Depersonalisation/derealisation symptoms and updating orientation in patients with vestibular disease

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Word count for main body text: 4056
ABSTRACT

Background: Patients with vestibular disease have an increased rate of reporting symptoms of depersonalisation/ derealisation (DD) and similar symptoms can be provoked in healthy subjects during caloric vestibular stimulation.

Objective: To assess the relationship between DD symptoms in patients with peripheral vestibular disease and their ability to update orientation in the environment.

Methods: Sixty healthy subjects and 50 patients with peripheral vestibular disease completed a DD questionnaire (Cox and Swinson, 2002) and the GHQ-12 (Goldberg and Williams, 1988). This was followed by a test of updating spatial orientation in which subjects were exposed to 10 manually driven whole body rotations of 45°, 90° or 135° in a square room, which contained distinctive features on the walls, in such a way that the features and corners subtended 45° with respect to the subject. After each rotation subjects reported which wall or corner they were facing. Estimation error was calculated by subtracting the reported rotation from the actual rotation.

Results: DD scores were significantly higher in vestibular patients than in healthy subjects (p<0.05, t test). In patients, the lowest symptom scores and the lowest estimation errors were found in those with a unilateral canal paresis without balance symptoms whereas the highest scores and largest estimation errors were found in those with bilateral vestibular loss (p<0.05, ANOVA). Across all patients, DD scores were related to estimation errors (adjusted $R^2= 0.25$, p<0.05, ANCOVA).

Conclusions: Patients with peripheral vestibular disease have a deficit in the ability to update orientation on the environment and a high prevalence of DD symptoms, which may imply a high order effect of the vestibular impairment. Derealisation symptoms in vestibular disease may be a consequence of a sensory mismatch between disordered vestibular input and other sensory signals of orientation.

Key words: derealisation, vestibular, orientation
INTRODUCTION

Altered perceptions of the self and the environment are termed "dissociation phenomena". Depersonalisation is defined as "an alteration in the perception or experience of the self so that one feels detached from, or as if one is an outside observer of, one's mental processes or body"; and derealisation as "an alteration in the perception or experience of the external world so that it seems strange or unreal" [1]. Depersonalisation/derealisation (DD) symptoms are commonly described accompanying a wide variety of psychiatric and neurological conditions [2-7]. Although most of the neurological conditions associated with DD have poor localisation value, different DD components have also been described accompanying localised lesions, which suggest that a differentiation of the mechanisms underlying depersonalization and derealisation has neurobiological validity [8].

Behaviour in the environment requires an internal representation of physical space. Evidence supports that the vestibular system is necessary for the perception of the head in space and consequently to maintain an adequate internal representation of the body in space; accordingly, after a unilateral vestibular lesion, patients can show a transient impairment in navigational accuracy, which can recover over a period of 1 month [9]. In addition, when humans change their orientation in the environment, the spatial updating of egocentric relations (self-to-object directions and distances) that takes place concurrently with the change of spatial relations depends upon the availability of proprioceptive and vestibular information [10, 11]. In the absence of visual cues, it is predominantly vestibular information that is used in the perception of angular displacements in the horizontal plane [12].

A recent study showed that vestibular dysfunction as well as caloric vestibular stimulation can induce DD symptoms [13]. The frequency and severity of DD symptoms were higher in vestibular patients than in age matched healthy subjects. Of note, healthy subjects undergoing caloric vestibular stimulation reported DD symptoms that they had not previously experienced, whereas vestibular patients reported that the DD symptoms induced by caloric stimulation were similar to their own spontaneous symptoms [13]. A functional imaging study of patients with depersonalisation disorder has suggested abnormalities in the sensory cortex and areas responsible for an integrated body schema [14], consistent with the proposal that the inferior parietal cortex is concerned with spatial orientation, visuo-motor and vestibular function [15]. Additional clinical evidence suggests that there is some degree of hemispheric specialization in the cortical processing of vestibular signals [16]. However, little is known about the relationship between orientation updating (based on vestibular signals and hence upon the quality of vestibular performance) and feelings of detachment from the environment (derealisation).

