Oscillopsia: visual function during motion in the absence of vestibulo-ocular reflex

A B Morland, A M Bronstein, K H Ruddock*, D S Wooding

Abstract

***Objectives*—To investigate (1) the effects of loss of vestibular function on spatio-temporal vision and (2) the mechanisms which enable labyrinthine defective (LD) patients to adapt to oscillopsia.**

***Methods*—Visual function and eye movements were assessed in seven normal subjects and four LD patients with oscillopsia due to absent vestibulo-ocular reflex. Temporal vision was assessed by measurement of threshold sensitivity for detection of a target which moved across a flickering, spatially uniform background field. Spatial vision was investigated by measurements of threshold sensitivity for the detection of a target moving across a spatially modulated background in the form of square wave gratings. Velocity discrimination was assessed with drifting gratings. All measurements were made under static conditions and during oscillatory movement of either the visual stimulus or the subject (1 Hz, peak velocity 50°/s).**

Results

***Temporal responses*—Normal subjects and LD patients exhibited similar responses while static and under body oscillation.**

***Spatial responses*—The two groups achieved similar results under static conditions but body oscillation reduced threshold sensitivities and shifted the spatial response function towards lower spatial frequencies in the LD patients only. Similar changes in the spatial responses were seen during oscillation of the visual stimulus but these occurred in both normal subjects and LD patients.**

***Velocity discrimination*—Two LD patients achieved normal velocity discrimination but the other two showed abnormal responses to visual stimulus movement; one displayed a loss of velocity discrimination during whole body oscillation, and the other mismatched the velocity of two moving grating stimuli.**

***Conclusions*—The changes in the spatial responses are attributed to the presence of retinal slip during visual stimulus motion in all subjects or body oscillation in the LD patients. It is concluded that any visual adaptation to oscillopsia achieved by the LD patients does not influence the measured spatial response functions, which arise at an early stage of visual processing. The abnormal velocity discrimination may relate to the progressive improvement in oscillopsia reported by LD patients.**

MRC Human Movement and Balance Unit (Section of Neuro-Otology), The National Hospital for Neurology and Neurosurgery and Physics Department (Biophysics), Imperial College, London, UK
A B Morland
A M Bronstein
*K H Ruddock
D S Wooding

Correspondence to:
Dr A Bronstein, MRC Human Movement and Balance Unit, The National Hospital for Neurology and Neurosurgery, 8-11 Queen Square, London WC1N 3BG, UK.

Received 13 May 1997 and in revised form 1 April 1998
Accepted 24 April 1998

*Deceased

(*J Neurol Neurosurg Psychiatry* 1998;**65**:828–835)

Keywords: labyrinthine defective; vision; vestibular; oscillopsia; eye movements

Patients with acquired bilateral loss of vestibular function often perceive an unpleasant blurring and jumping of the external world during active or passive head movements.[1 2] This symptom, known as oscillopsia, is a consequence of poor stabilisation of the retinal image during movement of the head.

Normally, image stability during head movements is maintained by slow phase compensatory eye movements generated by the vestibulo-ocular reflex. In its absence, labyrinthine defective (LD) patients rely on pursuit/optokinetic mechanisms[3 4] as well as cervico-ocular reflexes[5–8] to stabilise retinal images during head motion. However, as the frequency response and latency of these alternative systems is not sufficiently fast to compensate fully for the high frequency components of natural head movements,[3 9] retinal slip and oscillopsia often persist.

After the initially severe oscillopsia, LD patients develop compensatory mechanisms which reduce the intensity of this symptom.[3 6 10] The possible contributions of ocular motor mechanisms to the reduction of symptoms have been previously studied,[4 5] but, despite subjective improvement of the oscillopsia, eye movements never fully compensate for head motion and considerable retinal slippage of images persists.[6 10 11] Thus it has been suggested that in addition to improved ocular stability during head movements, LD patients may develop some perceptual adaptation to retinal slippage.[10] One of the aims of this study is to examine LD patients for possible visual adaptations which could mediate a subjective improvement in oscillopsia.

In addition to their clinical relevance, the experiments also have implications for normal visual function. Although it is accepted that optimum visual acuity requires a stable image on the retina,[12–15] the precise consequences of retinal slippage on spatial vision are not known. In particular, no studies have provided a direct comparison of spatial visual responses made during motion of the subject with those made during movement of the visual stimulus. In this study we report on visual responses attributed to peripheral and central visual stages in normal subjects and LD patients, studied under static conditions and under whole body and visual stimulus oscillation.

Methods

To assess peripheral visual responses, we measured the spatial and temporal response known as the spatiotemporal filter 1 (ST1) and spatiotemporal filter 2 (ST2) which reflect, respectively, the "sustained P" and "transient M" channels.[16–18] To assess central adaptation to retinal image motion we measured velocity discrimination.

