Neuroanatomy of space, body, and posture perception in patients with right hemisphere stroke

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ABSTRACT

Objective: To specify the neuroanatomical correlates of biases in the representations of the gravitational vertical (subjective vertical [SV]) and body axis (subjective straight ahead [SSA]), as well as postural difficulties, in patients with hemispheric stroke.

Methods: The analysis focused on right hemisphere lesions in 21 neglect patients and 21 non-neglect patients (using MRIcro software) and related performance in 2 experimental tasks (SV and SSA) and a clinical balance assessment. Voxel-based lesion-symptom mapping was used to highlight brain areas in which lesions best explained the severity of task biases ($p < 0.01$).

Results: The bias in the representation of body orientation was found to be strongly related to lesions of the anterior parietal cortex and the middle part of the superior temporal gyrus. The SV errors were associated with more widespread lesions of the posterior parietal and temporal cortices. Imbalance was preferentially associated with lesions of the posterior insula and the adjacent temporoparietal cortex.

Conclusion: This study evidenced a cortical dissociation for body-centered and gravitational representations biases, which may reflect the differential involvement of these brain regions in spatial information processing. The lesions involved in representation biases (especially of the SV) and postural difficulties overlapped to some extent in the temporoparietal, superior temporal, and posterior insular regions of the cortex. *Neurology*® 2013;81:1291–1297

GLOSSARY

LSI = least severely impaired; **MNI** = Montreal Neurological Institute; **MSI** = most severely impaired; **PASS** = Postural Assessment Scale for Stroke Patients; **PIVC** = parieto-insular vestibular cortex; **SSA** = subjective straight ahead; **SV** = subjective vertical; **VLSM** = voxel-based lesion-symptom mapping.

Spatial representations of the body midline and gravitational vertical guide actions within the peripersonal and extrapersonal spaces and the control of posture.[1,2] Brain damage can impair these representations.[3] Indeed, the subjective straight ahead (SSA) is deviated to the lesion side in neglect patients.[1,4–7] The subjective vertical (SV) is also affected, with a contralesional rotation in the frontal (roll) plane.[8–10] Moreover, links between spatial representation biases and postural disorders were evoked.[8,11] Importantly, the SV errors displayed in the first 3 months poststroke appeared predictive of imbalance and dependence of patients at 6 months,[11] and could thus represent an important factor for functional handicap in everyday life.

The anatomy of brain systems involved in the elaboration of these representations was studied in healthy subjects and rarely in patients. Functional MRI in normal subjects[12] revealed that SSA tasks activate a frontoparietal network with right-side predominance. During verticality judgments,[13] high-density electrical neuroimaging showed an early potential map in the right temporal-occipital cortex, followed by a bilateral map in the temporal-occipital and parietal-occipital cortices. However, verticality errors made by patients were linked to more anterior lesions involving the temporal-insular cortex.[9,14]

We used quantitative voxel-based lesion-symptom mapping (VLSM)[15] to determine and compare the anatomic correlates of biases of the SSA and SV and related body posture disorders, with the hypothesis of a partially different involvement of parietal and temporal cortices.

METHODS Subjects. We performed a retrospective cohort study of prospectively collected data in the neurorehabilitation department of the University Hospital of Lille, France. Patients were included in the first few months following a unilateral right cerebral stroke. We excluded patients with bilateral lesions, previous neurologic or psychiatric disorders, impaired primary visual perception (except for visual field defects), behavioral disorders, motor disorders of the right arm, or pusher syndrome (i.e., contralateral trunk deviation with active resistance to any external correction, as determined by the scale for contraversive pushing).[16]

The patients were tested for spatial neglect, which was defined as an abnormal performance in at least 2 of the 3 following tests: line bisection (cutoff: rightward deviation >11%),[17] scene copying (cutoff: score ≥1/4),[18] and bell cancellation (cutoff: left-right omissions ≥4/15).[19] We also presented the Catherine Bergego questionnaire[20] to probe behavioral neglect in daily living activities. Visual field defects were assessed clinically and instrumentally. In the clinical test, after a demonstration of 2-second finger movements in the unaffected hemifield, the patient had to discriminate movement (2 trials) and no movement trials (2 trials) in each quadrant. We considered that hemianopia was probable when a "no" response was produced repeatedly. The visual field was examined instrumentally using kinetic (Goldmann) perimetry (Metrovision, Pérenchies, France) under monocular conditions and with a chin support.

