doi:10.1093/brain/awq311 Brain 2010: 133; 3552–3563 | 3552

BRAIN
A JOURNAL OF NEUROLOGY

Humans use internal models to construct and update a sense of verticality

Julien Barra,[1,2] Adélaïde Marquer,[2] Roxane Joassin,[3] Céline Reymond,[2] Liliane Metge,[4] Valérie Chauvineau[5] and Dominic Pérennou[2,3,5]

1 Laboratoire de Psychologie et Neuropsychologie Cognitive, Université Paris Descartes, FRE 3292, France
2 Clinique MPR-CHU, Laboratoire TIMC-IMAG CNRS 5525 Equipe Santé-Plasticité-Motricité, Université Grenoble 1, Grenoble, France
3 Service de Rééducation Neurologique, CHU Dijon, France
4 Département d'Imagerie Médicale, CHU de Nîmes, France
5 Unité de Rééducation Neurologique, CHU de Nîmes, France

Correspondence to: Dominic Pérennou,
Clinique MPR- CHU,
Laboratoire TIMC-IMAG CNRS 5525 Equipe Santé-Plasticité-Motricité,
Université Grenoble 1,
Grenoble, France
E-mail: dperennou@chu-grenoble.fr

Internal models serve sensory processing, sensorimotor integration and motor control. They could be a way to construct and update a sense of verticality, by combining vestibular and somatosensory graviception. We tested this hypothesis by investigating self-orientation relative to gravity in 39 normal subjects and in subjects with various somatosensory losses showing either a complete deafferentation of trunk and lower limbs (14 paraplegic patients after complete traumatic spinal cord injury) or a gradient in the degree of a hemibody sensory loss (23 hemiplegic patients after stroke). We asked subjects to estimate, in the dark, the direction of the Earth vertical in two postural conditions—upright and at lateral whole body tilt. For upright conditions, verticality estimates were not different from the direction of the Earth vertical in normal (0.24° ± 1; $P = 0.42$) and paraplegic subjects (0.87° ± 0.9; $P = 0.14$). The within-subject variability was much greater in hemiplegic than in normal subjects (2.05° ± 1.15 versus 1.06° ± 0.4; $P < 0.01$) and greater in paraplegic than in normal subjects (1.13° ± 0.4 versus 0.72° ± 0.4; $P < 0.01$). These findings indicate that, even if vestibular graviception is intact, somaesthetic graviception contributes to the sense of verticality, leading to a more robust judgement about the direction of verticality when vestibular and somaesthetic graviception yield congruent information. As expected, when normal subjects were tilted, their verticality estimates were biased in the direction of the body tilt (5.55° ± 3.9). This normal modulation of verticality perception (Aubert effect), was preserved in hemiplegics on the side of the normoaesthetic hemibody (ipsilesional) (6.09° ± 6.3), and abolished both in paraplegics (1.06° ± 2.5) and in hemiplegics (0.04° ± 6.7) on the side of hypoaesthetic hemibody (contralesional). This incongruence did not exist in deafferented paraplegics who exclusively used vestibular graviception with a similar efficacy no matter what the lateral body position. The Aubert effect was not an on–off phenomenon since the degree of hemiplegics' somatosensory loss correlated with the modulation of verticality perception when they were tilted to the side of hypoaesthetic hemibody ($r = -0.55$; $P < 0.01$). The analysis of anatomical correlates showed that the Aubert effect required the integrity of the posterolateral thalamus. This study reveals the existence of a synthesis of vestibular and somaesthetic graviception for which the posterolateral thalamus plays a major role. This corresponds to a primary property of internal models and yields the neural bases of the Aubert effect. We conclude that humans construct and update internal models of verticality in which somatosensory information plays an important role.

Keywords: visual vertical; somatosensory information; representation; orientation; cerebral plasticity; postural control

Received April 18, 2010. Revised and Accepted September 27, 2010

Introduction

Internal models may represent a general neural process to resolve sensory ambiguity, to synthesize information from disparate sensory modalities, and to combine efferent and afferent information (Merfeld *et al*., 1999). They appear to be of primary importance in sensory processing (Glasauer, 1992; Lewald and Ehrenstein, 1998), sensorimotor integration (Bell *et al*., 1997) and motor control (Yasui and Young, 1975; Wolpert *et al*., 1995). Their involvement in both the representation of verticality and action with respect to gravity has been suggested (Pérennou *et al*., 2008), which would imply that efferent and afferent information with respect to verticality may be combined. Nevertheless, there is a limited amount of direct evidence of the existence of such internal models devoted to the construction and updating of a biological vertical supporting a sense of verticality. It would be built by synthesizing visual, somatosensory and vestibular information (Brandt *et al*., 1994; Bisdorff *et al*., 1996; Merfeld *et al*., 1999; Van Beuzekom and Van Gisbergen, 2000; Bronstein *et al*., 2003; Barbieri *et al*., 2008; Pérennou *et al*., 2008; Tarnutzer *et al*., 2009). The role of visual and vestibular information in verticality perception has widely been investigated (Dichgans *et al*., 1972; Gresty *et al*., 1992; Brandt *et al*., 1994; Merfeld *et al*., 1999; Zupan and Merfeld, 2003; Lorincz and Hess, 2008), whereas the contribution of somaesthetics to verticality perception is less well known. Attempts to address this question have been hampered by the difficulty of experimentally suppressing somaesthetic information, contrary to visual and vestibular information. Manipulation of somaesthetic information has clearly shown that it plays a role in verticality perception (Wade, 1973; Bisdorff *et al*., 1996; Perennou *et al*., 1998; Trousselard *et al*., 2003, 2004; Barbieri *et al*., 2008); the nature of this role and the way it might contribute to an internal model of verticality remain in need of full investigation.

There have been few reports of interplay between somaesthetic information and other sensory modalities in patients with somatosensory alterations in the construction of verticality representation. Yardley (1990) investigated the perception of visual vertical in a patient with poor somatosensory function below the neck as a result of the loss of a large myelinated sensory nerve function. When lying horizontally, the patient did not exhibit the substantial perceived tilt of the visual vertical in the direction of the body tilt (Aubert effect) as control subjects did. In terms of variability, both control and patients showed a similar increased variability of perception. Anastasopoulos and Bronstein (1999) provided the second case study. A 21-year-old female with chronic left hemihypoaesthesia due to a posterior thalamic infarct presented a normal estimate of the visual vertical when seated upright. Observation of the expected Aubert effect took place while she was lying on her right side, but this same Aubert effect was absent when she was lying on her hypoaesthetic side. They found a large increase in visual vertical variability settings after lateral tilts on both sides of control subjects. Anastasopoulos *et al.* (1999) qualitatively reported on the perception of verticality in patients with somatosensory deficits from disparate aetiologies (spinal cord tumours, cerebral haemorrhages, cerebral and spinal ischaemia, demyelination and polyneuropathy). The two patients with the most severe hemisensory loss experienced no Aubert effect when lying on the hypoaesthetic side. We learnt from these observations that the Aubert effect may be absent in subjects with altered somaesthetic graviception.

Although the Aubert effect may provide a window to better understand the interplay between vestibular and somaesthetic graviception, the impact of these studies was diminished by the limited number of patients investigated, the various topographies of sensory alteration due to disparate aetiologies, and the existence of a residual somatosensory sensitivity without precise quantification (Yardley, 1990; Anastasopoulos and Bronstein, 1999; Anastasopoulos *et al*., 1999). Does resistance to the Aubert effect correspond to an on–off phenomenon by way of a processing switch to an intact and reliable vestibular graviception? Or does it rather correspond to taking into account sensory information which would give more weight to the vestibular graviception when the somaesthetic graviception is less available? The existence of this latter mechanism would demonstrate that, to build a reference of verticality, the human brain synthesizes vestibular and somaesthetic graviception. As such, a synthesis of sensory modalities is a primary property of internal models, and this would provide support for the existence of an internal model of verticality in the human brain.

To go further, we investigated verticality representation in 76 subjects with clear somatosensory patterns: 39 normal subjects, 23 stroke subjects showing different degrees of hypoaesthesia pressure on the contralesional hemibody and 14 paraplegic subjects completely deafferented below a given metamere. The accuracy of verticality direction and variability were analysed in both upright and tilted postures. Our main hypothesis was that somatosensory graviception plays a major role in the construction and formation of internal models of verticality. In the conditions in which somaesthetic and vestibular graviception were congruent (upright), we predicted a better representation of verticality with intact vestibular and somaesthetic graviception than when only vestibular graviception was available. When somaesthetic and vestibular graviception were not congruent (tilted posture), we predicted that the Aubert effect would disappear in paraplegics and diminish as a function of the sensory loss in hemiplegics. This would imply that the modulation of the subjective visual vertical by lateral body tilts requires the integrity of neural structures involved in the conduction and integration of the somaesthetic information, especially the spinal cord, thalamus and somatosensory cortex. Another objective of this study was to analyse the correlates between brain structures and the modulation of the subjective visual vertical by lateral body tilts.