MEASUREMENT OF SPATIAL (ST1 SPATIAL) AND TEMPORAL (ST2 TEMPORAL) VISUAL RESPONSE FUNCTIONS

These functions were measured by the background modulation method.[16 19] In this task the luminance of a moving target at which it can just no longer be detected by the subject is measured. This threshold value of target luminance, I_t is measured for a target moving across a background field which was modulated either in time (flickered) or spatially, in which case it appeared as a grating (fig 1). Although the average luminance of the background is maintained at a constant level, the target threshold luminance, I_t, varies markedly with the frequency of the background modulation, and a plot of I_t against the background modulation frequency yields a frequency response curve. For normal observers, a plot of log I_t against the spatial periodicity of the background grating peaks at 3 to 4 cycles/°, falling on either side to give a well defined band-pass response (fig 2). This response was attributed to the spatial tuning of a visual mechanism with sustained temporal characteristics.[16] Similarly, the target threshold luminance, I_t, for detection of a target moving across a flickering background peaks at a frequency of about 20 Hz, to give a well defined band-pass response ("ST2 temporal" response,[16] fig 3).

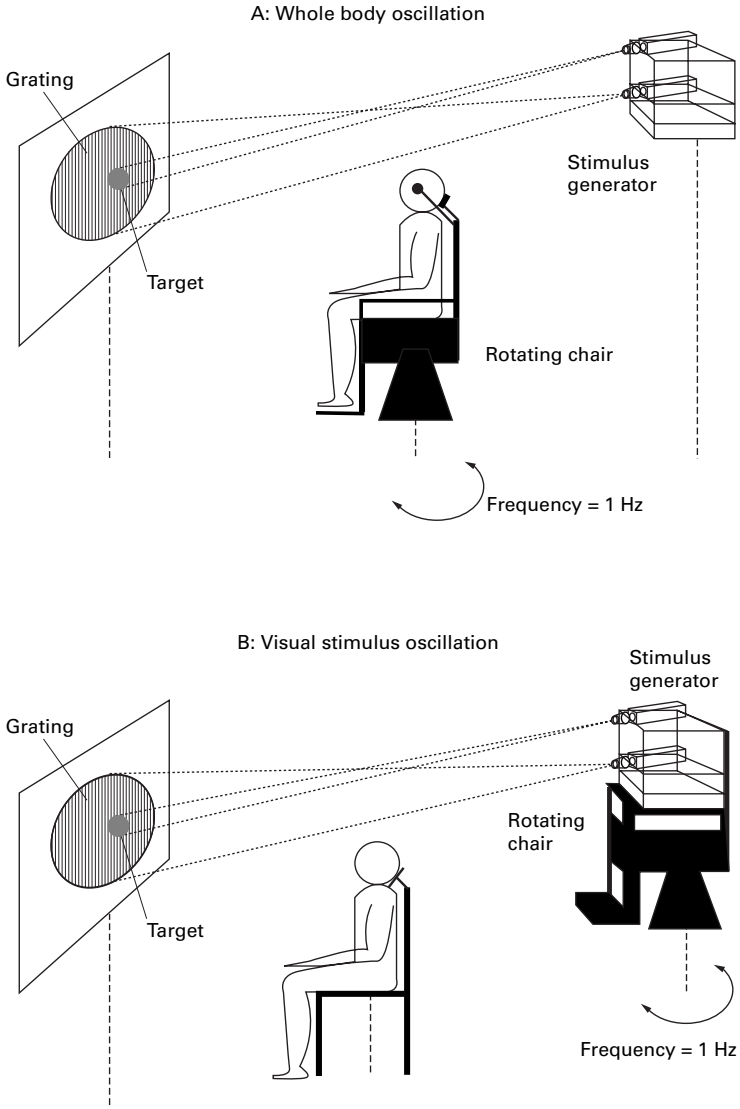

Figure 1 Experimental configuration used to determine the psychophysical response functions of spatial and temporal vision. (A) A schematic representation of the arrangement employed for subject motion and a static visual stimulus. The subject is seated in a rotating chair to which his or her head is clamped. The visual stimulus generator is static and provides the projected stimulus image on the screen. The stimulus image illustrated is that used to determine the ST1 spatial response, and consists of a grating background field, across which the target is moved. (B) experimental arrangement employed to generate visual stimulus motion with the subject static. The visual stimulus generator is fitted to the rotating chair and images the stimulus on to the reflective screen. The observer is seated on an office chair with a head rest. The distance between stimulus generator and screen was the same as that in A. The stimulus image shown is again that used to derive the ST1 spatial response. Measurements with both the subject and the visual stimulus stationary were made under both configurations A and B.

The light stimuli were generated by a purpose built optical system, incorporating two projectors providing independent optical channels, one for the target and the other for the background (fig 1). The images of the target and background were projected on to a white scattering screen. The circular target was imaged from a precision drilled metal aperture, driven mechanically across the object plane of the target projector to provide the target movement. The target luminance was controlled by two polaroid filters, one of which was fixed, whereas the other could be rotated. Spatial modulation of the background was achieved by placing a photographically produced, high contrast (>95%) square waveform grating in the object plane of the background projector. The luminance of the background field was kept constant with neutral density filters. Temporal modulation of the flickering background was achieved by a rotating sector disk, placed in a defocused position beyond the projector lens, and driven by a variable frequency motor. This arrangement provided high contrast (>90%) pseudosinusoidal modulation and relatively low harmonic distortion in the temporal frequency profile (<10%).

