Dissociated Horizontal Deviation After Surgery for Infantile Esotropia

Clinical Characteristics and Proposed Pathophysiologic Mechanisms

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Objective: To examine the results of reversed fixation testing in patients who develop consecutive exotropia after surgery for infantile esotropia.

Methods: The reversed fixation test was performed prospectively in 28 patients who developed consecutive exotropia after surgery for infantile esotropia. All patients were assessed for adduction weakness, latent nystagmus, dissociated vertical divergence, and neurologic disease.

Results: A positive reversed fixation test, indicating the presence of dissociated horizontal deviation, was found in 14 of 28 patients (50%) with consecutive exotropia. In patients with dissociated horizontal deviation, the exodeviation was usually smaller with the nonpreferred eye fixating, and smaller with the preferred eye fixating than during periods of visual inattention or under general anesthesia. Dissociated horizontal deviation correlated with the findings of dissociated vertical divergence, but not with asymmetric adduction weakness, latent nystagmus, or neurologic disease.

Conclusions: Dissociated horizontal deviation is a clinical expression of dissociated esotonus. The common clinical presentation of dissociated horizontal deviation as an intermittent exodeviation of 1 eye results from the superimposition of a dissociated esotonus on a baseline exodeviation.

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Dissociated horizontal deviation is defined as a change in horizontal ocular alignment, unrelated to accommodation, that is brought about solely by a change in the balance of visual input from the 2 eyes. Unlike in other forms of intermittent exotropia, the observed exodeviation is slow, variable, and asymmetrical in the 2 eyes.

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Dissociated horizontal deviation usually manifests as a spontaneous unilateral exodeviation or an exodeviation of greater magnitude in 1 eye during prism and alternate cover testing (Figure 1). In some instances, fixation with one eye evokes an esodeviation of the other eye during prism and alternate cover testing (Figure 2).

Many reports have described dissociated horizontal deviation in patients who have been treated with strabismus surgery for infantile esotropia. These patients characteristically show dissociated signs such as latent nystagmus and dissociated vertical divergence, as well as torsional eye movements, sensorial suppression, and a positive Bielschowsky phenomenon in the horizontal plane. These signs have led to the clinical inference that this unilateral or asymmetric exodeviation must constitute a variable abducting component of dissociated vertical divergence.

In this journal, we previously defined the critical role of reversed fixation testing in the diagnosis of dissociated horizontal deviation. This test allows the examiner to visualize a dissociated component without inducing any positional change in the fixating eye. It was first devised by Mattheus and colleagues as a clinical technique to visualize the dissociated component in patients with dissociated vertical divergence, and later recognized by Graf to be a decisive diagnostic test for dissociated horizontal deviation. The reversed fixation test is preceded by the prism and alternate cover test, which is used to neutralize the exodeviating eye. After the patient continues to fixate through the prism with the exodeviating eye for at least 5 seconds, the reversed fixation test is performed by shifting the occluder to again cover the
Jampolsky has observed that some patients with strabismus the nondominant eye on the size of the horizontal deviation. Also wished to assess the role of fixation with the dominant vs. signs of dissociated horizontal deviation are not apparent. We showed a positive reversed fixation test even when overt clinical soned that some patients with consecutive exotropia may have not been systematically examined. In this investiga-
tion, we used the reversed fixation test to elucidate the pathophysiology of dissociated horizontal deviation.

**METHODS**

We therefore assigned the preferred eye for fixation rather than the eye with greater visual acuity as the dominant eye.

Between January 1, 2003, and November 30, 2006, we prospectively examined 28 patients with consecutive exotropia for the presence of dissociated horizontal deviation. Consecutive exotropia was diagnosed when we found a constant or intermittent exodeviation that exceeded 12 prism diopters (°) at distance or near. Simultaneous prism and cover testing and the prism and alternate cover testing were used to measure the exotropia. Once consecutive exotropia was diagnosed, we used the prism and alternate cover test to determine the size of the exodeviation with each eye fixing in primary position. We then documented the presence and direction of any horizontal re-fixation movement that was evoked by the reversed fixation test. The reversed fixation test was performed during distance fixation at 6 m, and with the cycloplegic refraction in place, to eliminate any confounding effect of diminished accommodation in the nonpreferred eye.

We routinely recorded the presence or absence of adduction weakness, assigning a value of −½ to correspond to 90% adduction, −1 to correspond to 80% adduction, and −2 to correspond to 60% adduction. We documented the presence or absence of latent nystagmus and dissociated vertical divergence. Latent nystagmus was defined as a horizontal conjugate jerk nystagmus with a fast phase that beat in the direction of the fixating eye, with amplitude that increased when the fixating eye was rotated into the abducted position. When present under binocular conditions, it was diagnosed as manifest latent nystagmus. Dissociated vertical divergence was defined as an alternating hyperdeviation of the occluded eye, or a hyperdeviation of 1 eye without a corresponding hypodeviation of the contralateral eye. We documented the presence or absence and characteristics of any associated neurologic disease.

