Convergent-divergent pendular nystagmus:
Possible role of the vergence system

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Article abstract—We used the magnetic search coil technique to measure horizontal, vertical, and torsional components of convergent-divergent pendular nystagmus in three patients. All showed phase shifts of approximately 180° between the two eyes in the horizontal and torsional planes, but the vertical components were conjugate. Viewing a near target increased the oscillations threefold in one patient and by 60% in a second patient. The waveform was sinusoidal in one patient, but in the other two it was complex, resembling either a sum of several sine waves or a cycloid. When the predominant frequency of the nystagmus was low (1.8 Hz), oscillation of visually mediated vergence might have been responsible; when the frequency was high (6 Hz), the nystagmus might have arisen from an internal instability in connections between nucleus reticularis tegmenti pontis and cerebellar nucleus interpositus, which are important for vergence control.

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Acquired pendular nystagmus (APN) occurs most commonly in patients with demyelinating diseases or as a component of the syndrome of oculopalatal myoclonus.[1-3] The characteristics of this nystagmus are determined by the trajectories of the eye movements and their temporal relationships. The trajectory is a function of the amplitudes and phase shifts of the horizontal, vertical, and torsional components of the oscillation. For example, if the horizontal and vertical oscillatory components are at the same frequency and in phase, the trajectory of the nystagmus is oblique, but if they are out of phase, the trajectory will be elliptical or circular.[1] Although the frequency of oscillations is usually similar in both eyes, the amplitude may differ and there is often a phase shift, so that the eyes move in opposite directions for at least part of each cycle. Thus, it is the phase shift between the eyes that determines whether the oscillations are in opposite directions; when the phase shift is 180°, the oscillations are convergent-divergent. Few studies have attempted to analyze the temporal relationship of the APN oscillations in the two eyes, although this is now possible in all three planes using the magnetic search coil technique. In the last year, in the course of studying 10 patients who had APN, we found three in whom the nystagmus was convergent-divergent in character, and we sought to examine possible sources of such oscillation.

Case reports. Patient 1. A 41-year-old woman developed visual blurring in her right eye, progressive for 6 months; she noted illusory motion (oscillopsia) of words while reading. One year previously, multiple sclerosis (MS) had been diagnosed; the presenting feature was gait ataxia. MRI showed changes typical of MS. She was being treated with azathioprine. General neurologic examination showed increased tone in her lower extremities and bilateral extensor plantar responses. She had mild gait and limb ataxia. Corrected visual acuity was 20/40 OD and 20/30 OS, and J10 OD and J1 OS at near. There was slight color desaturation in the right eye, along with bilateral blind-spot enlargement. The pupillary responses were symmetric, and there was no overt disk pallor. Ocular motility testing demonstrated a full range of eye movements, bilateral slowing of adducting saccades (more so on the right), and an esodeviation of 10 prism diopters in viewing a target at distance. No nystagmus could be seen in primary position, but horizontal oscillations were noted in the right eye during convergence effort.

Patient 2. A 64-year-old woman was referred for evaluation of slowly progressive oscillopsia and vertical diplopia of about 2 years' duration. Three years previously, she had been diagnosed as having "progressive ataxia and palatal myoclonus" syndrome.[4] There was no history suggestive of stroke. CSF examination had revealed oligo-

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clonal bands. Brain MRI was within normal limits. Serologic tests, including those for anticerebellar antibodies, were negative. On examination, she had mild truncal ataxia, dysmetria, and dysdiadochokinesia, more on the right. Her speech was slightly slurred, with voice tremor. Symmetric rhythmic movements of the palate were present. The rest of the general neurologic examination was unremarkable. Corrected visual acuity was 20/30 OD and 20/50 OS, and J1 OD and J7 OS at near. Her optic fundi and pupils were normal. She had only rudimentary stereopsis at near, along with a moderate reduction of the convergence amplitude to 14 prism diopters. A low-amplitude torsional nystagmus was present, more evident in the left eye.

