Perception of Verticality After Recent Cerebral Hemispheric Stroke

Alain P. Yelnik, MD; Frederique O. Lebreton, MD; Isabelle V. Bonan, MD; Florence M.C. Colle, MD; Francesca A. Meurin, MD; Jean Pierre Guichard, MD; Eric Vicaut, MD

Background and Purpose—Perception of the subjective visual vertical (SVV) is affected by cerebral hemispheric lesions. Knowledge of this disturbance is of interest for the study of its possible relation to balance disturbances. There is still uncertainty about the possible effects of a visual field defect and of the side and site of the lesion. This study was conducted to assess SVV with the head upright or tilted and to explore its relation to a visual field defect, visuospatial neglect, and the site of lesion.

Methods—Forty patients with hemiplegia after a recent hemispheric stroke (20 with left and 20 with right stroke) were studied. The site of the lesion was determined on CT scan, with special attention focused on the vestibular cortex. A neurological examination with determination of the visual field and visual neglect was conducted before SVV was tested. Subjects sat in a dark room and adjusted a luminous rod to the vertical position. Measures were repeated with binocular and monocular vision and with the head upright or tilted to the right or left.

Results—SVV was abnormally deviated in 23 of 40 patients (57%). The deviation was significantly greater among patients with a right or left hemispheric lesion than among healthy controls (±2.2° and 1.5° versus 0.2°); the same applied to the range of uncertainty (7.6° and 4.7° versus 1.9°). SVV deviation was not significantly related to the location of the lesion but was closely related to visuospatial neglect. The “E” effect observed in controls with the head tilted, ie, an SVV shift in the direction opposite to the head tilt, was not observed in hemiplegic patients with the head tilted toward the nonparetic side.

Conclusions—Recent hemispheric stroke affects SVV perception, which is closely correlated to visuospatial neglect. It is suggested that the E effect might be mediated by the stretching of the somatosensory structure of the neck. (Stroke. 2002;33:2247-2253.)

Key Words: equilibrium ■ hemiplegia ■ stroke ■ visual verticality

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erurbed perception of the subjective visual vertical (SVV) may be one of the components underlying the difficulties hemiplegic patients experience in keeping their balance after a recent stroke. The present study constitutes the first stage of an investigation in which we sought to establish whether early clinical findings about SVV perception are predictive of balance difficulties in hemiplegic patients and can serve as a guide to setting up a rehabilitation program. Vestibular, visual, and somatosensory information all contribute to this perception. Assessment of the verticality of the environment in the upright position needs mainly vestibular and visual inputs. Somatosensory information is required when the head or the whole body is tilted. Because such tilting can make a vertical line look as if it were tilted to the opposite side, a mobile rod set in the vertical position shifts in the same direction as the head or body tilt. This is termed the “A” effect and was described by Aubert in 1861. When the tilt is moderate or restricted to the head, the apparent vertical often shifts in the direction opposite to the tilt. This is termed the “E” effect, described by Muller in 1916. It is of interest to assess SVV with the head tilted in hemiplegic patients to improve understanding of the mechanisms of this perception. Few studies report such assessment, and isolated loss of sensory perception does not seem to alter SVV in the upright position but does alter the A effect.

Peripheral pathological information, especially vestibular information, leads to disturbances in SVV perception. To our knowledge, no SVV disturbance due to peripheral vision deficiency has been reported. A central lesion of the sensory pathways also leads to SVV disturbances, but previous results are sometimes inconsistent, and the mechanisms underlying the observed perturbations of the SVV remain unclear. Contralateral tilt of the SVV after hemispheric lesions was reported long ago, especially after right hemispheric lesions, before precise localization of the lesion became possible. Since then, brain stem lesions have been demon-
TABLE 1. Patients’ Characteristics

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>No. of Patients</th>
<th>Age, y</th>
<th>Sex, M/F</th>
<th>Time Since Stroke, wk (Range)</th>
<th>Etiology, Infarction/Hemorrhage</th>
<th>Neglect</th>
<th>VFD</th>
<th>PASS</th>
<th>FIM</th>
<th>Motricity</th>
</tr>
</thead>
<tbody>
<tr>
<td>RHL</td>
<td>20</td>
<td>55.7±9.7</td>
<td>14/6</td>
<td>5.4±2.9 (2–12)</td>
<td>14/6</td>
<td>13</td>
<td>6</td>
<td>22.1±7.8</td>
<td>87.5±17.3</td>
<td>87.4/200±58.6</td>
</tr>
<tr>
<td>LHL</td>
<td>20</td>
<td>58.3±13.4</td>
<td>11/9</td>
<td>6.1±2.3 (3–12)</td>
<td>15/5</td>
<td>3</td>
<td>3</td>
<td>28.9±12.1</td>
<td>97.3±15.8</td>
<td>77.2/200±53.5</td>
</tr>
</tbody>
</table>

