Prospects for Treating Acquired Pendular Nystagmus with Servo-Controlled Optics

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Purpose. To determine whether a device featuring electronically controlled motor-driven prisms can reduce oscillopsia and improve acuity in patients with acquired pendular nystagmus (APN).

Methods. A device was developed that senses eye movements and, by the use of motor-driven prisms, oscillates the image of the world in lockstep with the pathologic nystagmus, to negate its deleterious visual effects. Unlike existing optical and surgical treatments for nystagmus, the device negates only the pathologic movements. Voluntary and normal reflex eye movements required for normal vision are unaffected. The benefits of the device were assessed by its impact on acuity in five patients with medication-refractory APN.

Results. All patients reported decreases in oscillopsia when the device was in operation. Averaged across patients, the device increased the percentage of time in which retinal image velocity was within ±4°/sec from 12.8% to 33.3%. Acuities improved in four of five patients, by an average of 0.21 logMAR units.

Conclusions. The symptoms of pendular nystagmus can be treated with a servomechanical device. Further refinements in the device should result in greater improvements in acuity, and a portable, wearable version is feasible using existing technologies. (*Invest Ophthalmol Vis Sci.* 2000;41: 1084–1090)

Acquired, involuntary sinusoidal oscillation of the eyes (acquired pendular nystagmus, APN) produces an illusion that the world is in motion (oscillopsia) and degrades the clarity of vision. Many drugs have been reported to treat this disorder, but efficacy is variable, often incomplete, and in some patients the condition does not respond to any agent.[1,2] Some patients respond but are unable to tolerate the medications on a daily basis because of adverse effects such as sedation or ataxia. Other treatment strategies, including weakening selected extraocular muscles with botulinum toxin or using spectacle–contact lens combinations to optically attenuate the visual consequences of the oscillation have been impractical and have not gained wide patient acceptance.[3,4] One of the disadvantages of existing surgical and optical treatments is that they impair normal reflex and voluntary eye movements as much as they reduce pathologic nystagmus. For instance, when using the spectacle–contact lens combination, the vestibulo-ocular reflex is nullified, and thus any head movements generate oscillopsia.

In the laboratory setting, acuity can be enhanced and oscillopsia prevented if the visual target (for instance, an acuity chart) is oscillated in lockstep with an APN patient's ocular oscillations.[5] This observation suggests a new way to treat APN through a goggles-mounted device that senses the patient's ocular oscillations and optically translates the image of the world, to stabilize the image on the moving retina. Using a prototype table-mounted device, we explored the feasibility of such an electromechanical–optical treatment for medication-refractory patients with APN. Preliminary results have been reported.[6]

Methods

We studied five patients with APN secondary to multiple sclerosis. All patients had previously received a variety of medical or surgical treatments for APN without resolution of nystagmus. All but patient 1 (see description later) had histories of optic nerve demyelination, and all had some degree of cerebellar ataxia. Four were wheelchair bound due to combinations of ataxia and weakness. This study conformed with the tenets of the Declaration of Helsinki, informed consent was obtained from subjects after explanation of the nature and possible consequences of the study, and the research was approved by the Institutional Review Board of the Cleveland Veterans Affairs Medical Center.

Device Electronics and Optics

The prototype device is capable of correcting vertical and horizontal sinusoidal oscillations in one eye. Its basic elements include a head-mounted eye position–sensing device; table-mounted, motor-driven prisms that allow the image of the world (as viewed through the prisms) to be shifted left-right and up-down; and an electronics package that uses the eye movement signals to control the prism motors. One of the authors models the device in Figure 1.

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FIGURE 1. One of the authors models the prototype device. He wears a pair of goggles that holds the infrared eye movement probe and looks through the table-mounted, motor-driven prism assembly. The edge of the support used to hold acuity test cards is visible in the foreground.

