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# Unilateral posterior parietal lobe lesions affect representation of visual space

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**Abstract**

This study assessed accuracy of visually perceived vertical and trunk median plane orientation in 41 subjects: 17 had unilateral brain lesions including the posterior parietal lobe (PPL), 8 had lesions outside PPL, and 16 were neurologically normal. Vertical perception errors clearly increased with size of unilateral lesions to PPL and posterior superior temporal gyrus (PSTG). Median plane perception errors increased only slightly with size of unilateral lesions to frontal lobe premotor areas and supramarginal gyrus. These results are compatible with the hypothesis that accurate visual vertical perception depends critically on intact PPL and PSTG in both cerebral hemispheres while accurate median plane perception likely involves a bihemispheric network that can compensate for lesions to one hemisphere.


## 1. Introduction

We used the human lesion method to assess the role of posterior parietal cortex in perception of object orientation. Lesions in the posterior cerebral hemispheres may shift the perceived orientations of visual representations with respect to different frames of reference (Fig. 1). For instance, damage to parieto-insular vestibular cortex (PIVC) may cause tilt of the visually perceived earth-fixed vertical axis away from the side of the lesion (Brandt, Dieterich, & Danek, 1994). Similarly, hemineglect due to acute or chronic lesions in frontal (Teuber & Mishkin, 1954) and posterior parietal and temporal cortices (Kerkhoff & Zoelch, 1998) may also cause tilt of visually perceived earth-fixed vertical axis and rotation (Karnath, Schenkel, & Fischer, 1991; Ventre, Flandrin, & Jeannerod, 1984) or displacement (Jeannerod & Biguer, 1987; Mark & Heilman, 1990) of the trunk midpoint (median plane) toward ipsilesional hemispace. Such displacements have been found using (a) kinesthetic "point straight ahead" tasks (Chokron & Bartolomeo, 1998; Heilman, Bowers, & Watson, 1983) and (b) visual tasks in which subjects stop a moving target directly in front of the body midline (e.g., Ferber & Karnath, 1999; Karnath, 1994; Rossetti et al., 1998), although a large study including 23 individuals with hemineglect did not confirm the results of these previous studies (Farne, Ponti, & Ladavas, 1998).

Neurologically normal observers perceive orientation (of objects and themselves) more accurately with respect to an earth-fixed vertical axis and a trunk-fixed anterior–posterior (*a–p*) axis than with respect to other head- or trunk-fixed axes (Fig. 1), suggesting these two axes may define a coordinate system for visual spatial perception of orientation of external objects (Darling, Butler, & Williams, 1996; Darling & Hondzinski, 1997). Studies of human brain activation (Galati et al., 2000; Vallar et al., 1999) suggest that percepts related to the trunk-fixed *a–p* axis (mid-sagittal plane direction) depend on a bilateral fronto-parietal network involving the angular gyrus of the posterior parietal lobe (PPL), superior occipital gyrus (area 19), lateral premotor cortex (area 6) and inferior frontal gyrus (area 44). In these studies, subjects pressed a button when a moving bar or spot of light

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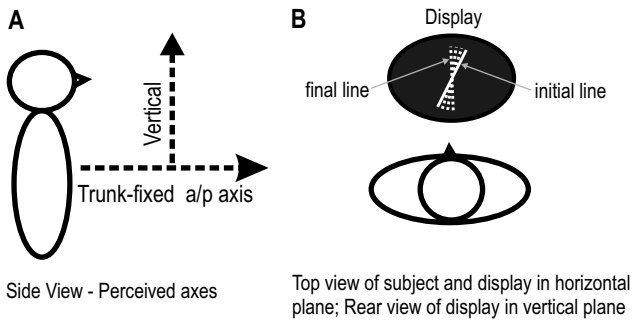


Fig. 1. Diagram of perceived axes (A) and experimental setup (B). The circle indicates the subject's head and ellipse the trunk. A side-view of the subject is shown in A with the trunk-fixed *a–p* axis and earth-fixed vertical axes shown. A top or rear (oblique) view of the subject is shown in (B) with the display and a line in an initial orientation (solid white line) and intermediate and final orientations (dashed white lines—note that the actual line remained solid and initial and intermediate line orientation did not remain in view while the subject rotated the line to the final orientation).

passed the mid-sagittal plane, indicating perceived trunk midline location (rather than direction of the trunk-fixed *a–p* axis). Percepts related to vertical may involve PIVC that is activated by galvanic stimulation of the vestibular otoliths and includes the posterior insula and supramarginal gyrus of the PPL (Bucher et al., 1998).

While research on impaired orientation of visual spatial representations has focused on subjects with hemineglect (e.g., Farne et al., 1998; Karnath, 1994; Kerkhoff & Zoelch, 1998), hemineglect may not be necessary for the orientation defect. Posterior regions of the right superior temporal gyrus (PSTG) implicated in hemineglect may also be important in visual representations of extrapersonal space (Karnath, Ferber, & Himmelbach, 2001). However, parietal lobe lesions may impair visual spatial representations of vertical and body-fixed axes, even in the absence of hemineglect. Hemineglect is well known to recover after an acute lesion of the parietal lobes (Anderson, 2003), while orientation perception defects may persist. Along these lines, Holmes (1946) suggested that judgment along the *a–p* axis (median plane or straight ahead) "requires integrations of impressions of various origins" (e.g., binocular vision and eye muscle proprioception) and can be impaired by lesions in parieto-occipital regions, angular gyrus and white matter impaired independently of hemispatial neglect.

We tested the hypothesis that unilateral lesions of the PPL cause impaired judgments of object orientation relative to earth-fixed vertical and trunk-fixed *a–p* axes with the head erect and aligned to the trunk and with head orientation varied. We used multiple regression analysis to assess whether lesions of different regions of the PPL (i.e., inferior and superior parietal lobules), posterior insula (PI), PSTG and lateral premotor areas (Brodmann's 6, 44) impair judgment of object orientation to earth-fixed vertical and trunk-fixed *a–p* axes. Furthermore, because unilateral cortical lesions may disrupt processing of visual information presented on one side of body midline, we tested whether perceptual errors were larger when the perceived object was displayed on the side opposite the lesion versus the side ipsilateral to the lesion.

## 2. Methods

### 2.1. Subjects

Sixteen neurologically normal controls (6 males, 10 females) aged 28–83 years [59.7 (mean) ± 16.1 (s.d.) years], 17 individuals with lesions involving the PPL (Table 1—age: 55.7 ± 15.1 years) and 8 individuals with cerebral lesions outside the parietal lobe (Table 2—age: 50.8 ± 15.9 years) participated in these experiments. All subjects with brain lesions were tested in the chronic phase of recovery, 4 months or more after the ictus. The lesion locations specified in Tables 1 and 2 were defined from MR or CT images according to standard techniques (Damasio & Damasio, 1989; Damasio & Frank, 1992).

To allow comparisons of perceptual errors among normal controls, subjects with cortical lesions outside PPL (to test whether brain lesions in general cause defects in representation of visual space) and subjects with unilateral PPL lesions, we divided subjects into four groups: (1) 16 neurologically normal controls (CON), (2) 8 subjects with cortical lesions not involving the PPL (Table 2—lesion controls—LCON), (3) 7 subjects with focal lesions including primarily the right PPL [RPPL—Table 1], (4) 10 subjects with focal lesions including the left PPL [LPPL—Table 1]. Note that in the LPPL and RPPL groups there were subjects with lesions limited to the inferior and superior parietal lobules and subjects with lesions involving both lobules. We assessed contributions of different regions of PPL using multiple regression analysis (see below). Visual fields information is also provided in Table 1 because previous research has shown that visual field defects can influence visual perception of subjective straight ahead (Ferber & Karnath, 1999). Notably, none of the subjects with PPL lesions had visual field defects and only one of the control lesion subjects had a visual field defect (left upper quadrantanopia).