Materials and methods

Participants

All patients enrolled in this study were recruited from a neurorehabilitation unit after giving their informed consent in accordance with the local ethics committee guidelines. Since paraplegics were statistically younger than hemiplegics [$t(35) = 3.07$; $P < 0.005$] and given the influence of age on verticality perception (Barbieri *et al*., 2010),

two different control groups were tested, matched in age with the patients groups.

Twenty-three subjects with hemisphere stroke (52.9 ± 11 years; 6 females, 17 males; 9 left- and 14 right-sided strokes, 16 infarcts and 7 haematomas) matched with 27 control subjects (54 ± 9 years) participated in the study. In order to be eligible for the study, patients had to have the inclusion criteria of a first and unique hemisphere stroke. Exclusion criteria included unstable status, neuropathy, psychiatric disorders, major comprehension problems due to aphasia, dementia or signs of vestibular disorders. Vestibular dysfunction signs were sought by a clinician trained in neuro-otology. Patients included had no dizziness, vertigo, spontaneous nystagmus or diplopia. Spontaneous head tilt and skew deviation were sought as signs of otolithic dysfunction (Gresty *et al.,* 1992). The head-shaking test was performed as semicircular canal test (Hain and Spindler, 1993). The patients' heads were moved in the horizontal plane 45° in both directions; a nystagmic response indicated the existence of an imbalance in the vestibular-ocular system. According to Tseng and Chao (1997), the head-shaking test is a simple clinical test that can reliably identify patients with unilateral vestibular dysfunction. The period of time from the stroke was 3.7 ± 2 months. Assessment of hypoaesthesia of the paretic side was carried out through pressure sensitivity with a set of 20 Semmes–Weinstein monofilaments (Semmes *et al*., 1960). As proposed for stroke patients (Pérennou *et al*., 1998), the force applied to the skin at the pulp of the big toe and the second metacarpo-phalangeal joint, needed for a given patient to perceive the stimulus, was subjected to log transformation to obtain a 20-point linear scale (values increasing with the hypoesthesia). We then averaged the values obtained in the upper and lower limbs. Lesion location and extension were analysed using MRI in 16 subjects or computed tomography (CT) scans in 7 subjects. In order to avoid an overestimation of lesion extension in haemorrhagic strokes, lesion location was analysed using MRI or CT performed ~2 months after the onset of stroke. MRI were performed with a 1.5 T Magnetom Vision Plus MRI (Siemens, Erhlangen, Germany) according to a protocol that comprised 20 slices (thickness: 6 mm, interslice gap: 1 mm) in T_2-weighted sequence (repetition time: 6600 ms, echo time: 128 ms) in the anterior commissure-posterior commissure plane; and 20 slices (thickness: 6 mm, interslice gap: 1 mm) in T_2-weighted fluid attenuated inversion recovery (repetition time: 9000 ms, echo time: 119 ms) sequence in the coronal plane. The field of view was 175–220 mm^2. CT scanning was performed using a CT SOMATOplus 4 (Siemens, Erhlangen Germany), with continued slices (thickness: 10 mm, time: 1.5 s, 140 kV, 111 mA) in a plane corresponding to the meato-orbital plane minus 10°. The lesions were reconstructed onto standardized brain templates by an operator who was naive to the results. All lesions were mapped using the free MRIcro (www.mricro.com) software distribution (Rorden and Brett, 2000). Since stroke images comprised CT scan slices in the axial plane, all lesions were drawn manually by one experimenter on axial slices of a T_1-weighted template MRI scan from the Montreal Neurological Institute (MNI) (www.bic.mni.mcgill.ca/cgi/icbm_view). The extension and location of lesion shapes of all patients were controlled (and possibly modified) by a second operator. Both operators were blind to patients' verticality estimates. The template used is oriented to match Talairach space (Talairach and Tournoux, 1988). Lesions were mapped onto the slices that correspond to *z*-coordinates −16, −6, 4, 14, 24, 34, 44 and 61 mm in Talairach coordinates.

Fourteen paraplegic subjects (41.6 ± 12 years, 11 males and 3 females) matched in age and gender with 12 control subjects (39 ± 10 years) participated in the study. Inclusion criteria were traumatic spinal cord injury resulting in a chronic paraplegic condition with a complete sensory loss below a given sensory level while the patient is medically stable. Exclusion criteria included: history of stroke, vestibular disorder or any other disorder affecting the central or peripheral nervous system, a pronounced visual deficiency or bedsoreness. The period of time from the traumatic spinal injury was 93.5 ± 51 months. The sensory level was established by a standard non-invasive neurological examination of touch, pain, vibration sensitivity and reflexes (American Spinal Injury Association, 2000). The sensory level, by convention the lowest segment where sensory function is normal on both sides, was the fourth thoracic vertebra in four subjects (deafferented below the nipple line), the fifth thoracic vertebra in two subjects, the sixth thoracic vertebra in three subjects (deafferented below the xiphisternum), the seventh thoracic vertebra in one subject, the eighth thoracic vertebra in one subject, and the 12th thoracic vertebra in three subjects (deafferented below the lower part of the trunk). The diagnosis was corroborated by available anatomical evidence on complete local destruction or interruption of the cord. The motor recovery was poor in all patients, none of them being able to stand up.

Tasks

The perception of visual vertical was assessed in complete darkness by visual adjustments of the direction of a luminous line (15 cm long, 2 mm wide, 1.5 m from the subject). Subjects indicated verbally how to reset the line to their subjective visual vertical. The subject's head, trunk and lower limbs were restrained in an upright sitting position within a tilting drum. The subjective visual vertical was investigated in the upright posture (0°, tested first) and in laterally-tilted postures.

Paraplegic subjects were tilted 50° to only one side since no asymmetric effect was expected on one hand, and the need to limit the experiment duration due to a high bedsore risk on the other hand. The side of the lateral tilt was counterbalanced between subjects (patients as controls) and visual vertical estimates were pooled after a sign transformation according to the lateral tilt side.

Hemiplegic subjects were 30° tilted on both sides because of their hemi-body sensory asymmetry. The angle of 30° was a compromise between tolerable discomfort (especially a possible shoulder pain when tilted to the paretic side), and optimizing the effects of tilt on perception. Visual vertical estimates were subjected to a sign transformation according to the lesion side.

The initial orientation of the luminous line was either 30° or −30°, and the order was randomly distributed over the 10 trials by condition. For lateral tilts the drum was manually rolled towards one side as smoothly as possible at ~1.5°/s. Tilt was measured by an inclinometer (accuracy 0.5°) and subjects were kept in steady position 5 min before starting trials.

Data analysis

Analysis of variance (ANOVA) was used to study the error of perceptual estimates relative to the objective orientation of the gravitational upright and the variability corresponding to the within-subject standard deviation computed from repetitive adjustments of the visual vertical. In order to obtain Gaussian distribution of the variability of visual vertical perception, we performed a logarithmic transformation of the data. *Post hoc* analyses used Tukey's Honestly Significant Difference and when needed, Spearman's correlations were computed.

Previous studies investigating the perception of visual vertical in hemiplegic subjects after hemispheric stroke (Brant *et al*., 1994; Kerkhoff and Zoelch, 1998; Yelnik *et al*., 2002; Perennou *et al*., 2008) reported that perception of visual vertical can be biased towards the contralesional side. In order to analyse the influence of somaesthesia on the visual

vertical estimates, we calculated the modulation of the visual vertical perception (Aubert effect) taking into account the possible initial subjective visual vertical bias in the upright posture. The modulation of the visual vertical perception was computed as the difference between visual vertical estimates after body tilt and visual vertical estimates in upright posture (visual vertical estimates after lateral body tilt—visual vertical estimates in upright posture). We tested the influence of clinical features on vertical estimates in upright patients as well as on visual vertical modulation by lateral body tilts using non-parametric Spearman's correlations.