Eye movements in one plane (horizontal or vertical), are used to control the operation of the device in both horizontal and vertical directions, which takes advantage of the fact that in APN, horizontal and vertical components of the nystagmus are almost always phase locked.[7,8] Horizontal and vertical eye movements are tracked by a battery-powered infrared (IR) reflectance system (Series 1000; Microguide, Downers Grove, IL). This system has a linear range of up to ±20° and ±10° in the horizontal and vertical planes, respectively, typical horizontal-vertical cross talk of less than 20%, and a bandwidth of DC-100 Hz. The IR probe is mounted in a modified snorkeling mask.

Figure 2 shows a simplified block diagram of the device electronics. The circuit can be driven by either the horizontal or vertical eye movement channel. In practice, we selected whichever channel carried the strongest pendular nystagmus signal. The input eye position signal is differentiated (D/DT) and fed to a phase-locked loop, an oscillator that generates an

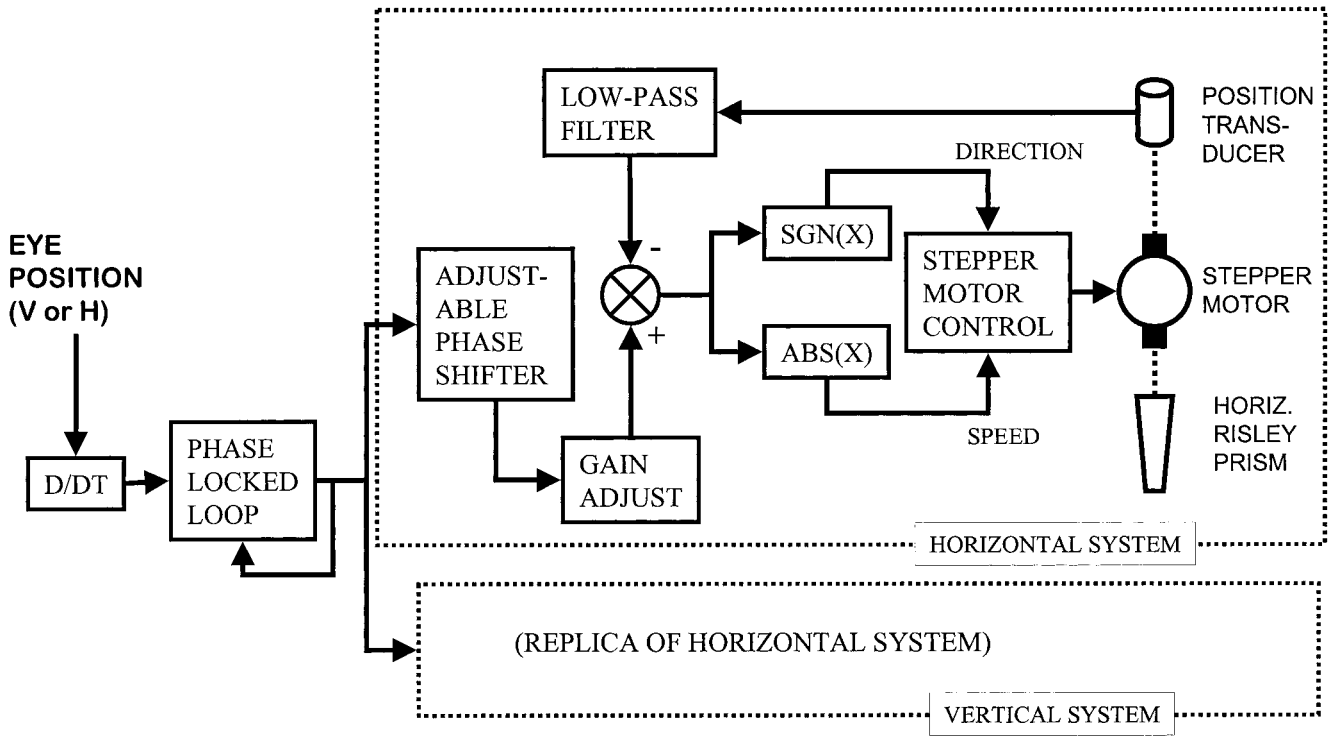

FIGURE 2. Simplified block diagram of the device electronics.