All subjects with brain lesions had complete neurological examinations including motor control, strength, gait, tone, station, reflexes, somatosensory ability including proprioception (joint position sense) and coordination—including visuomotor coordination, e.g., rapid alternating appendicular movements; moving the hand accurately to touch extrinsic targets (the examiner's finger) and intrinsic targets (nose, ear, other body

Table 1
Ages, lesion locations and neglect ratings for individuals with posterior parietal cortex lesions

| Subject/group[a] | Age | Sex | Visual fields[b] | Location[c], size[d] and cause[e] of lesion | Neglect ratings[f] | |
|---|---|---|---|---|---|---|
| | | | | | Acute | Chronic |
| LPPL-1 | 72 | M | F | P01(2); CVA | 1 | 1 |
| LPPL-2 | 42 | F | F | P01(2),P05(3),T08-9(2); CVA | 1 | 1 |
| LPPL-3 | 54 | F | F | P01(2),F06(3),F07(1),F08(3),-F12(1),BG1(2),BG2(3),BG3(2),-I01(3); CVA | 1 | 1 |
| LPPL-4 | 76 | M | F | P01(3),P02(3),F06(3),F08(2),-T03(3),T04(3),T07(3),T08(3),-T09(3),O04(2),O05(2),I01(3),-I02(3); CVA | 1 | 1 |
| LPPL-5 | 48 | M | F | P01(3),P02(3),F06(3),F08(2),-I01(3),I02(3); CVA | 1 | 1 |
| LPPL-6 | 52 | F | F | P01(3),P02(2),P05(3),T09(2); SR | 1 | 1 |
| LPPL-7 | 71 | M | FC | P01(3),P02(2),F06(2),F08(2),-T04(2),T07(3),T09(3),I01(3),-I02(3); CVA | 1 | 1 |
| LPPL-8 | 43 | F | F | Left: F02(3),P04(1),C02(2),C03(3); Right: F02(2),C02(2),C03(2); SR | 1 | 1 |
| LPPL-9 | 66 | M | F | P03(1-white); SR | 1 | NT |
| LPPL-10 | 48 | F | F | F02(2); F05(2); P04(2) | 1 | 1 |
| RPPL-1 | 83 | M | F | F06(2),F08(2),T08(3),T09(3),-T12(2),P01(2),T12(2),P01(2),-I01(3),I02(3); CVA | 1 | 1 |
| RPPL-2 | 52 | M | F | F06(2),F08(2),T03(2),T07(2),-T08(2),T09(2),T12(2),P01(2),-P02(1),BG2(3),BG3(3),IC1(2),I-C2(2),IC3(2),I01(3),I02(3); CVA | NT | 1 |
| RPPL-3 | 28 | F | F | T04(2),T06(2),T10(2),P01(2),-P02(2),O05(2),TH1(2),TH2(2),T-H3(2),TH4(2); CVA | 3 | 1 |
| RPPL-4 | 54 | M | FC | F01(2),F06(3),F07(2),F08(2),-F09(2),F11(2),T03(2),T04(2),-T05(3),T06(3),T09(3),T12(2),-P01(3),P02(2),P05(3),P06(2),-I01(3),I02(3); CVA | 3 | 3 |
| RPPL-5 | 60 | M | FC | F08(2),T09(2),P01(2),BG1(2),-I02(2); CVA | 3 | 1 |
| RPPL-6 | 49 | F | FC | F06(2),F08(2),T07(2),T08(2),-T09(2),P01(2),P02(2),I01(2),I02(2); CVA | 1 | 1 |
| RPPL-7 | 35 | F | FC | P04(3); CVA | NT | 1 |

[a] Group: L—left side, R—right side, PPL—posterior parietal lobe.
[b] Visual fields: F—no defect, tested; FC—full to confrontation, not tested; NT—visual fields not tested, LHH—left homonymous hemianopia, LUQ—left upper quandrantanopia.
[c] Location of lesions: as specified in Damasio and Damasio (1989): F—frontal, O—occipital, T—temporal, P—parietal, I—Insula.
[d] Size of damaged area in lesion is indicated by the number in parentheses after the location (1—less than 25%, 2—25–75%, 3—more than 75%).
[e] Cause of lesion: CVA—cerebrovascular accident or SR—surgical resection.
[f] Neglect rating is from an assessment of the presence and degree of neglect (1 = absent, 2 = mild, 3 = severe) based on neuropsychological testing (Rey–Osterreith complex figure test—copy version, a line cancellation task, a freehand drawing (of a clock), as well as pattern of performance on the Benton visual retention test and judgment of line orientation) completed shortly after the lesion (acute) or more than 3 months after the lesion (chronic); NT—not tested for neglect.

parts). These subjects also participated on a detailed battery of standard neuropsychological tasks, as part of an ongoing study of anatomical substrates of human behavior. This included standardized tests of attention, perception, language, memory and executive functions, and a specific assessment for hemineglect. Tests sensitive to neglect included line cancellation (LC), the Rey–Osterreith complex figure test (CFT), and extinction of left-side visual targets with double simultaneous stimulation (DSS) in both hemifields, Benton VRT and judgement of line orientation. Severe left hemineglect is operationally defined by failure to cross >30% of lines on the left side in the LC test and left-sided extinction to DSS. Moderate neglect corresponds to normal or near

Table 2
Ages and lesion locations for individuals with lesions outside posterior parietal cortex

| Subject/ group[a] | Age | Sex | Hemisphere of lesion[b] | Vis fields | Location[c], size[d] and cause of lesion[e] | Neglect rating[f] | |
|---|---|---|---|---|---|---|---|
| | | | | | | Acute | Chronic |
| LCON-1 | 27 | M | L | FC | T05(2); T10(1); T12(3) | 1 | 1 |
| LCON-2 | 49 | M | L | F | T03(1); T08(1); T09(2); CVA | 1 | 1 |
| LCON-3 | 66 | F | L | FC | T04(2) | 1 | 1 |
| LCON-4 | 56 | M | B | F | Left: F06(3); F07-8(2); F11(2); T04(3); T08(2); Right: F12(2); SR | 1 | 1 |
| LCON-5 | 58 | M | R | F | T08(3); T12(2); BG3(2); I01(3); I02(1); CVA | 1 | 1 |
| LCON-6 | 30 | F | R | F | T05(1); T10(3); T12(3); SR | 1 | 1 |
| LCON-7 | 67 | M | R | LUQ | T06(1); O01(2); O03(2); CVA | 1 | 1 |
| LCON-8 | 43 | M | R | FC | T03(1); T04(2); T08(2); T09(1); CVA | 1 | 1 |

[a] Group: CL—control lesion group.
[b] Hemisphere of lesion: L—left, R—right, B—bilateral.
[c] Location of lesions: as specified in Damasio and Damasio (1989): F—frontal, O—occipital, T—temporal, P—parietal, I—Insula.
[d] Size of damaged area in lesion is indicated by the number in parentheses after the location (1—less than 25%, 2—25–75%, 3—more than 75%).
[e] Cause of lesion: CVA—cerebrovascular accident or SR—surgical resection.
[f] Neglect rating is from an assessment of the presence and degree of neglect (1 = absent, 2 = mild, 3 = severe) based on neuropsychological testing (Rey–Osterreith complex figure test—copy version, a line cancellation task, a freehand drawing (of a clock), as well as pattern of performance on the Benton visual retention test and judgment of line orientation) completed shortly after the lesion (acute) or more than 3 months after the lesion (chronic); NT—not tested for neglect.

normal performance on LC, but significant asymmetry of missing components (L > R) on the CFT. Mild neglect is diagnosed if the subject performs normally on the LC test, but shows clear asymmetry (L > R) of missing components on the CFT. Based on these criteria, only one subject showed evidence of left neglect at the time this study was conducted (Table 1—RPPL-4). This subject had a right inferior parietal lobule (IPL) lesion that also extended into the mesial deep right superior parietal lobule (SPL) paraventricular area. All subjects gave voluntary written informed consent in accord with institutional and US HHS guidelines.

### 2.2. Procedure

The subject sat comfortably in front of a dim visual display (15″ computer screen positioned such that the middle of the screen was in line with body midline) with an obliquely oriented thin white line presented on a black background. The computer display was covered with a black elliptical mask to block view of the sides of the computer screen (Fig. 1). The experiments were carried out in a dark room to minimize environmental (allocentric) cues to vertical or the *a–p* direction. Luminance (measured with a Minolta LS-110 photometer) of the black background display was 0.0 cd/m$^2$ and of the thin white line was 0.05 cd/m$^2$. Room lights were turned on between each experimental condition to prevent dark adaptation. The subject was instructed to use arrow keys on the keyboard to align the white line to vertical (with the display positioned vertically) in one experiment or parallel to the trunk-fixed *a–p* axis (with the display positioned horizontally) in a second experiment (Fig. 1). When the subject perceived the line to be aligned to the desired axis, s/he pressed the space bar. The computer program ignored pressing of other keys by the subject. The arrow keys and space bar were covered with felt for easy identification by touch. The initial orientation of the line was varied randomly between 0.3–0.5 rad clockwise (cw) and counter-clockwise (ccw) from vertical or the true *a–p* axis. Vertical was described to the subjects as an imaginary line pointing directly upward and perpendicular to the floor. The trunk-fixed *a–p* axis was described as an imaginary line extending straight forward from the trunk or sternum. All subjects were able to understand these instructions. It should be noted that the task of aligning the line to the trunk-fixed *a–p* axis differs from the "subjective straight ahead" task used in previous research (e.g., Farne et al., 1998) in which subjects stop or position a spot of light directly in front of perceived body midline position. Specifically, the task used here is a line orientation task requiring specification of direction of the trunk *a–p* axis.