In all these experiments, the circular target was 3° in diameter and moved horizontally at 15°/s across the central 8° of background grating. The circular background was 17° in diameter, and of average luminance 1.8 log troland. The experimental procedure required the observer to respond "seen" or "not seen" in response to a single target presentation against the background, which was continuously visible. The target was set at a series of luminances around the threshold level, and a value of target threshold luminance was only recorded if at least three successive turning points (changes in subject response from "seen" to "not seen" or vice versa) were made within a luminance range of ±0.04 log units. This range also provides an upper limit of the experimental error. The luminances of all

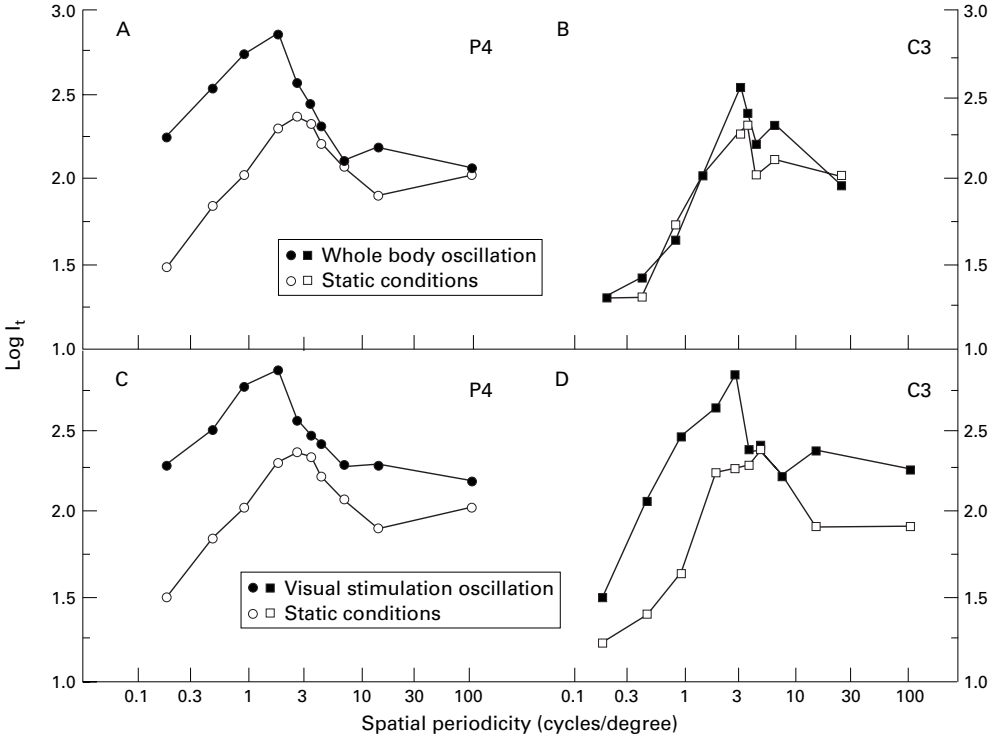

Figure 2 The ST1 spatial response measured for patient P4 and control C3. The logarithm of the threshold target luminance, log I_t, is plotted as a function of the spatial periodicity of the background grating. Data are given for whole body oscillation (filled symbols in A and B), and for visual stimulus oscillation (filled symbols in C and D). Responses obtained under static conditions are shown as open symbols in each panel.

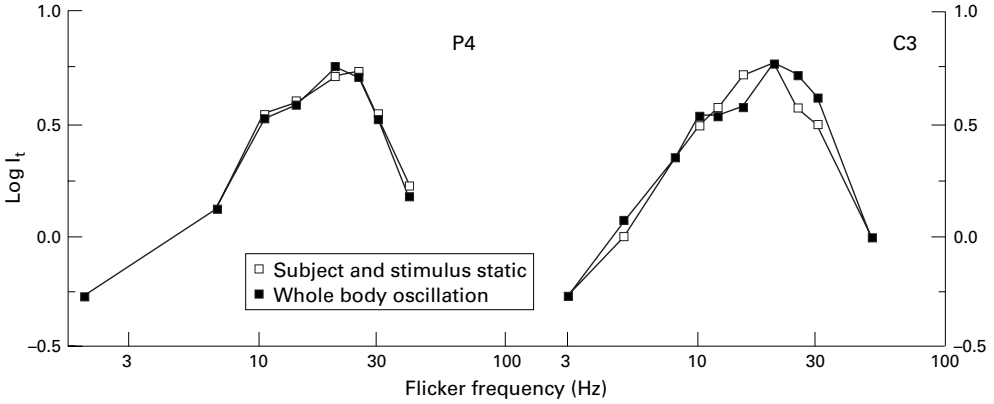

Figure 3 The ST2 temporal response for patient P4 and control C3. In each case the common logarithm of the threshold target luminance, log I_t, is plotted as a function of the background field flicker frequency. Open squares denote data obtained with both the subject and the visual stimulus static and filled squares denote data for the whole body oscillation condition.

stimuli were measured in situ with a Macam spectrophotometer/radiometer.

MEASUREMENT OF VELOCITY DISCRIMINATION

The light stimulus used for velocity discrimination measurements was a moving, sinusoidal grating of spatial frequency 0.72 cycles/°, which covered a circular area of 15° diameter. The stimulus was generated by an analogue grating generator driving a cathode ray tube (AVS-02 Faulkner Associates). A lens was positioned in front of the cathode ray tube so that the light stimulus was back projected on to a translucent screen, giving an average display luminance of 1 candela/m^2, and modulation depth 100%. The stimulus was arranged such that the grating bars were horizontal and moved vertically downwards. This grating orientation was chosen so that oscillation of the subject about a vertical axis produced no blurring of the grating bars in the image. Possible effects associated with the outer boundary of the stimulus were minimised by the introduction of a progressive reduction in the grating modulation from its maximum value at 1° from the edge of the field to 0% at the edge itself.