unmodulated (constant amplitude), quasi-sinusoidal signal with frequency and phase that are locked to the patient's sinusoidal nystagmus. The phase-locked loop is configured to lock to any oscillation in the approximate range of 2 to 10 Hz, with an acquisition time of two to three cycles. The loop's low-pass characteristics guarantee that momentary transients in the eye velocity signal (e.g., from small saccades) are not tracked. Likewise, slow eye movements related to the vestibulo-ocular reflex or smooth pursuit have essentially no effect on the output of the phase-locked loop. After the phase-locked loop circuitry, the control of vertical and horizontal movement channels of the device diverges. For each plane, the signal passes through an adjustable phase-shift network, which allows the phase of the prism motion to be adjusted to perfectly match the patient's oscillation. The phase-shifted signals are then fed to sign detection and absolute-value circuits, the outputs from which, respectively, supply the direction and speed commands to stepper motor controllers. Angular position of the prisms (which is transduced by potentiometers mechanically coupled to the motor shafts) is low-pass filtered (f_c = 0.8 Hz) and fed back to the control circuitry. The sign of this position feedback is contrived so that the circuit maintains average prism position centered at the point where angular deflection of the world (seen through the prisms) is nil.

The optics consist of two motor-driven Risley prism configurations, one for each axis (plane) of eye movement. Each Risley configuration consists of a pair of circular wedge prisms. When the prisms are counter-rotated around the optical axis, the image translates in the frontal plane. One pair of prisms is oriented to produce horizontal translations, and the other is oriented to produce vertical translations. The prototype system allows a circular field of view subtending approximately 10° of arc.

After passing through low-pass Bessel filters (corner frequency, 100 Hz), eye and prism position signals are digitized at 200 Hz with 16-bit resolution and recorded on a computer. Prism position signals are scaled to reflect the angular deflection of the world, as viewed though the prisms. The scale factor was determined by passing a laser beam through the prism assemblies, converting the linear displacement of the beam to angular displacement, and then correlating these angles with the amplitude of the position feedback signal.

Selection of Correction Axis and System Alignment

Although the prototype device is capable of simultaneously correcting both horizontal and vertical components of APN, in practice we corrected oscillations in one plane only (see the Discussion section). For each patient we chose the eye and treatment plane based on a number of considerations, seeking to study the eye in which the oscillations were most pronounced, most sinusoidal (i.e., minimally distorted by other motions such as superimposed jerk nystagmus), and closest to pure horizontal or vertical. We also wanted to avoid studying an eye that had severe degradation of vision due to concomitant optic nerve disease, although mild to moderate reduction of acuity due to optic nerve disease or refractive errors (patients did not wear correction during testing) should not affect the results, because they are based on the *change* of acuity produced by the device.

All eye movement calibrations and acuity determinations were performed as the patient viewed through the prism optics. The device's limited field of view required patients to restrict their head movements during testing. However, their heads were not restrained, and small head movements with consequent compensatory eye movements occurred. Throughout the testing, the untreated eye was patched. Vertical and horizontal calibrations were determined as the patient foveated targets at known angular positions. After calibration, the system phase was adjusted so that the prism and eye oscillations, as viewed on an oscilloscope, were in perfect registration. Next, the amplitude of the prism oscillation was adjusted in an iterative process until the patient reported optimum stability of the visual world.

Acuity Testing

After completion of the adjustment procedure described, the effect of the device was determined objectively by comparing patients' acuities with the device switched on or off. Acuity was tested using four-position, black-on-white Landolt C optotypes. Two different methods were used to present the optotypes. For subjects 2 and 4 and session 1 of subject 1, the optotypes were presented on cards, positioned 440 mm from the patient's eye. For subjects 3 and 5 and session 2 of subject 1, optotypes were displayed on the screen of a laptop computer (Powerpoint; Microsoft, Redmond, WA), positioned at distances of 120 to 150 cm from the patient. Twenty black optotypes of each size were presented on a white background, each for 3 seconds, with a fade-through-black transition between successive optotypes. For both methods of acuity testing, optotypes were converted to decimal acuity (taking into account the viewing distance), and the percentage of correct responses was scored.