In this study, we attempt to relate symptoms of derealisation, as experienced by vestibular patients, to their vestibular performance during an orientation task. We studied the relationship between self-ratings of DD symptoms and the ability to update egocentric relations to the environment after whole body passive rotations in the horizontal plane. Symptomatic and asymptomatic patients with both bilateral and unilateral lesions were studied. Patients with posterior canal Benign Paroxysmal Positional Vertigo (BPPV) and a normal caloric test result (which examines horizontal canal function) were also evaluated to control for the experience of vertigo without a horizontal canal defect.

METHODS

Subjects.
110 subjects gave their informed consent to participate in the study comprising:

- 60 healthy subjects, aged 21 to 77 years (29 females and 31 males), who gave the reference responses to the study protocol. Since subject age may influence the report of depersonalisation/derealisation symptoms, for comparison with the patients, an age-matched subgroup was also created from twelve of the healthy subjects, aged 30 to 77 years (6 females and 6 males). Their general characteristics are described in Table 1. All subjects denied having a history of dizziness,
- 50 patients with peripheral vestibular disease, aged 27 to 81 years (mean 55 ± 13), 26 females and 24 males (Table 2), with a history of vestibular disease from 1 month to 25 years. The vestibular disorder was diagnosed after neuro-otological evaluation that included: eye movement examination, positional manoeuvres, bithermal caloric testing and pure tone audiometry. Patients with bilateral vestibular dysfunction also had electronystagmography with rotational testing. Clinical diagnoses are shown in table 2. Twenty seven patients had unilateral canal paresis, which was defined as an asymmetry of ≥20% between right and left responses to 30° and 44° caloric stimuli [17], based on automated peak slow phase nystagmic velocity analysis. Thirteen of these 27 patients with canal paresis reported dizziness or imbalance at the time of evaluation and needed follow-up and treatment (= with balance symptoms group); asymmetry of caloric responses >50% was observed in 38% of patients with balance symptoms and 60% of patients without balance symptoms. Eleven patients had bilateral vestibular dysfunction of unknown origin, of whom 10 showed more than 90% reduction of vestibular function on the basis of rotational/caloric findings [18]. The remaining patient had a reduction of 70% [18]. In the 12 patients with posterior canal BPPV, the Dix-Hallpike manoeuvre was positive at the time of evaluation but the responses to caloric stimulation (which examines horizontal canal function) were normal. The main balance symptoms reported by patients with bilateral dysfunction were postural instability and oscillopsia, while the main complain of patients with unilateral canal paresis with symptoms and patients with BPPV was vertigo. The general characteristics of each subgroup are described in Table 1. None of the patients had ophthalmological disease other than corrected refraction errors. Eleven patients had mild hearing loss and 2 patients had moderate to severe hearing loss. In all cases, the hearing loss was concomitant to vestibular disease or due to presbyacusis. Patients with a history of migraine or other neurological or psychiatric disorders (submission to psychiatric care or psychopharmacological treatment) were not included in the study.

Patients were evaluated by 3 different collaborators: one performed the clinical evaluation; a second classified the patients according to their clinical history and a third administered the questionnaires and performed the orientation test.

Table 1. General characteristics of the vestibular patients and the healthy subjects who participated in the study.

