Latent Nystagmus
Release and Suppression at Will

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Purpose. The authors report the cases two unusual patients with infantile convergent strabismus and latent nystagmus.

Methods. Electronystagmography was used.

Results. The two patients were able to release and suppress their nystagmus at will. With voluntary effort, the nystagmus became as strong as it was when brought out by occlusion of the squinting eye and, in one of the patients, even stronger. The nystagmus beat toward the fixing eye, and the slow phases had slightly decreasing velocity or were linear. Both patients were able to evoke and to stop the nystagmus in front of visual contours and, one of them, also in the dark.

Conclusions. This phenomenon could be the result of voluntary control of the visual input contributed by the amblyopic eye and/or a direct influence of will on the slow eye movement and fixation systems. A mechanism related to vergence eye movements is less likely. The ability to release and suppress a latent nystagmus at will is unusual and, to the authors' knowledge, has not been described before. Invest Ophthalmol Vis Sci. 1993;34:1785-1792.

Latent nystagmus (LN) is one feature of the infantile strabismus syndrome.1-7 LN is a conjugate jerk nystagmus; its direction depends on the relative balance between the visual inputs conveyed by the right and left eyes. The more the visual input is dominated by one eye, the higher the velocity of the slow phases in the direction toward the opposite eye.6 Monocular occlusion is an effective means to shift the balance of visual inputs to the viewing eye. In alternating strabismus, a more subtle alteration of ocular dominance can occur, with both eyes viewing when fixation is switched to the other eye. In this instance, suppression of the image of the squinting eye suffices to change the balance of visual inputs so that a small amount of LN beating toward the fixing eye remains. This was described as manifest LN (MLN).8 The exact mechanisms underlying LN are not known, although both sensory and motor mechanisms seem to be important.9

We report the cases of two patients who were unusual because they could release and suppress a strong LN at will. Preliminary data on Patient 2, including a motion picture demonstration, was reported previously.2

METHODS

Eye movements were measured using direct current electro-oculography (band width, 0–30 Hz). Horizontal rotations were recorded for each eye separately from skin electrodes placed near the inner and outer canthi. Vertical eye deviations and blink artifacts were identified from vertical electrodes above and below the right eye. The electro-oculogram was displayed on a chart recorder with a time scale of 25 mm/sec. Calibration was obtained by refixations between two tar-
get spots that were 10° apart. Both eyes were left open during calibrations, which means that the dominant eye (the left eye in our two patients) fixed on the targets. Using the cover test, we ascertained that our patients never switched fixation to their amblyopic right eye, unless forced to do so during occlusion of their dominant left eye. Although only the dominant eye was taken for fixation, the calibrations could be used for both eyes because clinical examination revealed concomitance of the strabismus in both of our patients. The calibrations were repeated several times to minimize errors caused by changes in the corneoretinal potential. The slow-phase velocity (in degrees per second) was the index of nystagmus intensity. The maximum velocity of each of ten consecutive slow phases was measured by applying a straight edge to the eye position recording, and the average was calculated. The eye movements were recorded when the patient fixed on a target spot at a distance of 70 cm with (1) both eyes open and (2) one eye covered by an occluder or the palm of the patient’s hand.

The retinal area used for monocular fixation was determined by asking the patient to fixate a target in an ophthalmoscope. The relative alignment of the eyes was measured by comparing the recordings of the two eyes and by observing the eyes during the prism and cover test. The average of the end points of several rapid nystagmus phases was taken as the position of each eye.

Further tests included smooth pursuit of a target spot moving horizontally at 20 and 40°/s, optokinetic response to a full-field pattern of 7° wide stripes moving horizontally at 20°/s, vestibulo-ocular reflex in darkness with sinusoidal chair rotation over ± 20° at a maximum velocity of 40°/s, and fixation suppression of the vestibulo-ocular reflex with the same rotational stimulus.

The research followed the tenets of the Declaration of Helsinki. Informed consent was obtained from the two patients after the nature of the procedures had been explained fully. Approval by our institutional human experimentation committee was not deemed necessary because all our testing was part of the routine clinical examination necessary for the care of these two patients.

CASE REPORTS

Case 1

History. This was a 19-year-old man who complained of intermittent oscillopsia that had disturbed him as long as he could remember. The oscillopsia would last for a few seconds up to 2 min. To overcome the oscillopsia, the patient had developed three different strategies, described by “look steady!,” “strain!,” and “relax!” The patient’s right eye was amblyopic and had been squinting inward since early infancy. At the age of 3 yr, occlusion therapy had been tried. Otherwise, this young man had never had any serious illness.