Some patients with APN can temporarily attenuate the nystagmus by blinking, squinting, or making saccades.[7] Where applicable, patients were instructed to avoid these volitional maneuvers during acuity testing. Acuity testing can still be confounded in patients with APN because momentary attenuations of the nystagmus may still occur, allowing a patient to resolve an occasional optotype, particularly if each optotype is displayed for a prolonged time. These spurious correct responses make it difficult to assign a numerical acuity. The computerized optotype presentation was designed to reduce this problem in several ways: Each optotype was visible for only a short period (reducing the chance of a nystagmus arrest occurring), large numbers of optotypes were displayed (reducing the statistical effect of an occasional nystagmus arrest), optotypes were presented at a fixed position (obviating the need for foveating saccades that might induce a nystagmus arrest), and the gradual transition between optotypes eliminated temporal edges that might assist the patient in overcoming the blurring effect of the nystagmus.

Data Analysis

The frequency and amplitude of ocular and world (as viewed through the device) oscillations were determined by fitting sine waves to successive 1-second epochs of the record and the resultant frequencies and amplitudes averaged. Any epochs that generated spurious fits (due, for instance, to a saccade-associated shift in nystagmus phase occurring within the analysis epoch) were discarded. All saccades, blinks, and temporary cessations of nystagmus (generally occurring after blinks) were deleted before curve fitting.

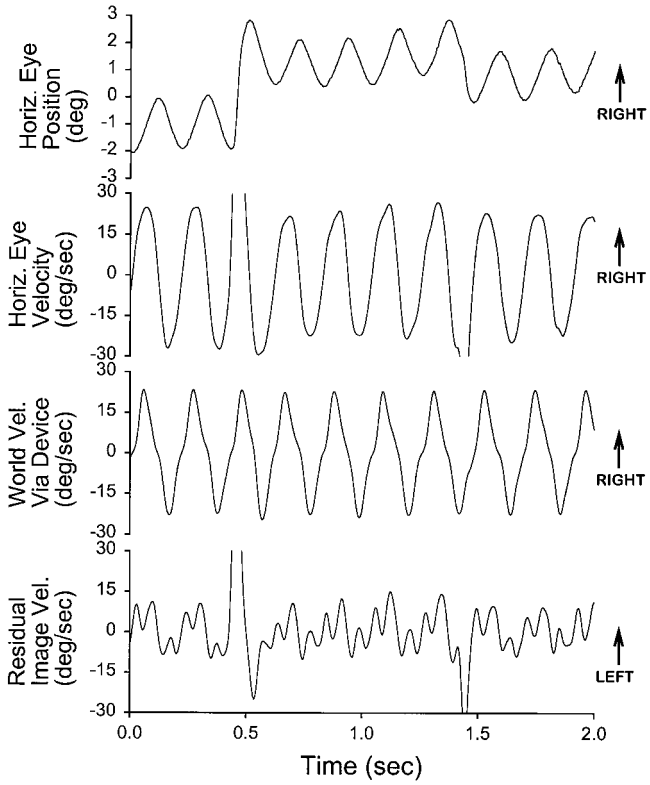

FIGURE 3. Record of horizontal eye and (apparent) world movements (due to the motion of the prisms) made during acuity testing in patient 1. Velocity scales are identical, to facilitate comparisons between traces. Velocities during the two small saccades exceed this scale and are thus truncated.

RESULTS

The most extensive testing was performed in patient 1, a woman with multiple sclerosis who had had APN for 6 years and had previously received little symptomatic relief from multiple treatments, including gabapentin, clonazepam, trihexyphenidyl, baclofen, and amantadine. The nystagmus was predominantly monocular, nearly perfectly sinusoidal, and essentially purely horizontal, with an average frequency of 4.7 Hz. Position amplitudes varied, tending to increase with fatigue. During testing, the 0-peak position amplitude averaged 0.7° (velocity amplitude of 21 deg/sec). The patient was not known to have ever had an episode of optic neuritis, and clinical examination revealed neither relative afferent pupillary defect nor optic disc pallor.