<table>
<thead>
<tr>
<th>VARIABLES</th>
<th>Vestibular patients</th>
<th>Healthy subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BPPV (N=12)</td>
<td>Unilateral</td>
</tr>
<tr>
<td></td>
<td>without symptoms</td>
<td>with symptoms</td>
</tr>
<tr>
<td>AGE (years)</td>
<td>59 ± 12.3</td>
<td>51.7 ± 16.1</td>
</tr>
<tr>
<td>Mean ± Standard Deviation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GENDER</td>
<td>7 / 5</td>
<td>6 / 8</td>
</tr>
<tr>
<td>Female/ male ratio</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDUCATION LEVEL</td>
<td>University 25%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>Secondary school 50%</td>
<td>57%</td>
</tr>
<tr>
<td></td>
<td>Primary school 25%</td>
<td>0%</td>
</tr>
<tr>
<td>EMPLOYMENT STATUS</td>
<td>Employed 25%</td>
<td>50%</td>
</tr>
<tr>
<td></td>
<td>Student 0%</td>
<td>7%</td>
</tr>
<tr>
<td></td>
<td>Retired 33%</td>
<td>43%</td>
</tr>
<tr>
<td></td>
<td>Unemployed 42%</td>
<td>0%</td>
</tr>
<tr>
<td>MARITAL STATUS</td>
<td>Single 33%</td>
<td>21%</td>
</tr>
<tr>
<td></td>
<td>Married 67%</td>
<td>58%</td>
</tr>
<tr>
<td></td>
<td>Divorced or widowed</td>
<td>0%</td>
</tr>
<tr>
<td>HEALTH HABITS</td>
<td>Smokers 25%</td>
<td>14%</td>
</tr>
<tr>
<td></td>
<td>No alcohol 25%</td>
<td>50%</td>
</tr>
<tr>
<td></td>
<td>1-5 units/week 42%</td>
<td>14%</td>
</tr>
<tr>
<td></td>
<td>6-10 units/week 8%</td>
<td>14%</td>
</tr>
<tr>
<td></td>
<td>&gt;10 units/week 25%</td>
<td>22%</td>
</tr>
</tbody>
</table>
Table 2. Clinical diagnoses of the patients with peripheral vestibular disease who participated in the study.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral canal paresis (symptoms/ no symptoms)</td>
<td>27</td>
</tr>
<tr>
<td>Vestibular neuritis</td>
<td>9 (5/4)</td>
</tr>
<tr>
<td>Vestibular neuroma before treatment</td>
<td>3 (0/3)</td>
</tr>
<tr>
<td>Meniére disease</td>
<td>4 (1/3)</td>
</tr>
<tr>
<td>Unknown origin</td>
<td>6 (3/3)</td>
</tr>
<tr>
<td>Canal paresis &amp; BPPV</td>
<td>5 (4/1)</td>
</tr>
<tr>
<td>Bilateral dysfunction</td>
<td>11</td>
</tr>
<tr>
<td>BPPV (no canal paresis)</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
</tr>
</tbody>
</table>

*The 3 of them with isplateral hearing loss & no central signs.

II. After completing the questionnaires, subjects performed a test of updating orientation. The test was specially designed to minimise the feeling of laboratory testing and to mimic the daily life situation of orienting ourselves relative to the location of the distinctive features of an unfamiliar room, with the possibility to use both geometric and non-geometric information (landmarks). Subjects were first familiarised with a room (2.1 x 2.5 m), which contained four fixed features, each positioned in the middle of each wall (Figure 1). They sat upright on a hydraulic barber’s chair, in the centre of the room and were asked to remember the location of each of the features while they were rotated in the light, to face each of the walls. Then, blindfolded and wearing sound occluding head phones, the subjects were exposed to 2 sets of 5 manually driven whole body rotations of 45°, 90° or 135° (circa 40°/s & 0.36Hz, 60°/s & 0.48 Hz and 70°/s & 0.24Hz peak velocity & peak frequency, respectively); chair rotational velocity was measured by a gyroscope mounted on the head rest which gave an analog display; rotations were performed to the right or to the left, in an unpredictable sequence balanced for amplitude, direction and order. Between each rotation, 10 seconds elapsed to allow for any post-rotatory sensation to fade so as not to interfere with subsequent responses. After the 5th rotation, the chair was returned to the start position, the eye mask was removed and the subjects had a short rest of 1 to 2 minutes before commencing the final set of rotations.