Forty-two right-handed patients were included, with a mean age of 58.3 ± 13 years and a mean time since stroke of 61.9 ± 35.2 days (table 1). The lesions were mostly in the territory of the middle cerebral artery (36 cases) and occasionally in the territory of the posterior cerebral artery (4 cases) or at the junction between the 2 territories (2 cases). Twenty-one patients had neglect and 20 had hemianopia (with 16 patients having both conditions). Visual

acuity was low (5/10) in one case and completely or relatively unaffected in all the other cases (>7/10). None of the patients presented with peripheral oculomotor disorders or nystagmus.

Neglect and non-neglect patients were intentionally matched in number (21) and compared with 15 matching (age, education) healthy control subjects.

Standard protocol approvals, registration, and patient consents. All participants gave their written informed consent to participation. This observational study was performed in accordance with the precepts of the Declaration of Helsinki.

Assessments of body-midline and vertical representations. We screened for body midline representation and vertical perception disorders by performing standard evaluations of the SSA[7] and the visuohaptic SV[10] (figure 1).

The participant lay in a semirecumbent position on a medical examination table. A metal rod (25 cm long and 1.5 cm × 1.5 cm in section) was placed 50 cm in front of the participant. The rod axis could rotate or slide (i.e., to give translation) along a horizontal slit. A potentiometer inserted into the rod axis gave the rotation angle: 0° corresponded to a sagittal orientation, a positive value to a clockwise tilt. Translation movements were coded by a second potentiometer. A value of 0 indicated that the center of the rod was in the subject's midsagittal plane, whereas a positive value corresponded to a rightward displacement.

Ten light-emitting diodes (length 2 cm; width 0.5 cm) were inserted into the rod. Adjustments were carried out in darkness, and the rod was grasped and manipulated at the axis level (figure 1) with the right hand. All the fixed parts of the apparatus were centered on the participant's midsagittal plane.

For each of the 2 following tasks, the participant was required to close his or her eyes during the setting of the rod initial location and rotation.

Subjective straight ahead test. The participant was instructed to imagine a line starting from the navel and extending away straight ahead of the trunk. He or she then had to adjust the rod position in the horizontal plane. Depending on the trial, the rod axis was initially translated by −15, 0, or +15 cm and rotated by −45°, 0°, or +45°. Two trials were run for each of the 9 initial positions (3 translations × 3 rotations). The mean final translation was used in the analysis.

Subjective visual vertical test. The participant had to set the rod to the gravitational vertical in the frontal plane. The rod axis was initially fixed on the midsagittal plane or 15 cm to the left or to the right and rotated by −45°, 0°, or +45°. Two trials were run for each of the initial positions. The mean final deviation was used in the analysis.

Body posture examination. Seven subtests from the Postural Assessment Scale for Stroke Patients (PASS) were selected[21]: sitting without support, standing with support, standing without support, supine to affected side, supine to nonaffected side, supine to sitting, and sitting to supine. Each was coded from 0 (impossible) to 3 (fair performance without help). The dependent variable was the overall score.

Lesion analysis. For each patient, the location and extent of brain damage was delineated on standardized brain templates with MRIcro software (www.mricro.com)[22] by a trained neurologist (M.R.) who was blind to the patient's performance. The standard slice thickness of MRI scans (performed in 34 patients; Achieva, 1.5 T, Philips Medical Systems, Best, the Netherlands) was 6 mm (interslice gap: 1 mm). For optimal identification of damaged areas, we used both T1- and T2-weighted images, including T2-weighted fluid-attenuated inversion recovery sequences. In all cases, we selected the brain