Figure 3 shows a 2-second segment of ocular oscillations in patient 1, recorded with the device in operation. In the absence of the device, the patient would experience a retinal slip velocity equal and opposite to her eye velocity. With the device in operation, the patient experienced only a residual image velocity (bottom trace), which was calculated from the difference between eye velocity and angular velocity of the world imparted by the movement of the prisms. Comparison of the eye and residual image velocity traces demonstrates how the device reduced peak-to-peak retinal image velocity by more than 50%. The record contains two small saccades, which had no effect on the ocular oscillation. Likewise, the prism oscillation was unaffected. This state of affairs is desirable, because the device should counter only the involuntary oscillations.

Patient 1 noted that the oscillopsia was consistently reduced, and the visual world appeared clearer when the device was operating. We assessed her acuity using Landolt C optotypes as described in the Methods section. Figure 4 plots the fraction of correctly identified optotypes as a function of optotype size expressed in terms of decimal acuity. The patient's accuracy was uniformly better when the device operated (filled squares) than when it was switched off (open circles). The accuracy data were fit with 3-term sigmoidal curves, and the points at which the fitted curves fell below 62.5% accuracy determined (62.5% being halfway between perfect accuracy and accuracy no better than chance). With the device on, accuracy declined below the 62.5% criterion at an optotype size of 0.64 versus 0.52 with the device off. This difference represents a logMAR change of 0.09 or a shift in the 62.5% accuracy point of just under one line on a logarithmic acuity chart.[9]

The results of device testing for all five patients (including two sessions performed by patient 1) appear in Table 1. For each patient and session, we tabulated the frequency of the pendular nystagmus, the 0-peak velocity amplitude of the corrected axis (either horizontal or vertical), and the amplitude of the other, nontreated axis. The device gain was calculated as the ratio of the velocity amplitudes of the prism and eye in the treated axis. In studies of the effects of retinal image slip, acuity begins to decline and oscillopsia is experienced when retinal slip exceeds 3 to 4 deg/sec.[5,10,11] For each subject, we selected portions of the eye movement records in which the device was continuously in lock and calculated the percentage of the recording in which the absolute value of eye (image) velocity was below 4 deg/sec. This calculation was also performed on residual image velocity (difference of eye velocity and world velocity). The device increased the percentage of time image velocity was below the criterion value in all patients by factors ranging from 1.4 to 4.8. The table also lists the percentage of cycles of the device that were in phase with a cycle of patient nystagmus, determined from a cycle-by-cycle review of 15 to

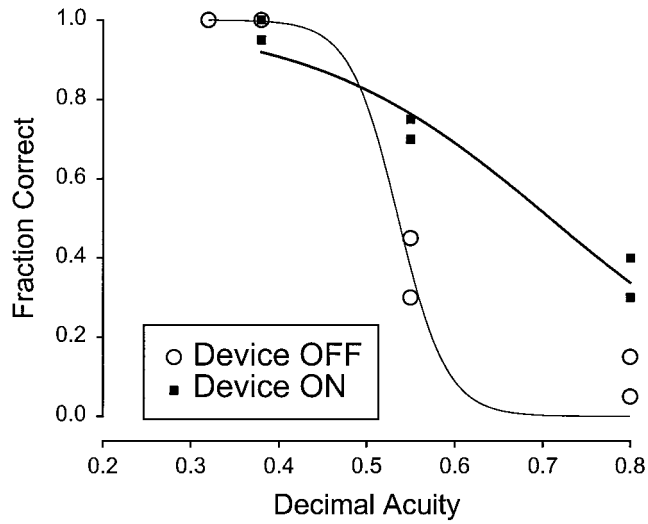

FIGURE 4. Plot of accuracy versus optotype size (expressed as decimal acuity) for patient 1, session 2. Three-term sigmoidal curves are fit to the data. The patient's acuity with device on or off is taken as the optotype size at which the fitted accuracy curve crosses 62.5% accuracy.

