A new optical treatment for oscillopsia

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SUMMARY A simple optical device (spectacles plus contact lens) enabling viewing of the real world with either partial or almost-complete retinal image stabilisation has been tested in patients with oscillopsia caused by nystagmus. The device gave a useful improvement in vision in two of 14 patients. Reasons for success and failure were clear and are discussed. Obvious contraindications include severe optic atrophy, titubation and dementia. Net benefit is also unlikely if there is a good null point or area to the nystagmus, or if acuity (corrected but unstabilised) is 6/9 or better. It is not suitable for the treatment of oscillopsia caused by failure of the vestibulo-ocular reflex.

Some, but not all, patients with nystagmus experience a sensation of continuous oscillation of the visual world (oscillopsia), which can result in a marked loss of acuity owing to continuous retinal image-slip. Oscillopsia of this sort is not uncommon in multiple sclerosis.¹ Up to now, no effective treatment has been available for oscillopsia, with rare exceptions. Good results have been reported in periodic alternating nystagmus, using baclofen.² Occasional patients treated with isoniazid for rubral tremor in multiple sclerosis³ have also reported some improvement. Surgical treatment by extraocular myotomy or recession is destructive and often ineffective;⁴ it is not often used. Treatment of oscillopsia using retrobulbar injections of botulinus toxin has also been described,⁵ but this procedure has obvious dangers.

We report results in an unselected group of patients referred with oscillopsia, using a recently-developed method⁶ for stabilising the retinal image, using a wearable device (fig 1) which allows viewing of the real world. It comprises a high-minus contact lens (fig 2a) combined with a high-plus spectacle lens (fig 2b). Depending on the power of the lenses, this combination has a greater or lesser effect in reducing retinal image-slip as the eye moves. The simplest way to visualise the principle of the device is to consider a plus spectacle lens whose power is such that (if the eye's own optics are disregarded for a moment) a distant object on the lens axis would be imaged at the centre of rotation of the eye. Then, all rays from that object are radial to the eye after passing through the

spectacle lens; so whichever way the eye looks, a ray from that object is traversing the optic axis of the eye, and reaches the same retinal point. The high-minus contact lens is then needed to restore image focus to the retina.

Nystagmus, oscillopsia and central cancellation

Some patients with nystagmus may report only slight, intermittent or no oscillopsia, so evidently the brain can under some circumstances "cancel out" the retinal image-slip, with the effect that the visual world remains steady. This may be done by mechanisms akin to those that preserve the stability of the seen world during saccades and the fast phase of phys-

Fig 1 Diagrammatic representation of the optical method for obtaining retinal image stabilisation (RIS). For full RIS, the spectacle lens S is placed with its secondary focal point F' at the centre of rotation of the eye. When viewing a distant object AB, the lens would then converge all rays (such as b1, b2) from the point of interest B on its optic axis, so that they are radial to the globe. Then, when the eye is turned, rays from B will continue to traverse the optic axis of the eye, so that it continues to view that same point in space. The strongly divergent contact lens C is now required to diverge the rays sufficiently so that the image is formed at the retina, rather than in the vitreous.

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iological nystagmus. Many observers believe that this normal "central cancellation" mechanism involves outflow monitoring of oculomotor signals (ref 7, pp 237–41). In many other patients with nystagmus an imperfect attempt at central cancellation may occur, so that the amplitude of oscillopsia is smaller than would be predicted from the amplitude of the nystagmus. This phenomenon (oscillopsia smaller than retinal slip) has been well described and recorded in oscillopsia caused by a deficiency of the vestibulo-ocular reflex (VOR),⁸ and in downbeat nystagmus,⁹ but it may be more general.¹⁰ Besides cancellation, there are other possible mechanisms for reduced oscil-

lopsia, such as a raised perceptual threshold, or reduced sensitivity.^{8,9}

Patients whose oscillopsia amplitude is less than would be predicted from the amplitude of nystagmus then require only partial retinal image stabilisation (RIS) in order to eliminate oscillopsia by supplementing their central cancellation. In them it is necessary to measure the angular size ("N") of the nystagmus, and the angular size ("O") of the oscillopsia and compare the two to see how much RIS is needed. The necessary observations of O and N can be done semi-quantitatively without special equipment, so as to calculate O/N approximately for each patient (see *Methods*). The amount of supplementary RIS required to abolish the remaining oscillopsia is then given directly by O/N; RIS is on a scale running from 0 (normal; no stabilisation) to 1 (retinal image fully stabilised).

There are several assumptions implicit in this argument. They are firstly that O/N is relatively constant with time and eye-position for a given patient. Secondly, it is assumed that O is *caused* by the retinal image-slip resulting from N, so that it can be eliminated by reducing the image-slip. Thirdly, it is assumed that any cancellation signal is not greatly phase-shifted in relation to N. Fourthly, it is assumed that supplementary RIS will not itself alter the amount of central cancellation. These assumptions seem to be roughly borne out in practice.

