ABSTRACT. The purpose of this study was to investigate the effects of caloric vestibular stimulation on the postural sway characteristics of hemiparetic patients. Two groups of 15 hemiparetic patients each (right and left) were compared to a group of 15 control subjects. Hemiplegic patients were selected for the study if they showed ability to stand without external support for at least 30 seconds. Posturographic evaluation was performed on a statokinesimetric platform just before and after a cold contralesional ear irrigation (20°C) during 60 seconds. Two quantitative parameters were analysed: the antero-posterior difference and the lateral difference, reflecting the asymmetry of standing in the antero-posterior and frontal planes, respectively. The results of the 3 groups studied were compared with a Student’s t-test. Before stimulation, as previously reported, left hemiparetic patients showed a predominant lateral displacement of the centre of pressure toward the side of the lesion, as compared to right hemiparetic patients. After vestibular stimulation, the lateral displacement was reduced in both patient groups, predominantly in the left hemiparetic group. After vestibular stimulation, the lateral displacement thus was not different in both patient groups and in the control group. Antero-posterior differences were not significantly different in the patient groups and in the control group before stimulation and were not affected by vestibular stimulation. The suggestion is made that greatest postural imbalance produced by right brain damage could reflect a persistent distorsion of a “spatial postural representation”. Vestibular stimulation may restore symmetrical activity in the cerebral structures involved in the generation of this “spatial postural representation”.

Key words: hemiplegia, posture, right hemisphere, spatial cognition, vestibular stimulation.

INTRODUCTION

Postural disturbances are common in patients with hemiplegia of vascular origin, mainly as a shift of body weight toward the non-paretic limb (2, 8, 9, 25, 30, 31, 37, 38). This postural asymmetry may be explained by the relative importance of proximal and motor deficiency as well as by somesthetic, visual or vestibular disturbances.

Previous data showed that postural asymmetry was larger in left hemiparetic patients, as compared to right hemiparetic patients, regardless of sensory or motor deficit (17, 19). This predominance of postural imbalance was likely to be related to the right hemisphere damage. The prevalence of visuospatial disorders with lesions of this hemisphere suggests its involvement in the integration of spatial informations, needed for the orientation of motor behaviour in space (15, 16).

Behavioural orientation in space implies building internal representations, resulting from the symmetrical activity of cortical and subcortical areas involved in multimodal sensory integration. Distorsions of these representations may be observed following a unilateral post-rolandic damage, as suggested by the shift of the midsagittal plane representation (26, 27) or the neglect of representational space in neglect patients (3). In a previous study, we therefore suggested that the predominance of a postural asymmetry in left hemiparetic patients could reflect a persistent distorsion of a “spatial postural representation” (29).

Distorsion of representation can be improved by sensory manipulation. Indeed, the shift of the midsagittal plane internal representation is improved after caloric vestibular stimulation (27), optokinetic stimulation (20), controlateral neck muscle vibration (22) or controlateral trunk rotation (21). The neglect of representational space may be also reduced through caloric vestibular stimulation (12, 28). These data suggest thus that sensorial stimulation may influence the mechanisms of central multimodal integration.
involved in the elaboration of the spatial mental representations.

If the predominance of postural imbalance in left hemiparetic patients is a disturbance of a “postural referential system”, we hypothesize that sensorial manipulation would reduce the predominance of postural imbalance in left hemiparetic patients, as compared to right hemiparetic patients. We thus investigated postural control with posturography before and after vestibular caloric stimulation in 2 groups of hemiparetic patients (right and left) and in a group of control subjects.

MATERIAL AND METHODS

Subjects
Thirty right-handed hemiparetic patients (as described in Table I) participated in this study. Fifteen subjects had a right hemiplegia (RH) and 15 had a left hemiplegia (LH). Hemiplegia was secondary to an ischaemic stroke in acute phase of recovery (within 6 months of onset). Patients were selected for the study if they (1) were medically stable without prevailing complications or drugs that may disturb posture, (2) had no history of neurologic, orthopaedic or vestibular diseases, (3) had potential for functional locomotion with rehabilitation, and (4) showed ability to stand up without external support for at least 30 seconds. In addition, 15 healthy volunteers (with no history of vestibular dysfunction) were tested (8 females and 7 males). The average age of control subjects was 31 years old (range: 22–42 years old).