During the test, the actual rotation sequence included 5 rotations to the right and 5 to the left; 4 rotations of 45°, 5 of 90°, and 1 of 135°; the sequence of small rotations followed by large rotations and vice versa were similar to the right and to the left. Whenever the first rotation was to the right then the 6th rotation (after the short rest) would be to the left and vice versa.

The subject's task was to report, after each rotation, which wall or corner they were facing. The error of each orientation estimate was calculated by subtracting the reported rotation from the actual rotation.

**Data processing and analysis**

The scores for the GHQ-12 and the DD inventory were calculated as the sum of the individual scores of each of the items. Statistical analyses were performed according to data distribution and scale of measurement (i.e. parametric when normally distributed) using Kolmogorov-Smirnov test, “t” test, Mann Whitney U test, Pearson and Spearman correlation coefficients, Discriminant Function Analysis, ANOVA, Tukey’s honestly significance difference test and ANCOVA; p values lower than 0.05 were considered significant.
RESULTS

Questionnaire data:

GHQ 12 score.
In the group of 60 healthy subjects and in the subgroup of 12 age-matched subjects, the GHQ-12 score ranged from 0 to 2 (median of 0). Across all vestibular patients, the range of the GHQ-12 score was from 0 to 10 (median of 3) with no significant difference among subgroups of patients (p> 0.05, Kruskal Wallis test). Compared to the age matched subgroup of healthy subjects, the GHQ-12 scores were significantly higher in each subgroup of vestibular patients (p< 0.05, Kruskal Wallis, Mann-Whitney U). No influence of hearing loss was observed on the GHQ-12 score.

DD score.
In the group of 60 healthy subjects the DD score ranged from 0 to 28 (average 4). The most frequent symptoms were: “Déjà vu” (51%) and “Difficulty concentrating” (46%), all the other symptoms were observed in less than one third of the subjects (Table 3, Figure 2). A similar result was observed in the subgroup of 12 age-matched subjects (Table 3). In the entire group, the total DD score was significantly related only to the age of the subjects, with younger subjects having higher scores than older subjects (r = -0.33, p= 0.01, Pearson Coefficient).

Across all patients, the range of the DD score was from 1 to 79 (average 19). The most frequent symptoms reported by the patients were arguably those closely related to their vestibular dysfunction, such as dizziness and the feeling of walking on shifting ground (Table 3). However, they also reported symptoms such as feeling “spacy” or feeling as though in a dream, which were more frequent than in healthy subjects (Figure 2). The DD score was not related to any of the general characteristics of the patients (described in Table 1), and no influence of hearing loss was observed.

Amongst the subgroups of patients, the DD scores were variable but this variability was related to the vestibular lesion subgroup (e.g. BPPV versus bilateral) and to the reporting of balance symptoms (Figure 3). The lowest frequency of symptoms and the lowest score among patients were found in patients with a unilateral canal paresis without balance symptoms, while the higher frequency of symptoms and the higher scores were found in patients with bilateral dysfunction. Compared to age-matched healthy subjects, all patients but those with unilateral canal paresis without symptoms showed higher scores (p< 0.01, ANOVA, Tukey honest significance difference) (Figure 3). However, no significant difference was observed between the DD scores of patients with BPPV and those with unilateral canal paresis without symptoms.
Table 3. Frequency of depersonalisation/derealisation symptoms reported by 60 healthy subjects and 50 vestibular patients.