Prescribing the correct amount of stabilisation

In theory, the amount of RIS applied should be O/N. In practice, owing to difficulties of lens manufacture, the choice available to our patients was limited to three values, approximately 0.3, 0.5 and 0.82. It is well known that perfect stabilisation (RIS = 1.0) of the retinal image leads to intermittent visual fading.¹¹ This problem does not arise with the RIS values used here. The nomogram (fig 3) shows the relation between contact lens power (Pc), spectacle lens power (Ps), separation of the principal planes of the lenses (Ds-c), and RIS, and it is used for prescribing the correct lenses. Given values for any two of the four variables, the other two can be read off the nomogram.

Methods

Lenses

The contact lenses (fig 2a) are made of conventional polymethyl methacrylate material, and are fitted to the cornea in the usual way. They are of 11.0–12.0 mm od, so as to extend almost to the limbus, for stability. The position of the contact lens has to be fixed relative to the cornea in spite of the nystagmus, if the RIS-system is to work. If the lens slips during eye movements, then retinal image-shift will occur, because of the prismatic effect of the contact lens. For the

Fig 2 (a) Contact lens. This example is -58 D, giving RIS = 0.82. It is made with a plane front surface, which is easy to form, and is made of conventional polymethyl methacrylate ($\mu = 1.5$). It weighs 55 mg. If the front surface is made to the largest radius available on a conventional contact lens maker's radius lathe, then the power is about -28 D (RIS = 0.5 approx). For this practical reason, lenses between -30 and -55 D have not been available to us. In order to obtain RIS = 1, the contact lens power would have to be about -80 D. If made of polymethyl methacrylate, this would require a concave front surface, which would be uncomfortable and fill with tears. Alternatively, the contact lens could be constructed to include an air lens. This would be difficult, bulky and expensive. Full RIS could be obtained using a contact lens with a plane front surface, if made of a material with μ approximately 1.8.

(b) Spectacles. For RIS = 0.82, spectacles of about $+32$ D are needed, and only a monocular view is possible (see text). A high-plus lens is put over the other eye both for appearance and so as to disable it.

Fig 3 A nomogram derived by geometrical optical calculation,¹² relating four variables: spectacle lens power (P_s), contact lens power (P_c), separation between spectacle and contact lenses (D_{s-c}), and RIS. It allows easy calculation of the spectacle and contact lenses required in order to achieve a given degree of stabilisation. The variable RIS as here used is related to rotational magnification (RM), as derived in ref 12, by:

$$RIS = \frac{RM - 1}{RM}$$

RIS is used rather than RM here, as RIS thus defined is (subject to assumptions discussed in the text) numerically the same as the fraction O/N , which is easily determined for each patient by observation of the amplitude of oscillopsia ("O") and the amplitude of nystagmus ("N"). To use the nomogram, the RIS required is calculated, and the distance from cornea to spectacle plane (D_{s-c}) is measured. Then, read along the line for the required RIS until it intersects the required D_{s-c} . At this point, read off the powers needed for the contact lens (P_c) and spectacle lens (P_s).

spectacle lens, the higher the power the more critical the positioning of the spectacle becomes, for focusing.

Measuring O and N

We use a simple semi-quantitative method for measuring O and N. The amplitude of nystagmus in the primary position is estimated ophthalmoscopically from the motion of the optic disc, assuming the disc diameter is 4–5°. The amplitude of oscillopsia is estimated using an Amsler chart (Hamblin's). If the chart is held at 29 cm, one square (0.5 cm) represents one degree. The patient is asked to say over how many squares the fixation mark appears to oscillate. This observation may not be practicable if corrected acuity is much worse than 1/60. Between 1/60 and 6/60 the fixation mark may need to be enlarged. The corneal radius is measured on a keratometer in the usual way. If O is about equal to N, then the appropriate 0.82 RIS contact lens from a trial set is inserted. A drop of local anaesthetic is not essential, but may help the patient to make careful observations of the effect of RIS on the oscillopsia, without distraction by initial contact lens discomfort. Acuity is measured (Snellen and reading charts), and residual oscillopsia if applicable (Amsler). If O is much smaller than N, then 0.3 or 0.5 RIS is tried first as appropriate. Over-stabilisation can result in reversed phase of oscillopsia.

Patients

The subjects reported here were an unselected series of 14 referrals (mainly from neurological clinics) of patients complaining of oscillopsia at rest. The table shows their nystagmus type, underlying conditions, corrected acuity, and acuity using the amount of RIS that best suppressed their oscillopsia. All patients gave their informed consent to the RIS trial procedure beforehand.

Results

Two of the 14 patients with oscillopsia gained worthwhile benefit from RIS (patients 4 and 14 in table). They both had a rapid pendular or rotary nystagmus present in all directions of gaze.