There were six experimental conditions to permit testing the effects of varying position of the displayed line relative to body midline (central—directly in front of body midline, left of midline, right of midline) and head orientation (erect or varied). Head orientation was varied by left/right lateral flexion (when perceiving

vertical) and left/right rotation about a vertical axis (when perceiving the trunk-fixed *a–p* axis). Subjects voluntarily maintained the head in the specified tilted or rotated orientation and in the standard erect position in the different conditions. Five trials were performed in each of the head erect conditions and 20 trials were performed in each of the conditions in which head orientation was varied. The line was always positioned directly in front of body midline, with the head maintained in an erect position, for the first set of trials. Order of performing in other conditions was varied among subjects.

### 2.3. Data recording

Orientation of the line on the screen was calculated from the pixel coordinates of the line endpoints when the subject pressed the space bar to indicate that the line was aligned to the desired axis. Head orientation was recorded using an electromagnetic system (Ascension Technologies minibird system). This system provides 6 degree of freedom information (3-dimensional location and orientation) of a receiver fixed to the body segment of interest relative to an electromagnetic transmitter. The receiver was fixed to an adjustable headpiece that was fitted snugly to the subject's head.

### 2.4. Data analysis

Perceptual errors were computed from the difference in angular orientation of the line from the orientation of the axis to be perceived (i.e., vertical or trunk-fixed *a–p* axis). Constant errors were computed as mean of the perceptual errors in each condition. Absolute constant errors were computed as the magnitude of the constant errors. Variable errors were computed as the standard deviation of the errors in each condition. Effects of age, head orientation, line position and brain lesions were examined using three-way repeated measures analysis of covariance with age as the covariate and factors of group (controls, lesion controls, left PPL lesions, right PPL lesions), head position (erect, varied) and line position (left, center right). We also assessed effects of age on absolute constant errors and variable errors by computing correlation coefficients for each of the six different experimental conditions. Geisser–Greenhouse epsilon values were examined to determine whether the assumption of sphericity was met when there were three or more levels in a repeated measures factor (i.e., location of presented line). Adjustments of degrees of freedom for $F$-tests were made on the basis of the epsilon values, resulting in adjusted $p$-values (see Section 3). Post hoc testing was carried out using Tukey's procedure. Regression analysis was used to determine whether the errors on individual trials (dependent variable) depended on head orientation or initial orientation of the line (independent variables) at the start of each trial. $Z$-transformed correlation coefficients were entered into repeated measures ANOVAs for comparison among the subjects in different groups and for different locations of the line relative to body midline. We also assessed whether subjects' perceptions were biased toward the head longitudinal axis (when aligning the line to vertical) and the head *a–p* axis (when aligning the line to the trunk-fixed *a–p* axis) using single linear regression as described in previous studies (Darling et al., 1996; Darling & Hondzinski, 1997).

To assess possible roles of different regions of various brain areas in generating representations of visual space, we used stepwise multiple regression to evaluate the relationship between perceptual errors and the location and size of the lesions in different brain areas. These regressions were performed for conditions in which the line was positioned directly in front of body midline with head orientation erect and with head orientation varied, except where noted in Section 3. Lesion volumes were estimated using the techniques of Damasio and Damasio (1989). Specifically, lesion volumes within each region of IPL (P01, P02), SPL (P03-P06), PSTG (T09), PI (I02), areas 6 and 44 (F06, F08—which also include primary somatosensory areas 1,2,3, primary motor area 4 and area 45) and total of all other lesioned cortical areas were quantified as in Tables 1 and 2 as 0 (no lesion), 1 (up to 25% of region), 2 (25–75% of region), or 3 (75–100% of region). Thus, lesion volumes of IPL ranged from 0–6, SPL from 0–12, F06/F08 from 0–6, and PI from 0–3. This is a well-established technique to estimate lesion volume; but emerging computer-based morphometric techniques may improve future estimates. The stepwise multiple regression analysis initially included total PPL lesion volume and volumes of lesions in F06/F08, T09, I02 and total volume of lesions outside these areas as independent variables. A second stepwise multiple regression was run if PPL lesion volume contributed significantly to prediction of the errors ($p < 0.05$). This regression used supramarginal gyrus, angular gyrus and SPL lesion volumes as independent variables (and other areas lesion volumes if significant in the initial stepwise multiple regression).

## 3. Results

### 3.1. Perception of vertical

Most subjects with lesions of the PPL made larger variable, but not constant, errors in perception of the earth-fixed vertical axis than control subjects (Fig. 2). All neurologically normal control subjects had accurate perception of verticality under all conditions. Subjects with lesions outside the PPL were only slightly less accurate than controls (Fig. 2). There were no statistically

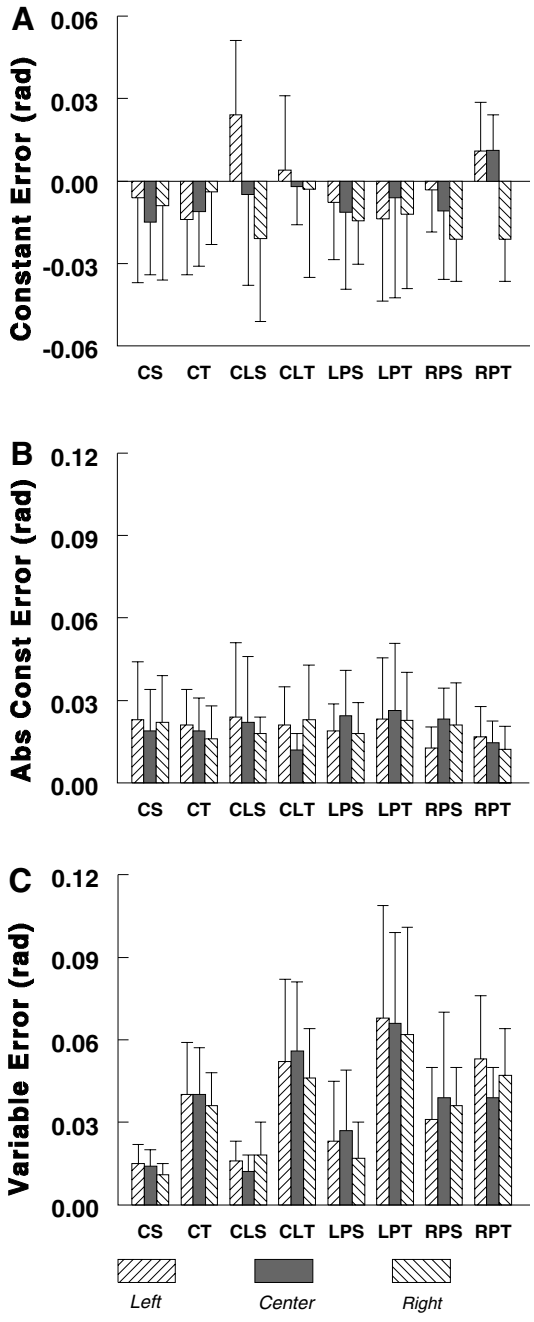


Fig. 2. Mean errors for visual perception of earth-fixed vertical. Average constant (A), absolute constant (B) and variable (C) errors are shown for each group of subjects. Abbreviations on the abscissae represent the group (C—controls, CL—lesion controls, LP—left PPL lesions, RP—right PPL lesions) and experimental conditions (S—standard head erect position, T—head tilted to left/right). Different hatched bars show errors when the line was presented on the left side of body midline (left), directly in front of midline (center), and on the right side of midline (right). Error bars are 1 standard deviation (s.d.).

significant differences among the four groups of subjects for constant ($p = 0.34$) or absolute constant ($p = 0.62$) errors. There were also no significant interactions involving the different subject groups in the absolute constant errors ($p > 0.3$ in all cases). However, constant errors differed between the right and left PPL groups when head orientation was varied, but not in other groups as shown by a significant group × condition interaction (Fig. 2A, $F_{3,33} = 3.34$, $p = 0.031$). Constant errors were counter-clockwise (ccw) in all groups, but subjects with right-sided lesions made clockwise (cw) errors when head orientation was varied in two of the three line locations (Fig. 2A). The age covariate did not significantly affect constant or absolute constant errors ($p > 0.5$).