Patient 3. A 64-year-old woman had sudden onset of blurred vision, horizontal oscillopsia, and tinnitus, which thereafter remained stable for about 2 years. Her medical history was significant for treated hypertension. Repeated brain MRI and screening for paraneoplastic syndromes, including anticerebellar antibodies, were negative. Audiometric evaluation disclosed mild high-frequency hearing loss, more on the left. The presumptive diagnosis was APN following a stroke, and she was treated with carbamazepine, clonazepam, and valproate without any appreciable benefit. Despite her visual complaints, her corrected visual acuity was 20/20 and J1 at near in both eyes, with normal optic fundi and pupils. Neurologic examination was normal apart from her eye movements. These consisted of pendular, horizontal oscillations of both eyes, changing to jerk nystagmus on lateral gaze.

Methods. *Eye movement measurements.* Horizontal, vertical, and torsional rotations of both eyes and of the head were recorded using the magnetic search coil technique.[5] With their heads stationary, the patients attempted steady fixation, with each eye in turn (the other being occluded), of visual targets located near the primary position and at eccentricities of ±20° horizontally and ±15° vertically at viewing distances of 1.3 m (far target) or 18 cm (near target). The effects of viewing a near target binocularly (attempted convergence in response to combined accommodative-fusional stimuli) were also measured. Horizontal and vertical saccades were made between the fixed target locations, and horizontal and vertical smooth pursuit were measured as the patients followed a small target moving through ±15°, either sinusoidally at 0.3 Hz or in a triangular wave at 0.5 Hz. The stability of gaze was measured while the patients made active horizontal or vertical head rotations, viewing first the far and then the near target. Data were filtered (bandwidth, 0 to 90 Hz) prior to digitization at 200 or 500 Hz. Analysis was performed using interactive programs written in the ASYST language.[6] Epochs of 5 to 10 seconds of the oscillations during attempted fixation, which were relatively free of saccades and blinks, were analyzed, and the mean amplitude of nystagmus in each plane was calculated for each fixating eye. The gain of compensatory eye movements during head rotations while the patient viewed a stationary target—the visually-enhanced vestibulo-ocular reflex (VVOR)—and of smooth pursuit were also determined, as previously described.[5] Because the frequency of the APN was at least six times greater than the frequency of pursuit target motion, it was possible to filter out the nystagmus using a Hamming window prior to measurement of pursuit gain. To compare the phase relationships between the three component movements of each eye, we carried out a 256-point fast Fourier analysis to determine the relative magnitude and phase of the predominant component in each eye.

Results. *Patient 1.* Fixation was disrupted by a predominantly horizontal pendular nystagmus, which was of greater amplitude in the patient's right eye (figure 1A). The waveform was sinusoidal, at 6 to 7 Hz. The oscillations were of convergent-divergent type and were about 180° out of phase in horizontal and torsional planes but conjugate vertically (table). The nystagmus changed little with different gaze angles, but the peak-to-peak amplitude of the right horizontal oscillations increased threefold, from 0.6° to 1.8°, during viewing of near targets (figure 1B). This increase in the nystagmus at near was accompanied by a decrease in frequency from about 7.1 Hz to about 6.2 Hz. The gain of the horizontal VVOR was 1.06 while the patient was viewing the far target and 1.4 while viewing the near target. The gain of the vertical VVOR was 1.07 at far and 1.39 at near. Adducting saccades were slowed, consistent with bilateral internuclear ophthalmoplegia. Smooth pursuit eye movements were well preserved (gain approximately 0.9). The patient was treated with spectacles that contained a 5-diopter base-in prism over the right lens, which improved near acuity to J3 OD.

Patient 2. Fixation was disrupted by a nystagmus that was predominantly cyclovergent, ie, convergent-divergent torsional, so that upper poles of the eyes moved toward and away from each other at a frequency of approximately 1.8 Hz (figure 1C). Although the waveform was smooth, it was not truly sinusoidal. The waveform usually appeared "cycloidal" (figures 1C and 2), and Fourier analysis indicated principal frequencies that did not appear to be harmonically related (eg, 1.96 Hz and 3.52 Hz). The nystagmus was greater in the patient's left eye, with small horizontal and vertical components (figure 1D). In the horizontal and torsional planes, her eyes were about 180° out of phase; in the vertical plane her eyes were conjugate (table). The amplitude of the left eye's torsional component was about 2° in primary position, and increased by about 25% in right gaze and by 60% during convergence effort. The gain of the horizontal VVOR was 1.07 while viewing a distant target, and 1.13 while viewing a near target. The gain of the vertical VVOR was 0.99 at far and 1.22 at near. The gain of the torsional VVOR was 0.48 at far and 0.51 at near. Mild vertical saccadic hypermetria was present, but smooth pursuit was normal (gain of approximately 0.9).