Each trial consisted of sequential presentation of two moving stimuli, the first at a fixed, reference velocity (v_r) of 5.75°/s, and the second moved at one of five different randomly selected test velocities (4, 5, 6, 7, and 8°/s). This was the highest velocity range which we could achieve with the equipment available and corresponds to the retinal slip velocities which occurred under whole body oscillation of the LD patients at 1 Hz (±50°/s). Each presenta-

tion was of 1 second duration, with a 1 second interval. During the interval separating the two grating presentations of each trial, and for periods between trials, the screen was set at a uniform luminance equal to the average grating luminance, which prevented the formation of after images. After each trial, the observer was asked to state whether the velocity, v_t, of the second grating was the "same as" or "different" from the first, reference velocity.

Velocity discrimination was quantified as the number of times the subject could not distinguish between the reference velocity and the test velocity (the number of "same" responses), as a function of the test velocity. This presentation of the data defines a number distribution in which the SD can be used as a representative value of the subject's performance. A subject with "ideal" motion discrimination would only reply "same" in response to a test velocity equal to the reference velocity (without variance in his response). Thus a low value of the SD indicates good velocity discrimination and vice versa.

EYE MOVEMENT RECORDINGS

Eye movements were recorded on a PC with DC coupled bitemporal electro-oculography (EOG). The EOG signal was filtered at 70 Hz and numerically differentiated with an interactive program.

The visual stimulus was one of the projected gratings used in the measurements of the spatial frequency response function (0.72 cycles/°), with a 0.1° fixation point at its centre. The observer was instructed to maintain fixation on this spot as accurately as possible and eye movements made during 30 cycles of subject and visual stimulus oscillation were recorded. Results were expressed as gain, the ratio slow phase peak eye velocity/peak stimulus velocity.

OSCILLATORY MOVEMENT OF THE SUBJECT AND THE VISUAL STIMULUS

Subjects were oscillated around a vertical axis by means of a chair mounted on a velocity controlled motorised turntable (Contraves-Goez, 120 Nm); head and trunk were firmly clamped in an upright position to the chair (fig 1 A). For oscillation of the visual stimulus, the optical projection system was locked to the rotating chair, with the observer seated stationary on an office chair fitted with a head rest (fig 1 B). In these experiments the observer to screen and projector to screen distances were equal to those employed when the observer was oscillated. The following motion conditions were investigated (1) Static: ST1 spatial, ST2 temporal, and velocity discrimination; (2) whole body oscillation: ST1 spatial, ST2 temporal, velocity discrimination, and EOG; (3) visual stimulus oscillation: ST1 spatial and EOG. All oscillations were at 1Hz, peak velocity 50°/s, and amplitude ±8°.

SUBJECTS

Three male patients P1, P2, and P3 (46, 63, and 31 years old respectively) and a 40 year old female patient (P4) were studied. P1, P2, and P3 sustained loss of vestibular function as a result of bacterial meningitis, P1 and P2 4 years before this study, P3 20 years before. P4 had neurofibromatosis type II and underwent acoustic neuromectomies 6 and 4 years before this study. The three patients who had acute vestibular loss due to meningitis initially reported severe oscillopsia which gradually lessened over time such that they no longer reported it voluntarily. The patient P4 had not experienced sudden onset of oscillopsia but, in common with the other patients, she reported it on specific questioning. Absence of vestibular function was established with velocity step rotation at 80°/s in the dark, and bithermal caloric tests, with and without visual fixation, at 30°C and 44°C. During the experiments subjects did not volunteer, nor were they questioned about the presence of oscillopsia. A total of 10 normal subjects (ages 25-55 years) were used as controls, a maximum of seven for each experiment. Subjects wore their refractive correction during the experiments, achieving visual acuity of 6/6 or better. Informed consent was obtained from all subjects.

Results

The results are presented in separate sections according to the visual function under investigation.

ST2 TEMPORAL RESPONSES

Representative ST2 temporal responses are shown for one of the three LD patients, P4, and a control subject, C3 (fig 3). Threshold luminance I_t, was plotted against the temporal frequency of the background flicker and the data show that the response functions were similar for patient and control subjects. They also established that there were no differences between the responses recorded under static conditions and under body oscillation for either the LD patients or controls.

ST1 SPATIAL RESPONSES

Representative data for the ST1 spatial responses are shown in fig 2. Responses are given for one patient, P4 (fig 2 A, C), and for a control, C3 (fig 2 B, D). Measurements made with the observer oscillating and the stimulus stationary were compared with those obtained with both the observer and stimulus stationary in fig 2 A, B. For the control, the spatial response functions obtained under the two conditions were very similar, with a peak in threshold luminance at around 3 cycles/° and similar frequency band widths. By contrast, threshold luminances for the patient are raised during whole body oscillation relative to the values obtained with the observer stationary. The increase is particularly marked at low spatial frequencies and causes a displacement of the peak response to lower spatial frequencies. The data obtained with the visual stimulus oscillated and the subject stationary are plotted in fig 2 B and D, together with the data measured with both the visual stimulus and subject stationary. It is clear that oscillation of the visual stimulus increases threshold

