Regional white and gray matter differences between visual vertigo patients and healthy controls: preliminary results.

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Introduction

Visual vertigo (VV) is a complex syndrome where patients experience discomfort, postural instability, spatial disorientation and symptom exacerbation in disorienting visual environments (e.g., supermarket aisles, crossroads, 3D movies, ...). Logically, this has a negative impact on the quality of life of these patients and their social activity. After an acute vestibular loss, the majority of patients rely on visual cues for posture control and they incorporate proprioceptive cues in a later phase, a process called sensory reweighting. However, a high visual dependency in VV patients interferes with this compensation process and therefore patients keep experiencing problems. The aim of this study was to investigate if there is a neural correlate for this hampered compensation process in VV patients.

Methodology

Subjects
- 5 VV patients (1 male, mean age: 48.4y ± 6.73y)
- 5 healthy controls (1 male, mean age: 50.6y ± 6.06y)

Data acquisition
- 3T MRI Tim Trio Siemens (32 channel head coil)
- Voxel size: 2.5 x 2.5 x 2.5 mm3, acquisition matrix = 96 x 96, TR = 8100 ms, TE = 116 ms.
- b-values: 700, 1000 and 2800 s/mm2, along 25, 45 and 75 non-collinear directions resp.
- Voxel-based morphometry (VBM) and Diffusion Tensor Imaging (DTI)

VBM data analysis
- Automated technique
- Whole-brain analysis
- Assessment of gray matter integrity.

DTI data analysis
- Manual region of interest (ROI) placement based on a priori hypothesis
- TrackVis® software
- White matter structures: splenium, inferior longitudinal fasciculus (ILF), inferior fronto-occipital fasciculus (IFOF) and the inferior, middle and superior cerebellar peduncles (ICP, MCP and SCP)

Results

Gray matter

![Image](https://www.uantwerp.be/en/staff/angelique-vanombergen/)

Fig 2: Coronal slices showing clusters of significant volume decrease between VV patients and healthy controls (controls>p=patients). Representative peak activations are listed in Table 1.

<table>
<thead>
<tr>
<th>Anatomical region</th>
<th>MNI coordinates</th>
<th>Cluster</th>
<th>p_uncorrected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior occipital lobe L</td>
<td>x = -43.5; y = -76.5; z = -9</td>
<td>127</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Angular gyrus R</td>
<td>x = 42; y = 61.5; z = 43.5</td>
<td>66</td>
<td>p &lt; 0.001</td>
</tr>
</tbody>
</table>

Table 1: Neuroanatomical regions showing a significant difference between VV patients and healthy controls, (numerical labels, x coordinates of sagittal slices; y coordinates of axial slices; z coordinates of coronal slices; L=left; R=right).

Discussion

- Shows a relation between diffusion parameters (brain connectivity) and clinical symptoms of vertigo.
- Decreased FA in the visuospatial network suggests a degradation of the interpretation of visual info.
- Increased FA in cerebellar projection tracts suggests inadequate compensation in the vestibulo-cerebellar regions?
- Differential diagnosis with psychogenic dizziness should be made carefully!

Conclusion

- Neurosensory mismatch that could explain the VV symptoms and overreliance on visual cues.
- Promising results for the use of DTI and tractography in vestibular patients: diffusion parameters may serve as biomarkers in visual vertigo patients.
- Clinical importance!!!
- Future studies (with larger n) should elaborate these findings.

References


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