VFD indicates visual field defect; PASS, Postural Assessment Scale for Stroke; and FIM, Functional Independence Measure.

strated to induce an SVV deviation toward the side of the lesion or, more precisely, contralateral to the lesion when it is rostral to the upper pons and ipsilateral when it is caudal. This effect is due to vestibular dysfunction. A contralateral tilt of the SVV has also been attributed to vestibular pathway lesion caused by thalamic infarction, especially paramedian infarction, or a lesion involving what is known as the parietoinsular vestibular cortex and the superior temporal gyrus. Sensory perturbation can also induce tilt of the SVV after thalamic infarction or after a lesion involving the sensory cortex. The presence of a visual field defect after right cerebral lesions would at least lead to a tilt of the SVV, but, according to Kerkhoff and Zoelch, this tilt is strongly correlated to visuospatial neglect and not to a visual field defect, whichever the side of the lesion.

Thus, different hemispheric lesion sites and different mechanisms can induce abnormalities of the verticality setting: these include thalamic lesions because of vestibular or sensory perturbations, cortical lesions involving either the vestibular cortex (whichever the side of the lesion) or the sensory cortex, and right posterior lesions leading to perturbed visuospatial analysis. However, opinions in this regard are still somewhat unclear because lesions could not be precisely located before the advent of the CT scanner and also because of the heterogeneous nature of the populations studied. Most authors have included stroke victims and a few examined patients with brain injury, or associated lesions. The time elapsing since the lesion is probably an important factor to take into account because of the spontaneous symptom regression shown after thalamic infarction. In previous series, the time since lesion varied, sometimes from months to years, even in the same study, but mostly this was not reported. Clinical data concerning the extent of the hemiplegia and the existence of a visual field defect or visual neglect are often also lacking or are not studied.

In the present investigation we included patients usually recruited in a physical and medicine rehabilitation department, ie, those disabled by motor and balance impairment. The time since stroke was not longer than 3 months (13 weeks). Patients were excluded if they had perturbed vigilance, a history of neurological disturbances, vertigo or vestibular dysfunction, amблиopia or diplopia, or severe aphasia. Before testing, we performed a complete neurological examination in which the following were evaluated: the Collins and Wade scale visual field, assessed at the bedside and confirmed by Goldmann camptometry when hemianopia was suspected; visuospatial neglect, using Albert’s test and Bergego’s scale, a test assessing daily behavior during meals, washing, and ambulation; balance, as estimated by the Postural Assessment Scale for Stroke; and FIM, Functional Independence Measure.

Forty consecutive patients were studied (Table 1), 20 with a right hemisphere lesion (RHL) and 20 with a left hemisphere lesion (LHL). The stroke was hemorrhagic in 11 patients and ischemic in 29. Among the ischemic lesions, posterior cerebral artery territory was never involved. Anterior cerebral artery territory was affected in 1 case, anterior choroidal artery territory in 7 cases (it was the only territory affected in 3), and middle cerebral artery territory in 28. Age, sex, etiology, and time since stroke did not differ in RHL and LHL patients. All patients suffered from hemiplegia. A visual field defect was observed in 6 RHL and 3 LHL patients. Visuospatial neglect, considered to be present when Albert’s test was different from zero or Bergego’s score exceeded 2, was noted in 13 RHL and 3 LHL patients. Visuospatial neglect and a visual field defect were associated in all 6 RHL patients and 2 of the 3 LHL patients.

The control group was composed of 20 healthy subjects, 9 men and 11 women, with a mean age of 42.9±5.8 years.