<table>
<thead>
<tr>
<th>Depersonalisation-derealisation symptoms</th>
<th>Vestibular patients</th>
<th>Healthy subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BPPV</td>
<td>Unilateral without symptoms</td>
</tr>
<tr>
<td>1. Surroundings seem strange and unreal</td>
<td>38%</td>
<td>14%</td>
</tr>
<tr>
<td>2. Time seems to pass very slowly</td>
<td>7%</td>
<td>14%</td>
</tr>
<tr>
<td>3. Body feels strange or different in some way</td>
<td>46%</td>
<td>42%</td>
</tr>
<tr>
<td>4. Feel like you’ve been here before (déjà vu)</td>
<td>30%</td>
<td>21%</td>
</tr>
<tr>
<td>5. Feel as though in a dream</td>
<td>46%</td>
<td>7%</td>
</tr>
<tr>
<td>6. Body feels numb</td>
<td>30%</td>
<td>7%</td>
</tr>
<tr>
<td>7. Feeling of detachment or separation from surroundings</td>
<td>30%</td>
<td>28%</td>
</tr>
<tr>
<td>8. Numbering of emotions</td>
<td>23%</td>
<td>14%</td>
</tr>
<tr>
<td>9. People and objects seem far away</td>
<td>23%</td>
<td>7%</td>
</tr>
<tr>
<td>10. Feeling detached or separated from your body</td>
<td>15%</td>
<td>7%</td>
</tr>
<tr>
<td>11. Thoughts seem blurred</td>
<td>61%</td>
<td>14%</td>
</tr>
<tr>
<td>12. Events seem to happen in slow motion</td>
<td>23%</td>
<td>7%</td>
</tr>
<tr>
<td>13. Your emotions seem disconnected from yourself</td>
<td>15%</td>
<td>14%</td>
</tr>
<tr>
<td>14. Feeling of not being in control of self</td>
<td>46%</td>
<td>28%</td>
</tr>
<tr>
<td>15. People appear strange or unreal</td>
<td>0%</td>
<td>7%</td>
</tr>
<tr>
<td>16. Dizziness</td>
<td>100%</td>
<td>78%</td>
</tr>
<tr>
<td>17. Surroundings appear covered with a haze</td>
<td>30%</td>
<td>14%</td>
</tr>
<tr>
<td>18. Vision is dulled</td>
<td>38%</td>
<td>7%</td>
</tr>
<tr>
<td>19. Feel as if walking on shifting ground</td>
<td>69%</td>
<td>28%</td>
</tr>
<tr>
<td>20. Difficulty understanding what others say to you</td>
<td>23%</td>
<td>14%</td>
</tr>
<tr>
<td>21. Difficulty focusing attention</td>
<td>46%</td>
<td>28%</td>
</tr>
<tr>
<td>22. Feel as though in a trance</td>
<td>23%</td>
<td>0%</td>
</tr>
<tr>
<td>23. The distinction between close and distant is blurred</td>
<td>38%</td>
<td>7%</td>
</tr>
<tr>
<td>24. Difficulty concentrating</td>
<td>46%</td>
<td>35%</td>
</tr>
<tr>
<td>25. Feel as though your personality is different</td>
<td>15%</td>
<td>14%</td>
</tr>
<tr>
<td>26. Feel confused or bewildered</td>
<td>23%</td>
<td>7%</td>
</tr>
<tr>
<td>27. Feel isolated from the world</td>
<td>15%</td>
<td>0%</td>
</tr>
<tr>
<td>28. Feel “spacy” or “spaced out”</td>
<td>38%</td>
<td>28%</td>
</tr>
</tbody>
</table>
Further comparison of the responses from patients with unilateral canal pareses without balance symptoms versus those with balance symptoms was obtained with discriminant function analysis, by using the rating of each of the 8 items of the DD inventory which we previously found useful for discriminating between vestibular patients and healthy subjects [13]. These items were 1. “Surroundings seem strange and unreal”, 3. “Body feels strange or different in some way”, 5. “Feel as though in a dream”, 7. “Feeling of detachment or separation from surroundings”, 10. “Feeling detached or separated from your body”, 14. “Feeling of not being in control of self”, 25. “Feel as though your personality is different” and 28. “Feel spacy or spaced out”. The combination of these items discriminated 93% of the patients without balance symptoms and 77% of the patients with balance symptoms (Wilk’s lambda of 0.41, squared Malahanobis distance of 5.71, p= 0.01), confirming our previous results [13]. In this analysis, to “feel as though in a dream” was the most important factor, discriminating 93% of the patients without balance symptoms and the 77% of the patients with balance symptoms (Wilk’s lambda of 0.65, squared Malahanobis distance of 2.1, p= 0.001). However, when the 4 groups of patients were considered in this analysis, the same items discriminated the 93% of the patients with a unilateral canal paresis without balance symptoms but only 60% of the patients of each of the other 3 groups. These results show that the low frequency and severity of the DD symptoms included in the analysis can distinguish the vestibular patients withoutbalance symptoms from those with balance symptoms, but cannot differentiate clearly among the subgroups of symptomatic patients.