Ocular Findings. Except for a hyperopia, both eyes were structurally normal. The fundi showed normal discs and maculas, and there was no afferent pupillary defect. Goldmann perimetry demonstrated normal visual fields. The patient wore fully correcting glasses throughout the examination. The cycloplegic refraction was +2.0 sphere in the right eye and +2.5 sphere in the left eye. Visual acuity was 0.1 in the right eye, 0.5 in the left eye, and 0.8 binocular. The right eye deviated inward (esotropia) with an angle of 2–15°. In addition, there was a right hypertropia of 6° that increased to 8° after occluding the right eye and decreased to 3° after placing a dark filter in front of the left eye (dissociated vertical deviation).

Eye Movements. With both eyes open, the patient could adopt one of four different viewing states or strategies. Each of them was associated with different degrees of nystagmus and esotropia (Table 1).

Most of the time, the patient could switch promptly between these four states at will and on command. For casual seeing, he used Strategy 4 (“relaxation”). When he wanted to see something in detail, ie, for visual resolution, he preferred Strategy 2 (“steadiness”). Occasionally, however, the patient was unable to suppress the oscillopsia by Strategy 2. He then switched to Strategy 3 (“strain”). Although this switch was accompanied by an increase of the esotropia, the pupil did not constrict nor was there an increase of accommodation, as checked by retinoscopy. Strategy 4 (“relaxation”) included not only looking 3° to the right past the target (Table 1) but also release of the near triad. Hence, when the patient looked at near, using Strategies 1, 2, or 3, and then relaxed to Strategy 4, accommodation was released, the pupils became larger, and the right eye diverged. Such a release of the near triad was occasionally also observed during Strategy 2 (steadiness).

The wave form of the nystagmus was saw-toothed. The slow phases were linear or with a slightly decreasing velocity (Fig. 1). The patient was further tested in a series of experimental conditions (Table 2). Monocular fixation by the right eye induced by occlusion of the left eye resulted in a nystagmus with slow phases directed to the left, and monocular fixation of the left eye induced by occlusion of the right eye resulted in a nystagmus with slow phases directed to the right. The retinal area used for monocular fixation was determined by asking the patient to fixate a target in an ophthalmoscope. In the amblyopic right eye, the end points of the rapid phases were 0–4° on the nasal side of the fovea, and in the dominant left eye, the end points of the rapid phases were in the fovea. With ei-
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TABLE I. Binocular Viewing States (Case 1)

<table>
<thead>
<tr>
<th>No.</th>
<th>Patient’s Report of His Subjective Sensation and Fixation Strategy</th>
<th>Velocity (deg/sec) and Direction of Slow Phases</th>
<th>Alignment of Eyes</th>
<th>Binocular Visual Acuity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Oscillopsia</td>
<td>39 R</td>
<td>RE esotropia 5°</td>
<td>0.4</td>
</tr>
<tr>
<td>2</td>
<td>Steadiness</td>
<td>12 R</td>
<td>RE esotropia 5°</td>
<td>0.8</td>
</tr>
<tr>
<td>3</td>
<td>Strain</td>
<td>15 R</td>
<td>RE esotropia 5°</td>
<td>0.8</td>
</tr>
<tr>
<td>4</td>
<td>Relaxation, looking to the right past target</td>
<td>0</td>
<td>LE looking 3° to the right of target, RE esotropia approximately 2°</td>
<td>Not measurable; “attention” resulted in strategy no. 2</td>
</tr>
</tbody>
</table>

When either eye occluded, the patient could not influence the nystagmus by voluntary effort. It did not matter whether the eye was occluded with the patient’s hand or with a black or an opaque cover. With either the right or the left eye occluded, large horizontal gaze deviations (> 20°) increased the nystagmus slightly when gaze was shifted to the side of the rapid phase (Alexander’s law). The difference of the nystagmus between right and left gaze was 5°/s or less. Smaller gaze deviations (≤ 20°) did not clearly influence the nystagmus intensity. In complete darkness, while imagining a visual target and “looking” at it with either both eyes open or with one eye covered by the patient’s hand, only occasionally were a few beats of nystagmus recorded.