Methods
Posturographic evaluation was performed using a Sereme + Spectral® statokinesimeter. It consisted of a statokinesimetric platform measuring 45 × 55 cm with four strain gauges placed on the diagonals of the platform. The strain gauges transformed pressure variations into variations in the current’s intensity. The accuracy of measurement on each detector was ± 0.5 kg. Information obtained from the strain gauges was transmitted to the central unit of a computer [for details, see (6) and (29)].

During the test, subjects stood barefoot in the upright position with their arms alongside their bodies and with their feet placed on the predesign site, 10 cm apart, centred in relation to the antero-posterior and lateral axis. Subjects kept their feet crossed with their right foot ahead of their left feet.

Table I. Right and left hemiparetic groups characteristics; standard error of the mean in parentheses

<table>
<thead>
<tr>
<th></th>
<th>RH group (n = 15)</th>
<th>LH group (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>55 (20–73)</td>
<td>56 (31–73)</td>
</tr>
<tr>
<td>Time from onset (months)</td>
<td>5.6</td>
<td>5.8</td>
</tr>
<tr>
<td>Male</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Female</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Weight (Kg)</td>
<td>69.4 (56–89)</td>
<td>68 (54–85)</td>
</tr>
<tr>
<td><strong>Paretic limb control</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isolated*</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Partial synergy†</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Full synergy‡</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td><strong>Sensory deficits</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypo-aesthesia</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Anaesthesia</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Absent</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td><strong>Visual field defects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortico–subcortical hypodense lesion:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>including the parieto-temporo-occipital carrefour</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>excluding the parieto-temporo-occipital carrefour</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Cortical hypodense lesion (parietal)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Neuropsychological disorders</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Unilateral neglect</td>
<td>0</td>
<td>9</td>
</tr>
</tbody>
</table>

* Ability to perform completely independent movements at the hip, knee and ankle;† ability to perform some independent joint movements while some were performed only in synergy with other joint movements;‡ all movements at each joint were performed only in synergy with motion at other joints.
their eyes open and remained silent. Statokinesimetric measurements recorded the weight supported by the right-anterior, left-anterior, right-posterior and left-posterior strain gauges for 30 seconds standing sampled at 100 Hz. For each measurement, the centre of pressure and the distribution of body weight were determined in kilogrammes. The mean distributions of body weight in the antero-posterior and lateral axes are called antero-posterior and lateral differences (see Fig. 1 in Rode et al. (29)).

These two parameters were obtained in patients and control subjects before and after caloric vestibular stimulation. For each parameter, mean and standard deviation in each group (control subjects = C, right hemiparetic patients = RH and left hemiparetic patients = LH) were assessed.

Statistical analysis

The two parameters (antero-posterior and lateral differences) were analysed. The effect of each group was analysed by comparing the 3 subject groups (RH, LH, C), either before and after vestibular stimulation, using a t-test. The effect of vestibular stimulation within each group was analysed by comparing the results before and after vestibular stimulation, using a t-test. Moreover, for the lateral difference parameter, the effect of vestibular stimulation between each group was analysed by comparing the delta difference (before–after) between the 3 subject groups (RH, LH, C), using a t-test.

Vestibular stimulation

An ENT examination was performed before caloric stimulation and showed normal results in patients and control subjects. Vestibular stimulation was performed using a cold ear caloric stimulation. The external ear canal was irrigated with 60 cc of cold (20°C) water for 30 seconds. In hemiparetic patients, the contralateral ear was irrigated; in control subjects, the left ear. During stimulation, the patient was blindfolded and was sitting on a chair. His head was tilted approximately 30 forward. Then he was asked to stand up on the platform.

In normal subjects, such a vestibular activation produces a horizontal nystagmus with a leftward slow phase lasting about 3 seconds and a marked sensation of vertigo. In patients, caloric stimulation produces a horizontal nystagmus with slow phase directed toward the irrigated ear during 2–3 minutes. However, only half of them experienced a sensation of vertigo.

Table II shows the means and standard deviations of each parameter in the 3 groups.

**Results**

**Antero-posterior difference**

Effect of group: before stimulation, no significant difference was shown between LH and RH groups ($t = 0.6; p = 0.5$), between LH and C groups ($t = 0.6; p = 0.5$) and between RH and C groups ($t = 0.04; p = 0.5$).