**Orientation updating data**

On the test of updating orientation, in the 60 healthy subjects, estimation errors were observed after 17.5% of all rotations (Figure 4); the typical magnitude of estimation errors was 45°, which was observed on 99% of all errors. Seventy percent of the subjects performed the test with up to 2 estimation errors; in which overestimation of rotation was more frequent than underestimation (84.7% versus 15.3%). The performance on the test was unrelated to the age or the gender of the subjects.

On updating orientation, across all vestibular patients, the average of the absolute values of the estimation errors was 27° (S.D. 34°). The average of the estimation errors of each subgroup of patients is shown in Figure 4. Patients with bilateral vestibular dysfunction had, as expected, the largest estimation errors of all participants; comparison among subgroups showed larger estimation errors in patients with bilateral vestibular dysfunction compared to any of the other subgroups of patients or to the age-matched subgroup of healthy subjects (p< 0.002, ANOVA, Tukey honest significance difference). However, patients with BPPV and those with unilateral canal paresis without symptoms showed similar results than healthy subjects.

In patients with unilateral canal paresis, estimation errors to the right or to the left did not show any particular trend according to the side of the canal paresis, this was probably because the passive rotations were within the range of rightwards-leftwards transduction of the intact canal [11, 23]. In all subgroups, overestimation of rotation was observed more frequently than underestimation, from 59% of the individual rotations with an estimation error of patients with bilateral dysfunction to 85% of the rotations with an estimation error of patients with a unilateral canal paresis without symptoms. The performance of patients with a unilateral canal paresis without balance symptoms and those with BPPV closely resembled the performance of healthy subjects (Figure 5). In these two subgroups, 68% of the patients performed the test with up to 2 estimation errors, with a typical error of 45° (96% of the time). However, none of the patients with a unilateral canal paresis with balance symptoms performed the test with fewer than 3 estimation errors. In this subgroup, estimation errors up to 180° were observed. In patients with bilateral dysfunction estimation errors were even larger (up to 270°). The performance on the test was unrelated to the age or the gender of the subjects.
Among subgroups of patients, the lowest symptom score and the lowest estimation errors were found in those with unilateral canal paresis without balance symptoms, and the bilateral vestibular loss patients showed the highest scores and the largest estimation errors. Across all patients, simple correlation analysis showed that the DD scores were related to estimation errors \((r = 0.49, p< 0.001, \text{Pearson Coefficient})\) and to the GHQ-12 score \((r = 0.3, p= 0.03, \text{Pearson Coefficient})\). However, covariance analysis showed a significant relation only with estimation errors (adjusted \(R^2 = 0.25, p= 0.002, \text{ANCOVA})

**DISCUSSION**

In this study, patients with peripheral vestibular disease showed higher DD scores and made larger errors in estimating their spatial orientation than healthy subjects. The highest scores and the largest errors in orientation were found in those patients who had bilateral vestibular dysfunction, followed by patients with a unilateral canal paresis and balance symptoms. A significant positive correlation was observed between DD scores and estimation errors; i.e. the more erroneous their spatial reorientation estimates were, the more the patient experienced DD symptoms. The deficit in vestibular signals alone could explain the difficulty on updating orientation without the assistance of vision. However, the relationship between estimating reorientation and the occurrence of DD symptoms suggests that the distorted vestibular transduction had a more wide ranging impact on the multisensory mechanisms involved with perceiving re-orientations in space.