The lesion extension was determined as the percentage of amount of hemisphere encroached on by the lesion (number of voxels for the lesion × 100/number of voxels for the hemisphere) (Karnath *et al*., 2004). In order to analyse the influence of stroke location on the visual vertical (in upright), groups were constituted according to the range of normality established in a previous study (Pérennou *et al*., 2008) of a larger population tested with the same procedure as the one used in the present study: from -2.5° to 2.5°. Lesion overlay maps of stroke areas for the nine subjects with normal visual vertical and the 14 subjects with biased visual vertical were constructed by superimposing their brain sections (Fig. 3A and B). The lesion reconstructions of left strokes were reversed to be combined with those of right strokes. To identify the structures that are specifically damaged in patients with visual vertical bias we subtracted the superimposed lesions of the group of patients without visual vertical bias from the overlap image of the patients with visual vertical bias revealing a percentage overlay plot. This created a map that highlights regions (and corresponding Talairach coordinates) frequently damaged in patients with a biased visual vertical (Fig. 3C). The same procedures were conducted on visual vertical modulation data in order to determine the brain structures specifically damaged in patients without visual vertical modulation (Fig. 4). When needed, the correspondence between given coordinates and a precise brain area was refined using the brain cartography in Talairach coordinates (www.talairach.org).

Results

Visual vertical accuracy

Visual vertical in upright and tilted postures

We first analysed the perception of visual vertical in upright posture using a one-sample *t*-test (against 0°). Subjective visual vertical was accurate (not different from 0°) in both control groups [$t(26) = 0.87$; $P = 0.4$ and $t(11) = -1.47$; $P = 0.17$] and in paraplegic subjects [$t(13) = 1.57$; $P = 0.14$], whereas a spontaneous contralesional subjective visual vertical tilt ($-4.7^\circ \pm 4.7$) was found in hemiplegic subjects [$t(22) = -4.82$; $P < 0.001$]. These findings mean that a somaesthetic deafferentation does not bias the perception of visual vertical.

Data in each group of patients were further investigated by a two-way ANOVA bearing on subjects' groups (control subjects, patients) and body positions (upright, lateral tilt).

As for paraplegic subjects (Fig. 1A), the ANOVA showed a body position effect [$F(1,24) = 57.47$; $P < 0.02$] and an interaction between both factors [$F(1,24) = 5.43$; $P = 0.028$] but no group effect [$F(1,24) = 3.37$; $P = 0.078$]. The *post hoc* analysis of the interaction showed the existence of a substantial Aubert effect in control subjects in whom visual vertical perception was biased in direction of the body tilt ($P = 0.014$). In contrast, paraplegic subject's visual vertical estimate was as accurate in tilted posture as in upright ($P = 0.998$). Furthermore, control and paraplegic subjects displayed similar subjective visual vertical accuracy in upright conditions ($P = 0.992$).

As for hemiplegic subjects (Fig. 1B), the ANOVA showed a group effect [$F(1,48) = 8.1$; $P < 0.01$], a body position effect [$F(2,96) = 50.12$; $P < 0.01$] and an interaction between both factors [$F(2,96) = 5.45$; $P < 0.01$]. The *post hoc* analysis of the

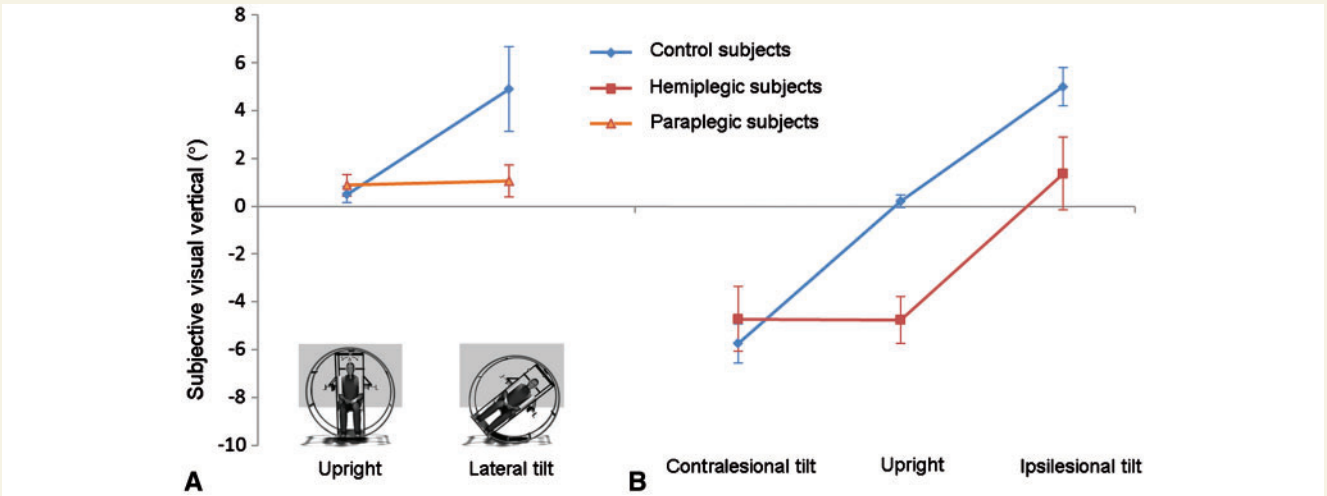

Figure 1 Comparison of visual vertical estimates for hemiplegic, paraplegic and control subjects in different body postures. Mean visual vertical accuracy (°) in 14 upright or tilted 50° paraplegic subjects and their 12 matched in age control subjects (**A**). A positive value corresponded to a deviation in direction of the lateral body tilt; (**B**) in 23 upright or 30° tilted hemiplegic subjects and their 27 matched in age control subjects. Positive value of visual vertical corresponded to a rotation relative to the objective direction (true vertical) towards the ipsilesional side (after sign transformation according to the lesion side) for the patient and towards the left shoulder of control subjects. The error bars correspond to the standard errors.

interaction showed a spontaneous deviation of visual vertical estimates in direction of the contralesional side (paretic) in hemiplegic subjects compared with controls subjects ($P < 0.01$). In control subjects, lateral tilts induced deviations of the subjective visual vertical in direction of the lateral body tilt compared with upright estimates (both sides $P < 0.05$), whereas in hemiplegic subjects this effect was only found in tilts towards the ipsilesional side ($P < 0.01$). No Aubert effect was found when hemiplegic subjects were tilted towards the contralesional side ($P = 0.99$). This resistance to the Aubert effect was further analysed. We found a strong link between the magnitude of subjective visual vertical modulation (visual vertical estimates after contralesional body tilt—visual vertical estimates in upright posture) and the degree of sensory loss (hypoaesthesia) on the impaired hemi-body ($r = -0.55$; $P < 0.01$; Fig. 2). The less somatosensory information was available on the contralesional side the less visual vertical perception was modulated. This finding proves the existence of a gradient of subjective visual vertical modulation depending on the degree of availability of the somatosensory information. In contrast, when hemiplegic subjects were tilted towards the ipsilesional side, the modulation of the visual vertical estimates (visual vertical estimates after ipsilesional body tilt-visual vertical estimates in upright posture) was not linked to the degree of hypoaesthesia ($r = 0.17$; $P = 0.4$).

Influence of stroke location and extension on verticality perception in the upright position

First, we checked that variations in visual vertical perception between patients were not due to the difference in elapsed time since the occurrence of stroke by testing the correlation between these two variables ($r = 0.05$; $P = 0.83$). The reconstruction of brain areas damaged in stroke subjects (Fig. 3) showed that lesions were more extended in those with a bias in the visual vertical than in those without bias in the visual vertical [17% ± 13 versus 7% ± 6; $t(21) = 2.14$; $P = 0.04$]. A significant correlation between lesion extension and the contralesional visual vertical tilt was also found ($r = 0.54$; $P < 0.01$): the longer the extension the more biased the visual vertical towards the contralesional side. Visual vertical was not different in magnitude in subjects with a right or a left stroke [$t(21) = -1.54$; $P = 0.19$]. This allowed left and right strokes to be superimposed on the same brain sections to identify structures specifically damaged in patients with a tilted visual vertical. The analysis of the overlay plot of the subtracted superimposed lesions of patients with visual vertical bias minus patients without visual vertical bias (Fig. 3C) showed that the most frequently and specifically damaged cerebral region in patients with biased visual vertical was centred on the insula ($X^2 = 12.4$; $P < 0.01$; $x = 34$, $y = 14$, $z = 4$) and the surrounding white matter. Lesions also frequently encroached on some adjacent areas: the parieto-rolandic opercular cortex ($X^2 = 8$; $P < 0.01$), the transverse temporal gyrus ($X^2 = 12.1$; $P < 0.01$) and the superior temporal gyrus ($X^2 = 11.4$; $P < 0.01$). This means that the insula is a key structure involved in the perception of the visual vertical.