Table 1	Demographic, experimental, and clinical data for the 42 patients with right brain damage	
	Spatial neglect, mean (SD)	No neglect, mean (SD)
Age, y	61.0 (14.4)	55.5 (11.1)
M/F	8/13	10/11
Etiology, infarct/hemorrhage	10/11	12/9
MRI/CT scan	16/5	18/3
Time since stroke, d	59.6 (33.7)	64.7 (37.3)
SV bias, °	−6.9 (4.7)	−3.0 (2.9)
SSA bias, cm	+4.7 (2.4)	+2.5 (1.8)
PASS score (/21)	12.2 (4.1)	15.5 (4.1)
Bell cancellation (total omission) (/35)	17.9 (10.1)	1.5 (2.2)
Line bisection (% deviation)	55.4 (24.0)	2.9 (5.8)

Abbreviations: PASS = Postural Assessment Scale for Stroke Patients; SSA = subjective straight ahead; SV = subjective vertical.

Figure 1 Participant's position and apparatus for investigating the subjective straight ahead (A) and the subjective vertical (B)
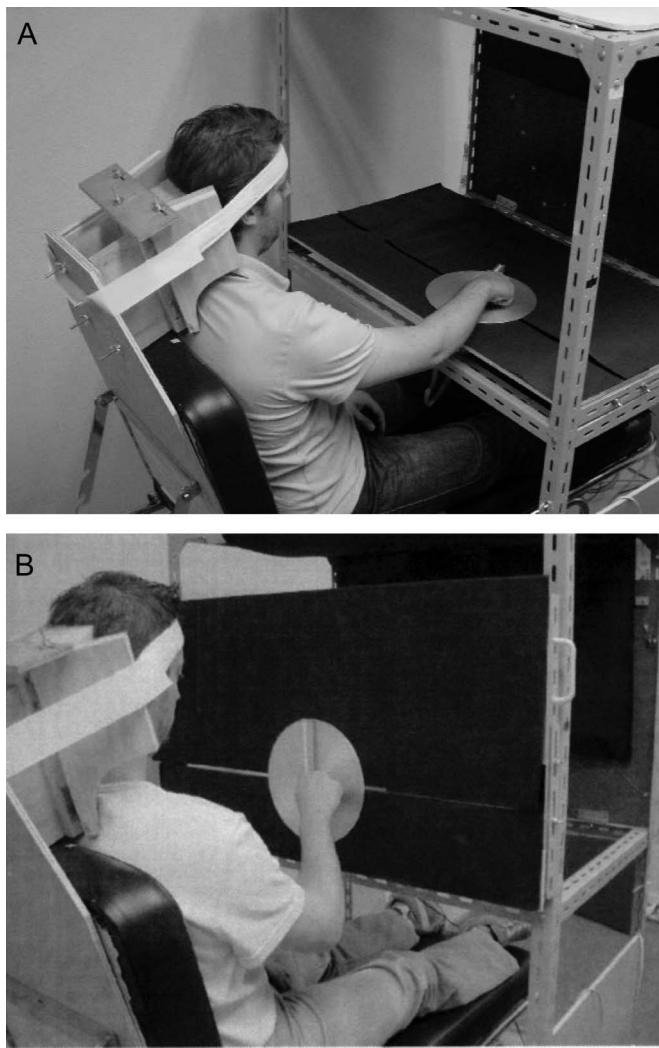
scans that showed the most widespread lesions. The standard slice thickness for the CT scan (8 patients; Somatom machine, Siemens Health care, Malvern, PA) was 10 mm.

We first examined the overlap between normalized lesion areas in specific subgroups of patients. For each test, a lesion subtraction method (MRIcro software) contrasted the 10 least severely impaired patients (LSI group) and the 10 most severely impaired patients (MSI group). This selection enabled us to obtain sharper intergroup contrasts.

Table 2 Correlations of SV and SSA biases and postural difficulties with clinical tests and lesion volume

	Line bisection	Scene copy	Bell test	CB questionnaire	Lesion volume
SV	−0.469[a]	−0.360[b]	−0.425[a]	−0.410[a]	0.501[c]
SSA	0.332[b]	0.383[b]	0.426[a]	0.368[b]	0.256
PASS	−0.240	−0.305[b]	−0.342[b]	−0.406[a]	−0.233

Abbreviations: CB = Catherine Bergego; PASS = Postural Assessment Scale for Stroke Patients; SSA = subjective straight ahead; SV = subjective vertical.
[a] $0.01 \geq p > 0.001$.
[b] $0.05 \geq p > 0.01$.
[c] $0.001 \geq p > 0.0001$.