Derealisation has been explained as the consequence of a mismatch between the models of memory and those of immediate experience [24]; where the latter can be modified by different circumstances, such as changing sensory input [25], drugs [26], alcohol [27], and social isolation [28], with consequences ranging from distortion of body image and space-time disorientation through to feelings of detachment from reality. In this view, it should not be surprising that patients with vestibular dysfunction experience DD symptoms since the vestibular system is the only sensory organ, which has evolved specifically to transduce orientation and reorientation in space. Accordingly, we observed that patients with peripheral vestibular disease may report symptoms of detachment from reality related to spatial disorientation, as evaluated with a simple task of estimating orientation with respect to the distinctive features of a room. Since updating visual space through vestibular signals can be inaccurate [29] the test was specially designed to minimise the feeling of laboratory testing, with no special devices, unusual instructions or motor response and thus resemble a daily life situation in which memorized visual information and vestibular signals are used for orientation. In agreement with previous studies on estimation of passive rotation, overestimation of rotation was more frequent than underestimation in the two groups, healthy subjects and vestibular patients [30, 31].

Evidence suggest that determination of the magnitude of head in space motions and coding a target position after such movements correspond to different cognitive tasks involving different neural substrates [29]. In this study, although reorientation estimates of vestibular patients were inaccurate they were not arbitrary; most disoriented patients gave responses which suggested that their perception of objects' spatial sequencing was preserved and that spatial memory persisted over disorientation. This result is consistent with the vestibular impairment causing a change in calibration of vestibular signals rather than a disorganisation of spatial processes at a more fundamental level.

The internal representation of space involves the integration of the different sensory inputs which yield reference frames [32, 33] and different brain regions, particularly the hippocampus [34, 35] and the posterior parietal lobe [36]. Studies in humans [37, 38] and primates [39] support the importance of a parieto-hippocampal network for spatial navigation. Representation of objects as well as allocentric locations are present in the hippocampus where neurons can respond to unique
combinations of objects and places [34]. Experimental evidence supports the notion that vestibular signals are critical for hippocampal spatial activity [40]. Specifically, human imaging studies showing that acquired chronic bilateral vestibular loss can be related to selective atrophy of the hippocampus [41] and spatial memory and navigation deficits [42]. The posterior parietal lobe is implicated in providing egocentric representations [36] by using different sensory inputs to determine the position of the body in space [43], with signals from the vestibular system and neck proprioceptors playing a primary role [32, 44].

A reorientation in space normally gives rise to a number of sensory signals (haptic, visual, vestibular), yielding reference frames that are not based on individual peripheral sensory codes [45], being organized instead in ego-centered (e.g. head, trunk, arm) and environment-centered coordinates [36]. As we execute a motor plan, the sensory feedback of the movement concurs with what we have learned to expect from experience [46, 47]. Accordingly, a veridical perception of orientation and reorientation movements is created which can guide effective sensory motor control. In the case of vestibular dysfunction, head movement provokes vestibular signals that are poorly calibrated to actual movement having a different dynamic range from normal and possibly rightwards-leftwards asymmetries. Thus, patients’ vestibular signals of reorientation indicate different magnitudes of body displacement and velocity than their visual and somatosensory signals. Unless the patient "ignores" the distorted vestibular signals from the process of evaluating spatial orientation [48] a sensory incongruity or mismatch is created and the perception of orientation becomes less sure. Our data suggest that such mismatch might lead to a sense of derealisation; in words patients often use, ‘being not completely in touch with the world’.