Variable errors were generally larger in individuals with PPL lesions, especially in subjects with left PPL lesions when head orientation was varied (Fig. 2). Errors differed among groups (Fig. 2, $F_{3,33} = 3.71$, $p = 0.021$) and increased by 2–3× when head orientation was varied (Fig. 2, $F_{1,34} = 48.0$, $p < 0.001$). However, subjects with right PPL lesions did not exhibit greater variable errors when head orientation was varied, resulting in a strong trend for a dependence of variable errors on both group and head orientation main effects (Fig. 2, $F_{3,34} = 2.61$, $p = 0.07$). Post hoc comparisons showed that all groups had larger variable errors when head orientation was varied ($p < 0.029$) except subjects with right PPL lesions ($p = 0.94$). Furthermore, subjects with left PPL lesions had larger variable errors when head orientation was varied than normal controls ($p < 0.006$) but subjects with right PPL lesions did not ($p = 0.98$). When the head was erect, variable errors were similar in all groups ($p > 0.82$) except subjects with right PPL lesions who had larger variable errors than controls (Fig. 2, $p = 0.1$). Effect of the age covariate was not significant ($p = 0.33$).

Varying position of the displayed line relative to body midline had little influence on perceptual errors (Fig. 2). Constant, absolute constant and variable errors were not statistically different for different positions of the line (adjusted $p > 0.2$) and there were no significant interactions involving the line position effect (Fig. 2, $p > 0.14$).

There was no evidence that direction of constant errors depended on the side of the PPL lesion. Constant errors were usually ccw except that subjects with right PPL lesions had cw constant errors when head orientation was varied (Fig. 2A). Thus, ccw tilts of subjective visual vertical observed previously in individuals with acute hemineglect or "vestibular cortex" lesions (Brandt et al., 1994; Kerkhoff & Zoelch, 1998), were not observed in most subjects with chronic phase PPL lesions without hemineglect. Indeed, only one subject with a right IPL lesion (RPPL-5—Table 1) made large ccw constant errors (0.17 rad with the line displayed centrally). RPPL-5 had left hemineglect in the acute phase of brain injury, but RPPL-2 also had this condition in the acute phase (Table 1) and had low constant errors (<0.05 rad) in all conditions. RPPL-4, the only subject with chronic hemineglect (Table 1), had small cw

constant errors in all conditions. Thus, hemineglect in the acute or chronic stage injury does not necessarily cause long-lasting defects in visual perception of vertical.

Perception of vertical was strongly related to varied head orientations in most subjects (Fig. 3). Correlation coefficients exceeding 0.7 between errors and head orientation were commonly observed and average coefficients (computed using Fisher $Z$-transformations) exceeded 0.6 (Fig. 3B). The correlation coefficients were usually lower for subjects with RPPL lesions than controls (Fig. 3B, $F_{3,32} = 4.04$, $p = 0.015$, $p = 0.013$ for post hoc comparison of RPPL and CON groups) but were similar for the CON, LCON and LPPL groups ($p > 0.19$). The correlation coefficients were also similar for different line positions relative to body midline ($p = 0.6$) and the interaction of group by line position was not significant ($p = 0.75$). Many subjects (about 70%) showed some indication of perceptual bias toward or away from the head longitudinal axis (i.e., a significant positive correlation between perceptual errors and head longitudinal axis orientation in the frontal plane would indicate bias toward the head, a negative correlation would indicate bias away from the head—Darling & Hondzinski, 1997). However, about equal percentages of controls and subjects with PPL lesions showed such biases.

Variations in orientation of the line at the start of each trial had little effect on errors for aligning the line to vertical. Although the correlation coefficients were statistically significant in most cases, they were usually below 0.4 (Fig. 3D). There were no differences in correlation coefficients among the different groups ($p = 0.63$) or different line positions, although a trend for lower coefficients when the line was positioned on the left side of the screen was observed (adjusted $p = 0.051$).

Variable and constant errors depended on volumes of lesions of the PPL and PSTG, but not lesions to frontal lobe premotor areas. Multiple regression analyses showed that when the head was erect variable errors increased with increasing volumes of SPL (cells P03-6) and PSTG (cell T09) lesions (Fig. 4, $R = 0.77$, $p < 0.001$; $p < 0.05$ for regression coefficients for SPL and PSTG lesion volumes; $p > 0.2$ for lesion volumes of

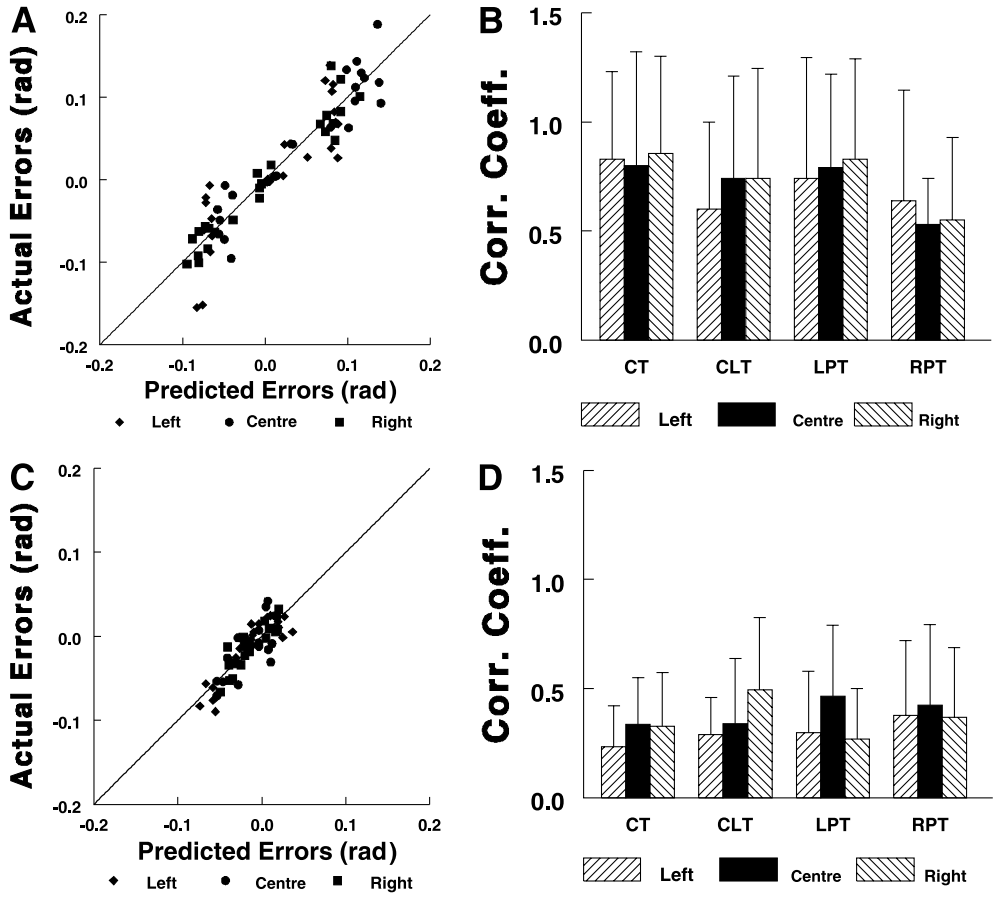


Fig. 3. Dependence of vertical perception errors on head orientations and initial line orientations. The scatterplots show single trial errors (ordinate) plotted against errors predicted from head orientation (abscissae) for CE who has a left IPL lesion (A) and a control subject (C). The bar graphs show mean correlation coefficients across subjects in each group for predictions of errors from head orientations (B) and starting orientation of the line (D). Error bars are 1 s.d.