Patient 3. Fixation was disrupted by a horizontal-torsional, low-amplitude oscillation of both eyes that was greater in the right eye (about 0.3°). In the horizontal and torsional planes, the oscillations were convergent-divergent, being about 180° out of phase (figure 1E), and in the vertical plane the eyes were conjugate (table). Two independent wave-

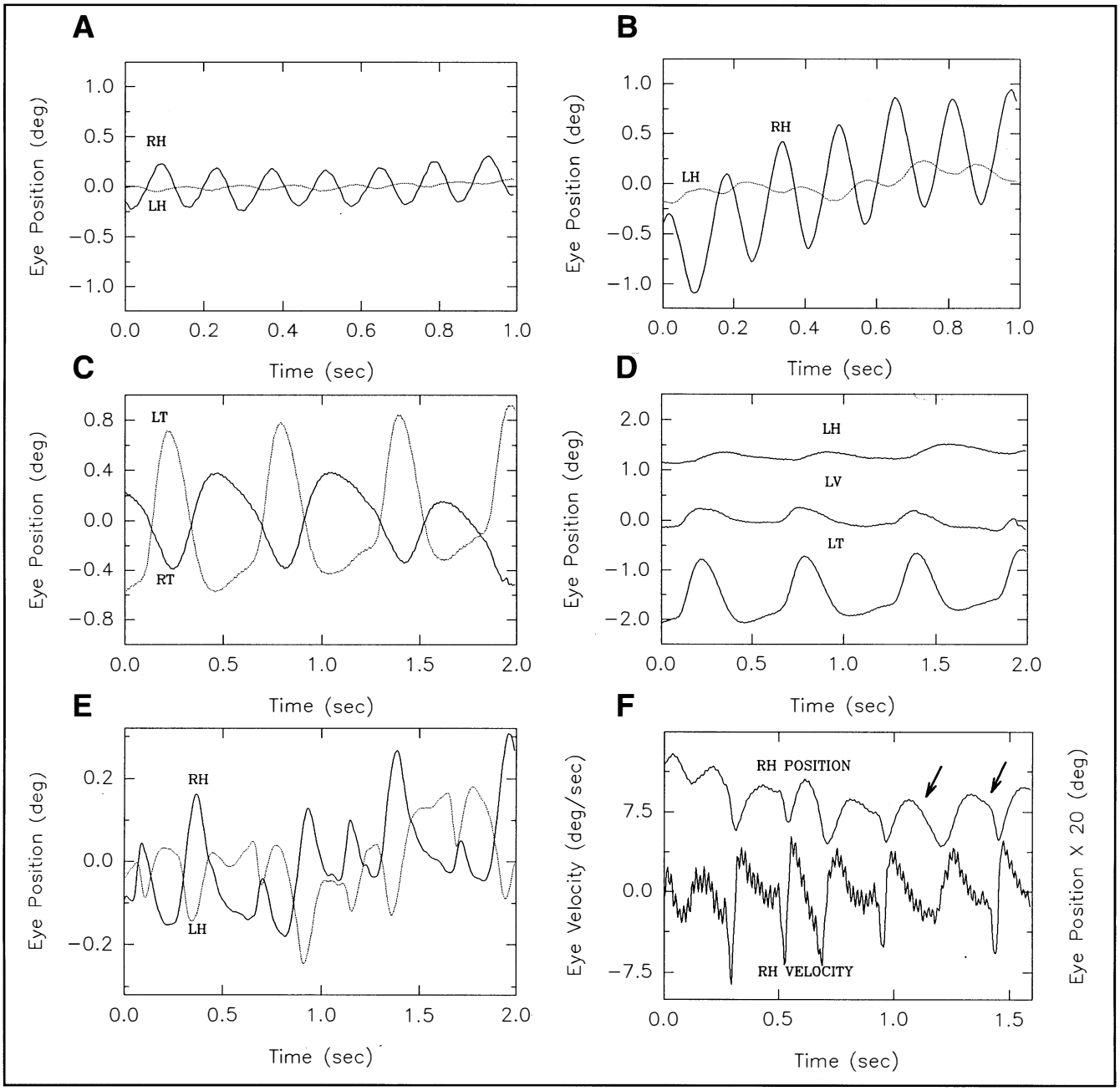

*Figure 1. Scleral magnetic search coil recordings. H, V, and T indicate horizontal, vertical and torsional planes of oscillation of the right (R) and left (L) eyes. (*A and B*) Patient 1. A 1-second segment of binocular recording in the horizontal plane while the patient viewed a target located at far (A) and near (B). The amplitude of the nystagmus is greater in the right eye, with the eyes moving in opposite directions. At near, the amplitude is markedly increased. Note the high frequency of the nystagmus. (*C and D*) Patient 2. A 2-second segment of binocular torsional (C) and monocular three-dimensional (D) recording. The nystagmus is mainly torsional; it is bigger in the left eye, with the eyes moving in opposite directions, ie, cycloverging. Note that the oscillations in the horizontal and torsional planes are about 90° out of phase with respect to each other (D). (*E and F*) Patient 3. A 2-second segment of horizontal right and left eye positions (E), and a segment of right eye horizontal position and velocity (F). The amplitude of the nystagmus is slightly greater in the right eye, with the eyes moving in opposite directions. Note the low amplitude of the nystagmus. The waveform is complex, with portions of the record resembling cycloids (arrows). The corresponding velocity trace shows that the movement contains no rightward saccades, although occasional small (up to 6 deg/sec) leftward saccades occur.*

forms could be distinguished in all planes of oscillation with peaks in the Fourier spectrum at 3.4 Hz and 5.9 Hz. Occasionally, the waveform appeared cycloidal, with frequency of about 4 Hz (figures 1F and 2). The relative dominance of these two waveforms varied inconsistently during the course of the recording session. The amplitude of the nystagmus was unaffected by assuming different convergence or gaze angles. The gain of the horizontal VVOR was 0.94 while viewing a distant target and 1.04