We looked for changes in the angle of the exodeviation during periods of patient inattention to determine how the exodeviation changed when fixation was suspended. In patients who were subsequently treated with additional horizontal strabismus surgery, we examined the position of the eyes under nondepolarizing paralyzing anesthesia to determine how the clinical measurements corresponded to the baseline exodeviation of the eyes when innervational forces were suspended.

We excluded patients with congenital nystagmus with or without associated sensory visual loss to avoid the potential con-
founding effects of active convergence blockage. We also ex-
cluded patients in whom the reversed fixation test could not be performed because of age, short attention span, ocular or central nervous system disease causing reduced vision in 1 or both eyes, or dense amblyopia precluding central fixation in 1 or both eyes. Statistical analysis was performed with the Fisher exact test. This study was approved by the institutional review board of the University of Arkansas for Medical Sciences and Arkansas Children’s Hospital, Little Rock.

**RESULTS**

Fourteen of 28 patients (50%) with consecutive exotropia were found to have dissociated horizontal deviation, as demonstrated by a positive result on the horizontal reversed fixation test (Table 2). In these patients, the measured amplitude of the final refixation movement ranged from 2° to 10°. In 10 of the 14 patients, the final step of the reversed fixation test showed an abduction saccade of the preferred eye (Table 3). In 3 of 14 pa-
tients, the final step of the reversed fixation test showed...
an adduction saccade of the preferred eye. In the remaining patient, the movement of the preferred eye was not examined in the final step of the reversed fixation test. In all 8 patients in whom it was tested, the reversed fixation test showed an adduction saccade of the nonpreferred eye. In the reversed fixation test, a significantly greater number of patients displayed abducting saccades than adducting saccades of the preferred eye (P = .02, Fisher exact test).

Primary position measurements of the exodeviation were unequal when the right and left eyes were fixating in 12 of the 14 patients (86%) with dissociated horizontal deviation and in 3 of the 8 patients (38%) without dissociated horizontal deviation in whom primary position fixation with both eyes was measured (P = .05, Fisher exact test). Seven of the 14 patients with dissociated horizontal deviation had a greater exodeviation when fixating with the preferred eye in primary position (this calculation includes patient 20, who had an esodeviation at distance with a smaller esodeviation of the fixating eye). Two patients with dissociated horizontal deviation (cases 27 and 28) had the same exodeviation with either eye fixating in primary position because of a greater adduction lag in the nonpreferred eye. The remaining 5 patients with dissociated horizontal deviation (cases 4, 11, 12, 23, and 24) had a greater exodeviation when fixating with the nonpreferred eye in primary position. Three of these 5 patients (cases 4, 23, and 24) had a greater adduction lag in the nonpreferred eye, which explained the greater exodeviation when the nonpreferred eye was used for fixation. One patient (case 11) had dissociated horizontal deviation and a greater exodeviation when fixating with the nonpreferred eye. This patient showed an abduction saccade of the nonpreferred eye in the last step of the reversed fixation test, indicating that the nonpreferred eye was generating less esotonus. The last patient with dissociated horizontal deviation (case 12) showed an abduction saccade of the preferred eye with no asymmetry in adduction between the 2 eyes.

Primary position measurements of the exodeviation were obtained with each eye fixating in 8 of the 14 patients without dissociated horizontal deviation. In the remaining 6 patients, primary position measurements were obtained with only the preferred eye fixating. Five of the 8 patients in whom we had obtained bilateral primary position measurements (cases 7, 9, 14, 17, and 22) showed an equal exodeviation with each eye fixating, whereas 3 (cases 5, 8, and 19) showed unequal exodeviations.

The 3 who showed unequal exodeviations, 1 had a greater exodeviation with the preferred eye fixating secondary to a greater adduction lag in the nonpreferred eye (case 5), 1 had a greater exodeviation with the nonpreferred eye fixating despite equal adduction movements (case 8), and 1 had a greater exodeviation with the preferred eye fixating despite equal adduction movements (case 19).

We found asymmetric adduction in 6 of 14 patients with dissociated horizontal deviation (cases 4, 20, 23, 24, 27, and 28) vs 3 of 14 patients without dissociated horizontal deviation (cases 1, 5, and 6) (P = .42, Fisher exact test). Seven of these 9 patients with asymmetric adduction (cases 1, 4, 6, 20, 23, 24, and 27) had stronger adduction in the preferred eye, indicating that, in patients with asymmetric adduction, the eye with greater adduction tends to be the preferred eye. The remaining patients (cases 5 and 28) showed stronger adduction in the nonpreferred eye.