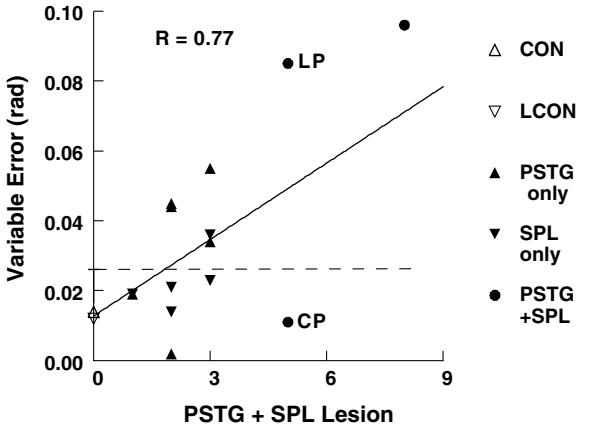


Fig. 4. Dependence of variable errors for perception of earth-fixed vertical on volume of PSTG and superior parietal lobule lesions. The scatterplot shows variable errors for individual subjects with focal lesions of PSTG only, superior parietal lobule only and PSTG + superior parietal lobule plotted against lesion volumes. Mean variable errors for neurologically normal (CON) and lesion (LCON) controls are also shown on the ordinate axis. The dashed line indicates 2 s.d. above the mean of normal controls. Variable errors of subject RPPL-4 and LPPL-2 are shown with initials beside the symbols.

supramarginal gyrus, angular gyrus, PI, F06/F08 and other cortical areas). When head orientation was varied, variable errors showed no significant dependence on lesion volumes of PPL, PI, PSTG, F06/F08 or other cortical areas ($R = 0.34$, $p = 0.09$). This is consistent with the finding that subjects with right posterior parietal lobule lesions did not have larger variable errors when head orientation was varied. However, excluding subjects with right posterior parietal lobule lesions produced a significant relationship between variable errors and lesion volumes of left PSTG and cortical areas outside of PPL and PSTG ($R = 0.46$, $p = 0.021$; $p > 0.1$ for lesion volumes of other areas). Absolute constant errors showed no dependence on lesion volumes of posterior parietal, posterior insula, F06/F08 or other brain areas ($R = 0.22$, $p = 0.87$) when the head was erect. In contrast, when head orientation was varied these errors increased with larger volumes of angular gyrus lesions ($R = 0.39$, $p = 0.012$), but not of any other brain areas ($p > 0.07$).

Fig. 4 also shows that two subjects with identical classifications for SPL and PSTG lesions (LPPL-6, LPPL-2—Table 1) had very different variable errors. LPPL-2, who had a small variable error of 0.011 rad, had a small IPL lesion that involved only the supramarginal gyrus while LPPL-6, who had a much larger variable error of 0.085 rad, had a larger IPL lesion involving both angular and supramarginal gyri. Notably, lesions of PSTG without accompanying PPL lesions did not cause large variable errors. LCON-2 and LCON-8 of the lesion control group had small PSTG lesions (Table 2) but had variable errors less than 0.02 rad (Fig. 4).

### 3.2. Perception of the trunk-fixed a–p axis

Constant errors were usually ccw in subjects with right and left PPL lesions, but were quite variable among subjects in all groups (Fig. 5). Some subjects with right PPL lesions (RPPL-4, RPPL-5, RPPL-2, RPPL-6)

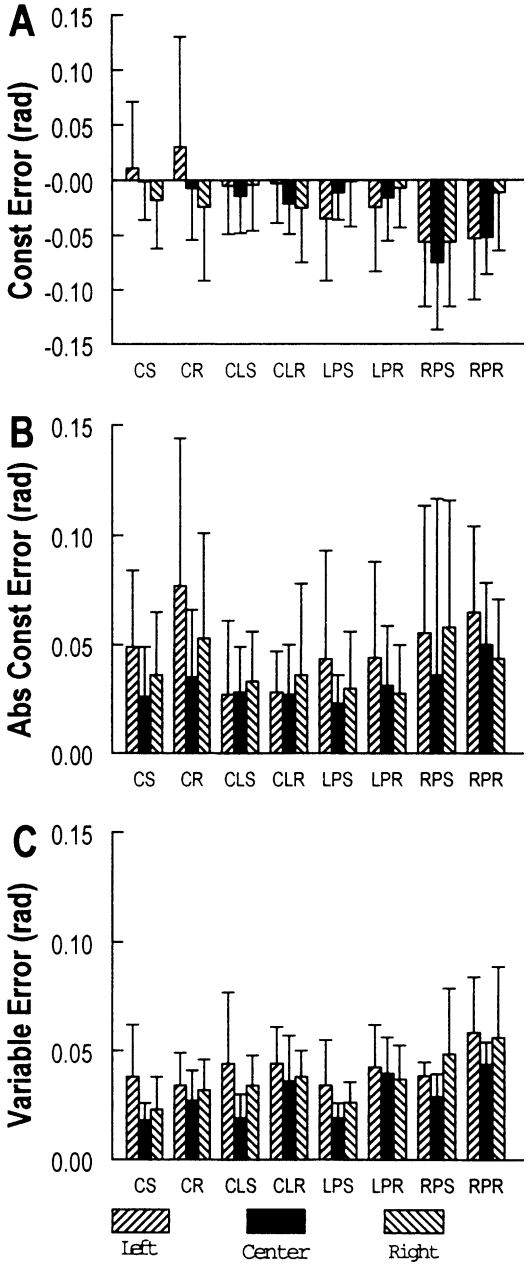


Fig. 5. Mean errors for visual perception of the trunk-fixed *a–p* axis. Average constant (A), absolute constant (B) and variable (C) errors are shown for each group of subjects. Abbreviations on the abscissae represent the group (C—controls, CL—lesion controls, LP—left PPL lesions, RP—right PPL lesions) and experimental conditions (S—standard head erect position, R—head rotated left/right relative to trunk). Different hatched bars show errors when the line was presented on the left side of body midline (left), directly in front of midline (center), and on the right side of midline (right). Error bars are 1 s.d.

exhibited very large ccw perceptual constant errors (Fig. 5), consistent with rotation of the trunk-fixed *a–p* axis away from the side of the lesion. However, subjects with left PPL lesions and lesion controls also usually had ccw constant errors (Fig. 5). In contrast, controls exhibited both cw and ccw constant errors. Effect of the age covariate was not significant (Rao's $R = 0.787$, $p = 0.587$). There were differences in constant errors among the different groups ($F_{3,33} = 2.9$, $p = 0.049$) and a group by line location interaction ($F_{6,68} = 2.41$, adjusted $p = 0.062$). Post hoc analysis showed that subjects with right PPL lesions performed similarly to all other groups when the line was displayed directly in front of body and to the right of midline ($p > 0.28$). However, when the line was displayed to the left of body midline, subjects with right PPL lesions had ccw constant errors that differed from controls ($p < 0.001$) but not from the other groups who also tended to have ccw constant errors ($p > 0.79$). Notably, three subjects with right PPL lesions and left hemineglect in the acute or chronic phases (Table 1—RPPL-5, RPPL-2, RPPL-4) made large ccw constant errors of 0.1–0.15 rad. However, LPPL-1 who had a left PPL lesion and no evidence of hemineglect (Table 1) also had a large leftward constant error of 0.16 rad when the line was displayed on the left side.

Constant errors were usually larger in subjects with right PPL lesions, especially when the line was positioned directly in front of body midline. However, there were no significant differences in absolute constant errors among the four groups (Fig. 5, $p = 0.23$). Effect of the age covariate was not significant ($p = 0.51$). There was a strong trend indicating a group by head orientation interaction effect ($F_{3,30} = 2.84$, $p = 0.053$). Post hoc tests showed that absolute constant errors for the RPPL group were larger than those for the LPPL group ($p = 0.036$) and controls ($p = 0.025$) with head orientation constant but when head orientation was varied errors were similar among these three groups ($p > 0.45$). The absolute constant errors were usually larger when the line was positioned on the left of body midline than when the line was in front of and to the right of midline (Fig. 5, $F_{2,68} = 5.9$, adjusted $p = 0.004$; $p < 0.015$ for post hoc tests) and were similar when the line was positioned centrally and to the right of body midline ($p = 0.85$). Interactions involving line location effects were not significant ($p > 0.27$).

Variable errors differed among the four groups (Fig. 5, $F_{3,33} = 4.24$, $p = 0.012$), with the largest errors by subjects with right PPL lesions. Post hoc analyses showed that errors by the RPPL group were significantly larger than those of normal controls ($p = 0.01$) and there was a trend for larger errors than in lesion controls ($p = 0.11$). Interactions involving group effects were not significant (Fig. 5, $p > 0.13$). Also, the age covariate was not significant (Rao's $R = 0.432$, $p = 0.851$).