Table. Nystagmus phase shift between the right and left eyes*

Pt no./Age/ Diagnosis	Horizontal component	Vertical component	Torsional component
1/47/Multiple sclerosis	175	8	170
2/64/Palatal myoclonus	172	0	186
3/64/Brainstem infarction	198	5	197

* Phase shifts were calculated using a 256-point fast Fourier transform on the nystagmus components of each eye, expressed in degrees.

while viewing a near target. The gain of the vertical VVOR was 0.95 at far and 1.13 at near. Saccades were conjugate, with normal metrics, and smooth pursuit gain was approximately 0.5 when the patient was tracking the triangular target motion.

Discussion. Using precise binocular measurements of the horizontal, vertical, and torsional eye rotations, we identified three patients with a convergent-divergent form of APN. All exhibited oscillations that were about 180° out of phase in the horizontal and torsional planes; the vertical components, however, were conjugate. In patient 2, the torsional oscillations were the greatest in amplitude—cyclovergent nystagmus which, to our knowledge, has not been previously reported. In patients 1 and 2, the nystagmus was affected by convergence. Patients 2 and 3 appeared to have complex waveforms that were not truly sinusoidal and which, at times, resembled cycloids. Our findings raise two issues: (1) What ocular motor system is responsible for the convergent-divergent form of APN? and (2) What could account for the peculiar, nonsinusoidal oscillations of the two patients?

Convergent-divergent APN is considered rare, and only a few cases have been reported previously. Convergent-divergent APN has been described in patients with MS,[7] brainstem stroke,[2] Arnold-Chiari malformation,[8] and cerebral Whipple's disease.[9] It is important to distinguish between convergent-divergent APN and convergence-retraction nystagmus that occurs with dorsal pretectal lesions; the latter is not a true nystagmus but rather a saccadic disorder, initiated by adducting saccadic movements.[1] It is possible, however, that convergent-divergent varieties of APN are underdiagnosed and would be detected more readily with reliable records of eye movements. For example, the oscillations of our patient 1 were visible only when she was viewing the near target and only in the right eye; in patient 2, the oscillations were also not clinically obvious.