Monocular optokinetic stimulation yielded only weak responses. Nasally moving stripes (“temporal–nasal optokinetic stimulation” in Table 2) minimally increased, and temporally moving stripes minimally decreased the nystagmus manifested by monocular occlusion (Table 2). Monocular tracking of a target spot moving horizontally at 20 and 40°/s showed superposition of pursuit on the nystagmus present during fixation.

The horizontal vestibulo-ocular reflex was tested with sinusoidal chair rotation over ± 20° at a maximum velocity of 40°/s while the patient was asked to “look” at an imagined target in darkness. The reflex movements were smooth and largely compensatory. Suppression of the vestibulo-ocular reflex was tested by sinusoidal rotation of a fixation target together with the patient’s body. With binocular fixation, suppression of the vestibulo-ocular reflex was nearly perfect, possibly because the patient used his “relaxation strategy” during this test. With monocular fixation, a strong nystagmus appeared when the patient was rotated toward the fixating eye.

Retinal Area of the Amblyotic Eye Decisive for Nystagmus Suppression. To find out whether a certain part of the retina of the right (amblyopic) eye was decisive for suppression of the left-beating nystagmus, the patient was examined at the synoptophore with identical

FIGURE 1. Modulation of nystagmus at will with both eyes open. In Patient 1, shortly after the command “wiggle!,” a strong nystagmus with slow phases to the right (upward deflection of trace) appears. After the command “stop!,” the nystagmus is markedly reduced. The slight divergence of the right eye (approximately 4°) that occurs together with the strong nystagmus is probably caused by a release of the near triad (target at a distance of 70 cm). The dashed lines indicate the approximate position of the target in relation to the right and left eyes. The asterisks mark the occurrence of blinks.
TABLE 2. Velocity (deg/sec) and Direction of Slow Phases Under Various Experimental Conditions (Case 1)

<table>
<thead>
<tr>
<th>Viewing Condition</th>
<th>Velocity (deg/sec)</th>
<th>Direction of Slow Phases</th>
<th>Visual Acuity</th>
</tr>
</thead>
<tbody>
<tr>
<td>RE monocular fixation</td>
<td>35 L</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>LE monocular fixation</td>
<td>43 R</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Darkness, &quot;looking&quot; at imagined target</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Darkness, wiggle!</td>
<td>Mostly 0,</td>
<td></td>
<td>Mostly 0,</td>
</tr>
<tr>
<td>Darkness, dominant LE “looking”</td>
<td>0</td>
<td>20 L</td>
<td></td>
</tr>
<tr>
<td>(LE covered by patient’s hand)</td>
<td>Mostly 0,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RE temporal-nasal optokinetic stimulation (20 deg/sec)</td>
<td>36 L</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RE nasal-temporal optokinetic stimulation (20 deg/sec)</td>
<td>23 L</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LE temporal-nasal optokinetic stimulation (20 deg/sec)</td>
<td>30 R</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LE nasal-temporal optokinetic stimulation (20 deg/sec)</td>
<td>24 R</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

("fusional") images. For suppression of the nystagmus, the image had to be placed on the macula or on the paracentral temporal retina. When the patient looked at a spot on a white screen with both eyes open (left eye fixing), a black dot of approximately 5° visual angle mounted on a small stick was moved closely in front of his amblyopic right eye. For suppression of the nystagmus, this dot had to be imaged on the macula or on the paracentral temporal retina. Imaging of the dot on the nasal half of the right retina was not effective. Changes of the angle of squint were not observed during this “perimetric” search.

Binocular Vision. When tested with striated glasses,10 the streak of the (dominant) left eye traversed the fixation light and was seen without a gap. It was difficult for the patient to see the streak of the amblyopic right eye because he could not see the central part (central suppression scotoma approximately 10° in diameter). Trying to extrapolate the whole streak from its ends to the center, the streak seemed displaced to the right by 5°, corresponding to the esotropia of 5° present under this condition. Switching between the strategies listed in Table 1 did not clearly change the appearance of the streak.

Surgical Correction and Postoperative Findings. We advised surgery in the expectation that suppression of the nystagmus might be facilitated by transposing the image of the fixation target onto the macula of the amblyopic right eye. This expectation was also based on the experience of others.11,12 A 6-mm recession of the right medial rectus was performed. During the first days after surgery, the right eye could not adduct completely. Six months after surgery, the unilateral cover test did not reveal a deviation in most viewing conditions, and binocular vision could be demonstrated with Bagolini’s striated glasses (right central suppression scotoma of 1° diameter but no displacement). Only occasionally, when the dissociated vertical deviation became manifest, did the suppression increase. With the eyes orthotropic, there was a slight MLN (slow phases 5°/s right), and the patient reached a binocular visual acuity of 0.8 without oscillopsia. Under this condition of orthotropia, the patient was no longer able to reproduce the strong nystagmus that he had been able to elicit before surgery when his right eye had been esotropic (Strategy 1 in Table 1). However, when the preoperative location of the right retinal image was re-established by applying prisms, the MLN increased to 10°/s, and the patient could augment it to 40°/s at will. Monocular occlusion resulted in nystagmus just as before surgery.