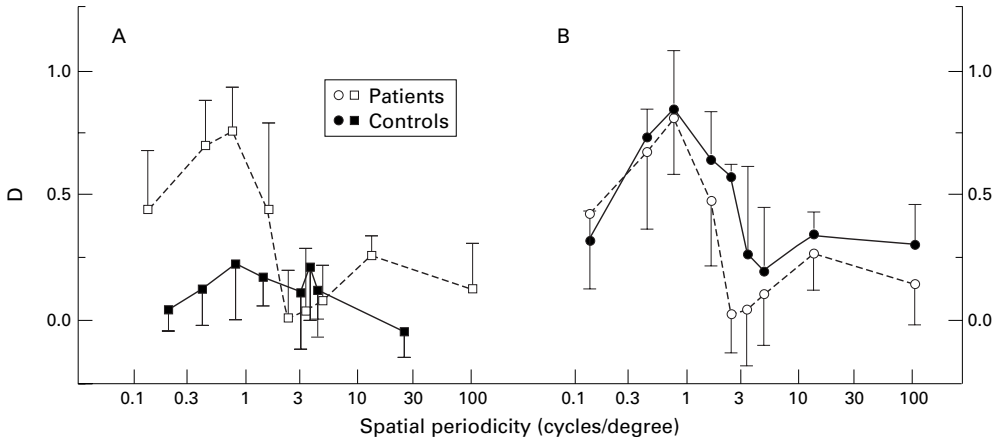

Figure 4 (A) Difference values, D, between the logI_t value measured under whole body oscillation and that measured under static conditions. Mean values for the four patients (open squares) and four controls (full squares) are plotted with SD as a function of spatial frequency. (B) As (A), but in this case data points are the difference between logI_t values for visual stimulus oscillation and static viewing conditions.

luminances for both the LD patient and the control subject, and that these increases are greatest for low spatial frequencies.

In fig 4 we present group averaged data of the differences between the pair of log I_t values under motion and static conditions, at each spatial frequency. The means and SD of the values for the four LD patients and those for the four controls were plotted against spatial frequency. During whole body motion (fig 4 A), the patients have higher thresholds than under static conditions. This increase in threshold is particularly marked for spatial frequencies below 3 cycles/°. There is little change in the threshold values obtained for the control group when whole body motion is compared with static conditions (fig 4 A). Figure 4 B shows comparisons between differences in thresholds measured during visual stimulus oscillation and stationary viewing conditions. The means and SD of the values for the two subject groups are very similar. Of particular note is the close similarity between the two functions in fig 4 B and the patients' function of fig 4 A.

To determine the effect of the changes in the ST1 spatial function during whole body oscillation on the patients' visual acuity, we generated a spatially filtered acuity chart. The filtering was achieved by the Fourier methods described in the appendix. The calculation was undertaken for patient P3 who was presented with the original chart shown in fig 5 A. His acuity under oscillation was 6/6, corresponding to the line OCMR, whereas when static he could read all lines of the normal chart, which defines 6/4 acuity. He was shown the blurred chart (fig 5 B) under static conditions and achieved 6/6 acuity, identical to that found under whole body oscillation with the original chart. The spatially filtered chart in fig 5 B, therefore, provides a reasonable representation of the blurring experienced by the patient during body motion at 1Hz.

VELOCITY DISCRIMINATION

Table 1 lists the velocity discrimination data as the SDs of the velocities of the test grating which seem to match that of the reference

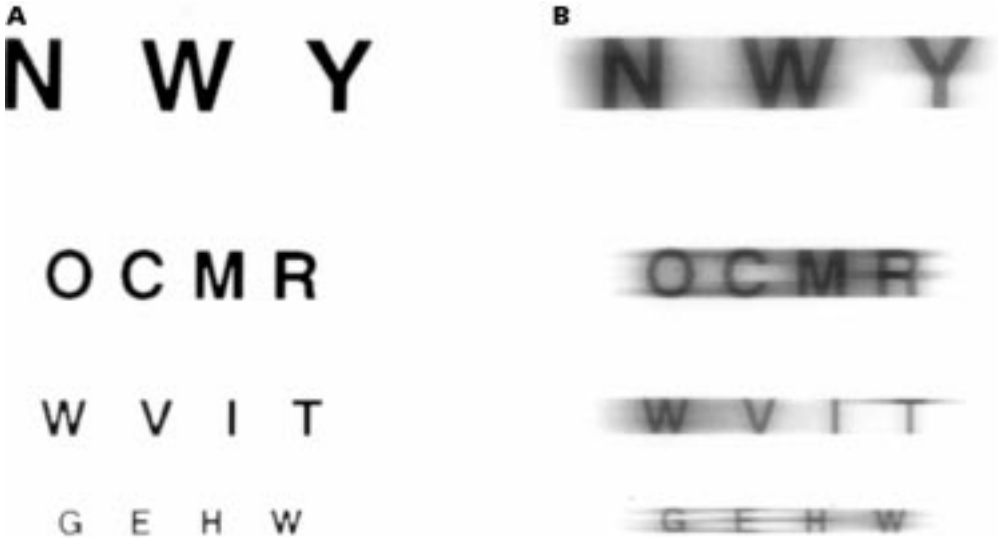

Figure 5 Computer generation of an acuity chart in original unfiltered format (A) and blurred (B) to take into account the change in the ST1 spatial response brought about by whole body oscillation for patient P3 (see appendix). The lines of the acuity chart correspond to 6/9, at the top, followed by 6/6, 6/5, and finally 6/4 at the bottom.