Procedure

The experiment was performed in a dark room with the subjects sitting in a wheelchair or normal chair. A 30-cm-long luminous rod was placed directly in front of each one at a distance of 2 meters. Subjects were asked to adjust the rod to the vertical position by manipulating a box held in the healthy hand, with 2 switches easily handled by the thumb; there was no time limit. As in previous studies, SVV was assessed with binocular and monocular vision. The head was not fixed but freely held upright. The manipulation of vestibular and somatosensory input by tilting the head affects the SVV in a way that, as already explained, depends on the angle of the tilt. We therefore assessed SVV with the head tilted to observe the possible effects of this sensory stimulation. The investigator stood behind the patient, with 1 hand on the patient’s head, to help her or him to sustain a tilt of approximately 20°. Two series of measurements were made in all cases: for the first, the rod was initially tilted 60° to the left of the vertical objective, and the patient had to adjust it with a clockwise movement; for the second, the rod was initially tilted 60° to the right of the vertical objective for a counterclockwise adjustment. The measures were then repeated with the head in the upright, right-tilted, and left-tilted positions, and after each positioning, the observer read the deviation in degrees (clockwise deviation, +; counterclockwise deviation, –). The relatively few trials conducted were chosen to avoid distortion of the results due to fatigue, since patients were deliberately examined in a subacute stage after stroke. The following data were analyzed: mean SVV (V value) for the clockwise and counterclockwise measurements in each position, and the range of uncertainty (U value), defined as the maximum difference between the clockwise and counterclockwise adjustments. Because the binocular and monocular V and U values with the head held upright were not different for controls and hemiplegic patients, the data were combined and
recorded as $V_{\text{upright}}$ and $U_{\text{upright}}$. With the head tilted, SVV was only measured with binocular vision. Therefore, $V_{\text{upright}}$ and $U_{\text{upright}}$ refer to upright head data, $V_{\text{righttilt}}$ and $U_{\text{righttilt}}$ refer to rightward-tilted head data, and $V_{\text{lefttilt}}$ and $U_{\text{lefttilt}}$ refer to leftward-tilted head data.

Identification of Site of Lesion

The site of the lesion was determined on CT scans to explore possible correlations between the abnormalities recorded and particular lesion sites. CT scans were done with a Picker unit, with a slice thickness of 4 mm up to the midbrain and 8 mm more rostrally. The stereotaxic baseline was used according to the Duvernoy atlas.23 If the imaging plane was not parallel to the anterior/posterior commissure plane, we attempted to optimize the localization of the projected lesion. Anatomic lesions were noted, with particular attention focused on the recognized regions of interest in the vestibular cortex. These regions are the parietoinsular vestibular cortex and areas 2v, 3a, and 7, as described by Brandt et al.16 The parietoinsular vestibular cortex lies deep in the posterior part of the insula, area 2v is located at the tip of the interparietal sulcus, area 3a at the anterior bank of the central sulcus, and area 7 at the inferior parietal cortex. The main cerebral areas involved are given in Table 2.

Statistical Analysis

Since the distribution of the variables studied was nongaussian, data were summarized by median, interquartile distance (IQD), and extreme values. Comparisons were made by distribution-free tests, ie, the Mann-Whitney and Wilcoxon tests for unpaired and paired groups, respectively. All tests were 2-sided; significance level was adjusted with the use of the Bonferroni procedure according to the number of comparisons made for each hypothesis tested. Thus, because 4 comparisons were made to analyze the possible changes of deviation or uncertainty from the upright position, the significance level was fixed at $P=0.0125$ for these types of comparisons. Because 3 comparisons between groups were made for each position, the significance level was fixed at $P=0.0166$ for these types of comparisons. All tests were done with the use of StatXact from Cytel Software.

Results

Controls

For the controls, no significant difference was observed between binocular, right eye, and left eye vision (0.2° [IQD 1.1°], 0.2° [IQD 0.8°], and 0° [IQD 0.9°], respectively). The median tilt of the SVV with the head upright ($V_{\text{upright}}$) was 0.35° (Table 3). Maximal individual deviations were 2.9° on the left and 2.2° on the right. Consequently, the results for each patient were considered different from control values when the deviation exceeded 3° for $V_{\text{upright}}$, which was clearly larger than the deviation for the controls, and similar to the normal range previously reported.14 The median range of uncertainty for $U_{\text{upright}}$ was 1.9° (IQD 1.3°), and the maximum intrindividual difference was 6°. Therefore, for $U_{\text{upright}}$, 8° was considered significantly abnormal. With the head tilted, the E effect was observed: thus, the SVV setting was significantly tilted to the opposite side, and $V_{\text{righttilt}}$ and $V_{\text{lefttilt}}$ were −2° and 1.2°, respectively. Differences between $V_{\text{righttilt}}$ and $V_{\text{upright}}$ reached the adjusted significance level ($P=0.0048$) but not those between $V_{\text{lefttilt}}$ and $V_{\text{upright}}$ ($P=0.023$). The ranges of uncertainty for $U_{\text{righttilt}}$ and $U_{\text{lefttilt}}$ did not differ from $U_{\text{upright}}$.