Varying head orientation and positioning the line to the right and left of body midline increased variable errors (Fig. 5). Increases in variable errors due to varying head orientation were small but consistent (Fig. 5, $F_{1,34} = 17.7$, $p < 0.001$). Variable errors were smaller when the line was positioned directly in front of midline than when placed on either side ($F_{2,68} = 12.2$, adjusted $p < 0.001$), but there were no differences between variable errors with the line positioned on the left or right side of midline ($p = 0.75$). These results were consistent among the four groups as there were no significant interaction effects with groups ($p > 0.13$).

Errors in perception of *a–p* axis direction were strongly correlated with variations in head orientation in both controls and individuals with PPL lesions. This is clearly shown in scatterplots of perceptual errors versus errors predicted from head orientations on individual trials (Fig. 6). Multiple correlation coefficients exceeding 0.8 were commonly observed with average coefficients exceeding 0.5 (Fig. 6). There were no statistical differences in the correlation coefficients among the four groups of subjects or among different line positions (Fig. 6, $p > 0.56$), although there was a strong trend for interaction of these main effects ($p = 0.088$, but post hoc tests found no significant differences). Also, about 1/3 of the subjects (29%) showed evidence of perceptual bias toward or away from the head *a–p* axis (i.e., a positive correlation between errors and head rotation angle would indicate bias toward the head axis, a negative correlation for bias away from the head axis—Darling et al., 1996). However, about equal percentages of controls and subjects with PPL lesions showed such biases. The correlation coefficients associated with different line positions differed among the groups (i.e., a significant interaction effect—$F_{6,50} = 2.43$, $p = 0.039$) but there were no statistically significant differences among individual group means for different line positions according to post hoc tests ($p > 0.1$).

Errors by subjects with right PPL lesions showed a greater dependence on initial line orientation than controls. However, perception of the trunk-fixed *a–p* axis was only weakly related to varied initial line orientations in most subjects because correlation coefficients were usually less than 0.4. The RPPL group had significantly larger correlation coefficients than controls ($F_{3,33} = 3.18$, $p = 0.037$, $p = 0.032$ for comparison of RPPL and CON groups) and lesion controls ($p = 0.09$), but had similar coefficients to those of the LPPL group ($p = 0.39$). Displaying the line in different positions relative to body midline did not affect the correlation coefficients ($p = 0.97$) and the group by line position interaction was not significant ($p = 0.71$).

Size of variable, but not constant, errors showed some dependence on volumes of lesions of F06/F08 cells (which include areas 6 and 44). Absolute constant errors showed no dependence on lesion volumes when the head

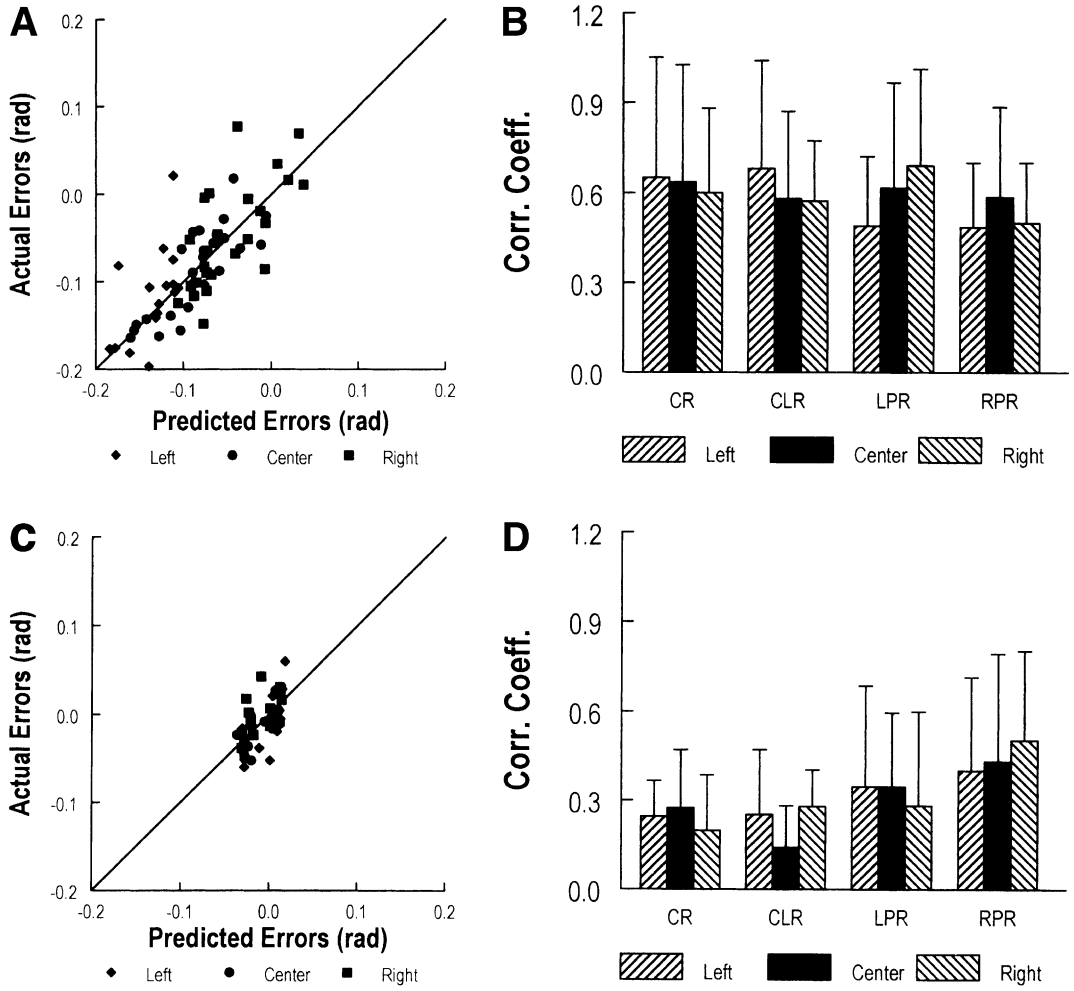


Fig. 6. Dependence of errors for perception of the trunk-fixed *a–p* axis on head orientation and starting position of the line. The scatterplots show single trial errors (ordinate) plotted against errors predicted from head orientation (abscissae) for CE who has a left IPL lesion (A) and a control subject (C). The bar graphs show mean correlation coefficients across subjects in each group for predictions of errors from head orientations (B) and starting orientation of the line (D). Error bars are 1 s.d.

was erect ($R = 0.22$, $p = 0.87$) or when head orientation was varied ($R = 0.24$, $p = 0.84$). In contrast, variable errors were correlated with volume of F06/F08 lesions when the head was erect ($R = 0.387$, $p = 0.013$) and with volume of supramarginal gyrus lesions when head orientation was varied ($R = 0.357$, $p = 0.022$).

## 4. Discussion

We find that individuals with unilateral lesions of right and left PPL that include the IPL had abnormally large variable errors in judging the alignment of visual targets to the earth-fixed vertical axis. These defects were present four or more months after lesion onset, suggesting that these areas play a critical role in generating accurate visual perception of vertical. Size of variable errors depended on the side and volume of lesions. In contrast, judgments of alignment of visual targets to the trunk-fixed *a–p* axis were only slightly less accurate in subjects with unilateral lesions of PPL but were much less accurate in subjects with lesions including premotor areas 6 and 44 (F06/F08 lesions). Changes in head orientation significantly increased variable errors for perception of vertical in controls and subjects with lesions, but had surprisingly little effect (a) in subjects with right PPL lesions and (b) on variable errors for perception of the trunk-fixed *a–p* axis in all subjects. Moreover, location of the line relative to body midline had little effect on judgments of line orientation along vertical or *a–p* axes, compatible with no chronic lateralized defect of spatial representation in most individuals with chronic unilateral PPL lesions. The observed defects in perception of line orientation were not due to visual field defects because none of the subjects with left or right PPL lesions had visual field defects. We conclude that persistent hemineglect is necessary to produce the large biases (constant errors) of visuospatial

perception reported previously, but unilateral PPL lesions without hemineglect produce defective visual perception characterized by large variability of perceived orientation.

### 4.1. Lesion effects on perception of vertical

The SPL and PSTG (components of the presumed PIVC) of both hemispheres appear to play a critical role in judging the earth-fixed vertical orientation of visually defined objects. We found that the magnitude of variable errors was correlated with the volume of lesions in right and left PSTG and SPL when the head was held erect ($R = 0.77$, $p < 0.001$). This finding is consistent with the idea that lesions of the right PSTG contribute to defects in visuospatial awareness (Karnath et al., 2001). Combination of IPL lesions with SPL and PSTG lesions may have the greatest influence on perception of vertical as subjects LPPL-6 (left hemisphere lesion) and RPPL-4 (right hemisphere lesion) both had lesions encompassing all these areas and also had the largest variable errors (Table 1, Fig. 4). In contrast, subject LPPL-2 had similar lesions of PSTG and SPL but with a spared IPL and had much lower variable errors than LPPL-6 and RPPL-4 (Table 1, Fig. 4).