What mechanism can produce convergent-divergent movements? There are two possibilities: (1) a phase shift between the eyes, produced by dysfunction in the normal yoking mechanisms, and (2) the vergence system, operating without pathologic phase shift. In other words, convergent-divergent APN can be viewed either as abnormal version (180° out of phase) or as vergence oscillations. The latter is a more likely explanation, since our pa-

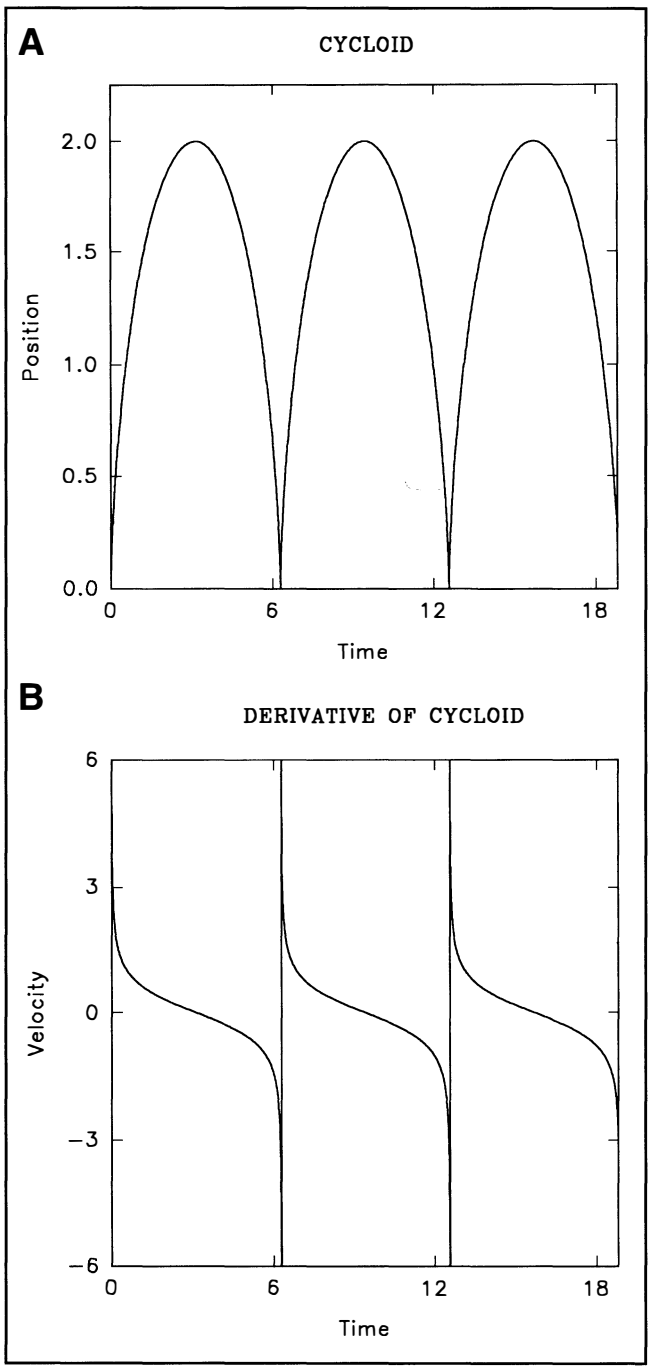

Figure 2. Computer-generated cycloid waveform (position, A) and its derivative (velocity, B). These waveforms are similar to those shown by patients 2 and 3 (see especially figure 1F). A cycloid is defined by the following parametric equations: $x = k(t - \sin t)$ and $y = k(1 - \cos t)$ where x and y are the coordinates on the Cartesian plane, k is a constant, and t is a variable (here, time).[26]

tients showed no phase shift (ie, were conjugate) vertically, and the relationship between the horizontal and torsional oscillations was similar to that occurring during normal vergence movements (excyclovergence with horizontal convergence).[10] Cyclovergence, seen in patient 2, is a disjunctive torsion movement; it is normally evoked by cyclodisparities between the two eyes[11] and is a component of horizontal vergence.[10,12]

The first to suggest that convergent-divergent APN may be oscillations of the vergence system were Sharpe et al.[7] This view was further developed by Schwartz et al,[9] who coined the term "pendular vergence oscillations" for patients with oculomasticatory myorhythmia and Whipple's disease. In their three patients, the low velocities and frequencies (1 Hz) of the oscillations and the involvement of vertical gaze led the authors to suggest that these oscillations were generated in the rostral midbrain by a "nearby component for convergence-divergence movements."[9]