Patients

As in the controls, no significant difference was observed between binocular or monocular vision (Table 3).

Right Hemispheric Lesions

Values outside the normal range were recorded for 13 of 20 RHL patients (65%); this was the case for both $V_{\text{upright}}$ and $U_{\text{upright}}$ in 5 patients, for $V_{\text{upright}}$ in 3, and for $U_{\text{upright}}$ in 5. SVV was significantly tilted toward the side contralateral to the lesion ($P<0.007$ for $V_{\text{upright}}$). Of greater interest was the range of uncertainty, which was dramatically higher in patients than controls ($P<0.001$ for $U_{\text{upright}}$ and $P<0.001$ for $U_{\text{lefttilt}}$). Seven patients were very disturbed by the initial position of the rod and were unable to adjust it to the vertical: thus, $V_{\text{upright}}$ reached 47.5° for 1 of them and 44°, 25.4°, 22.5°, 13°, 12.7°, and 9.7° for the 6 others. As in the controls, the E effect was observed when the head was tilted toward the side opposite to the lesion, ie, for right-side lesions, the leftward tilt of the head tended to compensate for the leftward shift of the SVV: $V_{\text{lefttilt}}$ differed significantly from $V_{\text{upright}}$ ($P<0.003$). There was no difference between the ranges of uncertainty $U_{\text{lefttilt}}$ and $U_{\text{upright}}$. But with the head tilted rightward, no change in $V_{\text{righttilt}}$ or $V_{\text{righttilt}}$ was observed in relation to the upright position, and there was apparently no E effect.

Left Hemispheric Lesions

Values outside the normal range were recorded for 10 of 20 LHL patients (50%); this was the case for both $V_{\text{upright}}$ and $U_{\text{upright}}$ in 2 patients, for $V_{\text{upright}}$ in 6, and for $U_{\text{upright}}$ in 2. SVV was significantly tilted toward the side contralateral to the lesion ($P=0.001$ for $V_{\text{upright}}$). The range of uncertainty was wider than for the controls, for $U_{\text{upright}}$ ($P<0.001$) and $U_{\text{righttilt}}$ ($P=0.002$). Head tilts tended to produce the same effect in LHL as RHL patients; as in the controls, the E effect was observed when the head was tilted toward the side opposite to the lesion, ie, for left-side lesions, the rightward tilt of the head tended to compensate for the rightward shift of the SVV: $V_{\text{righttilt}}$ differed, but not significantly, from $V_{\text{upright}}$. There was no difference between the ranges of uncertainty $U_{\text{righttilt}}$ and $U_{\text{upright}}$. But with the head tilted leftward, no change in $V_{\text{lefttilt}}$ or $U_{\text{lefttilt}}$ was observed in relation to the upright position, and again the E effect was apparently absent.

Differences Between Right and Left Hemispheric Lesions

The apparent difference between $V_{\text{upright}}$ values in the RHL and LHL groups (SVV was tilted to the left for RHL patients and to the right for LHL patients) was not a real difference when expressed in absolute values. The range of uncertainty, $U_{\text{lefttilt}}$, was wider in RHL than LHL patients (median, 7.6° versus 4.7°), but the difference did not reach significance. $U_{\text{righttilt}}$ was >8° for 10 RHL patients (mean, 20.3°) but for only 4 LHL patients (mean, 16.9°). $U_{\text{upright}}$ was >20° for 4 RHL patients and 1 LHL patient and >10° for 7 RHL and 3 LHL patients.