Influence of lesion location and extension on verticality modulation

First we analysed the influence of the sensory level of paraplegic subjects on their resistance to the Aubert effect. No correlation was found ($r = 0.04$; $P = 0.88$). The resistance to the Aubert effect was similar in the three subjects deafferented from and below the pelvis (12th thoracic vertebra) and the four subjects deafferented up to the nipple line (fourth thoracic vertebra): −0.6° ± 0.6 versus −0.9° ± 1.2. This result highlights the critical role of the sensory information provided below the metamere of the 12th thoracic vertebra (the buttocks area and the lower limbs), to determine while sitting the direction of the vertical. No correlation

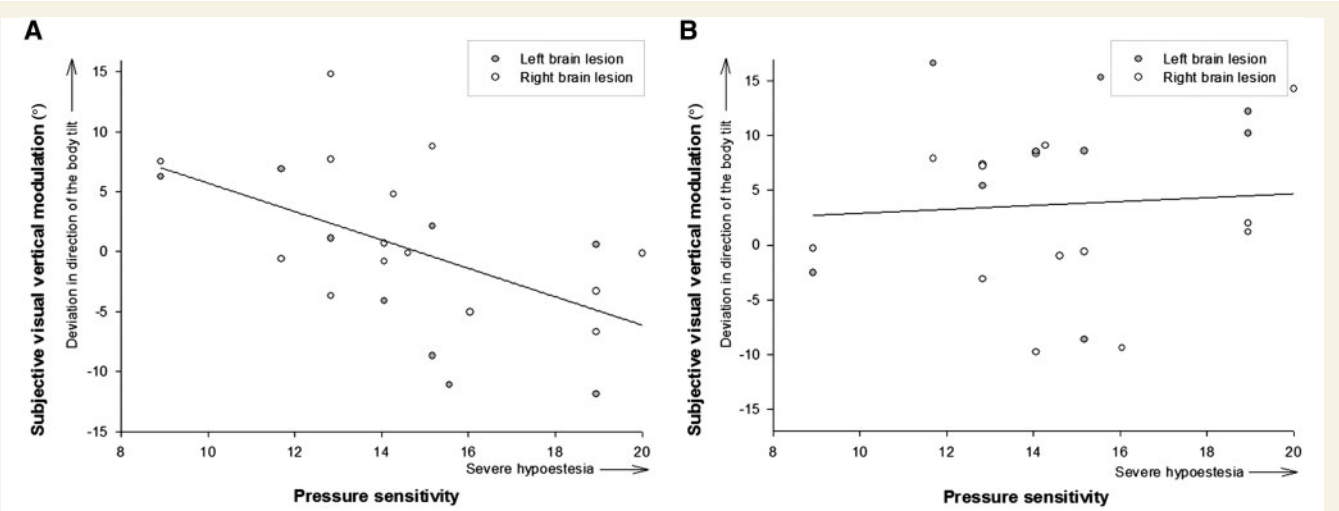

Figure 2 Modulation of visual vertical accuracy (visual vertical estimates after lateral tilt—visual vertical estimates in upright posture) and pressure sensitivity loss in 23 hemiplegic subjects (**A**) after ipsilesional body tilt; (**B**) after contralesional body tilt. Positive values correspond to a deviation of the visual vertical in direction of the body tilt.

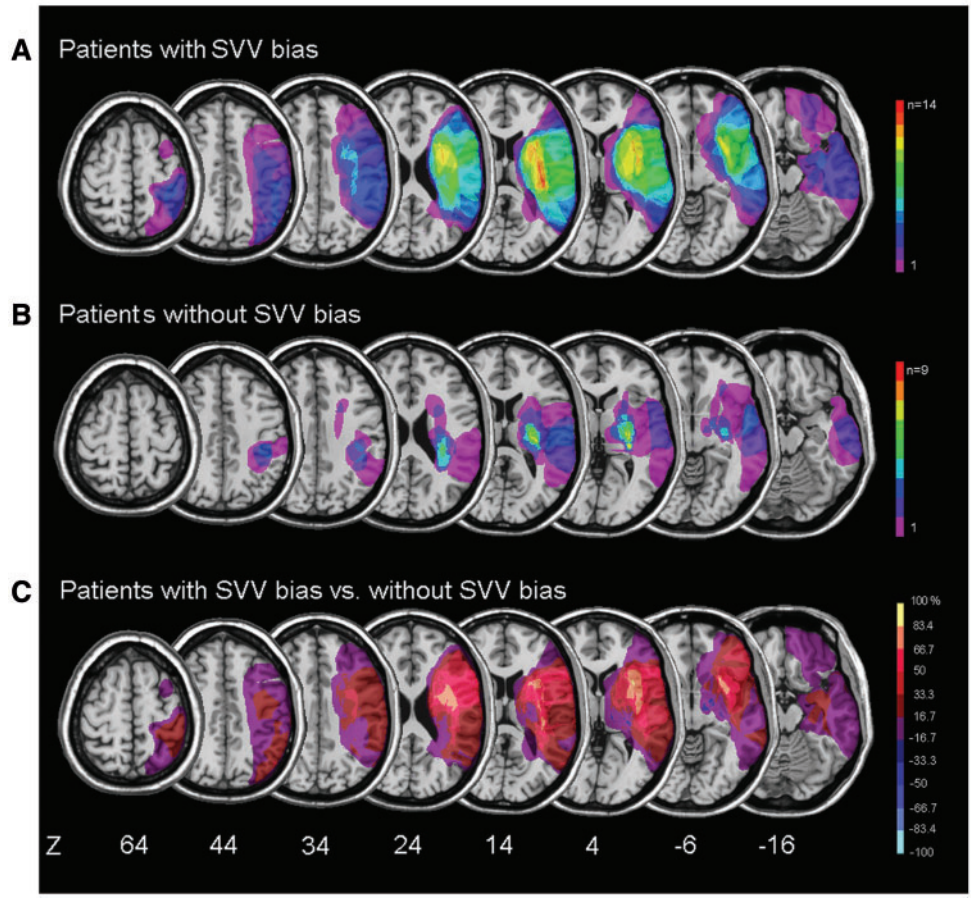

Figure 3 (**A**) Overlay lesion plot of the patients with a visual vertical bias ($n = 14$). The number of overlapping lesions is illustrated by different colours coding increasing frequencies from violet ($n = 1$) to red ($n = 14$). (**B**) Overlay lesion plot of patients without visual vertical bias ($n = 9$). Number of overlapping lesions is illustrated by different colours coding increasing frequencies from violet ($n = 1$) to red ($n = 9$). (**C**) Overlay plot of the subtracted superimposed lesions of the patients with visual vertical bias minus the patients without visual vertical bias. The percentage of overlapping lesions of the group with visual vertical bias after subtraction of the patients without bias is illustrated by five different colours coding increasing frequencies from dark red (16.67–33.34%) to yellow (83.35–100%). Each colour represents 16.67% except the violet bar that represent 33.34% (from −16.67 to +16.67%). The colours from dark blue (from −16.67 to 33.34%) to light blue (from −83.35 to −100%) indicate regions damaged more frequently in the patient without visual vertical bias than in patients with visual vertical bias. Talairach *z*-coordinates of each transverse slice are given (Talairach and Tournoux, 1988). The figure illustrates that the anatomical area related to subjective visual vertical (SVV) bias is centred on the insula.

was found between the time elapsed since the paraplegia onset and the resistance to the Aubert effect ($r = 0.25$; $P = 0.39$). As the modulation of visual vertical by an ipsilesional tilt is not different from that found in controls, we only analysed the influence of stroke location and extension on the modulation of the visual vertical by contralesional body tilt. The eight subjects with visual vertical modulation were compared with the 15 subjects without visual vertical modulation. We considered a modulation of visual vertical as substantial when it was superior to 1.2° [controls' mean modulation − 1 standard deviation (SD): 5.36 − 4.15 = 1.2°]. Analysis of a reconstruction of brain areas damaged in stroke subjects (Fig. 4) showed that the lesions were not more extended in those with visual vertical modulation by contralesional body tilt than in those without visual vertical modulation (15% ± 14 versus 9% ± 7; $t(21) = 1.2$; $P = 0.24$). In addition, no correlation was found between lesion extension and visual vertical modulation by contralesional body tilt ($r = 0.09$; $P = 0.69$). Visual vertical modulation by contralesional body tilt was not different in right and left strokes [$t(21) = -1.35$; $P = 0.2$]. Furthermore, the lateralization of the lesion did not influence the presence of a visual vertical modulation (five rights/three lefts with visual vertical modulation versus nine rights/six lefts without visual vertical modulation; $X^2 = 0.01$; $P = 0.9$). This allowed superimposition of left and right strokes on same brain sections to identify the structures specifically damaged in patients with resistance to the Aubert effect. Analysis of the overlay plot of the subtracted superimposed lesions of the patients without visual vertical modulation minus the patients with visual vertical modulation (Fig. 4C) showed that the most frequently damaged area in patients without visual vertical modulation corresponded to a small area in the posterolateral thalamus, encroaching mostly on the ventro-posterior lateral nuclei ($X^2 = 15.2$; $P = 0.003$; $x = 17$, $y = -20$, $z = 4$). No link