In trying to determine whether the oscillations of convergent-divergent APN are generated by the vergence system, the frequency of oscillations is an important factor. The responses of the vergence system are usually thought to be slow.[1] Under experimental conditions, the vergence system has been made to oscillate at frequencies of up to 2.5 Hz.[13,14] In general, negative feedback systems, such as the visually mediated vergence system, will break into oscillation on one or more of the following conditions: (1) increase in gain or delay within the system's internal feedback loops; (2) external oscillation imposing upon the system. In our patient 2 and in reported patients with Whipple's disease, the frequency is similar to that of experimentally induced vergence oscillations. However, in two of our patients, the frequency was higher than theoretically predicted by vergence system models. To explain nystagmus in these patients by oscillation of the vergence system, one has to suggest that the system must either be pathologically changed (in terms of its gain or delay) or is influenced by other system oscillations imposing upon it. Any additional delay within the system loops will produce an oscillation at a lower frequency; only decreasing the delay while increasing the gain might elevate the frequency of oscillation. It is easier to imagine pathologic lesions that cause prolongation of the delay, eg, slowing of conduction due to demyelination. Nevertheless, shortening of the delay within the system might also occur, for example, due to loss of inhibitory connections; when such a system becomes unstable, it is capable of producing a higher frequency oscillation. This oscillation would become evident only when the vergence system is operating, ie, during near vision. In fact, this could account for convergence-induced nystagmus in our patient 1. Another possibility is that the nystagmus is produced by oscillators external to the brainstem ocular motor subsystems, perhaps of cerebellar origin. Our patient 3, with two independent waveforms, might represent an example of the latter, with two hypothetical oscillators projecting onto the vergence system. Alternatively, a single, nonlinear oscillator could also produce waveforms with multiple frequencies, particularly if the observed frequencies are harmonically related. Furthermore, time variance of such an oscillator might produce frequencies that appear not to be harmonically related.

Convergence has been reported to make nystagmus better or worse, to change its waveform, to induce nystagmus, or just to be impaired in patients with pendular nystagmus.[2,3,7,15-18] For example, in the series of Gresty et al,[2] all 16 patients with APN had "convergence failure." In contrast, our patients 1 and 3 were able to converge normally for their age; the insufficiency of convergence in patient 2 was probably long-standing. In the series of Barton and Cox[3] of patients with APN in MS, convergence was somehow involved in 14 of 19 patients examined for its effect: in six it damped the nystagmus and in five it increased it, and three patients could not converge. Convergence may convert one form of nystagmus to another, such as upbeat to downbeat and downbeat to upbeat.[15,17] Occasionally, nystagmus can be provoked by convergence, as in our patient 1.[7,15]

Convergence can exert its influence in a number of ways. Most commonly, the damping action of convergence on the existing nystagmus is ascribed to its peripheral effects, arising from lateral and medial recti co-contraction,[19] although this has never been formally examined. In other instances, eg, convergence-induced nystagmus, central mechanisms are implied. Sharpe et al[7] suggested that, in one of their patients who had conjugate, congenital pendular nystagmus, convergence had induced an oscillation of the smooth pursuit system, whereas in the other patient with disconjugate (180° out of phase) APN, the vergence system itself was oscillating. Each of the versional eye movement subsystems can be affected by convergence. For example, it is known that convergence effort increases the gain of VVOR,[20] and such modulation might explain changes in central vestibular types of nystagmus (eg, upbeat or downbeat) at near, as mentioned above. Alternatively, Gamlin et al[21] recently demonstrated that, in monkeys, experimental internuclear ophthalmoplegia increases baseline convergence tone. The same may be true for some patients with internuclear ophthalmoplegia; in such patients, vergence gain might be elevated to the point of instability during attempted convergence, leading to oscillations. This might be the case with our patient 1, who had bilateral internuclear ophthalmoplegia and esotropia.

The neural substrate for convergent-divergent APN is unknown. In Whipple's disease, the ocular oscillations are frequently associated with lesions in the rostral midbrain and cerebellum, with a predilection for the periaqueductal gray and tegmentum.[9] In our three patients, neuroimaging

failed to provide anatomic correlates for their ocular motor findings. In patient 2, who had oculopalatal myoclonus, the cerebellar signs were mainly contralateral to the eye with more prominent nystagmus. This might imply involvement of the brachium conjunctivum after its decussation, near the mesencephalic portion of the central tegmental tract, and in the vicinity of structures important for convergence.