A role for the IPL in visual perception of vertical is consistent with previous reports that subjects with chronic left or right neglect due to parieto-temporal lesions (which probably included the IPL) exhibit large constant errors when aligning a line to vertical, horizontal and 45° angles (Kerkhoff & Zoelch, 1998). It is also clear in Fig. 1 of the work by Kerkhoff and Zoelch (1998) that subjects with neglect had large variable errors in the alignment task, although these were not quantified. Thus, PPL damage with persistent neglect causes large constant and variable errors for visual perception of vertical (Kerkhoff & Zoelch, 1998), but in PPL damage without neglect large variable errors persist. These large variable errors may reflect difficulty in task performance by subjects with PPL damage. However, with the head erect only subjects with right PPL damage had larger variable errors than controls (Fig. 2). Thus, it seems doubtful that difficulties in performing the task due to brain lesions can explain the large variable errors in only one of the lesion groups. An alternative explanation is that recovery after the lesion is better in subjects who do not exhibit persistent neglect so that constant errors return to the normal range but large variable errors persist because of difficulty integrating visual and graviceptive (vestibular) inputs to accurately define the vertical axis. The finding that perceptual errors depended strongly on head orientation support this explanation.

Contributions of the IPL and PSTG of both hemispheres to visual perception of vertical were further demonstrated when head orientation was varied. Under these conditions the magnitude of constant errors showed a dependence on volume of lesions to right and left angular gyri ($R = 0.39$, $p = 0.01$) and variable errors were correlated with volume of lesions to left PSTG ($R = 0.46$, $p = 0.021$). Thus, these regions probably integrate vestibular and visual inputs to specify the earth-fixed vertical axis for the visual system. Larger single trial errors in perception of vertical were correlated with variations in head orientation in all subjects, suggesting that vestibular compensations for head tilt are imperfect and lead to larger errors in perception of vertical. Such effects were much larger in subjects with left PSTG lesions who had the largest variable errors when head orientation was varied. These findings on effects of head tilt contrast somewhat with previous work showing that subjects with anterior brain lesions due to penetrating head wounds exhibit larger constant and variable errors in visual perception of vertical than control subjects when the body is tilted (Teuber & Mishkin, 1954). However, the brain lesions were not precisely described in the Teuber's work due to lack of precise neuroimaging methods and were likely larger than the brain lesions of the subjects studied here because of the extensive brain damage usually caused by such wounds (Holmes, 1946; Rizzo & Tranel, 1996). We did not find any evidence of frontal lobe contributions to perception of vertical in that larger perceptual errors were not correlated with volume of lesions to frontal lobe premotor areas.

We found that head orientation variations did not consistently increase variable errors in subjects with right PPL lesions as was observed in all other groups. Examination of individual RPPL subject data showed that variable errors decreased for some and increased or were unaffected by head orientation variations in others with right hemisphere lesions. There were no obvious lesion or age differences that could account for these findings. Further research in a larger group of subjects with right hemisphere lesions is needed to address this unexpected finding.

Functional MRI findings of activation of PI during galvanic vestibular stimulation (Bucher et al., 1998; Lobel et al., 1999) and lesion studies (Brandt et al., 1994) have suggested that PIVC, including the PI, plays a critical role in perception of the earth-fixed visual vertical axis. However, we found no evidence in our multiple regression analyses that lesions of PI impair perception of earth-fixed vertical, even when head orientation is varied and processing of vestibular inputs would be needed for precise estimates of earth-fixed vertical. Moreover, four of our subjects with moderate—large PI lesions (LCON-5, RPPL-2, LPPL-5, RPPL-1—Table 1) had accurate perception of vertical (with constant and variable errors below 0.05 rad for erect and varied head orientations; see Tables 1 and 2 for lesion anatomy). Although the PI may process inputs from the

vestibular otoliths, our findings do not indicate that this area plays a critical role in visual perception of the earth-fixed vertical axis. This is consistent with findings that PIVC neurons are strongly activated by body/head rotation, but not by static tilt (Akbarian et al., 1988). PSTG, which is commonly damaged in association with PI lesions is considered part of the PIVC (see Table 1 and Brandt et al., 1994), and may be involved in processing otolith information based on our multiple regression analyses. Brandt et al. (1994) found large constant errors in perception of vertical primarily in the acute phase following the brain injury but follow-up studies into the chronic phase in some subjects found much smaller errors in perceived vertical.

Some individuals with acute right hemisphere lesions and left hemiparesis stand and sit with the body tilted toward the left side and actively push away when a therapist attempts to correct the body tilt, predisposing them to falling toward the paretic side (Karnath, Ferber, & Dichgans, 2000). Yet, these subjects have accurate visual perception of vertical and can align the body to the vertical axis when vision is allowed but show a pronounced postural tilt to the left when asked to align the body to vertical without vision. In contrast, individuals with acute unilateral vestibular disturbances show disturbed visual perception of vertical but normal postural vertical, suggesting different roles for central neural pathways and peripheral receptors. Visual vertical may be derived from head-fixed (vestibular) receptors and postural vertical may be derived from trunk-fixed receptors (statoliths of kidneys, blood volume receptors—Mittelstaedt, 1992). The individuals with brain lesions that we studied did not exhibit tilted postures or, in most cases, tilted visual vertical indicating that both pathways and receptors were functioning. However, the higher variability of perceived visual vertical associated with lack of compensation for varied head orientation suggests disrupted processing of vestibular signals.

### 4.2. Lesion effects on perception of the trunk-fixed *a–p* axis

Our findings suggest that accurate perception of the trunk-fixed *a–p* axis direction depends on intact areas 6 and 44 in both hemispheres. Larger variable errors for perception of the trunk-fixed *a–p* axis with lesions that include areas 6 and 44 are consistent with previous reports that implicate a bihemispheric network involving the angular gyrus and occipital and frontal areas for generating percepts of the "mid-sagittal plane" (Vallar et al., 1999). However, we found only weak correlations between perceptual variable errors and lesions involving these cortical areas (in the head held erect condition). These weak correlations may reflect that the F06/F08 cells that contain areas 6 and 44 (implicated in perception of the mid-sagittal plane—Vallar et al., 1999) also contain areas 1–4 and 45, which are not implicated in perception of the mid-sagittal plane (although areas 1–3 receive somatosensory input that might help define trunk orientation). Accurate judgments of orientation of the trunk-fixed *a–p* axis despite unilateral right and left PPL and PSTG lesions suggest a bilateral representation of this axis such that bilateral lesions may be necessary to produce large perceptual defects and unilateral lesions can be adequately compensated.

Of note, different static head rotation positions (sensed primarily by neck proprioceptors) did not increase variable errors for judgment of orientation along the trunk-fixed *a–p* axis. This is consistent with previous findings in young neurologically normal adults (Darling et al., 1996). In contrast, differing amounts of head tilt (sensed by both the neck proprioceptors and vestibular otoliths) produced large variable errors for perceiving visual vertical orientation, suggesting that otolith inputs have a greater effect on visual perception than neck proprioceptive inputs. Unilateral PPL lesions do not appear to disrupt processing of neck proprioceptive inputs for specifying orientation of the trunk *a–p* axis, based on the finding of similar relationships between perceptual errors and head orientation in both controls and subjects with lesions.

### 4.3. Perceptual judgments in contralesional and ipsilesional sectors of egocentric space

We found no clear evidence that unilateral PPL lesions produced a defect in representation of earth-fixed vertical or the trunk-fixed *a–p* axis in the region of visual space contralateral to the lesion. Subjects with PPL lesions showed no evidence of larger perceptual constant errors with the perceived axis directed toward ipsilesional space or of larger variable errors for aligning the line to the desired axes in contralesional versus ipsilesional space. Thus, visual representation of direction in different regions of space appears to be unaffected by unilateral PPL lesions in the absence of visual neglect, except in a small number of such subjects. However, all groups usually had lower absolute constant and variable errors when the line was positioned directly in front of body midline. In this position, the task is probably simpler because the endpoints of the line can be aligned to perceived body midline. The current findings provide suggest that the neural representation of visual space is not distorted in a consistent manner by unilateral parietal lobe lesions, although such lesions produce greater variability in visual judgments of orientation. Similarly, Karnath and Ferber (1999) recently reported that individuals with neglect perceived horizontal plane distances accurately in both neglected and non-neglected hemispace.

