The world jumps up and down when I move
Oscillopsia and Gaze stabilisation in Multiple Sclerosis

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Background and Aim

- Gaze stabilisation mechanisms allow us to perceive a stable visual world when we move.
- The stabilisation of gaze in the light involves several mechanisms including the vestibulo-ocular reflex (VOR) as well as the smooth pursuit and optokinetic reflex.
- Gaze stabilisation can be assessed using clinical and laboratory based tests.
- Multiple Sclerosis (MS) causes dizziness in 63% of cases with true rotational vertigo being reported in 6-20% of cases. Demyelination within the brainstem and adjacent structures can affect the gaze stabilisation pathways.
- Poor gaze stabilisation will result in the symptom of oscillopsia when the visual world moves with head motion.

This study aimed to describe the prevalence of subjective reports oscillopsia in people with MS reporting dizziness and explore the relationship of oscillopsia to clinical and laboratory based measures of gaze stabilisation.

Methods

People were recruited from local Multiple Sclerosis clinics in the South West, UK. People were eligible if they scored 1-5 on Patient determined disease steps and reported one of the following at least 4 times/month:

- feeling that things are spinning or moving around
- a feeling of being light-headed, “swimmy” or giddy
- feeling unsteady and about to lose balance

Dynamic Visual Acuity

Binocular visual acuity was assessed using an EDTRS chart 2 m away.

Visual acuity was assessed with the head static and with manual passive motion of the head (40^o amplitude , 1.5 Hz).

The test was stopped when participants reported x3 consecutive incorrect.

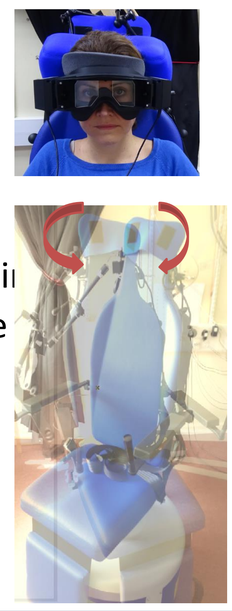
The logMAR score was calculated from the point of test cessation and included any previous incorrect scores.

The change in scores between static and head motion conditions was calculated.

A change of >0.2 is considered abnormal.

Gaze stabilisation using VNG

Eye movements were recorded using a videonystagmography (VNG) system (Micromedical,USA). Participants were rotated sinusoidally (40^o amplitude, 0.2 Hz) for 8 cycles. Participants were in complete darkness for testing in the dark and viewed a single dot for testing in the light. The gain of the response (eye movement velocity / chair velocity) was calculated.



Analysis

Differences in tests of gaze stablisation in people with and with out oscillopsia were assessed using an unpaired t –test.
Associations between the DVA and assessment of VOR in the light and dark were assessed using linear regression

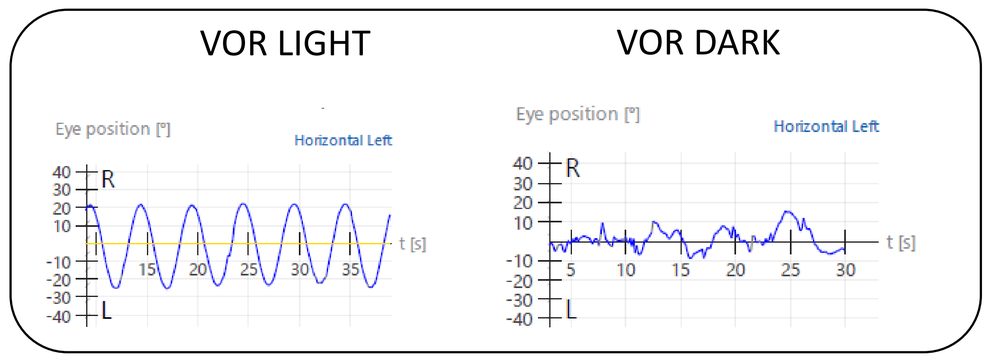
Results

- Oscillopsia was subjectively reported in 55% (n=15) of the 27 participants tested.
- The DVA resulted in an increase in of greater than 0.2 (2 lines on the EDTRS chart) in 85% (n=23) of participants.
- The gain of the VOR in the light was 103.7 (+/- 14.1) and in the dark was 60.7(+/- 20.07).
- People with oscillopsia had a larger DVA scores (indicating worse gaze stabilisation) than those who did not report oscillopsia (p=0.05). There was no difference in VOR-LIGHT and DARK scores in those that did / did not report oscillospia.
- The DVA score showed a weak association with the VOR light score (R^2=0.27 p<0.05).

Age(yrs) Mean±STD	55.70 (±10.58)
Diagnosis	21 RRMS; 3 PPMS; 3 SPMS
Male: Female	8:19
PDDS (median , Range)	3 (1-6)

Participant Characteristics

RR= relapsing Remitting; PP= Primary progressive SP= Secondary Progressive



Examples of eye movement response to chair rotation in the light and dark

Discussion and Conclusions

The DVA has been shown to be a reliable method of assessing gaze stabilisation. The DVA and VOR-LIGHT both measure the effects of the VOR as well as other gaze stabilising reflexes (smooth pursuit and optokinetic reflex). Without the visual reflexes the gain of the VOR decreases .Differences in the frequency of head motion used in the clinical and laboratory tests may underlie the weak association between the tests and different responsive to the presence of subjective oscillopsia. The DVA may be able to predict the presence of oscillopsia. Additional tests of smooth pu rsuit and optokinetic reflex are required to fully understand the causes of gaze stabilisation deficits in the light.

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