Table 1 Standard deviations (SD) of the number of distributions of the test velocities which the subjects perceived as the same as the reference velocity (data are given for each of the labyrinthine defective subjects, as well as the mean and range of the seven normal controls)

Subject	Static SD	Moving SD
P1	0.77	1.34
P2	0.65	0.84
P3	0.71	0.64
P4	0.52	0.72
Control mean	0.71	0.72
Control range	0.60 to 0.75	0.60 to 0.83

Table 2 Slow phase eye movement gain (peak eye velocity/peak stimulus velocity) obtained during oscillation of the visual stimulus (smooth pursuit) or of the labyrinthine defective subject (whole body oscillation)

Subject	Smooth pursuit Mean (SD)	Whole body oscillation Mean (SD)
P1	0.74 (0.12)	0.70 (0.10)
P2	0.85 (0.07)	0.82 (0.08)
P3	0.79 (0.04)	0.82 (0.07)
P4	0.86 (0.04)	0.88 (0.06)

grating (5.75°/s). Data are given for each of four LD patients and for the control group of seven. For three of the LD patients (P2, P3, and P4) the SD values were close to the normal range and, in common with the normal controls, did not change during body oscillation. The only significant increase in the SD value relative to those in the normal control group was found for P1 during body oscillation, indicating a loss of velocity discrimination (increased SD) during body oscillation. The distribution of velocities which were matched to the reference velocity had a low mean value for P4, a feature not seen in any of the other subjects. The mean values for P4 were 4.60 and 4.76°/s during stationary and whole body oscillation conditions respectively, whereas the total range of mean values were 5.69 to 6.27°/s (stationary condition) and 5.70 to 6.00°/s (whole body oscillation) for the other LD patients and controls. The mean values for P4 are, therefore, below the normal range for both experimental conditions.

EYE MOVEMENTS

Eye movement gains are presented in table 2. Due to the absence of vestibular function in the LD patients, compensatory eye movements in response to whole body oscillation can only be generated by the pursuit/optokinetic systems. It was expected, therefore, that gains during oscillation of the visual stimulus and of the body would be similar. The data in table 2 show that this was so for all of the LD patients.

Discussion

The rationale of our experimental procedure was to establish the change in visual function associated with the loss of the vestibulo-ocular reflex and identify any visual adaptation to oscillopsia. Thus, we initially determined visual responses, which are consistent with the sustained (P type) and transient (M type) visual mechanisms, to quantify the patients' visual loss. The transient and sustained information streams project to the visual cortex at which stage motion is first processed. So, in addition to the quantification of visual loss, the measurement of the spatiotemporal visual responses is important in localising the site of any visual adaptation as being beyond the dorsal lateral geniculate nuclei (dLGN). Measurements of velocity discrimination were selected to investigate if adaptation to oscillopsia involved central visual processing of motion.

The finding that the ST2 temporal response is unaffected by body oscillation in normal and LD patients is not unexpected as the flickering background is spatially uniform. This finding is important, however, because it establishes that detection of the moving target itself is not influenced by whole body oscillation. In addition, motion processing requires appropriate transient information to be encoded in the visual pathway. Adaptation to visual motion must therefore be expressed in mechanisms beyond the dLGN.

The ST1 spatial responses measured under static conditions for normal and LD patients are similar to those published previously in normal subjects, with a band-pass spatial response peaking at 3 to 4 cycles/°.[19 17] Holliday and Ruddock[16] associated this response with low level visual filters with receptive field organisation similar to that seen in X or P type retinal ganglion cells and neurons of the dLGN.[20 21] Oscillation of the subjects induced a large change in the spatial response functions in the four patients (figs 2 A and 4 A), with displacement in response peak towards lower spatial frequencies and increase in the luminance required to detect the target at and below spatial frequencies of 1 cycle/°(fig 2 A). This corresponds to a coarsening of visual responses—that is, greater sensitivity to coarse spatial frequencies than fine spatial frequencies. The result of being differentially more sensitive to the coarse spatial structure is modelled in the acuity chart (fig 5). In effect, the fine spatial information is swamped by the increase in sensitivity to the coarse, low spatial frequencies.

Oscillation of the visual stimulus (figs 2 C, D and 4 B) degraded spatial responses of normal subjects and LD patients alike. In this respect our results are in general agreement with measurement of threshold contrast of sine wave gratings during retinal slip.[22 23] In the patients, the retinal image motion caused by whole body and visual stimulus oscillation produced the same changes in threshold (fig 4). Because the relative motion between observer and visual display was identical in these two conditions it is concluded that for the patients, no compensation for the loss of the vestibulo-ocular reflex is effective in modifying this spatial response and the spatial response is, therefore, determined by the retinal image slip. This conclusion is consistent with the eye velocity gains of LD patients shown in table 2, essentially identical under body and visual stimulus motion, and with the drop in visual acuity when viewing a simulated modification of an acuity chart based on the changes in the ST1 spatial visual response (fig 5). This confirms that the spatial visual performance is directly limited by retinal image motion. Differences between the ST1 spatial responses during self and visual motion, reported in a pilot study in one patient[24] (P1 in this study), were

due to differences in the visual motion stimulus, which was viewed behind a static window in a Maxwellian optical system. The static window is likely to have reduced compensatory optokinetic eye movements[25 26] which in turn would degrade spatial vision.