Table 1. Summary of Patient Characteristics and Results

Patient, Sex, Age	APN (y)	Eye*	Optic Rx†	Freq‡ (Hz)	Treated Axis, Amplitude§ (deg/sec)	Other Axis, Amplitude‖ (deg/sec)	Gain¶	Cycles Lock# (%)	Device Off (% < 4 deg/sec)**	Device On (% < 4 deg/sec)**	Acuity, Device Off††	Acuity, Device On††	logMAR Change
1 (session 1), F, 46	6	OD	+0.25	4.69 ± 0.09	H, 22.5 ± 1.2	V, 2.5 ± 0.6	0.86	97.0	11.0	52.5	0.25	0.38	0.18
1 (session 2)				4.65 ± 0.15	H, 19.5 ± 1.9	V, 5.7 ± 1.1	0.86	100.0	13.1	44.8	0.52	0.64	0.09
2, M, 41	9	OS	NA	3.76 ± 0.25	H, 25.2 ± 4.2	V, 15.4 ± 3.1	0.74	78.8	10.3	26.0	0.094	0.144	0.18
3, F, 50	NA	OD	−3.50	4.51 ± 0.22	V, 11.8 ± 2.3	H, 4.6 ± 1.4	1.09	68.5	21.0	50.0	0.121	0.136	0.05
4, F, 48	4	OS	−7.00	3.36 ± 0.10	V, 41.9 ± 8.1	H, 47.9 ± 5.1	0.17	63.2	7.8	10.6	0.046	0.135	0.47
5, F, 34	14	OD	−4.25	4.02 ± 0.11	H, 14.1 ± 2.4	V, 16.4 ± 2.9	0.57	53.9	13.0	31.0	0.151	0.154	0.01

V, vertical; H, horizontal; NA, not available.
* Eye that viewed through the device, drove the circuitry, and to which the tabulated nystagmus and acuity data apply.
† Optical correction normally worn by the patient (but not worn during device testing).
‡ Nystagmus frequency (mean ± SD).
§ Axis (vertical or horizontal) and 0-peak velocity amplitude of the nystagmus that the device attempted to nullify.
‖ Axis perpendicular to the treated axis.
¶ Ratio of world (viewed through the moving prisms) and eye angular velocities.
# Percentage of cycles of prism oscillation that were in phase with a cycle of patient nystagmus.
** Percentage of time in which the absolute value of residual image velocity fell within ±4 deg/sec.
†† Optotype size (expressed as decimal acuity) at which the patient read at 62.5% accuracy.

180 seconds of recorded data for each patient. The table also lists the optotype size (expressed as decimal acuity) at which patient accuracy decreased below 62.5%, based on curves fit to the plots of accuracy versus decimal acuity (as in Fig. 4). Patients 1, 2, 3, and 4 improved their acuities by logMAR values ranging from 0.05 to 0.47.

Patient 5 did not experience any improvement in acuity despite her reporting a decrease in the horizontal component of the oscillopsia and our having increased the percentage of time in which horizontal image velocity was below 4 deg/sec from 13% to 31%. The lack of improvement may stem from any of several causes. As shown in Figure 5, the pendular nystagmus was contaminated by a leftward–downward jerk nystagmus that would not be nullified by the device. The patient also had a tendency to blink frequently, breaking the device lock (only 54% of the cycles of prism oscillation were locked to a cycle of her nystagmus). The oscillation in the vertical plane, which we did not correct, was as large as the oscillation in the horizontal plane. The relatively low amplitude of nystagmus, together with the fact that she preferred a lower device amplitude setting, resulted in a low prism amplitude (8.0 deg/sec). Because of backlash in the prism mechanics, tracking fidelity decreased as prism amplitude declined into these low ranges.

