Multiple lesion mapping and functional imaging studies undoubtedly showed that the posterior insular cortex (IC) and neighboring peri-insular region are central structures of the human vestibular cortical network.1-5 However, it seems that small lesions exclusively affecting the IC might not be sufficient to cause vestibular otolith deficits, such as tilt of subjective visual vertical (SVV).2 Presumably, because of the rareness of isolated insular infarctions, there are no data reporting about vestibular parameters in patients with isolated infarctions of the IC. The approach of this study focused on a strong hypothesis on the exclusive role of the IC in vestibular otolith processing. Thus, parameters of otolith dysfunction consisting of the triad of head tilt, skew deviation, and ocular torsion, as well as tilt of SVV as its perceptual parameter were investigated in 10 patients with acute unilateral stroke restricted to the IC.

Methods

Neurological Examination

We identified 10 patients (5 left-sided and 5 right-sided lesions) with a first ever acute stroke and MRI lesion restricted to the IC among our database of 475 stroke patients, which were tested for tilt of SVV, skew deviation, head tilt, and ocular torsion, who were admitted to our department between 2003 and 2012. Because vascular lesions exclusively affecting the IC are extremely rare, our sample consisted only of a small amount of patients with circumscribed damage to the posterior IC (7 female, 3 male; mean age, 71 years; SD, 9.3 years). Mean time between stroke and testing was 5 days (SD, 1.4 days). None of the patients tested showed clinical signs of neglect such as orienting toward the ipsilesional side when addressed from the front or the left, or ignoring contralateral located people or objects.6 The subjects gave their informed consent to participate in the study, which was performed in accordance with the ethical standards outlined in the 1964 Declaration of Helsinki and approved by the local ethics committee.

Vestibular Testing

SVV, as a measure of tonic vestibular otolith perception, as well as ocular torsion, skew deviation, and head tilt were tested as described previously.7,8 The mean of static SVV was determined by means of 7 adjustments from a random offset position. A mean deviation of >2.5° of the static SVV—determined binocularly—was considered as abnormal tilt.

MRI Scans

In all patients, MRI scans were performed with a mean time interval of 6 days between lesion onset and MRI (SD, 6.5 days). We used diffusion-weighted imaging (DWI) within the first 48-h poststroke and T2 or fluid-attenuated inversion-recovery (FLAIR) sequences when imaging was conducted 48 h or later. Lesion mapping using the normalization algorithm of SPM5 (http://fil.ion.ucl.ac.uk/spm/) was conducted as described previously.2,9 Statistical

Background and Purpose—In previous imaging studies, the posterior insular cortex (IC) was identified as an essential part for vestibular otolith perception and considered as a core region of a human vestibular cortical network. However, it is still unknown whether lesions exclusively restricted to the posterior IC suffice to provoke signs of vestibular otolith dysfunction. Thus, present data aimed to test whether patients with lesions restricted to the IC showed vestibular otolith dysfunction.

Methods—We studied 10 acute unilateral stroke patients with lesions restricted to the IC which were tested for signs of vestibular otolith dysfunction, such as tilts of subjective visual vertical, out of 475 stroke patients.

Results—None of the patients was with stroke exclusively affecting the IC-specified vertigo as a symptom. In addition, neither showed a deficit in the perception of verticality (subjective visual vertical tilts) nor showed any further vestibular otolith deficits, such as ocular torsion or skew deviation.

Conclusions—It seems that lesions of the posterior IC might have to be combined with lesions of adjacent regions of the cortical and subcortical vestibular network to cause vestibular otolith deficits. (Stroke. 2013;44:00-00.)

**Key Words:** insula ■ lesion ■ stroke ■ subjective visual vertical ■ verticity ■ vestibular system
analysis was conducted using SPSS version 15.0 for Windows (SPSS Inc, Chicago, IL).