Studies in patients with oculomotor disorders[27] or those with congenital nystagmus[28] suggested that dissociations between retinal slippage, oscillopsia, and visual function could occur. Some of the proposed strategies which patients may adopt to overcome the effects of retinal slip involve the exploitation of short periods of image stability, for example when the head is momentarily static in LD patients[6] or when the eye is static in patients with congenital nystagmus.[28] These previous studies, however, assessed spatial vision with high contrast acuity charts, which are continuously visible and give the patients opportunities to foveate during such short intervals of relative image stability. In our experiments, the moving target was presented transiently (0.5 s) which is likely to give subjects little chance of using special foveating strategies. We conclude that any central adaptation to oscillopsia does not influence the spatial responses measured by our methods either because the ST1 spatial response arises at an early (peripheral) processing stage in the visual system or because central adaptation is restricted to responses involving visual motion.

To assess central adaptation of visual function in the patients, velocity discrimination was measured under static and self motion conditions. The data for normal controls and patients P2, P3, and P4 indicate no difference in velocity discrimination when static or under self motion. One patient (P1), however, had a significant reduction in velocity discrimination when he was oscillated (table 1). It is unlikely that his loss of discrimination is due to retinal slip during whole body oscillation, because motion of the drifting grating was vertical and that of the subject horizontal. Thus any blurring would be perpendicular to the axis of the spatial structure. This conclusion is supported by the finding that two of the patients had entirely normal velocity discrimination despite the retinal slip caused by body oscillation. A further patient (P4) had normal velocity discrimination but she nevertheless displayed an unusual characteristic in this test. The distribution of velocities which she reported to be the same as the reference velocity was displaced to abnormally low speeds under static and self motion conditions. The data could be accounted for by a non-linear response leading to perception of all velocities below a critical value as equivalent to each other. We suggest that the reduction in velocity discrimination during self motion (P1) and the non-linear response (P4) make these two patients less sensitive to visual motion and thus represent forms of adaptation to oscillopsia. Using a different technique, similar conclusions were reached for patients with retinal slippage due to ocular motor disorders.[29] The differences in velocity discrimination among our patients indicate that the magnitude and type of the adaptation to long standing oscillopsia must be variable among LD patients. This finding has a counterpart in studies describing idiosyncratic ocular-motor mechanisms also compensating for bilateral vestibular loss.[3–5 10] In addition, occasional improvement in oscillopsia may be due to direct recovery of vestibular function.[30]

We are indebted to the patients and control subjects who participated in the study. We acknowledge the award by the Wellcome Trust of a research grant to KHR, which supported this study.

Appendix

We have used the ST1 spatial responses measured in our experiments to show the appearance of spatially structured images viewed by an LD patient undergoing whole body oscillation at 1Hz. The results are illustrated with reference to the acuity chart shown in fig 5. As motion is restricted to the horizontal (x) direction, we restrict analysis to the one dimensional case.

The problem is to calculate the image distribution $I'(x)$, which appears to a stationary patient identical to the original image, $I(x)$, viewed under oscillation at 1Hz with peak velocity of 50°/s. We assume that the only source of image degradation under motion is that due to filtering by the ST1 spatial mechanism. Let the ST1 spatial frequency response measured under static conditions be $F(s)$, and that measured under oscillation at 1Hz be $F'(s)$, where s is the frequency variable corresponding to the spatial variable x. Let the spatial frequency distributions corresponding to $I(x)$ and $I'(x)$ be $I(s)$ and $I'(s)$ respectively. Then:

$$F(s)I'(s) = F'(s)I(s) \qquad 1$$

gives the condition that the image $I'(x)$ viewed under static conditions, will give the same output from the ST1 spatial filter as the image $I(x)$, viewed under oscillation at 1Hz. Thus, we require the distribution

$$I'(s) = \frac{F'(s)}{F(s)} I(s) \qquad 2$$

$F(s)$ and $F'(s)$ are provided by the experimental data and, as noted by Barbur and Ruddock[19], these responses are linearly related to image contrast, so can be used in linear analysis. It is, however, necessary to calculate $I(s)$ from the image distribution $I(x)$ and this was achieved by Fourier transform methods.

To achieve this the acuity chart was defined as a 512×512 pixel grid and the intensity of a pixel at position x in a row was defined as $I(x)$, where x takes values of N between 1 and 512. The Fourier transform of the row can be calculated as:

$$I(s) = \sum_{x=1}^{N} I(x) \cdot e^{\frac{2\pi i(x-1)(s-1)}{N}} \qquad 3$$

The calculation was iterated for each row to give the complete 512×512 image reconstruction. Similarly, the spatial distribution $I'(x)$ corresponding to the frequency distribution I'(s) can be computed from the inverse Fourier

transform of $I'(s)$ given by:

$$I'(x) = \frac{1}{N}\sum_{s=1}^{N} I'(s) \cdot e^{\frac{2\pi i(x-1)(s-1)}{N}} \qquad 4$$

The acuity charts shown in fig 5 correspond to the light distributions I(x) and I'(x) calculated for patient P3.