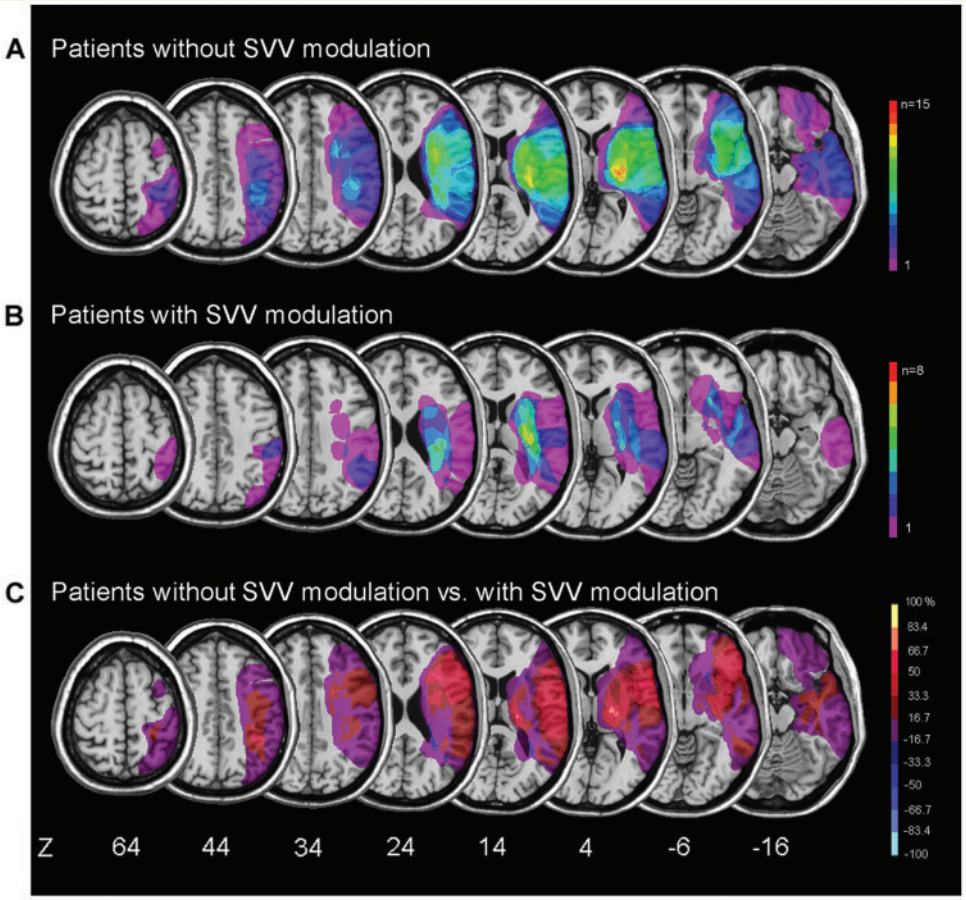

Figure 4 (**A**) Overlay lesion plot of the patients without visual vertical modulation ($n = 15$). The number of overlapping lesions is illustrated by different colours coding increasing frequencies from violet ($n = 1$) to red ($n = 15$). (**B**) Overlay lesion plot of patients with visual vertical modulation ($n = 8$). Number of overlapping lesions is illustrated by different colours coding increasing frequencies from violet ($n = 1$) to red ($n = 8$). (**C**) Overlay plot of the subtracted superimposed lesions of the patients without visual vertical modulation minus the patients with visual vertical modulation. The percentage of overlapping lesions of the group without visual vertical modulation after subtraction of the patients with visual vertical modulation is illustrated by five different colours coding increasing frequencies from dark red (16.67–33.34%) to yellow (83.35–100%). Each colour represents 16.67% except the violet bar that represent 33.34% (from − 16.67 to + 16.67%). The colours from dark blue (from −16.67 to 33.34%) to light blue (from −83.35 to −100%) indicate regions damaged more frequently in the patient with visual vertical modulation than in patients without a visual vertical modulation. Talairach *z*-coordinates of each transverse slice are given (Talairach and Tournoux, 1988). The figure illustrates that the most frequently damaged area in patients without visual vertical modulation corresponded to a small area in the posterolateral thalamus, encroaching mostly on the ventro-posterior lateral nuclei. SVV = subjective visual vertical.

was found between time elapsed since the onset of stroke and visual vertical modulation by contralesional tilt ($r = 0.19$; $P = 0.4$).

Visual vertical variability

Data in each group of patients were analysed by a two-way ANOVA bearing on subject groups (control subjects, patients) and body positions (upright, lateral tilt). As for paraplegic subjects (Fig. 5A), this ANOVA showed a body position effect [$F(1, 24) = 18.54$; $P < 0.001$], an interaction between position and group factors [$F(1, 24) = 13.82$; $P < 0.001$], without group effect [$F(1, 24) = 0.1$; $P = 0.97$]. *Post hoc* analyses showed that the subjective visual vertical variability was higher in paraplegic subjects than in control subjects in upright ($P < 0.01$) but higher in control subjects than in paraplegic subjects in tilted posture ($P < 0.01$). These findings indicate that, even if the vestibular graviception is intact, the somaesthetic graviception contributes to the sense of verticality, leading to a more robust judgement about the direction of verticality provided that vestibular and somaesthetic graviception yield congruent information. In other words, in upright posture the sense of verticality is better when combining vestibular and somaesthetic graviception than with the vestibular graviception alone. While a lateral body tilt dramatically increased subjective visual vertical variability in normal subjects ($P < 0.001$), subjective visual vertical variability was as good in tilted posture as in upright paraplegic subjects ($P = 0.96$). Again this finding shows the important contribution of somaesthetic graviception to the sense of verticality in healthy subjects. This alters the robustness

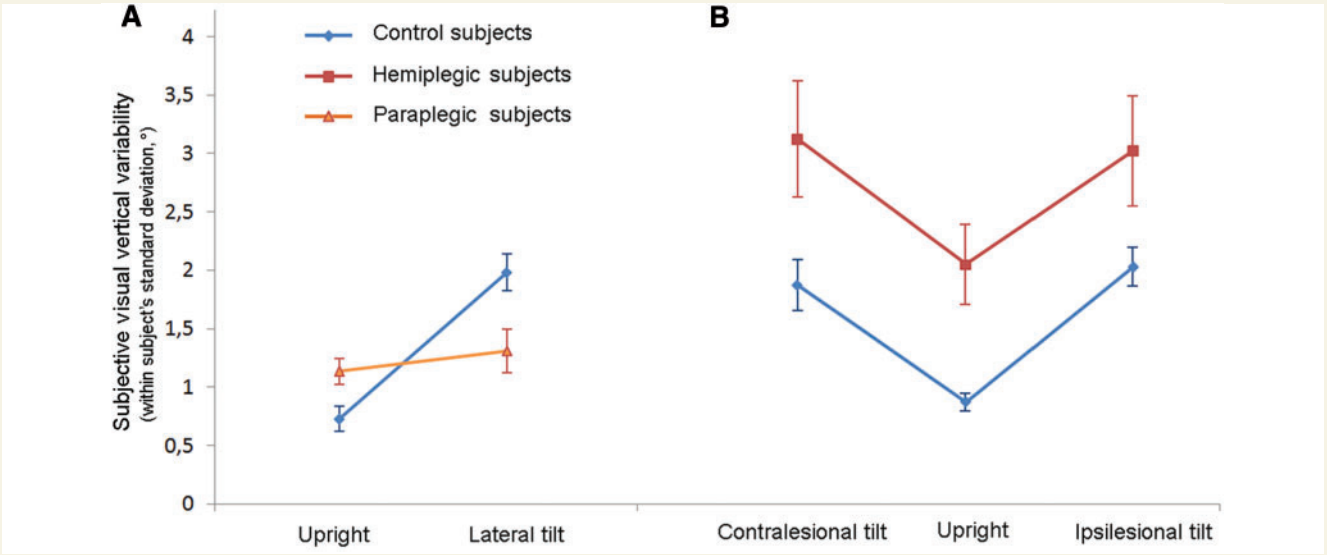

Figure 5 Comparison of the variability of visual vertical estimates for hemiplegic, paraplegic and control subjects in different body postures. Mean visual vertical variability (within-subject standard deviations of visual vertical estimates); (**A**) in 14 upright or tilted paraplegic subjects and their 12 matched in age control subjects; (**B**) in 23 upright or tilted hemiplegic subjects and their 27 matched in age control subjects. The error bars correspond to the standard errors.

of the judgement about verticality direction when verticality directions yielded by somaesthetic graviception and by vestibular graviception are not congruent. This incongruence does not exist in deafferented paraplegics who exclusively use vestibular graviception with a similar efficacy no matter what the lateral body position.