In a second step, we used a procedure applied to the analysis of brain lesion-behavior relationships.[15] The VLSM method analyzes the relations between continuous variables (such as the SSA deviation) and the presence or absence of a lesion in each voxel of the brain maps. All 3D lesion maps were fed into the MATLAB-based VLSM module[15,22] with the corresponding behavioral scores: SV bias, SSA bias, or PASS score. In each voxel, the VLSM separated the subjects into 2 groups, depending on the presence or absence of a lesion. Next, a *t* test was conducted comparing the behavioral scores of patients with or without lesion in that voxel. We used a false discovery rate correction ($p < 0.01$) for multiple comparisons. Areas showing significant relationships with behavioral disorders were identified. For each variable, the resulting statistics were mapped onto a standardized brain template from the Montreal Neurological Institute (MNI) and coded according to a color scale. Identification of lesion site was performed by confrontation to the cortical maps included in the MRIcro software and the 3D description of subcortical tracts.[23]

Statistical analyses. We used the Student *t* test for comparing the means of patient and control groups and the Pearson test for investigating correlations between variables. The threshold for statistical significance was set to $p \leq 0.05$.

RESULTS Patient performance. Neglect patients showed greater biases than non-neglect patients for the SV ($p = 0.001$) and the SSA ($p = 0.014$) (table 1). Both groups of patients differed from control subjects for the SV (-0.47 ± 0.79; $p = 0.0001$ and $p = 0.038$) and SSA ($+0.10 \pm 0.37$; $p = 0.0001$ and $p = 0.001$). The control of balance was less efficient in neglect patients than in non-neglect patients ($p = 0.013$).

Hemianopic patients showed greater biases than non-hemianopic patients in the SV ($-7.1°$ vs $-3.1°$, $p = 0.002$) and SSA ($+4.5$ vs $+2.7$ cm, $p = 0.013$) tasks. Both groups differed from control subjects for the SSA ($p = 0.0001$ and $p = 0.001$). Only hemianopic patients showed impairment for the SV ($p = 0.0001$ and $p = 0.076$). No balance deficit (PASS) was associated with hemianopia ($p = 0.853$).

In the whole patient group, the SSA correlated with the SV ($r = 0.38$, $p = 0.014$) and the PASS ($r = -0.34$, $p = 0.027$). However, the SV did not correlate with balance disorders ($r = 0.05$, $p = 0.767$). The SV, the SSA, and the PASS correlated with the severity of neglect in visuospatial tests (table 2), but only the SSA correlated with the volume of lesions.

Lesion overlap analysis. When SV errors were considered (figure 2), areas of greatest overlap for the MSI group were in the inferior parietal lobe (MNI coordinates: 47, −49, 50), the superior temporal gyrus (MNI coordinates: 65, −40, 15), and the middle occipital gyrus (44, −72, 1). For the LSI, the areas of overlap were the thalamus (20, −23, 1) and putamen (28, −14, 8). For SSA errors, areas of overlap for the MSI group were located in the middle frontal gyrus (35, 15, 45), the parietal lobe within the supramarginal gyrus (60, −22, 34), and the anterior part of the superior temporal gyrus (42, 11, −23). In the LSI