Patient 4 had waxing–waning ptosis as well as deep-set eyes, which together degraded the transduction of the ocular oscillations and caused the device to break lock intermittently. Consequently, we tested her acuity using the printed rather than computerized optotypes, and we advanced the optotypes only after she had had approximately 3 seconds of viewing time with the device in lock. The periods of lock, which were associated with a rhythmic oscillation of the prism assembly, were easily detectable by ear. Patient 4 reported clearer vision, and her acuity improved despite her preference for a low device gain and the consequently minor increase in the percentage of time during which image velocity decreased below the criterion speed of 4 deg/sec. Her preference for a low device gain may reflect the fact that, unlike the other patients presented in this study, she experienced only blurred vision, not oscillopsia, due to APN. Increasing the device gain above approximately 0.20 actually produced oscillopsia and thus limited the degree of compensation that could be used.

Discussion

Our experience with the prototype device demonstrates its potential for treating patients with APN. The four patients with oscillopsia reported reduction of the illusory motion, and all but one of the five patients experienced acuity increases. These results are particularly significant in light of the fact that all the patients had previously failed or exhibited only incomplete responses to adequate trials of multiple medications for nystagmus. The advantage of an "active optics" treatment for nystagmus is that it should reduce retinal slip in any patient with APN, provided the predominant source of the retinal slip

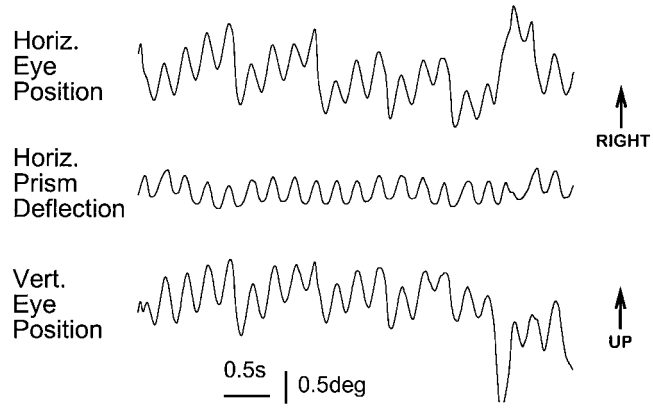

Figure 5. Record of eye and prism movement in patient 5, the one patient in whom the device did not improve acuity, although it reduced oscillopsia. Note the superimposed left–down jerk nystagmus and the comparable amplitudes of the treated (horizontal) and untreated (vertical) ocular oscillations. The patient experienced diagonal oscillopsia.

is a consistent sinusoidal oscillation. The device differs from existing nondrug treatments for APN (i.e., the spectacle–contact lens combination or weakening of extraocular muscles by surgery or botulinum toxin), in that it nullifies only the pathologic eye movement. As they viewed the acuity test, patients could shift gaze or move the head normally (within limits imposed by the 10° field of view of the table-mounted optics), because saccades and low-frequency (i.e., frequency content lying below the capture range of the phase-locked loop oscillator) vestibulo-ocular reflex movements were unaffected.

Although the prototype device is table mounted, most of the elements can be miniaturized easily, suggesting that a portable, wearable, battery-operated device could be developed. The infrared eye movement sensor is already battery powered, operates under any lighting conditions, and has circuitry that fits in a small box that could be worn by the patient on a belt. The prism-control electronics would fit easily in the same box and run from the same power source. Only the motor-operated prisms present a problem, because of their weight, power requirements, and need for bulky heat sinks. However, the problem could be solved by replacing the motor-driven Risley prisms by variable power prisms such as those used in stabilized binoculars (e.g., Vari-Angle prisms, Canon, Lake Success, NY). These assemblies are light, quiet, and operate from battery power. The variable prism is also more compact (longitudinally) than the Risley configuration and thus would allow a larger field of view for any given prism diameter.

The device should be operable by a patient without assistance. For the purposes of these experiments, it was important to obtain interpretable eye movement recordings, and thus the IR probe had to be positioned correctly and calibrated. However, in practice, no calibration would be performed, and alignment would be noncritical, because all that is required is a nystagmus signal of sufficient quality for the circuitry to achieve phase lock. Because nystagmus amplitude varies through the day, the patient would need to readjust the vertical and horizontal prism amplitudes periodically to maintain the preferred system gain. However, because the phase relationships between vertical and horizontal nystagmus components tend to remain stable from day-to-day in a given patient, the system phase controls would need only periodic adjustments, which may be performed in a physician's office.