In this study, as judged by both DD scores and estimation errors, symptomatic patients with unilateral canal paresis were more impaired than asymptomatic patients. Of note, the latter had a similar performance to that of healthy subjects, despite their abnormal caloric responses. These findings suggest that derealisation may persist with incomplete clinical recovery. However, the finding that BPPV patients had similar DD scores than those observed in patients with unilateral canal paresis with or without symptoms, but all of them had lower scores than patients with bilateral vestibular dysfunction, supports the notion that both the experience of movement hallucination (e.g. vertigo, oscillopsia) and the primary vestibular dysfunction may have an impact on the occurrence of derealisation symptoms. Still, one must consider that, since only updating orientation in the axial plane was evaluated, patients with BPPV and normal caloric responses had higher DD scores than healthy subjects, but similar estimation errors. Other limitations of the study are that we evaluated the patients just at one point in time during the clinical evolution of their disease and that we did not measure the functional or physiological vestibular compensation on each of the subgroups of patients. Although, in patients with vestibular impairment evidence has shown poor correlation between the physiologic and functional measures of self-perceived disability/ handicap [49, 50] research on vestibular compensation has concentrated mostly on reflex and brainstem function. The results of this study indicate the need for further research on the cortical basis of recovery from a peripheral vestibular lesion and on the perceptual and cognitive processes related to vestibular function.

In summary, patients with peripheral vestibular disease have a deficit in the ability to update orientation on the environment and a high prevalence of DD symptoms, which may imply a high order effect of the vestibular impairment. We propose that a failure to effectively re-calibrate the disordered vestibular signal in the sensory integration that underlies stable perception of orientation is a major factor in the emergence of depersonalisation/ derealisation symptoms in vestibular patients.
Acknowledgements. This research was supported by the Medical Research Council of the UK. KJR was supported by Instituto Mexicano del Seguro Social and DG by the Dix foundation.

No competing interests

Procedures in the study were approved by Riverside Research Ethics Committee of The Hammersmith Hospitals Trust (RREC 3642)

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FIGURE LEGENDS

Figure 1
To perform the orientation updating test, subjects sat upright in a rotating chair, in the centre of a square room, which contained visually distinctive features positioned in the middle of each wall.

Figure 2
Comparison between vestibular patients (n=50) and healthy subjects (n=60) of the 10 most frequent depersonalisation/ derealisation symptoms reported by vestibular patients; *= p<0.05.

Figure 3
Mean, standard error of the mean and 95% confidence interval of the mean of the depersonalisation/ derealisation score of 12 healthy subjects and patients with BPPV (n=12), bilateral vestibular dysfunction (n=11), unilateral canal paresis with balance symptoms (n=13) and unilateral canal paresis without balance symptoms (n=14).

Figure 4
Mean, standard error of the mean and 95% confidence interval of the mean of the average estimation error of 12 healthy subjects and patients with BPPV (n=12), bilateral vestibular dysfunction (n=11), unilateral canal paresis with balance symptoms (n=13) and unilateral canal paresis without balance symptoms (n=14).

Figure 5
Mean and standard deviation of the mean of the estimation error after each rotation of patients with BPPV (n=12), bilateral vestibular dysfunction (n=11), unilateral canal paresis with balance symptoms (n=13) and unilateral canal paresis without balance symptoms (n=14). The data for 12 healthy subjects are also shown.
References


POSTER OF A BABY

DOOR

COMPUTER

ROTATING CHAIR

BLACK CURTAIN
Dizziness as if walking on shifting ground

Difficulty concentrating

Thoughts seem blurred

Feel "spacy"

Difficulty focusing attention

Feel as though in a dream

Detachment from surroundings

Not being in control of self

Body feels strange

Feel "spacy"

As if walking on shifting ground

Dizziness
Depersonalisation/derealisation symptoms and updating orientation in patients with vestibular disease

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J Neurol Neurosurg Psychiatry published online June 19, 2007

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