1 Bender MB. Oscillopsia. *Arch Neurol* 1965;**13**:204–13.
2 Ford FR, Walsh FB. Clinical observations upon the importance of the vestibular reflexes in ocular movements. *Bulletin of Johns Hopkins Hospital* 1936;**58**:80–8.
3 Gresty MA, Hess K, Leech J. Disorders of the vestibulo-ocular reflex producing oscillopsia and mechanisms compensating for loss of labyrinthine function. *Brain* 1977; **100**:693–716.
4 Huygen PLM, Verhagen WIM, Theunissen EJJM, *et al*. Compensation of total loss of vestibulo-ocular reflex by enhanced optokinetic response. *Acta Otolaryngol Suppl (Stockh)* 1989;**468**:359–64.
5 Kasai T, Zee DS. Eye-head coordination in labyrinthine defective human beings. *Brain Res* 1978;**144**:123–41.
6 Chambers BR, Mai M, Barber HO. Bilateral vestibular loss, oscillopsia, and the cervicoocular reflex. *Otolaryngol Head Neck Surg* 1985;**93**:403–7.
7 Bronstein AM, Hood DJ. The cervico-ocular reflex in normal subjects and patients with absent vestibular function. *Brain Res* 1986;**373**:399–408.
8 Takahashi M, Saito A, Okado Y, *et al*. Recovery of gaze disturbance in bilateral labyrinthine loss. *ORL J Otorhinolaryngol Relat Spec* 1989;**51**:305–10.
9 Leigh JR, Dell'Osso LF, Kosmorsky GS. Relationships among oscillopsia, the vestibulo-ocular reflex and nystagmus. In: Sharpe JA, Barber HO, eds. *The vestibulo-ocular reflex and vertigo*. New York: Raven Press, 1993.
10 Bronstein AM, Hood DJ. Oscillopsia of peripheral origin: central and cervical compensatory mechanisms. *Acta Otolaryngol (Stockh)* 1987;**104**:307–14.
11 Zee DS, Yee RD, Robinson DA. Optokinetic responses in labyrinthine-defective human beings. *Brain Res* 1976;**113**: 423–8.
12 Westheimer G. McKee SP. Visual acuity in the presence of retinal-image motion. *J Opt Soc Am A* 1975;**65**:847–50.
13 Murphy BJ. Pattern thresholds for moving and stationary gratings during smooth eye movement. *Vision Res* 1978;**18**: 521–30.
14 Barnes GR, Smith R. The effects on visual discrimination of image movement across the stationary retina. *Aviat Space Environ Med* 1981;**52**:466–72.
15 Abadi RV, Sandikcioglu M. Visual resolution in congenital pendular nystagmus. *American Journal of Optometry and Physiological Optics* 1975;**52**:573–81.
16 Holliday IE, Ruddock KH. Two spatio-temporal filters in human vision. *Biol Cybernetics* 1983;**47**:173–90.
17 Grounds AR, Holliday IE, Ruddock KH. Two spatio-temporal filters in human vision II selective modification in amblyopia, albinism, and hemianopia. *Biol Cybernetics* 1983;**47**:191–201.
18 Coleston DM, Chronicle E, Ruddock KH, *et al*. Precortical dysfunction of spatial and temporal visual processing in migraine. *J Neurol Neurosurg Psychiatry* 1994;**57**:1208–11.
19 Barbur JL, Ruddock KH. Spatial characteristics of movement detection mechanisms in human vision. I achromatic vision. *Biol Cybern* 1980;**37**:77–92.
20 Enroth-Cugell C, Robson JR. The contrast sensitivity of retinal ganglion cells of the cat. *J Physiol (Lond)* 1966;**187**: 517–52.
21 De Monasterio FM, Gouras P. Functional properties of ganglion cells of the rhesus monkey retina. *J Physiol (Lond)* 1975;**251**:167–95.
22 Flip se JP, Wildt VD, Rodenburgh JG, *et al*. Contrast sensitivity for oscillating sin wave gratings during ocular fixation and pursuit. *Vision Res* 1988;**28**:819–26.
23 Burr DC, Ross J. Contrast sensitivity at high velocities. *Vision Res* 1982;**22**:479–84.
24 Bronstein AM, Ruddock KH, Wooding DS. Spatial vision during motion in the absence of vestibular functions in men. *J Physiol* 1992;**452**:P40.
25 Hood JD. Observations upon the role of the peripheral retina in the execution of eye movements. *ORL J Otorhinolaryngol Relat Spec* 1975;**37**:65–73.
26 Van Die GC, Collewijn H. Control of human optokinetic nystagmus by the central and peripheral retina: effects of partial visual field masking, scotopic vision and central retinal scotomata. *Vision Res* 1986;**383**:185–94.
27 Wist ER, Brandt Th, Krafczyk S. Oscillopsia and retinal slip. *Brain* 1983;**106**:153–68.
28 Dell'Osso LF, Leigh RJ. Foveation period stability and oscillopsia suppression in congenital nystagmus. *Neuroophthalmology* 1992;**12**:169–83.
29 Dieterich M, Brandt Th. Impaired motion perception in congenital nystagmus and acquired ocular motor palsy. *Clinical Vision Sciences* 1987;**1**:337–45.
30 Bronstein AM, Morland AB, Ruddock KH, Gresty MA. Recovery from bilateral vestibular failure: Implications for visual and cervico-ocular function. *Acta Otolaryngol (Stockh)* 1995; **Suppl 520**:405–7.