Figure 2   Lesion overlapping difference between the 10 most severely and the 10 less severely impaired patients
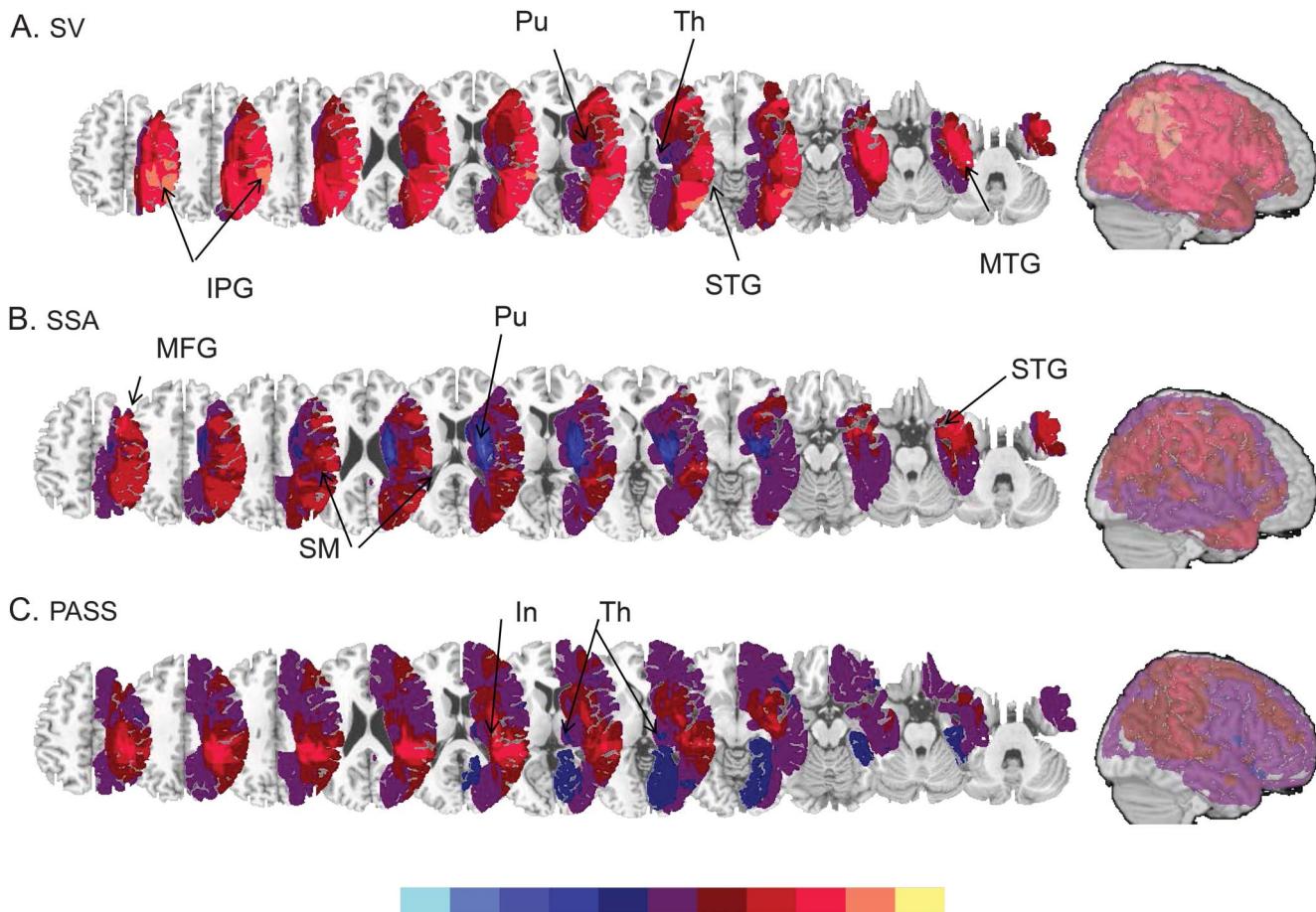
Presentation of the differences for the subjective vertical (A: SV), the subjective straight ahead (B: SSA), and the posture (C: Postural Assessment Scale for Stroke Patients [PASS]). The color range indicates the positive (yellow, red) and negative (blue) differences between groups for each voxel. The coordinates of the axial slices range from z −48 to −32 in Montreal Neurological Institute space. In = insula; IPG = inferior parietal gyrus; MFG = middle frontal gyrus; MTG = middle temporal gyrus; Pu = putamen; SM = supramarginalis gyrus; STG = superior temporal gyrus; Th = thalamus.

group, they were in the putamen (28, −8, 14). Finally, for balance disorders, the area of greatest overlap was in the posterior insula (37, −26, 17) for the MSI group, and in the thalamus (18, −24, 4) for the group with good balance.