Role of Visual Field Defect

Statistical analysis showed that a visual field defect had no effect on $V_{\text{upright}}$ or $U_{\text{upright}}$, irrespective of the side of the lesion. Nevertheless, among RHL patients, $V_{\text{upright}}$ tended to be greater when visual field defect was present, and $U_{\text{upright}}$ was >8 for 5 of the 6 patients with visual field defect compared with 5 of the 14 patients without visual field defect. Note that all the RHL patients did not differ from UHL in RHL and LHL patients (SVV was tilted to the left for RHL patients and to the right for LHL patients) was not a real difference when expressed in absolute values. The range of uncertainty, $U_{\text{lefttilt}}$, was wider in RHL than LHL patients (median, 7.6° versus 4.7°), but the difference did not reach significance. $U_{\text{righttilt}}$ was >8° for 10 RHL patients (mean, 20.3°) but for only 4 LHL patients (mean, 16.9°). $U_{\text{upright}}$ was >20° for 4 RHL patients and 1 LHL patient and >10° for 7 RHL and 3 LHL patients.

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patients with visual field defect and all but 1 of the LHL patients with visual field defect also had visuospatial neglect.

**Role of Visuospatial Neglect**
When all 40 patients were considered together, visuospatial neglect was strongly correlated with low $V_{\text{upright}}$ and $U_{\text{upright}}$ values ($P=0.002$ and $P=0.003$, respectively). The effect of visuospatial neglect seemed to be the same whichever the side of the lesion, but no statistical analysis could be conducted separately for LHL patients with visuospatial neglect because they were so few. None of the 16 patients with visuospatial neglect had a normal SVV setting. Two
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TABLE 3. SVV (V) and Range of Uncertainty (U), in Degrees, for Healthy Controls and Patients With Recent RHL or LHL

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>RHL</th>
<th>LHL</th>
</tr>
</thead>
<tbody>
<tr>
<td>V_uPr</td>
<td>0.35 (0.7)</td>
<td>-2.3 (6.6)</td>
<td>1.6 (3.2)</td>
</tr>
<tr>
<td>U_uPr</td>
<td>1.9 (1.3)</td>
<td>7.6 (8.8)</td>
<td>4.7 (3.4)</td>
</tr>
<tr>
<td>V_right</td>
<td>-2 (2.5)*</td>
<td>-2.7 (7)</td>
<td>-2.3 (8.5)</td>
</tr>
<tr>
<td>V_left</td>
<td>-2.6 (2.2)</td>
<td>5.8 (8.1)</td>
<td>4.7 (7.8)*</td>
</tr>
<tr>
<td>U_right</td>
<td>2.6 (2.2)</td>
<td>15 (15.8)*</td>
<td>0.9 (4.4)</td>
</tr>
<tr>
<td>U_left</td>
<td>1.2 (2.7)</td>
<td>6.7 (12.7)*</td>
<td>2.0 (4.4)</td>
</tr>
<tr>
<td>Median (QD)</td>
<td>1 to 0.9</td>
<td>-13.1 to 3.2</td>
<td>-3.6 to 9.3</td>
</tr>
<tr>
<td>Range</td>
<td>0.7 to 6.0</td>
<td>2.6 to 47.5</td>
<td>0.1 to 24.9</td>
</tr>
</tbody>
</table>

*Significantly different from V_uPr (significance level adjusted for multiplicity).
†Significantly different from controls (significance level adjusted for multiplicity).

RHL and 4 LHL patients without visuospatial neglect exhibited a tilt of the SVV, but it was always mild, as V_uPr was <7° and U_uPr was <16°.

Role of Site of Lesion

The frontotemporal lobule was affected by the stroke in 12 patients; the lesion was localized in the frontal areas alone in only 2 of these patients, who did not exhibit an abnormal SVV setting; the remaining 10 had large lesions of the middle cerebral artery, and only 2 of them had normal data. The thalamus was involved in only 2 patients, 1 of them with perturbed SVV, especially for the range of uncertainty (44°) and the values for both the leftward- and rightward-tilted head positions (V_right tilt = -8.3°, V_left tilt = +16.9°). Lenticular nuclei were affected by the lesion in 24 patients; 7 of them had an isolated lenticular lesion, 2 had abnormal SVV with the head upright and tilted, and 2 others only had abnormal SVV with the head tilted. The internal capsule was affected in 18 patients, 5 of whom had an isolated lesion and 2 of whom had slightly perturbed SVV. The occipital cortex was never affected. The vestibular cortex was affected in 12 patients, who included 7 RHL and 5 LHL patients. None of the lesions was limited to the vestibular cortex. The deviation of the SVV was not significantly different when the vestibular cortex was involved and when it was not (V_uPr = 3.0° and 2.3°, respectively), and the same applied to the range of uncertainty (U_uPr = 8.3° and 4.7°, respectively). This was because strong perturbations were also observed in some patients with lesions at other sites. In fact, the vestibular cortex was only involved in 6 of the 11 patients with strong perturbations for the range of uncertainty (U > 10°). The other lesion sites were the lenticular nuclei in 3 patients and the thalamus and internal capsule in 1 patient each.

