mesulam, M.-M. (1999). The large-scale neural network for spatial attention displays multifunctional overlap but differential asymmetry. *NeuroImage*, *9*(3), 269–277.

Kotchabhakdi, N., & Walberg, F. (1978). Primary vestibular afferent projections to the cerebellum as demonstrated by retrograde axonal transport of horseradish peroxidase. *Brain Research*, *142*, 142–146.

Lidvall, H. F. (1961). Vertigo and nystagmus responses to caloric stimuli repeated at short intervals. *Acta oto-laryngologica*, *53*, 33–44.

Lobel, E., Kleine, J. F., Bihan, D. L., Leroy-Willig, A., & Berthoz, A. (1998). Functional MRI of galvanic vestibular stimulation. *Journal of Neurophysiology*, *80*, 2699–2709.

Mennemeier, M., Vezey, E., Chatterjee, A., Rapcsak, S. A., & Heilman, K. M. (1997). Contributions of the left and right cerebral hemispheres to line bisection. *Neuropsychologia*, *35*, 703–715.

Mesulam, M.-M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of Neurology*, *10*, 309–325.

Milner, A. D., & Goodale, M. A. (1995). *The visual brain in action*. Oxford, England: Oxford University Press.

Penfield, W., & Jasper, H. (1954). *Epilepsy and the functional anatomy of the human brain*. Boston: Little, Brown.

Petit, L., & Beauchamp, M. S. (2003). Neural basis of visually guided head movements studied with fMRI. *Journal of Neurophysiology*, *89*, 2516–2527.

Philbeck, J. W., Behrmann, M., & Loomis, J. M. (2001). Updating of locations during whole-body rotations in patients with hemispatial neglect. *Cognitive, Affective, and Behavioral Neuroscience*, *1*(4), 330–343.

Rode, G., Charles, N., Perenin, M.-T., Vighetto, A., Trillet, M., & Aimard, G. (1992). Partial remission of hemiplegia and somatoparaphrenia through vestibular stimulation in a case of unilateral neglect. *Cortex*, *28*, 203–208.

Rode, G., & Perenin, M.-T. (1994). Temporary remission of representational hemineglect through vestibular stimulation. *Neuroreport*, *5*, 869–872.

Suzuki, M., Kitano, H., Ito, R., Kitanishi, T., Yazawa, Y., Ogawa, T., et al. (2001). Cortical and subcortical vestibular response to caloric stimulation detected by functional magnetic resonance imaging. *Cognitive Brain Research*, *12*, 441–449.

Tropper, J., Melvill Jones, G., Bloomberg, J., & Fadlallah, H. (1991). Vestibular perceptual deficits in patients with parietal lobe lesions: A preliminary study. *Acta Otolaryngologica (suppl.)*, *481*, 528–533.

Vitte, E., Derosier, C., Caritu, Y., Berthoz, A., Hasboun, D., & Soulie, D. (1996). Activation of the hippocampal formation by vestibular stimulation: A functional magnetic resonance imaging study. *Experimental Brain Research*, *112*, 523–526.

Wilson, B., Cockburn, J., & Halligan, P. (1987). *Behavioral inattention test*. Flempton, Bury St. Edmunds, Suffolk, England: Thames Valley Test Company.

Yardley, L., Gardner, M., Lavie, N., & Gresty, M. (1999). Attentional demands of perception of passive self-motion in darkness. *Neuropsychologia*, *37*, 1293–1301.