Voxel-based lesion-symptom mapping. Mapping of the SV bias revealed an involvement (figure 3) of the posterior part of the inferior parietal lobe (Brodmann area 39), posterior part of the horizontal segment of the intraparietal sulcus, the posterior superior temporal gyrus (Brodmann areas 41, 42, and 22), and the posterior middle temporal gyrus (Brodmann area 37), with extension into the adjacent dorsolateral occipital lobe (Brodmann area 19). Subcortically, the lesions involved the superior longitudinal fasciculus.

The SSA errors were associated with lesions of the anterior parietal lobe, including the primary somatosensory area (Brodmann areas 1 and 2), the anterior part of the inferior parietal lobe (Brodmann area 40), the intraparietal sulcus (vertical and horizontal segments), plus the middle part of the superior temporal

gyrus (Brodmann areas 41 and 42), and, to a lesser extent, the posterior part of the superior frontal gyrus (Brodmann area 6; dorsolateral premotor cortex). The superior longitudinal fasciculus was not affected.

Balance disorders (according to the PASS score) were associated with lesions of the posterior insula, upper part of the temporal cortex (Brodmann area 22), lower part of the parietal cortex (area 39), and, to a lesser extent, the somatosensory cortex (Brodmann areas 3, 1, 2). Many of these lesions extended subcortically to the superior longitudinal fasciculus.

Damaged areas involved in SV bias and balance disorders comprised the superior temporal lobe, the temporoparietal junction, the inferior parietal gyrus, and the subcortical longitudinal fasciculus. Those involved in SSA errors and balance disorders were limited and included the primary somatosensory cortex and the superior temporal cortex.

DISCUSSION Patients showed biases in the SSA and the SV, but the VLSM method dissociated the