Case 2

History. In this boy, a convergent squint had been noticed when he was 3 mo old. From the age of 9 mo, he was treated for infantile esotropia in our department. Intermittent occlusion was necessary to prevent strabismic amblyopia. At the age of 19 mo, the angle of squint was corrected surgically (left medial rectus recession 5 mm plus left lateral rectus resection 5 mm). When the boy was 9 yr old, it was discovered that he could entertain his school mates by wiggling his eyes at will (Fig. 2).

At the age of 14 yr, another operation was performed to correct an A-incomitance with a convergent strabismus in upgaze (right medial rectus resection 5 mm with upward transposition 4 mm plus right lateral rectus resection 5 mm). The last examination was performed when the boy was 15 yr old. The findings obtained at this time were as follows.

Ocular Findings. Both eyes were structurally normal. The fundi showed normal discs and maculas, and there was no afferent pupillary defect. Goldmann perimetry demonstrated normal visual fields. Cycloplegic refraction was +0.5 sphere in both eyes. Visual acuity was 0.4 right eye, 0.63 left eye, and 1.0 binocular.

Eye Movements. During casual viewing, there was no obvious nystagmus, and the eyes appeared parallel, as confirmed with the unilateral cover test. The alternating cover test revealed a dissociated vertical deviation of 4°. Monocular fixation of a target in an ophthalmoscope, with the other eye occluded, revealed a LN with the rapid phases ending with the image of the target at the fovea. When, during binocular vision, the
boy was asked to demonstrate his ability to wiggle his eyes, he released a strong left-beating nystagmus, accompanied by oscillopsia. The nystagmus was initiated by a conjugate leftward saccade, corresponding to an indistinct sensation of looking slightly leftward past the target. During the nystagmus released at will, the target was imaged on the temporal retina of the dominant left eye between eccentricities of 5 and 0°, by contrast with the LN during occlusion of the right eye in which the fixation target was imaged on the nasal retina. During the first two or three cycles of the nystagmus, the right eye slowly deviated to a 4° upward position, according to the dissociated vertical deviation. There was, however, hardly any, and certainly not a systematic change in horizontal ocular alignment, nor was there a change in pupil size or in accommodation (as determined by retinoscopy). Although the boy could evoke, sustain for at least 10 sec, and stop the left-beating nystagmus whenever he was asked to do so, even in complete darkness, he was unable to produce a right-beating nystagmus at will. On monocular occlusion of one eye, he could markedly augment the LN that appeared with fixation of his dominant left eye (left beating) but could hardly change the LN that appeared with fixation of his amblyopic right eye (right beating). The quantitative data obtained by electroneystagmography are summarized in Table 3.

In complete darkness, while “looking” casually at an imagined target with either both eyes open or with the dominant left eye (amblyopic right eye covered by the patient’s hand), there was a weak nystagmus (slow phases 2°/s to the right). With the command “wiggle,” this nystagmus could be markedly augmented (slow phases 17 and 13°/s, respectively). The change in nystagmus intensity was not accompanied by a vergence movement. With the amblyopic right eye “looking” (left eye covered by the patient’s hand), there was no nystagmus, not even after the command “wiggle.”

Monocular optokinetic stimulation from temporal to nasal yielded normal responses. Stimulation from nasal to temporal of the right eye did not result in a nystagmus. Stimulation from nasal to temporal of the left eye “looking” (left eye covered by the patient’s hand) evoked a weak nystagmus. This nasal to temporal response required a large-field stimulation; small-field stimulation with a hand-held tape did not evoke a nystagmus in this direction. Monocular tracking of a target spot moving horizontally at 20 and 40°/s showed superposition of pursuit on the nystagmus present during fixation.

The horizontal vestibulo-ocular reflex was tested with sinusoidal chair rotation at a maximum velocity of 40°/s while the patient was asked to “look” at an imagined target in darkness. The reflex movements were smooth and largely compensatory. Suppression of the vestibulo-ocular reflex was tested by sinusoidal rotation of a fixation target together with the patient’s body. With binocular fixation, suppression of the vestibulo-ocular reflex was nearly perfect. With monocular fixation, a strong nystagmus appeared when the patient was rotated toward the fixating eye.