Table Patients

Patient	Age (yr)	Sex	Nystagmus type	Cause	N	O	VAU	VAS	RIS	Comments
1	35	F	Disconjugate pendular	MS	4	4	1/60	1/60	0.82	Severe optic atrophy
2	60	M	INO (coarse)	MS	0.5	0.5	6/18	6/18	0.82	Null area
3	41	M	Coarse pendular	CN	Not recorded		6/18	6/12	0.82	Oscillopsia only when tired
4	30	F	Fine rotary/pendular	MS	0.5	0.25	6/18	6/9	0.5*	Oscillopsia recovered
5	36	M	Rotary coarse	CN	4	1	6/36	6/18	0.5	Compensatory titubation
6	68	F	MSWJ, VP	PSNP	1	1 (VP)	6/24	6/24	0.82	Confusion
7	66	F	Gaze-paretic, coarse	BLUC	5	5	6/6	6/9	0.82	Null point (30 deg elevation)
8	26	F	Gaze-paretic, coarse	BLUC	4	2	6/9	6/9	0.5	Null point (30 deg left)
9	38	M	INO, coarse	MS	2	1	6/36	6/18	0.5	Optic atrophy, titubation
10	42	F	PAN	BLUC	2	1	6/4	6/9	0.82	Null point (swinging)
11	26	F	Pendular + INO	MS	2	2	6/36	6/36	0.82	Optic atrophy, titubation
12	33	F	Pendular + INO	MS	1	1	6/36	6/12	0.82	Optic atrophy, titubation
13	63	F	Gaze paretic, coarse	MS	2	0.5	6/9	6/9	0.5	Null point
14	43	M	Pendular + INO	MS	1	0.5	1/12	6/36	0.5*	Optic atrophy, titubation (mild)

*RIS of practical use.

Abbreviations:

INO—internuclear ophthalmoplegia with ataxic nystagmus; PSNP—progressive supranuclear palsy; BLUC—brainstem lesion, unknown cause; CN—congenital nystagmus; PAN—periodic alternating nystagmus; MSWJ—macro square wave jerks; VP—vertical pendular; O—oscillopsia amplitude (degrees); N—nystagmus amplitude (degrees); VAU—visual acuity, unstabilised (Snellen, corrected, better eye); VAS—visual acuity, stabilised.

Patient 4 had a fine (5–6 Hz), disconjugate rotary/pendular nystagmus of 2 months duration. RIS enabled her to read finer print (improved from N24 to N10), and she used it for watching TV for up to 2 hours at a time. After 4 months use, she suffered another brainstem lesion clinically, with vertigo, diplopia, increased weakness of the legs, and incontinence. Following ACTH she improved, and subsequently found that her nystagmus and oscillopsia had almost disappeared (N had fallen from 0.5 to 0.2°).

Patient 14 has had continuous oscillopsia for 10 years, caused by a 4 Hz pendular nystagmus present on all directions of gaze. He uses RIS mainly for reading the newspaper (reading acuity N20 without and N12 with RIS), and for watching TV (up to 4 hours a day; 8 months follow-up).

In the other 12 patients it was usually clear at the first trial that RIS was unlikely to help, and the reasons were all such as would be evident on routine neuro-ophthalmological examination. They are listed at the right of the table. Unfavourable features were: (1) Severe optic atrophy (patients 1, 11). Improvement in acuity with RIS was insufficient to enable reading. These patients had an associated relative central field defect and poor colour vision. (2) Severe titubation (patients 5, 9). Such patients need their VOR to stabilise their eyes; RIS would relieve them of oscillopsia due to nystagmus, but replace it with oscillopsia due to titubation. (3) Near-normal acuity, without RIS. Some of these patients had a coarse nystagmus with a correspondingly slow oscillopsia, which they could "see through" (patients 2, 7, 8, 9, 13). In addition, those with an ataxic (2, 9), gaze-paretic (7, 8, 13) or periodic alternating nystagmus (10), had a good null point or area, which they had learned to capture and use. Patient 3 with a coarse pendular congenital nystagmus had only slight or intermittent oscillopsia in spite of his obvious nystagmus. Patient 5 also with congenital nystagmus was amblyopic. Although patients with oscillopsia and near-normal acuity would welcome an end to their oscillopsia, the advantage to them does not in practice seem to outweigh all the visual side-effects of RIS, at least as seen by the patient on initial trial. (4) Dementia. Patients must gain useful function from RIS if they are to be expected to use it. Considerable insight and learning ability is needed to be able to learn to use vision as modified by RIS.