Figure 3 Voxel-based lesion-symptom mapping study: Cortical correlates of the subjective vertical and subjective straight ahead biases and of postural difficulties (Postural Assessment Scale for Stroke Patients)
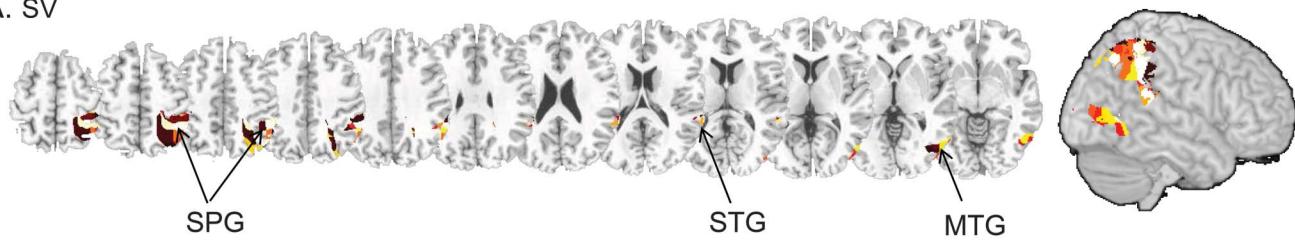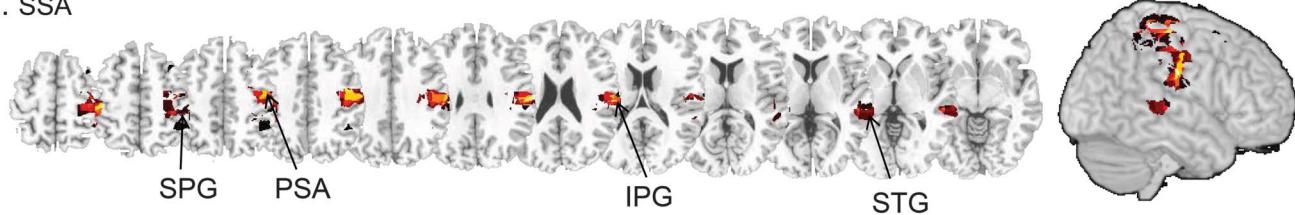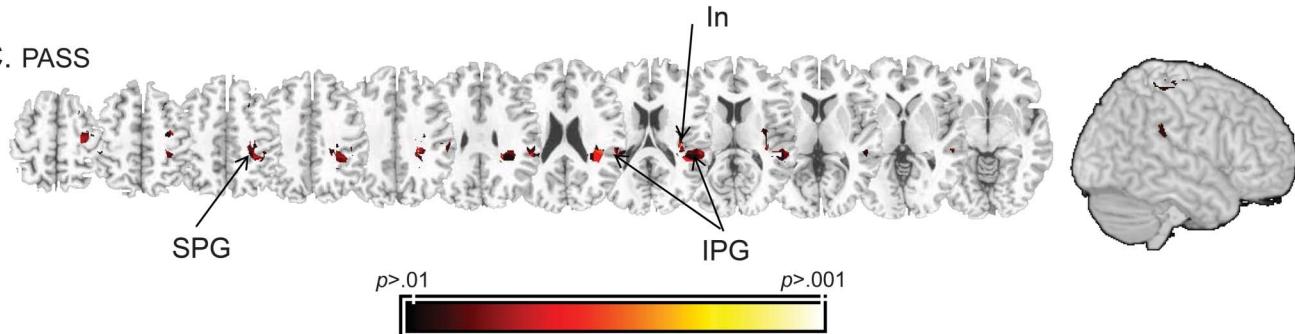
(A) The leftward tilt of the subjective vertical (SV) was associated with lesions of the inferior parietal gyrus, parieto-occipital junction, intraparietal sulcus, and posterior temporal lobe. (B) The rightward deviation of the subjective straight ahead (SSA) correlated with lesions of the anterior parietal lobe, the intraparietal sulcus, and the middle part of the superior temporal gyrus. (C) Postural disorders, as scored on the Postural Assessment Scale for Stroke Patients (PASS),[21] were best explained by lesions of the posterior insula and subcortical structures (superior longitudinal fasciculus). The color range indicates t test values. The axial slices displayed range from z coordinates -6 to $+64$ (A, B, and C) in Montreal Neurological Institute space. In = insula; IPG = inferior parietal gyrus; MTG = middle temporal gyrus; PSA = primary somatosensory area; SPG = superior parietal gyrus; STG = superior temporal gyrus.

cortical and subcortical areas likely to be critically involved in the elaboration of these spatial representations. These biases were influenced by spatial neglect, a finding well-documented for both the SSA[1,4] and the SV.[10] They were also influenced to a lesser degree by hemianopia.[24] However, in contrast with spatial neglect, hemianopia did not aggravate the postural disorders.

The SSA is thought to play a major role in actions within the peripersonal space and its deviation in patients with hemisphere lesions may contribute to difficulties in various egocentric tasks.[25] Here, the SSA bias was mainly related to anterior parietal lesions within and behind the primary somatosensory area (including the intraparietal sulcus), the superior temporal gyrus, and the dorsolateral premotor cortex. In fact, the damaged areas in the anterior parietal lobe were close to those incriminated in personal neglect.[26] The more posterior parietal regions (particularly the horizontal portion of the intraparietal sulcus) and

more anterior regions (especially in the premotor cortex) proved to be activated by egocentric tasks performed by normal subjects.[12] The adjustment of a movable rod in front of the body midline requires participants to develop a representation of their body midline, then to place the object with respect to the representation. One can legitimately hypothesize that the primary somatosensory area has a prime role in the elaboration of the body representation. However, other structures are involved in the multimodal integration necessary to the elaboration of this representation. Indeed, the supramarginalis gyrus (behind the lower part of the somatosensory area)[27] has been put in relation with personal neglect.[28] The superior parietal gyrus and adjacent intraparietal sulcus could also contribute.[29] This latter receives convergent visual, somatosensory, and vestibular afferences,[30] and proved to be activated by egocentric tasks.[27,29] In fact, it is an excellent candidate for the development of spatial egocentric representations. Importantly, the

insula region was unaffected. The lesions to the upper dorsolateral premotor region participated to a lesser degree in the SSA bias. This frontal involvement has been suggested in normal subjects[12,29] and may concern the intentional and motor-directional parts that are present in many spatial tests.[31] In fact, the SSA task conveys both a representational component and a motor component. The rightward shift of the SSA classically observed in patients with neglect can be enhanced by a leftward directional hypokinesia, caused by premotor lesions.