Experimental evidence supports the view that convergent-divergent nystagmus may result from vergence system dysfunction. Two brainstem levels are involved in vergence control—mesencephalic (pretectum) and pontine (nucleus reticularis tegmenti pontis [NRTP], which lies dorsal to the pontine nuclei and is contiguous with the paramedian pontine reticular formation). The NRTP is one of the precerebellar reticular nuclei of the cerebropontocerebellar pathway. There is accumulating evidence of its importance in ocular motor control.[22] It receives afferents with visual input from frontal eye fields, superior colliculi, and pretectal regions as well as from oculomotor, vestibular, and cerebellar nuclei.[23] Its efferents terminate exclusively in the cerebellum via the brachium pontis. The NRTP has reciprocal connections with the interpositus and dentate nuclei via the brachium conjunctivum and with the fastigial nucleus via the Hook bundle.[22] Gamlin and Zhang[24] showed that neurons in the cerebellar nucleus interpositus, which lies between the dentate and fastigial nuclei, play an important role in vergence control in rhesus monkeys. The effect could have been exerted through reciprocal connections with the NRTP. Selective, experimental lesions of the NRTP in monkeys impair the ability to sustain a converged eye position, a finding attributed to a "leaky convergence integrator."[25] In addition, such NRTP lesions produced pendular oscillations of convergence-divergence type at about 2 Hz,[25] similar to those predicted for vergence system oscillation by control system analysis.[13] If the NRTP is indeed part of a feedback loop involving the cerebellar nucleus interpositus, instability in such a local circuit could make the vergence system susceptible to oscillation, thus producing convergent-divergent nystagmus, although, as discussed above, a reduction in delays would be needed to account for the higher frequency oscillations.

One issue raised by this study is that the waveforms encountered were sinusoidal in patient 1 but more complex in patients 2 and 3. In studying other patients with nonconvergent forms of APN, we have often been struck by the unusual waveforms. One explanation for this might be that the waveform represents a "sum of sine waves," each component having a different frequency and amplitude. Particularly when the components are not harmonically related, this could imply more than one oscillatory mechanism or a single oscillator imposing on several ocular motor subsystems. In patients 2 and 3, however, the waveform at times appeared to be cycloid—ie, like the curve described by a point on the rim of a rolling wheel.[26] The similarity between the computer-generated cycloid and the record from patient 3 (figures 1F and 2) is apparent. (Cycloid should be differentiated from pseudocycloid waveform; the latter is reported mainly in congenital nystagmus and is characterized by small saccades followed by decelerating slow phases.[27]) The pathogenesis of cycloid waveform remains to be determined, but whatever the explanation may be, our findings further emphasize the complexity of the nystagmus-generating mechanism.

Each of our patients complained of oscillopsia, even though the amplitude of their nystagmus was small and hard to detect. In patient 1, the oscillopsia may be attributed mainly to the high velocities caused by the 6- to 7-Hz oscillations. However, in patient 3, whose maximal amplitude was 0.3° (velocity up to 4 deg/sec), and in patient 2, whose main component of nystagmus was in the torsional plane at 1.8 Hz, this did not seem likely. With such small amplitudes of oscillation the presence of oscillopsia is surprising, especially when the main component of nystagmus is torsional. The latter is considered the least important for steady vision of the world on the retina.[28] Our patients' oscillopsia might result from the marked disconjugacy between the eyes in the horizontal and torsional planes, with the eyes constantly moving in opposite directions.

Although no uniformly reliable measures for treatment of acquired nystagmus exist,[29] those patients whose nystagmus is decreased by convergence may benefit from base-out (converging) prisms, and those (like patient 1) whose nystagmus increases with convergence may benefit from base-in (diverging) prisms; such optical treatments may diminish nystagmus and improve the patient's vision.

In summary, we have described three patients with unusual, convergent-divergent form of APN. We postulate that vergence system oscillation is responsible, with the NRTP or its cerebellar connections being the likely sites of lesions. The complex waveforms observed may reflect several sources contributing to the oscillation or one source influencing different ocular motor subsystems.

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