As for hemiplegic subjects (Fig. 5B), the ANOVA showed a group effect [$F(1, 48) = 14.20$; $P < 0.001$] and a body position effect [$F(2, 96) = 14.94$; $P < 0.001$] but no interaction between both factors [$F(2, 96) = 0.40$; $P = 0.66$]. The variability of visual vertical estimates after ipsilesional and contralesional tilts did not differ ($P = 0.93$) and were much higher than that found in the upright condition (both $P < 0.001$).

In addition, in upright subjective visual vertical, variability was much higher in hemiplegic subjects than in paraplegic subjects [$t(35) = 2.82$; $P < 0.01$]. The subjective visual vertical variability was much lower in control subjects than in patients.

Discussion

We designed this study to test our hypothesis that somatosensory graviception plays a major role in the construction of internal model of verticality, and to reveal whether or not the resistance to the Aubert effect described in subjects with an altered somaesthesia responds to selection or ponderation mechanisms between vestibular and somaesthetic graviception. We selected 76 subjects with clear somatosensory patterns (39 controls, 23 hemiparetics, 14 paraplegics), and asked them to perform a similar paradigm of verticality perception, both in upright and tilted postures. Neural correlates underlying biases in visual vertical were analysed in patients.

Visual vertical in the upright position

Many studies have shown that the visual vertical is normally extremely precise in the upright position, an argument in favour of a robust representation of the vertical in the normal brain. Because the variability of the visual vertical estimates was greater in paraplegic than in control subjects, the first novelty revealed by our study is that somatosensory information may improve the stability of verticality perception. As no direct stimulus of gravity exists, the brain uses all information available to estimate the earth vertical and when information is congruent (upright posture), the more information available the stronger the construction of an internal model of verticality. The upright posture is ecological and corresponds to the position required for subjects to get a precise and stable orientation for action.

To the best of our knowledge, our study is one of the first to investigate verticality perception among a group of paraplegic subjects. As expected, no bias was found in visual vertical since the brain was intact and the somatosensory deficit was bilateral (Mazibrada *et al*., 2008).

Many subjects with a hemisphere stroke show an altered verticality representation, tilted to the contralesional side (Brandt *et al*., 1994; Kerkhoff and Zoelch, 1998; Yelnik *et al*., 2002; Barra *et al*., 2008; Pérennou *et al*., 2008) and have greater within-subject variability than control subjects (Yelnik *et al*., 2002). These troubles are the consequences of a cerebral lesion, and are greatly influenced by its location and extension. The present study provides statistical confirmation of the initial description by Brandt *et al*. (1994) whose investigation of 14 patients with well delimited temporal infarcts showing pathological subjective visual vertical tilts revealed that the area in most cases included the long insular gyrus and the adjacent superior temporal gyrus or the transverse

temporal gyrus. Verticality can be perceived through different modalities—the 'visual' perception of the vertical that relies on visuo-vestibular information, the 'postural' perception of the vertical derived from graviceptive-somaesthetic information, and the tactile (haptic) vertical (Pérennou *et al*., 2008). These three modalities can be concomitantly altered by a lesion involving the parietal cortex, and can be subjected to dissociated biases for other lesion locations (Pérennou *et al*., 2008). The primary role of the insula in the visual perception of the vertical emphasizes the major contribution of the vestibular information to the visual vertical (Brandt *et al*., 1994; Bronstein *et al*., 2003) and the major role of the insula in the high-order spatial integration of the vestibular information (Brandt *et al*., 1994; Brandt and Dieterich, 1999). Our study is the first to show an influence of the lesion extension on the frequency and magnitude of visual vertical tilts, a relationship previously found for the postural vertical with a quantification of lesion extension less sensitive than in the present study (Pérennou *et al*., 2008). Together with the fact that—in hemisphere strokes—lesion locations other than the insula may induce tilts of the visual vertical (Dieterich and Brandt, 1993*a*; Pérennou *et al*., 2008) or of the postural vertical (Pérennou *et al*., 2008), especially the temporal cortex, the parietal cortex and the thalamus (Dieterich and Brandt., 1993*b*; Pérennou *et al*., 2008), the relationship between lesion extension and abnormalities in verticality perception indicates that verticality representation depends more on the competencies of neural circuits than on the specificity of a given brain structure. We postulate that these neural circuits might cover the thalamo-parietal projections for the somaesthetic graviception (Pérennou *et al*., 2008) and the thalamo-insular projections for the vestibular graviception. Other mechanisms such as ocular torsions might be involved in visual vertical bias in upright patients after lesions of the cerebellum or of the brain stem (Dieterich and Brandt, 1993*a*; Bronstein *et al*., 2003; Pérennou *et al*., 2008; Baier *et al*., 2009).

The Aubert effect responds to properties of an internal model

The Aubert effect often manifests itself when many normal subjects are tested by being tilted laterally. This Aubert effect consists of a deviation of visual vertical estimates in direction of the body tilts (Aubert, 1861). It is supposed to result from a compromise that the brain reaches to get a precise and stable representation of verticality in a tilted posture, from the integration of the vestibular and the somaesthetic graviception (Mittelstaedt, 1983). It is known to be stronger for angles $>60^{\circ}$ (Day and Wade, 1969; Parker *et al*., 1983), but it has been reported in previous studies for angles similar to those used in our study ($<60^{\circ}$) (Witkin and Asch, 1948; Wade, 1972; Van Beuzekom and Van Gisbergen, 2000; Trousselard *et al*., 2003, 2004). Our results also confirmed the observations of case studies where subjects with no somatosensory function below the neck (Yardley, 1990) and subjects suffering from a hemisensory loss lying on their anaesthetized side (Anastasopoulos *et al*., 1999) did not exhibit the expected Aubert effect when tilted in the way that normally brought about an Aubert effect. In hemiplegic subjects, we also confirm that tilting patients towards the ispilesional side (non-paretic) could be a way to recalibrate the sense of verticality (Barra *et al*., 2008).

The primary goal of our study consisted of investigating the modulation of the visual vertical and analysing the link between the pattern and the degree of somatosensory loss, precisely quantified, and the modulation of verticality perception by lateral body tilts. Our results revealed that the Aubert effect is not an on–off effect responding to a selection by the brain of a given piece of sensory information, but rather a progressive effect responding to the modulation of the visual vertical perception as a function of somatosensory availability. This modulation was abolished in deafferented subjects, reduced in subjects with hypoaesthesia, and preserved when subjects were tilted to a side with a normal sensitivity. This demonstrates the existence of an internal model of verticality in human, with a biological (subjective) vertical constructed by synthesizing available sensory information.

Neural basis of the Aubert effect

Our study is the first to analyse the neural correlates of the Aubert effect. In subjects with stroke suffering a contralesional body tilt we found no correlation between lesion extension and visual vertical modulation, but a critical role of the posterolateral thalamus, more specifically an area encroaching on the ventro-posterior lateral nuclei. The existence of an Aubert effect requires the integrity of the posterolateral thalamus, which plays a functional role in the processing of both the vestibular (Dieterich *et al*., 2005) and the somaesthetic (Pérennou *et al*., 2008) graviception. This role is supported by the existence, in humans, of projections from the anterior part of the pulvinar and from the ventro-posterior lateral nuclei to the parieto-insular, temporal and parietal cortices (Behrens *et al*., 2003; Zhang *et al*., 2010). Studies on the monkey have shown most ascending vestibular fibres in the ventro-posterior lateral nuclei to be in close contact, intermingled with somaesthetic relay cells (Buttner and Lang, 1979). This proximity creates the conditions for synthesizing vestibular and somaesthetic graviceptions. Because no brain area other than the posterolateral thalamus was found to be involved in the resistance to the Aubert effect, and because the resistance to the Aubert effect was unrelated to lesion extension, we conclude that the posterolateral thalamus acts as an integrative complex in the synthesis of vestibular and somaesthetic graviceptions. This synthesis is a condition for developing an internal model of verticality, which consequently involves the thalamus. It might account for the critical role of the thalamus in orienting the body against gravity (Masdeu and Gorelick, 1988; Karnath *et al*., 2005; Pérennou *et al*., 2008).