**Binocular Vision.** When tested with striated glasses,10 the streak of the (dominant) left eye tra-
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We described the cases of two patients with the patho-
DISCUSSION
his eyes at will, the streak of the right eye disappeared
versed the fixation light and was seen without a gap.
TABLE 3. Velocity (deg/sec) and Direction
of Slow Phases Under Various Viewing
Conditions (Case 2, at the Age of 15)

<table>
<thead>
<tr>
<th>Viewing Condition</th>
<th>Velocity (deg/sec)</th>
<th>Direction of Slow Phases</th>
<th>Visual Acuity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Binocular fixation</td>
<td>0</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Binocular fixation, wiggle!</td>
<td>28 R</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>RE monocular fixation</td>
<td>5 L</td>
<td>0.4</td>
<td></td>
</tr>
<tr>
<td>RE monocular fixation, wiggle!</td>
<td>8 L</td>
<td>0.4</td>
<td></td>
</tr>
<tr>
<td>LE monocular fixation</td>
<td>5 R</td>
<td>0.63</td>
<td></td>
</tr>
<tr>
<td>LE monocular fixation, wiggle!</td>
<td>28 R</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Darkness, “looking” at imagined target</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Darkness, wiggle!</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Darkness, amblyopic RE “looking” (LE covered by patient’s hand)</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Darkness, amblyopic RE “looking” (LE covered by patient’s hand), wiggle!</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Darkness, dominant LE “looking” (RE covered by patient’s hand)</td>
<td>2 R</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Darkness, dominant LE “looking” (RE covered by patient’s hand), wiggle!</td>
<td>13 R</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RE temporal-nasal optokinetic stimulation (20 deg/sec)</td>
<td>19 L</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LE temporal-nasal optokinetic stimulation (20 deg/sec)</td>
<td>7 R</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LE nasal-temporal optokinetic stimulation (20 deg/sec)</td>
<td>18 R</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

fort, the nystagmus became as strong as when brought out by occlusion of the squinting eye and, in Patient 2, even stronger. When the patients intended to suppress their nystagmus, there was only a weak MLN, such as typically encountered in patients with LN,\textsuperscript{8,17} or no nystagmus at all.

The LN released at will was distinct from the well-known “voluntary nystagmus” that occurs in otherwise normal subjects. Voluntary nystagmus consists of to and fro saccades without an intervening pause.\textsuperscript{18} The oscillation reaches frequencies as high as 25 cycles per second.

The mechanism that allowed our patients to release and control their LN in the presence of visual contours before both eyes is open to speculation. We propose that they were able to admit or suppress the visual input from their amblyopic eye at will. The more they suppressed their amblyopic eye, the more they shifted the balance of visual inputs toward an absolute dominance of the other eye. This resulted in a release of the nystagmus, comparable to an occlusion of the amblyopic eye. The more they admitted the visual contribution of the amblyopic eye, the more were the inputs of the two eyes balanced. This resulted in a damping of the nystagmus.

Our hypothesis is supported by the following three observations.

1. Using striated glasses as a test for binocular vision, the streak of the amblyopic eye disappeared whenever the nystagmus was released at will. This was clearly reported by Patient 2. Patient 1 could observe the streak of the amblyopic eye consistently only after surgical alignment of the eyes, and then he was no longer able to release the nystagmus at will.

2. In Patient 1, after surgical correction of the esotropia, repositioning of the retinal image to the preoperative location by prisms resulted in reappearance of MLN and a recurrence of the patient’s ability to modulate his nystagmus at will.

3. For control of the nystagmus, visual input through the amblyopic eye was crucial. In Patient 1, the relevant field could be specified by presenting a black dot of approximately 5° visual angle to various parts of the retina in the amblyopic eye. It was the macula or the temporal retina that had to be stimulated. The ineffectiveness of stimulation of the nasal retina may have been the result of constant suppression because, in esotropic eyes, the suppression tends to be greater on the nasal than on the temporal retina.\textsuperscript{19}

Compatible with our hypothesis is the fact that both of our patients could only evoke a nystagmus that beat toward the dominant eye but not toward the am-
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In keeping with data from binocular perimetry in strabismic eyes, we would expect that suppression of the amblyopic eye is easier than suppression of the dominant eye.