Effect of vestibular stimulation: the comparison of the antero-posterior difference, before and after stimulation, showed no significant difference in LH group ($t = 1.3; p = 0.2$), RH group ($t = 1.1; p = 0.3$) and C group ($t = 0.8; p = 0.4$).

**Lateral difference**

Effect of group: before stimulation, the LH group showed a greater value than the C group ($t = 3.6; p < 0.01$) and than the RH group ($t = 3.9; p < 0.01$). Results showed also a significant difference between the RH and C groups ($t = −5.1; p < 0.01$). The results revealed that the hemiparetic patients had an inability to shift body weight onto the paretic limb. This asymmetry in posture predominated in the group of left hemiparetic patients (Fig. 1).

Effect of vestibular stimulation: lateral difference before and after stimulation was quite similar in the RH group and the C group but only reaches significance in the RH group (RH group: $t = 2; p < 0.05$; C group: $t = 1.8; p > 0.05$). In the LH group, the comparison of the lateral difference, before and after stimulation, showed a significant difference ($t = 3.9; p < 0.01$). Furthermore, after stimulation no significant difference was shown between LH and RH groups ($t = 0.1; p = 0.9$), between LH and C groups ($t = 1; p = 0.3$) and between RH and C groups ($t = 0.8; p = 0.4$).

Table II. Mean values and standard deviations (in parentheses) of antero-posterior and lateral differences in the 2 groups of hemiparetic patients and the group of control subjects, measured with a statokinesimetric platform, before and after caloric vestibular stimulation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>C group Before VS</th>
<th>C group After VS</th>
<th>RH group Before VS</th>
<th>RH group After VS</th>
<th>LH group Before VS</th>
<th>LH group After VS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antero-posterior difference</td>
<td>8.3 (17.2)</td>
<td>4.2 (21.4)</td>
<td>8.6 (14)</td>
<td>5.9 (21)</td>
<td>13 (20.9)</td>
<td>11.1 (20.3)</td>
</tr>
<tr>
<td>Lateral difference</td>
<td>10.6 (4)</td>
<td>2.9 (16.6)</td>
<td>-14 (16.3)</td>
<td>-8.5 (18.6)</td>
<td>29.6 (18.2)</td>
<td>9.7 (15.5)</td>
</tr>
</tbody>
</table>

C: control subjects; RH: right hemiparetic patients; LH: left hemiparetic patients; VS: vestibular stimulation.

Scand J Rehab Med 30
Lastly, the comparison of delta lateral difference (i.e. the difference between mean score of each group before and after vestibular stimulation) showed a significant difference between LH and RH groups ($t = 3.9; p < 0.01$), between LH and C groups ($t = 3.4; p < 0.01$) but no difference between RH and C groups ($t = 1.1; p = 0.25$) (Fig. 2).

The results revealed that the predominant shift of body weight toward the non-paretic limb, showed by left hemiparetic patients, was significantly reduced through vestibular stimulation (Fig. 1).

**DISCUSSION**

While no change in antero-posterior difference (i.e. no anterior or posterior displacement of the centre of pressure) was found in either group of hemiparetic patients, before or after vestibular stimulation, as compared to control subjects, some interesting results were found in the lateral difference and may be summarized as follow: (1) hemiparetic patients showed a lateral displacement of the centre of pressure toward the side of the lesion; (2) this postural asymmetry was predominant in the LH group; and (3) vestibular caloric stimulation reduced this asymmetry and was more effective in the LH group.

As previously reported, hemiparetic patients showed modifications of postural sway with a lateral displacement of the centre of pressure toward the side of the lesion, suggesting decreased postural balance (2, 8, 9, 25, 30, 31, 37, 38). This postural imbalance predominated in patients with right hemispheric lesions as compared to patients with left hemispheric lesions (17, 29). In our two groups of hemiparetic patients, clinical characteristics such as lower limb power, sensory disturbances and visual field defects were comparable and cannot explain this difference.