Our study shows that although both hemispheres are competent to perform this synthesis, it is performed in the hemisphere that processes the most relevant somatosensory information. In a subject laterally tilted, the most relevant somatosensory information is given by the hemibody submitted to the pressure of the body tilt. Therefore it is processed by the opposite hemisphere. For instance, a right hemisphere stroke involving the posterolateral thalamus perturbs the synthesis of the somaesthetic graviception given by the left hemibody and of the vestibular graviception given by the left otolith. This perturbation abolishes the Aubert

effect when the subject is tilted to the left whereas the Aubert effect is present when the subject is tilted to the right. In this latter position the synthesis of vestibular and somaesthetic graviceptions by the left hemisphere is not perturbed.

Although resistance to the Aubert effect was not significantly different for right and left strokes, the present study cannot make a definitive conclusion about a possible lateralization for the synthesis of the vestibular and somaesthetic graviception in the posterolateral thalamus. This lateralization would be in line with the predominance of the right vestibular cortex (Dieterich *et al*., 2005) and of the right thalamo-parietal projections involved in processing the somaesthetic graviception (Pérennou *et al*., 2008). Alternatively, the existence of a bilateral direct ascending vestibular projection from the vestibular nuclei to the inferior part of the insula, which bypasses the posterolateral thalamus and is stronger in the right hemisphere (Dieterich *et al*., 2005), could explain a relative symmetry in the synthesis of the vestibular and somaesthetic graviceptions by the left and right thalamus, and at the same time be compatible with a strong right hemisphere dominance for the representation of verticality (Pérennou *et al*., 2008). Further studies will be needed to clarify this point.

Nature of somaesthetic graviception

To the best of our knowledge, our study is the first that assessed the perception and variability of visual vertical in patients with a gradient of somatosensory loss precisely quantified. This provides the opportunity to test the validity of Mittelstaedt's model (1983) for explaining the Aubert effect. According to Mittelstaedt (1983), the Aubert effect expresses a vectorial resultant between a gravitational vector (veridically sensed by the otolith organs) and the main longitudinal axis of the body (the idiotropic vector). In our study, the orientation of the idiotropic vector was constant (the angle of the body tilt) whereas the length of the idiotropic vector varied with the degree of hypoaesthesia. The more pronounced the hypoaesthesia, the shorter the idiotropic vector. The resultant gravitational and idiotropic vectors were close to the gravitational vector in severe hypoaesthaesia that encounters the resistance to the Aubert effect in deafferented patients. This demonstrates the validity of Mittelstaedt's model (1983) for explaining the Aubert effect.

Patterns of pressures within and at the surface of the body (Pérennou *et al*., 1998), proprioceptive or visceral cues from the trunk (Trousselard *et al*., 2003), have an important role in constructing an internal model of verticality. From experiments with paraplegics subjected to centrifugation in lying, Mittelstaedt (1992) suggested the existence of at least two components of somatic graviceptive information, one 'truncal system' that is lost with destruction of the last two thoracic segments of the cord (11th and 12th thoracic vertebras) and another identified as a possible 'vascular' graviceptive system operative with lesion up to sixth cervical segment. This view is contradicted by the data of our study with sitting paraplegic subjects. We found that the resistance to the Aubert effect was similar in the three patients deafferented from and below the pelvis (12th thoracic vertebra) and the four patients deafferented up to the nipple line (fourth thoracic vertebra). This result stresses the critical role of the sensory information provided below the metamere T12 to determine the direction of the vertical in sitting patients. This role of the pressure distribution under the buttocks in the transmission of information to the brain regarding the vertical direction position has already been put forward (Pérennou *et al*., 1998, 2002). The present study seems to confirm this view. The proprioception of the lower limbs could also play an important role (Dietz *et al*., 1992; Massion, 1992; Barbieri *et al*., 2008).

Possible limitations of our study

Our findings were probably not induced by a methodological bias. The paradigm required large lateral body tilts but due to patients' clinical limitations (e.g. high bedsore risk, tolerable discomfort on the hemiplegic side) the angles were limited and not identical in both groups of patients. This difference in body tilt angles should not have affected the results since expected Aubert effects were observed in control subjects with both angles and since both groups of patients were never compared directly but compared with their specific control groups.

We did not measure ocular torsions that could theoretically induce a subjective visual vertical bias in some upright patients. There was no reason to expect ocular torsion in paraplegic subjects whereas few patients with a hemisphere stroke could have displayed an ocular torsion due to central vestibular disorders (Brandt *et al*., 1994). However, ocular torsion happens less often in hemisphere strokes than in brainstem strokes (Dieterich and Brandt, 1993*a*; Brandt *et al*., 1994). Patients with signs of vestibular disorders were not included in the study that limited the inclusion of patients with ocular torsion. The main results of the study in stroke subjects concern the modulation of the subjective visual vertical, and computation took into account the possible initial bias of visual vertical (irrespective of its nature).

The time elapsed since lesion varied greatly in patients. Could it have had an impact on the results through reorganization processes? The absence of any statistical link between the visual vertical modulation and time elapsed for both the stroke and the paraplegia presents a case against this possibility. The time elapsed since the spinal cord lesion was much longer than the time elapsed since the brain lesion. It is likely that this long period of time (93.5 ± 51 months) may have facilitated the sensory reorganization, increasing the weight of the vestibular information. Further studies testing paraplegic and stroke subjects at various phases of recovery could be interesting to determine more information.

Conclusion

By showing how vestibular and somaesthetic graviceptions are synthesized in the brain, and by revealing that the posterolateral thalamus plays a major role in this synthesis, which is a condition for an internal model, our study yields one of the first direct pieces of evidence of the existence of an internal model of verticality. It also reveals the neural bases of the Aubert effect, and shows that the somaesthetic information plays an important role in verticality representation. When both somaesthetic and vestibular graviceptive signals are congruent (upright posture), somatosensory

information may improve the stability of verticality representation (even in normal subjects). Somatosensory signal may also represent a rescue graviceptive signal in case of vestibular loss. When somaesthetic and vestibular graviceptive signals are not congruent (tilted posture), the brain synthesizes somaesthetic and vestibular graviceptive signals to reach a precise and stable representation of verticality that is a compromise between directions given by allocentric and egocentric coordinate systems. Our study shows that this compromise is influenced by the degree of availability of the somatosensory information, which demonstrates the validity of Mittelstaedt's model (1983) for explaining the Aubert effect. However, we did not find the distinction made by Mittelstaedt (1992) regarding the nature of the somaesthetic graviception from the upper and lower parts of the trunk.

Funding

La Fondation de l'Avenir; programs ETO-300 and ETA-382 (to D.P.).

References

American Spinal Injury Association. International Standards for Neurological Classifications of Spinal Cord Injury. Revised edn. Chicago, IL: American Spinal Injury Association; 2000. p. 1–23.

Anastasopoulos D, Bronstein AM. A case of thalamic syndrome: somatosensory influences on visual orientation. J Neurol Neurosurg Psychiatry 1999; 67: 390–4.

Anastasopoulos D, Bronstein A, Haslwanter T, Fetter M, Dichgans J. The role of somatosensory input for the perception of verticality. Ann NY Acad Sci 1999; 871: 379–83.

Aubert H. Eine scheinbare bedeutende Drehung von Objekten bei Neigung des Kopfes nach rechts oder links. Virchows Arch 1861; 20: 381–93.

Baier B, Stoeter P, Dieterich M. Anatomical correlates of ocular motor deficits in cerebellar lesions. Brain 2009; 132: 2114–24.

Barbieri G, Gissot AS, Fouque F, Casillas JM, Pozzo T, Pérennou D. Does proprioception contribute to the sense of verticality? Exp Brain Res 2008; 185: 545–52.