Discussion

The aim of this study was to assess perception of the SVV among recent hemispheric stroke patients and its relation to a visual field defect, visual neglect, and the site of the lesion. Knowledge of the visual perception of verticality early after stroke might be useful for establishing a balance rehabilitation program. It seems that there is not a direct relation between disturbed SVV and disturbed body posture, as shown in acute peripheral vestibular disorders and in hemiplegic patients with contraversive pushing. Nevertheless, a correlation between SVV and functional activities and walk cannot be ruled out and might have some bearing on balance rehabilitation programs. This is our general line of research, and the present study is the first stage of our investigations on this point. Twenty healthy control subjects and 40 patients, 20 with RHL and 20 with LHL, were studied. Although the controls were younger than the patients, it was useful to have control data obtained with the same test protocol.

Our protocol was a regularly used procedure, with patients sitting in a dark room and adjusting a luminous rod to the vertical position. Since deviation of the SVV and of the visual subjective horizontal are strongly correlated, only SVV was measured. As in previous studies, SVV deviation was assessed in degrees, but in addition we included an original data set called the range of uncertainty, defined as the maximum difference between the SVVs observed during clockwise and counterclockwise adjustment. The amplitude of the uncertainty is indicative of the difficulty some patients experience in assessing the visual vertical. As in previous studies, our patients adjusted the position of the luminous rod themselves. Therefore, the task they performed was a sensorimotor task, which differed from the tasks in other studies in which the investigator adjusted the position of the rod, making the task purely perceptive. Although in this study the task was easy and none of the patients seemed unable to do it, its sensorimotor nature may have affected the results and must be taken into account.

Among studies of the perception of verticality by hemiplegic patients, only the more recent ones specifically concerned poststroke patients. Time since stroke (or another lesion) varied greatly: it was sometimes not stated, ranged from days to years, or ranged from 3 to 15 months in the same study. However, at least after thalamic lesions, SVV perturbations regress spontaneously in a few months. We therefore focused our study on a specifically homogeneous population of recent stroke patients. Mean time since stroke was 6 weeks and ranged from 2 to 13 weeks, which is representative of hemiplegic patients undergoing care in a physical medicine and rehabilitation department. However, this also limited the scope of the study, although both controls...
and patients were subjected to the same procedure because only 1 series of recordings could be made for each test to avoid fatigue-related impairment of performance among patients.

The perception of the vertical, whether with monocular or binocular vision, was not different for controls and patients, unlike the results observed among patients with brain stem lesions for whom SVV deviation was shown to be greater for the eye on the side of the lesion.2 In these patients, the difference in SVV between the 2 eyes may be due to the ocular torsion and skew deviation often observed in brain stem lesions but not in hemispheric lesions.

We therefore analyzed 2 main sets of data: perception of SVV with the head in the upright position and the range of uncertainty for all data recorded in that position. The main findings were that, in accordance with the literature, the setting of the SVV among recent hemispheric stroke patients deviates significantly toward the side opposite to the lesion, that this deviation is closely correlated to the presence of visuospatial neglect, and that the E effect was not observed when the head was tilted toward the nonparetic side.

In healthy subjects, tilting the body or the head to 1 side causes an illuminated vertical line to appear to deviate toward the opposite side (the A effect).6–8 This effect seems to be mediated mainly by sensory information because it is absent after complete proprioceptive sensory loss below the neck due to polyneuropathy5 and is absent in patients with central hemianesthesia when they lie on the hypoesthetic side,3,4 even though vestibular signals help to counteract the disorientating stimuli conveyed by the visual or proprioceptive system.26 When the tilt is moderate, the opposite effect is observed, and a vertical line appears to deviate toward the same side as the body or head tilt. In that case, when the SVV is set in the dark with a luminous rod, it deviates toward the side opposite the tilt. This is the E effect,8,9 which was observed in our control subjects with a 20° tilt of the head. In the hemiplegic patients, this effect was only observed when the head was tilted toward the side contralateral to the lesion, ie, toward the hemiplegic side. When it was tilted toward the ipsilateral side, ie, the nonhemiplegic side, no E effect was observed. This meant that when RHL patients tilted their head to the left, the SVV setting tilted to the right, but when they tilted their head to the right, the tilt of this setting did not differ from the tilt observed with the head upright. If somatosensory information is essential to the perception of verticality during body or head tilt, as suggested by previous studies,3–5 our results suggest that the E effect might be mediated by the stretching of the somatosensory receptors of the neck. These findings can be considered together with those of Perennou et al,27 who showed that the postural bias observed in hemiplegic patients with spatial neglect can be improved by applying transcutaneous electric nerve stimulation to the neck on the side contralateral to the lesion.