Our patients were able to control their nystagmus to a much greater degree than the usual patient with LN who shows a small amount of MLN during binocular viewing. Often, our patients were able to suppress their nystagmus completely. Patient 2 did not need a special trick for this; Patient 1 had to "look slightly to the right" to reach full relaxation and the absence of oscillopsia. Indeed, our recordings confirmed that he looked 3° to the right past the target under this condition. We speculate that this strategy allowed him to "equalize" the visual inputs from both eyes. On the one hand, the input from the dominant left eye was decreased by imaging the target away from the fovea. On the other hand, the input from the amblyopic, esotropic right eye was increased by imaging the target closer to the fovea.

Another explanation for the ability to suppress the MLN completely is that the patients employed the "fixation system," a physiologic subsystem recently emphasized. The fixation system appears to have properties that distinguish it from smooth pursuit (eg, the presence of oscillations during pursuit but not during fixation). Voluntary manipulations of the fixation system has also been suggested as a mechanism for the ability to release and inhibit, at will, congenital nystagmus.

The ability to release and suppress a LN in the presence of visual surroundings is unusual and, to our knowledge, has not been described before. In the dark, however, cognitive influences on LN are well known. By "looking" at an imagined target and mentally switching from right to left eye "fixation," some patients are able to reverse the slow phases of their nystagmus. This phenomenon can best be elicited when the patient alternately occludes the right or left eye with their hands in darkness. A remarkable cognitive influence on LN in the dark was demonstrated in a patient who had been blind in the right eye from birth because of a malformation of that eye. The right eye had been enucleated. In darkness, the eyes open, both patients hardly showed any nystagmus. With the instruction to wiggle the eyes and with one or the other eye covered by his hand, only Patient 2 showed a consistent effect, ie, with the amblyopic right eye covered, he had slow phases to the right and was able to enhance their velocity from 2 to 13°/s. He could do so also with both eyes open. Only occlusion of the better left eye precluded his ability to evoke a nystagmus in darkness.

Although the ability of both of our patients to modulate their LN in the presence of visual contours can be explained by admittance or suppression of the visual input from the amblyopic eye, the ability of Patient 2 to modulate his LN in complete darkness requires us to postulate a nonvisual influence of will on the slow eye movement and fixation systems. The influence of will on LN may be related to the ability of normal subjects who, after appropriate training, can evoke an optokinetic nystagmus by visual imagination of a moving pattern in total darkness.

We also considered that our patients modulated their LN by a motor mechanism, in particular by vergence. This could be the case in Patient 1 when he applied his strain strategy, which increased his esotropia to 15° (No. 3 in Table 1). However, convergence was certainly not the only and most effective mechanism that allowed him to control his nystagmus. With the steadiness strategy (No. 2 in Table 1), the nystagmus diminished from 39 to 12°/s, while the esotropia remained 5°. With the relaxation strategy (No. 4 in Table 1), the nystagmus disappeared completely while the esotropia decreased to approximately 2°. Thus, his nystagmus could change dramatically with no or very little change in ocular alignment.

When Patient 2 released his nystagmus at will, his amblyopic eye drifted to a 4° upward position, according to his dissociated vertical deviation. The drift was slow, and the dissociated vertical deviation developed gradually, although the nystagmus had full intensity from the beginning. Although we cannot exclude the possibility that the motor signal responsible for the dissociated vertical deviation also caused the nystagmus, we rather assume that the deviation became manifest because of (voluntary) suppression of the visual input to the amblyopic eye, ie, was caused by the same sensory mechanism that also released the nystagmus. A systematic change in horizontal alignment was not recorded together with changes of nystagmus intensity in Patient 2.

In conclusion, we suggest an important role for sensory, rather than motor, mechanisms for modulation of latent nystagmus by will. Although our two patients were similar in many respects, they showed three differences that we cannot explain.

1. Patient 1 was disturbed by an occasional outbreak of oscillopsia and needed a special effort to control his nystagmus. Patient 2 never developed oscillopsia spontaneously; he used his ability to evoke a nystagmus to entertain his friends.
2. Patient 2 was able to elicit nystagmus in the dark; Patient 1 was not.
3. In Patient 1, monocular occlusion led to a stronger nystagmus when the dominant eye was viewing than when the amblyopic eye was viewing. This asymmetry was opposite to that normally encountered in patients with LN.

**Key Words**
nystagmus, latent nystagmus, strabismus

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**References**