Vestibular stimulation was effective in reducing postural asymmetry in both groups of hemiparetic patients. This result could be related to vestibular brainstem reflexes. Indeed, cold ear irrigation, through ipsilateral inhibition of vestibular afferences, acts as a contralateral vestibular activation and produces ipsilateral slow deviation of the eyes (vestibulo-ocular reflex), of the limb and of posture (vestibulo-spinal reflex). Therefore, changes in axial muscular tonus due to activation of contralateral vestibulo-spinal reflex could explain the minimal effect obtained in the RH group and the C group. However, the simple peripheral vestibular response to caloric stimulation is not sufficient to explain the large reduction of postural asymmetry in the LH group. Therefore, we suggest that the reduction of postural asymmetry in the LH group is a consequence of both a peripheral and a central effect. This

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**Fig. 1.** Diagrammatic representation of mean distribution of body weight in the antero-posterior and lateral axes in the 2 groups of hemiparetic patients and the group of control subjects before (white) and after (black) caloric vestibular stimulation, according to the side of the lesion. The shift toward the lesion side, showed by hemiparetic patients, which clearly predominated in left hemiparetic patients, was significantly reduced through vestibular stimulation. C: control subjects; RH: right hemiparetic patients; LH: left hemiparetic patients; LD: lateral difference; APD: antero-posterior difference; IL: ipsilesional; CL: contralesional.

**Fig. 2.** Mean values and standard deviations (in parentheses) of delta lateral difference in the 2 groups of hemiparetic patients and the group of control subjects. C: control subjects; RH: right hemiparetic patients; LH: left hemiparetic patients.
central effect could be related to a cortical integration of vestibular informations, involved in the mechanisms of spatial cognition.

Indeed, it is often postulated that spatial cognition needs integration of vestibular, visual and somatosensory inputs (33). Neurons fulfilling this criterion of plurimodal integration were recorded in several parts of the brain [see review in (18)]. Although vestibular cortical integration is a quite new concept, several regions of cerebral cortex in monkeys that respond to natural vestibular stimulation have been identified in the past 20 years (23). These regions include portions of the intraparietal cortex (area 2v) (5), the central sulcus (area 3av) (24), the posterior end of the insula (PIVC—posterior insular vestibular cortex) (1), and the medial superior temporal visual area (MST) (32). These areas responding primarily to vestibular stimulation were also found to respond to either visual (optokinetic) or somatosensory (proprioceptive) stimulations or both (13, 14).

Extrapolating such results from monkeys to human is not easy because of the anatomical cerebral differences between species. However, functional neuro-imaging disclosed increased cortical signals during caloric vestibular stimulation in distributed areas similar to those of monkeys (4, 11).

The existence of cortico-vestibular pathways that could control vestibulo-spinal reflex would provide also strong arguments in favour of an hypothesis of a central postural control. Indeed, cortical control of vestibular function has been shown in several experiments. In cats and monkeys, temporo-parietal cortical ablation produced asymmetrical changes in the vestibulo-ocular reflex (35, 36). Anterograde tracing technique in monkeys showed the existence of direct projection from the parietal cortex to the vestibular nuclei (10). Furthermore, vestibulo-ocular reflex abnormalities have been correlated to postural imbalance in hemispheric stroke (7).

There are strong arguments in favour of central integration of vestibular and other sensorial stimuli, and of a cortical control of vestibular outputs like posture. These vestibular informations, as visual, proprioceptive informations about the position of eyes, head and body, could be used by the brain to produce multiple higher-order (e.g. egocentric) representations of space subserving accurate spatial behaviour. Rather than a concept of a unitary representation of space or egocentric reference initially proposed by Ventre et al. (35) and Jeannerod & Biguer (19), the concept of multiple representations in spatial cognition is debated, especially to account for the many manifestations of neglect (34).

We thus suggest that the vestibular stimulation may directly influence neural mechanisms involved in the generation of these spatial internal representations. In our study, vestibular stimulation may affect the spatial representation, used for the adjustment of posture. In normal subjects, this representation would be symmetrical, and the modifications showed by subjects after vestibular stimulation may be explained by the peripheral vestibulo-spinal responses.

In hemiparetic patients, especially following right hemisphere damage, this representation can be distorted. The postural misrepresentation may be positively affected by vestibular stimulation, through restoration of a symmetrical activity in cerebral structures involved in spatial cognition. Similar results reporting the reversibility of other spatial misrepresentation by vestibular stimulation, like the reduction of shift of the midsagittal plane representation or the remission of representational neglect in right brain damaged patients argue in favour of this idea (26, 28).

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