### 4.4. Methodological issues

Although our tasks involved a motor component, they were devised to minimize any motor bias. Subjects pressed buttons on a keyboard with fingers of one hand to adjust the orientation of the line, perhaps causing a motor bias in the perceptual errors. Subjects did not move the hand in different directions or move the left versus right hand to produce line rotation in different directions, which would be more likely to produce motor-biased responses. A motor bias should result in opposite direction constant errors for right versus left hemisphere lesions but there was no indication of such opposite errors when the head was erect (Figs. 2 and 5).

Another issue is that when aligning the line to the trunk-fixed *a–p* axis subjects may have focused their attention on the endpoint of the line closer to or farther from them. If so, the large ccw error by four of the individuals with right PPL lesions could be due to attending to the proximal endpoint of the line which would be in the right hemifield for a centrally located line rotated ccw (i.e., position the proximal endpoint to the right of midline as observed previously in subjects with hemineglect—Ferber & Karnath, 1999; Rossetti et al., 1998). However, this explanation cannot account for the ccw errors observed when the line was presented in the right and left fields because both endpoints of the line were contained within one field. Also, when the line was presented centrally, the proximal endpoint would begin in the right field on some trials and in the left field on others, yet the constant errors were ccw for subjects in both RPPL and LPPL lesion groups (Fig. 5).

## 5. Conclusions

The present work shows that the right and left posterior parietal lobe and posterior region of the superior temporal gyrus are critical for generating accurate visual perception of the earth-fixed vertical axis. Accurately defining the earth-fixed vertical axis is important for balance control as discussed earlier and for control of upper limb movements to acquire visual targets (e.g., Bertenthal & Von Hofsten, 1998; Flanders, Tillery, & Soechting, 1992). Contributions of the superior and inferior parietal lobules to visually perceived vertical differed in that lesions of the SPL increased variable errors for perception of vertical when the head was upright whereas IPL lesions increased magnitudes of constant errors when head orientation was varied. Lesions to posterior regions of the left superior temporal gyrus increased variable errors under both conditions.

Accurately defining the median plane or trunk-fixed *a–p* axis orientation is also critical for control of upper limb movements to acquire objects. However, lesions of the inferior and superior parietal lobules cause only small defects in visual perception of this axis. Instead, frontal premotor areas may be necessary for accurate perception of the straight ahead direction with the supramarginal gyrus of the IPL also involved when head orientation is varied. Thus, subjects with PPL lesions might be expected to have large defects related to inaccurately specifying object vertical orientation and position but only small defects for errors related to horizontal plane orientation or right/left position of the target.

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## References

Akbarian, S., Berndl, K., Grusser, O. J., Guldin, W., Pause, M., & Schreiter, U. (1988). Responses of single neurons in the parieto-insular vestibular cortex of primates. *Annals of the New York Academy of Sciences, 545*, 187–202.

Anderson, S. W. (2003). Neuropsychologic rehabilitation for visuoperceptual impairments. In J. J. S. Barton, & M. Rizzo (Eds.), *Neurologic clinics of North America series: Vol. 21. Neuro-opthalmology: Vision and the brain*. Philadelphia: W.B. Saunders.

Bertenthal, B., & Von Hofsten, C. (1998). Eye, head and trunk control: The foundation for manual development. *Neuroscience and Biobehavioral Reviews, 22*, 515–520.

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., & Brandt, T. (1998). Cerebral functional magnetic resonance imaging of vestibular, auditory, and nociceptive areas during galvanic stimulation. *Annals of Neurology, 44*, 120–125.

Chokron, S., & Bartolomeo, P. (1998). Position of the egocentric reference and directional arm movements in right-brain-damaged patients. *Brain and Cognition, 37*, 405–418.

Damasio, H., & Damasio, A. (1989). *Lesion analysis in neuropsychology*. New York: Oxford University Press.

Damasio, H., & Frank, R. (1992). Three-dimensional in vivo mapping of brain lesions in humans. *Archives of Neurology, 49*, 137–143.

Darling, W. G., Butler, A. J., & Williams, T. E. (1996). Visual perceptions of head-fixed and trunk-fixed anterior/posterior axes. *Experimental Brain Research, 112*, 127–134.

Darling, W. G., & Hondzinski, J. M. (1997). Visual perceptions of vertical and intrinsic longitudinal axes. *Experimental Brain Research, 116*, 485–492.

Farne, A., Ponti, F., & Ladavas, E. (1998). In search of biased egocentric reference frames in neglect. *Neuropsychologia, 36*, 611–623.

Ferber, S., & Karnath, H. O. (1999). Parietal and occipital lobe contributions to perception of straight ahead orientation. *Journal of Neurology, Neurosurgery and Psychiatry, 67*, 572–578.

Flanders, M., Tillery, S. I. H., & Soechting, J. F. (1992). Early stages in a sensorimotor transformation. *Behavioral and Brain Sciences, 15*, 309–361.

Galati, G., Lobel, E., Vallar, G., Berthoz, A., Pizzamiglio, L., & Le Bihan, D. (2000). The neural basis of egocentric and allocentric coding of space in humans: A functional magnetic resonance study. *Experimental Brain Research, 133*, 156–164.

Heilman, K. M., Bowers, D., & Watson, R. T. (1983). Performance on hemispatial pointing task by patients with neglect syndrome. *Neurology, 33*, 661–664.

Holmes, G. (1946). *Introduction to clinical neurology*. Edinburgh: E & S Livingston Ltd.

Jeannerod, M., & Biguer, B. (1987). The directional coding of reaching movements. In M. Jeannerod (Ed.), *A visuomotor conception of spatial neglect* (pp. 87–113). Amsterdam: North-Holland.

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., & Ferber, S. (1999). Is space representation distorted in neglect? *Neuropsychologia, 37*, 7–15.

Karnath, H. O., Ferber, S., & Dichgans, J. (2000). The origin of contraversive pushing: Evidence for a second graviceptive system in humans. *Neurology, 55*, 1298–1304.

Karnath, H. O., Ferber, S., & Himmelbach, M. (2001). Spatial awareness is a function of the temporal not the posterior parietal lobe. *Nature, 411*, 950–953.

Karnath, H. O., Schenkel, P., & Fischer, B. (1991). Trunk orientation as the determining factor of the 'contralateral' deficit in the neglect syndrome and as the physical anchor of the internal representation of body orientation in space. *Brain, 114*, 1997–2014.

Kerkhoff, G., & Zoelch, C. (1998). Disorders of visuospatial orientation in the frontal plane in patients with visual neglect following right or left parietal lesions. *Experimental Brain Research, 122*, 108–120.

Lobel, E., Kleine, J. F., Leroy-Willig, A., Van de Moortele, P. F., Le Bihan, D., Grusser, O. J., & Berthoz, A. (1999). Cortical areas activated by bilateral galvanic vestibular stimulation. *Annals of the New York Academy of Sciences, 871*, 313–323.

Mark, V. W., & Heilman, K. M. (1990). Bodily neglect and orientational biases in unilateral neglect syndrome and normal subjects. *Neurology, 40*, 640–643.

Mittelstaedt, H. (1992). Somatic versus vestibular gravity reception in man. *Annals of the New York Academy of Sciences, 656*, 124–139.

Rizzo, M., & Tranel, D. (1996). *Head injury and postconcussive syndrome*. New York: Churchill Livingstone, p. 6.

Rossetti, Y., Rode, G., Pisella, L., Farne, A., Li, L., Boisson, D., & Perenin, M. T. (1998). Prism adaptation to a rightward optical deviation rehabilitates left hemispatial neglect. *Nature, 395*, 166–169.

Teuber, H.-L., & Mishkin, M. (1954). Judgment of visual and postural vertical after brain injury. *Journal of Psychology, 38*, 161–175.

Vallar, G., Lobel, E., Galati, G., Berthoz, A., Pizzamiglio, L., & Le Bihan, D. (1999). A fronto-parietal system for computing the egocentric spatial frame of reference in humans. *Experimental Brain Research, 124*, 281–286.

Ventre, J., Flandrin, J. M., & Jeannerod, M. (1984). In search for the egocentric reference. A neurophysiological hypothesis. *Neuropsychologia, 22*, 797–806.