Deficiencies of the Prototype Device

These experiments have identified a number of deficiencies in the prototype device that could be corrected, possibly leading to greater improvements in efficacy. The field of view was quite limited, subtending only 10° of arc. As noted, the field of view could be enlarged somewhat by using a more compact prism device. In addition, the constraints of the small field of view would be considerably mitigated by a head-mounted device. Based on the literature on adaptation to aperture goggles, we anticipate that patients would rapidly learn to substitute eye–head saccades for eye-only saccades, thereby maintaining visual targets within the field of view.[12–14]

Another deficiency of the prototype is apparent in Figure 3, in which it is clear that the velocity profile of the prisms is more triangular than sinusoidal; subtraction of this waveform from the more perfectly sinusoidal ocular oscillation produces a residual image velocity waveform with prominent ripples. As the device gain was increased toward 1.0, these ripples became apparent to subjects as a high-frequency jitter in the image and probably account for most patients' preferring a device gain significantly less than 1.0. The distortions stem from several sources, including mechanical backlash in the Risley prism gears and nonlinearities contributed by the behaviors of the absolute-value circuit around 0 volts, the simple resistor–capacitor phase-delay network, and the limited speed resolution of the stepper motor controller. Modifications to the system to increase the fidelity with which it mirrors eye velocity would reduce this jitter and may allow higher gains. The tracking inaccuracy may also explain why operating both prism axes simultaneously did not improve vision in patients with prominently circular–elliptical nystagmus (e.g., patients 2, 4, 5). Summation of the high-frequency errors from both axes would generate a complex two-dimensional jitter with high-image velocities. If tracking fidelity were improved, we should be able to operate both axes, again with potential for further improvements in efficacy. A third, rectifiable deficiency of the prototype was that it had no provision to disable its oscillation when it was out of lock. Thus, during transient losses of lock due to blinks, large saccades, or nystagmus arrests, the prisms continued to oscillate and actually increased retinal slip velocities. Disabling prism oscillation during these moments should improve measured acuities somewhat. A fourth deficiency of the device was that it did not permit patients to wear their optical correction. A practical device would be designed to incorporate the patient's optical prescription.

Experience with the prototype also revealed problems that will render challenging the creation of practical, wearable APN-nullifying goggles. First, the circuitry required several unbroken cycles of nystagmus to acquire complete phase lock. In some patients the pendular oscillation was constantly interrupted by blinks, saccades, and jerk nystagmus, and the percentage of in-lock time was reduced. Second, in some patients (e.g., patient 2), the nystagmus amplitude varied constantly. To maintain consistent compensation for the ocular oscillation, the current prism-control circuitry would require continuous amplitude adjustments. Both these problems could be addressed by replacing the phase-locked loop circuit with a more sophisticated predictive tracking mechanism, probably based on microprocessor and digital signal processor technology. A more sophisticated circuit could also adjust for changes in nystagmus amplitude, thus maintaining a consistent system gain. The need for this and other refinements must be clarified during testing of a head-mounted prototype. Other issues that should be considered in developing a practical treatment include the degree to which the device tolerates compensatory eye movements, the design and usability of any patient-operated controls, and the mean time to failure under realistic conditions of use.

Implications for Treating Disorders of Eye Movements in General

Acquired pendular nystagmus is only one of several disorders of eye movements that impair vision.[2] We chose to design a device to treat APN, because the characteristics of the nystagmus waveform are simple, and thus relatively simple circuitry sufficed to detect and selectively negate the ocular oscillations. With more sophisticated control circuitry, the general approach of using servo-controlled prisms to shift the image of the world could be used to treat nonsinusoidal ocular oscilla-

tions (i.e., jerk nystagmus), acquired strabismus, and, with the addition of a head movement sensor, vestibular insufficiency.

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