Discussion

This optical device magnifies the image as well as stabilising it, and the stabilised field of view (for nearly-complete RIS) is limited to about 30°. Using our lenses, there is an unstabilised view of the far periph-

ery, and a ring scotoma. The angular magnification (which amounts to about 1 Snellen line for $RIS = 0.5$, and nearly 2 lines for $RIS = 0.82$) is not taken into account in the acuity figures given here.

Normal saccades and head motion both cause perceived instability when using RIS, but for different reasons. Saccades are "doubly cancelled" (by the brain and then by RIS); so there is an illusion of movement in the direction of the saccade, but no retinal image-slip. The vestibulo-ocular reflex (VOR) on the other hand is disabled by RIS, so that during head movements the compensatory eye movements fail to prevent retinal image-slip. Vergence movements are also disabled, so in practice almost-complete RIS can only be used monocularly.

Some of these problems are much less severe if RIS of 0.5 or less is sufficient to abolish oscillopsia. Apparent motion during saccades quickly becomes surprisingly slight, perhaps through rapid adaptation of the cancellation mechanism by parametric feedback (ref 7, pp 244–6). Binocular vision is practicable. However, induced motion with head movement is still prominent, probably because it is not possible for the VOR gain to increase sufficiently to compensate.^{13–15} The VOR gain required for stability during partial RIS is numerically the same as the rotational magnification (for a definition of rotational magnification, see ref 12). The relation between RIS and rotational magnification is defined in the legend to fig 3.

It is our view that when RIS is considered in patients with oscillopsia at rest, there should be a specific functional aim. Such aims might be to restore the ability to obtain employment or enjoyment from activities such as reading, writing, recognising faces or watching TV. RIS can abolish oscillopsia, but it does not restore the visual world to normal. It is not useful as an aid to mobility, and should never be worn while driving. It is harmless, easy and quick to try out, and does not require expensive equipment.

The precise class or pathophysiology of the underlying nystagmus does not itself seem to matter, except in so far as it causes oscillopsia and disables vision. Ataxia of the arms is not necessarily a contraindication if the patient has help with placing and removing the contact lens. Favourable indications seem to be a VA of 6/18–6/60 in the better eye, with improvement of more than 2 lines on stabilisation.

Amongst patients with continuous oscillopsia who do not have the relative contraindications discussed, the success rate for optical RIS should be higher than in this unselected series of consecutive referrals.

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References

- 1 Brickner R. Oscillopsia: a new symptom commonly occurring in multiple sclerosis. *Arch Neurol Psychiat (Chic)* 1936;**36**:586-9.
- 2 Halmagyi GM, Rudge P, Gresty MA, Leigh RJ, Zee DS. Treatment of periodic alternating nystagmus. *Ann Neurol* 1980;**8**:609-11.
- 3 Satra AF, Hallett M, Sudarsky L, Mullaly W. Treatment of action tremor in multiple sclerosis with isoniazid. *Neurology* 1977;**32**:912-13.
- 4 Spalton DJ, Sanders MD, Gresty M, Rosen ES. Ocular myoclonus and oscillopsia. A problem of treatment. *Trans Ophthalmol Soc UK* 1980;**100**:507-8.
- 5 Crone RA, de Jong PT, Notermans G. Behandlung des Nystagmus durch Injektion von Botulinustoxin in die Augenmuskeln. *Klin Monatsbl Augenheilkd* 1984;**184**:216-7.
- 6 Rushton DN, Rushton RH. An optical method for approximate stabilization of vision of the real world. *J Physiol (Lond)* 1984;**357**:3P.
- 7 Carpenter RHS. *Movements of the Eyes*. London: Pion Press, 1977.
- 8 Wist ER, Brandt TH, Krafczyk S. Oscillopsia and retinal slip. *Brain* 1983;**106**:153-68.
- 9 Buchele W, Brandt T, Degner D. Ataxia and oscillopsia in downbeat-nystagmus vertigo syndrome. *Adv Oto-Rhino-Laryng* 1983;**30**:291-7.
- 10 Rushton DN. Oscillopsia and retinal image stabilization in patients with nystagmus. *J Neurol Neurosurg Psychiatry* 1986;**49**:729.
- 11 Ditchburn RW, Ginsborg BL. Vision with a stabilized retinal image. *Nature* 1952;**170**:36-7.
- 12 Rubin M. *Optics for Clinicians*. Gainesville: Triad Scientific Publishers, 1974:247-8.
- 13 Cannon SC, Leigh RJ, Zee DS, Abel LA. The effects of the rotational magnification of corrective spectacles on the quantitative evaluation of the VOR. *Acta Otolaryngol* 1985;**100**:81-8.
- 14 Gauthier GM, Robinson DA. Adaptation of the human vestibuloocular reflex to magnifying lenses. *Brain Res* 1975;**92**:331-5.
- 15 Gonshor A, Melvill Jones G. Short-term adaptive changes in the human vestibulo-ocular reflex arc. *J Physiol (Lond)* 1976;**256**:361-414.