In comparison with the SSA, the SV bias was related to more posterior temporoparietal lesions. In fact, these posterior areas correspond closely to those found to be activated in normal subjects during a SV task,[13] with bilateral activity at the temporo-occipital and parieto-occipital junctions. The anterior part of the damaged area comprised the posterior, superficial part of the parieto-insular vestibular cortex (PIVC),[30,32] but the insula and the adjacent transverse temporal gyrus[9,14] were spared. The lesion also comprised the posterior part of the inferior parietal lobule (area 39), which receives vestibular afferences.[33] The SV task used in the present study depended on visual input. The participants handled the rod with their healthy right hand, so that there were also sensory and motor components related to left hemisphere activity. Overall, our data suggest that the building of gravity representations for visual or visuo-haptic tasks is closely related to the posterior inferior parietal and the middle temporal gyri. The differences between areas involved in the elaboration of the SSA and SV seem to reflect the perceptual systems most deeply involved in each representation. Both representations are elaborated via the integration of somatosensory, vestibular, and visual information.[2,9,10] However, the weight of somatosensory information appears predominant for the body midline representation, whereas the visual system has a greater influence when the sense of verticality is explored through visual input.

Besides, postural disorders were mainly associated with lesions of the posterior insula, the superior temporal lobe, and the lateral parietal cortex. Many different lines of evidence suggest that these areas are strongly involved in postural control, especially in the right hemisphere.[8,33] The predominant participation of the right hemisphere to axial movements is also in accord with the preservation of these movements in patients with severe aphasia and limb apraxia.[34] From an anatomical point of view, the posterior insula, the parietal operculum, and the adjacent superior temporal lobe are the main cortical areas for vestibular afferences (human PIVC).[32,35] Patients with focal stroke in the posterior insula show dizziness, trunk ataxia, and gait instability.[36,37] Furthermore, the superior parietal cortex (area 7) and the adjacent intraparietal sulcus are multimodal areas that are strongly involved in spatial coding.[27,29] Damage in this region may provoke rotational vertigo and feelings of body tilt.[38] At subcortical level, we found a participation of the superior longitudinal fasciculus lesions in both postural disorders and SV bias. This tract is involved in perceptual-motor tasks and its lesion could contribute to postural difficulties exhibited by patients with subcortical neurodegenerative diseases.

In patients with hemisphere lesions, postural control disorders have been associated with both biased representations of the body midline and errors in the allocentric representation of the gravitational vertical.[8,11] Our study suggests that they are more related to a biased representation of the body axis. However, we also found that the anatomy of postural disorders overlapped with that of spatial representation biases, especially the SV bias (superior temporal lobe, temporoparietal junction, inferior parietal gyrus, and longitudinal fasciculus).

One limitation of the study was that all the patients had a right hemisphere stroke. It remains to be explored whether relationships are similar in left lesions. We did not investigate the neuroanatomical correlates of difficulties in daily living activities related to efficient trunk control, transfers, walking, and stairs climbing. The comparison with actual results would also be of clinical interest.

Identifying representational errors and postural difficulties after a stroke is of main prognostic value as regards patient recovery and dependence.[11,21] Identification of such lesion correlates could participate in the early evaluation of prognosis factors, and orient the rehabilitation efforts to be implemented.[39]

AUTHOR CONTRIBUTIONS

Dr. Rousseaux: study concept and design, analysis and interpretation of data, and drafting the manuscript. Dr. Honoré: study design, analysis and interpretation of data, revision of the manuscript for intellectual content. Pr. Vuillemier: revision of the manuscript for intellectual content. Dr. Saj: study concept and design, analysis and interpretation of data, and revision of the manuscript for intellectual content.

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DISCLOSURE

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