Results
In our sample of 475 patients with stroke tested for otolith dysfunction, we only found 10 patients with acute stroke lesions affecting the posterior IC (2%; lesion size, 0.2 to 2.9 cm³). This low number indicates that patients with lesions restricted to the IC are extremely rare. Neither of the 10 patients specified vertigo as a symptom nor had an abnormal tilt of SVV, i.e., a deviation of >2.5° (mean, 1.3°; SD, 0.81°; Table). The 1-sample t-test implies that the sample mean of the tilt of SVV of our 10 patients with insular strokes differs from our test value of 2.5° (t=−4.652; P=0.001). No other signs of otolith dysfunction such as ocular torsion, skew deviation, or head tilt in the acute phase 5 days after stroke onset were seen. The other symptoms of the patients are listed in the Table. The Figure shows the lesion distribution of the 5 right-sided (Figure A) and the 5 left-sided lesion patients (Figure B).

Discussion
To our knowledge, this is the first case series showing that patients with acute lesions restricted to the IC do not show any pathological signs of vestibular otolith dysfunction nor showed any sign of vertigo. Therefore, lesions of the IC alone do not seem to be sufficient to cause otolith deficits, and thus, do not play exclusively a role in abnormal tilt of SVV.

Previous data are not necessarily contradictory because these investigated larger infarctions of the MCA territory, whereas we focused on very small lesions exclusively affecting the IC. Obviously, cortical lesions outside the IC affecting the inferior frontal gyrus, the superior temporal gyrus, the peri-insular operculum, as well as white matter regions, such as the superior occipitofrontal fascicle or the inferior occipitotemporal fascicle, might, in addition, be important for the perception of verticality. One possible explanation could be that larger lesions affecting more parts of the vestibular network might lead to a more severe tilt of SVV in a higher percentage of patients, or in other words, otolith deficits because of lesions restricted to the IC might be compensated by vestibular mechanisms achieved in other neighboring regions within the cortical vestibular network. This finding confirms previous data indicating that not only the IC, but also its surrounding regions, such as the inferior frontal gyrus, the superior temporal gyrus, and the Rolandic operculum, play a role in SVV tilts. As a conclusion, not only the posterior part of the IC, but also surrounding regions might represent the main entrance of vestibular otolith signals to the cortex—but it might be not the only one.

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Table. Demographic and Clinical Data in the Acute Phase of All Patients With Isolated Lesions to the Insula

<table>
<thead>
<tr>
<th></th>
<th>Right-Brain Damage</th>
<th>Left-Brain Damage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Age (y), mean (SD)</td>
<td>69 (8.2)</td>
<td>74 (10.1)</td>
</tr>
<tr>
<td>Sex (w/m)</td>
<td>3 w, 2 m</td>
<td>4 w, 1 m</td>
</tr>
<tr>
<td>Lesion volume (in cc), mean (SD)</td>
<td>1.1 (1.1)</td>
<td>0.6 (0.5)</td>
</tr>
<tr>
<td>Contralateral paresis (MRC scale), median (range)</td>
<td>3 (0–5)</td>
<td>5 (3–5)</td>
</tr>
<tr>
<td>Tilt of SVV (absolute values in degrees), mean (SD)</td>
<td>1.3 (1.0)</td>
<td>1.4 (0.6)</td>
</tr>
<tr>
<td>Ocular torsion, % present</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Skew deviation, % present</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Head tilt, % present</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aphasia, % present</td>
<td>0</td>
<td>80</td>
</tr>
<tr>
<td>Dysarthria, % present</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>Somatosensory deficit of contralateral side (touch), % present</td>
<td>40</td>
<td>0</td>
</tr>
</tbody>
</table>

m indicates men; MRC, Medical research council; SVV, subjective visual vertical and w, women.
Disclosures

None.

References

Insular Strokes Cause No Vestibular Deficits
Bernhard Baier, Julian Conrad, Peter zu Eulenburg, Christoph Best, Wibke Müller-Forell, Frank Birklein and Marianne Dieterich

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