No relationship between SVV tilt and the existence of a visual field defect was observed in our study, contrary to previously reported results.12 However, the number of patients with such a defect was too small to allow separate analyses for right and left hemiplegic patients. The existence of such a relationship is in fact questionable, in the light of the work of Kerkhoff and Zoelch,17 who were the first to report impairment of spatial orientation in the frontal plane in patients who had visual neglect but no visual field defect, after a lesion of the parietal lobe, irrespective of the side of the lesion. Previous authors who stressed the relationship between RHL and perturbation of the SVV setting11–13 did not test for visuospatial neglect, which is known to be more frequent after RHL than LHL.28 This correlation between the presence of visuospatial neglect and a perturbed SVV setting is strong. In the series of Kerkhoff and Zoelch,17 only 1 of the 13 RHL patients with visual neglect, and none of the 3 LHL patients, had a normal SVV setting. Moreover, no tilt of the SVV was observed in patients without visual neglect. In our study none of the patients with visuospatial neglect had a normal SVV setting. Some of those without visual neglect exhibited a tilt of the SVV, but with mild perturbations. Analysis of the range of uncertainty Uupright is also of interest because it is indicative of a patient’s hesitation. Some of our patients were unable to assess the visual vertical. Patients experiencing such difficulty generally have visual neglect, and this was the case for 8 of the 10 RHL patients and both the LHL patients with Uupright > 8°. This wider variability in RHL patients than in controls and LHL patients was previously observed by others.41 Thus, visuospatial neglect is strongly associated with marked impairment of SVV perception.

How visual neglect leads to the perturbation of spatial orientation in the roll plane is unclear. Kerkhoff and Zoelch17 proposed 2 scenarios: (1) either a focal lesion of anatomically very close or overlapping regions in the inferior parietal lobule and parietoinsular vestibular cortex or (2) a network of several corticovestibular areas involved in the perception of axis orientation in the roll plane. The second possibility is interesting because vestibular stimulations are known to reduce visuospatial neglect29 or postural asymmetry in left hemiplegic patients.30 In our study, as in previous ones, patients without anesthesia sitting in a chair were unable to correct the erroneous visual feedback given by the tilted luminous rod, even though their sensory inputs were good or adequate and vestibular pathways had sustained no direct lesion. These are additional arguments for the existence of a strong functional link between the vestibular, sensory, and visual inputs involved in the construction of the gravitational referential. The role of the polymodal sensory cortex, especially the temporoparietal junction involved in balance control,31 is probably crucial. This junction, which has not yet been defined clearly, occupies almost the same territory as the vestibular cortex described by Brandt et al.16 These authors stressed the role of the vestibular cortex in the perception of verticality. This role was not clearly apparent in our study, but this is probably not meaningful because our patients had large lesions, and the vestibular cortex was almost never specifically involved. Our population was representative of patients regularly treated in a physical medicine and rehabilitation department for severe deficits and large lesions, whereas those studied by Brandt et al had very mild deficits. This is perhaps why these authors did not mention any perturbation of the SVV in patients with visuospatial disturbance.
Because the SVV is based on vestibular, somatosensory, and visual inputs, it is quite normal to observe that different kinds of lesions can lead to perturbed orientation in the roll plane. These include lesions involving the central vestibular pathways (brain stem, thalamus, or cortex) on either side,14–16 lesions involving sensory pathways,3,4 and lastly, lesions involving regions involved in visuospatial disturbances such as parietal lesions, and more specifically right parietal lesions,17 as observed here.

References
Perception of Verticality After Recent Cerebral Hemispheric Stroke
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