Barbieri G, Gissot AS, Pérennou D. Ageing of the postural vertical. Age 2010; 32: 51–60.

Barra J, Benaim C, Chauvineau V, Ohlmann T, Gresty M, Pérennou D. Are rotations in perceived visual vertical and body axis after strsoke caused by the same mechanism? Stroke 2008; 39: 3099–101.

Behrens T, Johansen-Berg H, Woolrich M, Smith S, Wheeler-Kingshott AM, Boulby P, et al. Non-invasive mapping of connections between human thalamus and cortex using diffusion imaging. Nat Neurosci 2003; 6: 750–7.

Bell C, Bodznick D, Montgomer J, Bastian J. The generation and subtraction of sensory expectations within cerebellum-like structures. Brain Behav Evol 1997; 50: 17–31.

Bisdorff A, Wolsley C, Anastasopoulos D, Bronstein A, Gresty MA. The Perception of body verticality (subjective postural vertical) in peripheral and central vestibular disorders. Brain 1996; 119: 1523–34.

Brandt T, Dieterich M. The vestibular cortex. Its locations, functions, and disorders. Ann NY Acad Sci 1999; 871: 293–312.

Brandt T, Dieterich M, Danek A. Vestibular cortex lesions affect the perception of verticality. Ann Neurol 1994; 35: 403–12.

Bronstein AM, Pérennou DA, Guerraz M, Playford D, Rudge P. Dissociation of visual and haptic vertical in two patients with vestibular nuclear lesions. Neurology 2003; 61: 1260–2.

Buttner U, Lang W. The vestibulocortical pathway: neurophysiological and anatomical studies in the monkey. Prog Brain Res 1979; 50: 581–8.

Day RH, Wade NJ. Mechanisms involved in visual orientation constancy. Psychol Bull 1969; 71: 33–42.

Dichgans J, Held R, Young LR, Brandt T. Moving visual scenes influence the apparent direction of gravity. Science 1972; 178: 1217–9.

Dieterich M, Bartenstein P, Spiegel S, Bense S, Schwaiger M, Brandt T. Thalamic infarctions cause side-specific suppression of vestibular cortex activations. Brain 2005; 128: 2052–67.

Dieterich M, Brandt T. Ocular torsion and tilt of subjective visual vertical are sensitive brainstem signs. Ann Neurol 1993a; 33: 292–9.

Dieterich M, Brandt T. Thalamic infarctions: differential effects on vestibular function in the roll plane (35 patients). Neurology 1993b; 43: 1732–40.

Dietz V, Gollhofer A, Kleiber M, Trippel M. Regulation of bipedal stance: dependency on "load" receptors. Exp Brain Res 1992; 89: 229–31.

Glasauer S. Interaction of semicircular canals and otoliths in the processing structure of the subjective zenith. Annl NY Acad Sci 1992; 656: 847–84.

Gresty MA, Bronstein AM, Brandt T, Dieterich M. Neurology of otolith function peripheral and central disorders. Brain 1992; 115: 647–73.

Hain TC, Spindler J. Head-shaking nystagmus. In: Sharpe JA, Barber HO, editors. The vestibulo-ocular reflex and vertigo. New York: Raven Press; 1993. p. 217–28.

Karnath HO, Berger MF, Küker W, Rorden C. The anatomy of spatial neglect based on voxelwise statistical analysis: a study of 140 patients. Cerebral Cortex 2004; 14: 1164–72.

Karnath HO, Johannsen L, Broetz D, Küker W. Posterior thalamic hemorrhage induces "pusher syndrome". Neurology 2005; 64: 1014–9.

Kerkhoff G, Zoelch C. Disorders of visuospatial orientation in the frontal plane in patients with visual neglect following right or left parietal lesions. Exp Brain Res 1998; 122: 108–20.

Lewald J, Ehrenstein W. Influence of head-to-trunk position on sound lateralization. Exp Brain Res 1998; 121: 230–8.

Lorincz EN, Hess BJ. Dynamic effects on the subjective visual vertical after roll rotation. J Neurophysiol 2008; 100: 657–69.

Masdeu JC, Gorelick PB. Thalamic astasia: inability to stand after unilateral thalamic lesions. Ann Neurol 1988; 23: 596–603.

Massion J. Movement, posture and equilibrium: interaction and coordination. Prog Neurobiol 1992; 38: 35–56.

Mazibrada G, Tariq S, Pérennou D, Gresty M, Greenwood R, Bronstein AM. The peripheral nervous system and the perception of verticality. Gait Posture 2008; 27: 202–8.

Merfeld DM, Zupan L, Peterka RJ. Humans use internal models to estimate gravity and linear acceleration. Nature 1999; 15: 615–8.

Mittelstaedt H. A new solution to the problem of the subjective vertical. Naturwissenschaften 1983; 70: 272–81.

Mittelstaedt H. Somatic versus vestibular gravity reception in man. Ann NY Acad Sci 1992; 656: 124–39.

Parker DE, Poston RL, Gulledge WL. Spatial orientation: visual-vestibular-somatic interaction. Percept Psychophys 1983; 33: 139–46.

Pérennou DA, Amblard B, Laassel el M, Benaim C, Herisson C, Pelissier J. Understanding the pusher behavior of some stroke patients with spatial deficits: a pilot study. Arch Phys Med Rehabil 2002; 83: 570–5.

Pérennou DA, Amblard B, Leblond C, Pélissier J. Biased postural vertical in humans with hemispheric cerebral lesions. Neurosci Lett 1998; 252: 75–8.

Pérennou DA, Mazibrada G, Chauvineau V, Greenwood R, Rothwell J, Gresty MA, et al. Lateropulsion, pushing and verticality perception in hemisphere stroke: a causal relationship? Brain 2008; 131: 2401–13.

Rorden C, Brett M. Stereotaxic display of brain lesions. Behav Neurol 2000; 12: 191–200.

Semmes J, Weinstein S, Ghent L, Teuber H-L. Somatosensory changes after penetrating brain wounds in man. Cambridge: Harvard University; 1960.

Talairach J, Tournoux P. Co-planar stereotaxic atlas of the human brain 3-dimensional proportional system: an approach to cerebral imaging. Stuttgart: Thieme; 1988.

Tarnutzer AA, Bockisch C, Straumann D, Olasagasti I. Gravity dependence of subjective visual vertical variability. J Neurophysiol 2009; 102: 1657–71.

Trousselard M, Barraud PA, Nougier V, Raphel C, Cian C. Contribution of tactile and interoceptive cues to the perception of the direction of gravity. Brain Res Cogn Brain Res 2004; 20: 355–62.

Trousselard M, Cian C, Nougier V, Pla S, Raphel C. Contribution of somesthetic cues to the perception of body orientation and subjective visual vertical. Percept Psychophys 2003; 65: 1179–87.

Tseng HZ, Chao WY. Head-shaking nystagmus: a sensitive indicator of vestibular dysfunction. Clin Otolaryngol Allied Sci 1997; 22: 549–52.

Van Beuzekom AD, Van Gisbergen JA. Properties of the internal representation of gravity inferred from spatial-direction and body-tilt estimates. J Neurophysiol 2000; 84: 11–27.

Wolpert D, Ghahramani Z, Jordan M. An internal model for sensorimotor integration. Science 1995; 269: 1880–2.

Yardley L. Contribution of somatosensory information to perception of the visual vertical with body tilt and rotating visual field. Percept Psychophys 1990; 48: 131–4.

Yasui S, Young L. Perceived visual motion as effective stimulus to pursuit eye movement system. Science 1975; 190: 906–8.

Wade NJ. Effect of forward head inclination on visual orientation during lateral body tilt. J Exp Psychol 1972; 96: 203–5.

Wade NJ. The effect of water immersion on the perception of the visual vertical. Br J Psychol 1973; 64: 351–61.

Witkin HA, Ash SE. Studies in space orientation III. J Exp Psychol 1948; 38: 603–14.

Yelnik AP, Lebreton FO, Bonan IV, Colle FMC, Meurin FA, Guichard JP, et al. Perception of verticality after recent cerebral hemispheric stroke. Stroke 2002; 33: 2247–53.

Zhang D, Snyder AZ, Shimony JS, Fox MD, Raichle ME. Noninvasive functional and structural connectivity mapping of the human thalamocortical system. Cereb Cortex 2010; 20: 1187–94.

Zupan LH, Merfeld DM. Neural processing of gravito-inertial cues in humans. IV. Influence of visual rotational cues during roll optokinetic stimuli. J Neurophysiol 2003; 89: 390–400.