Nineteen of the 28 patients (68%) had dissociated vertical divergence, and 7 of the 28 patients (25%) had latent nystagmus. The prevalence of dissociated vertical divergence was significantly higher in patients with dissociated horizontal deviation (13 of 14 [93%]) than in patients without dissociated horizontal deviation (6 of 14 [43%]) (P = .01, Fisher exact test). The prevalence of latent nystagmus was not significantly higher in patients with dissociated horizontal deviation (5 of 14 [36%]) than it was in patients without dissociated horizontal deviation (2 of 14 [14%]) (P = .38, Fisher exact test).
Table 2. Clinical Findings in Patients With Consecutive Exotropia

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>VA³</th>
<th>PACT</th>
<th>RFT</th>
<th>Adduction</th>
<th>DVD</th>
<th>LN</th>
<th>Neurologic Disease</th>
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<tbody>
<tr>
<td>1/M/15 OD 20/30 OS 20/20</td>
<td>16° RXT, 10° RhT</td>
<td>NT</td>
<td>OD −1</td>
<td>+</td>
<td>−</td>
<td>CP, CVL, PVL</td>
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<tr>
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<td>10° RXT</td>
<td>25° RXT, 10° RhT</td>
<td>OS −½</td>
<td>NT</td>
<td></td>
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<td>30° LXT</td>
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<tr>
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<td>25° LXT(T), 5° LH(T)</td>
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<tr>
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<td>NT</td>
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<tr>
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<td>20° LXT, 3° LH</td>
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<td>2° RXT, 14° RhT</td>
<td>9° LXT, 20° LH</td>
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<tr>
<td>13/M/4 OD 20/20 OS 20/20</td>
<td>10° RXT</td>
<td>16° LXT, 3° LH(T)</td>
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<tr>
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<td>18° LXT, 3° LH</td>
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<td>15/M/17 OD 20/20 OS 20/20</td>
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<td>18° RXT</td>
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<tr>
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<td>10° LXT, 10° LXT</td>
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<tr>
<td>17/F/8 OD 20/25 OS 20/25</td>
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<td>14° LXT, 1° LH</td>
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<tr>
<td>18/M/12 OD 20/25 OS 20/25</td>
<td>NT</td>
<td>25° LXT, 5° LH(T)</td>
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<tr>
<td>19/M/23 OD 20/40 OS 20/20</td>
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<td>40° LXT, 5° LH(T)</td>
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<td>10° RET</td>
<td>8° LET</td>
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</tbody>
</table>

(continued)
Fisher exact test). It was common for patients with a positive reversed fixation test to have an exodeviation that appeared to be much larger on casual inspection and under nondepolarizing general anesthesia than it measured during prism and alternate cover testing.

A history of neurologic disease was elicited in 6 of 14 patients (43%) with dissociated horizontal deviation and in 3 of 14 patients (21%) without dissociated horizontal deviation (P = .42, Fisher exact test) (Table 2).

Several lines of evidence support our hypothesis that monocular fixation augments esotony in patients with dissociated horizontal deviation.15,19 First, we have observed that, when patients are under nondepolarizing general anesthesia, the eyes of patients with and without dissociated horizontal deviation display a large baseline exodeviation that far exceeds the measured exodeviation (Figure 3).1 Given that suspension of tonic innervation results in a large-angle exotropia, it follows that fixational innervation must augment esotony. Such periods of “nonfixation” partly unmask the baseline exodeviation that would be present under nondepolarizing paralyzing general anesthesia. Third, dissociated horizontal deviation does not manifest only as an intermittent exodeviation. Spielmann’s original report25 of dissociated horizontal deviation in a cohort of patients with intermittent esodeviation of 1 eye (Figure 4) demonstrates how this dissociated esodeviation can be superimposed on any baseline horizontal position. Spielmann’s use of bilateral translucent occluders to equalize visual input and block fixation revealed a baseline orthoposition in the absence of sensory dissociation.20 The fact that dissociated horizontal deviation can manifest as a dissociated esodeviation (when the baseline position of the eyes is straight) or a dissociated exodeviation (when the baseline position of the eyes is one of exodeviation) again shows that fixational innervation must augment esotony. The slow velocity of the exodeviation is explained by the “braking” effect of dissociated esotony, while its variable amplitude probably reflects momentary fluctuations in fixational effort and depth of suppression.

### Table 2. Clinical Findings in Patients With Consecutive Exotropia (cont)

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>VA</th>
<th>PACT</th>
<th>RFT</th>
<th>Adduction</th>
<th>DVD</th>
<th>LN</th>
<th>Neurologic Disease</th>
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<tbody>
<tr>
<td>21/F/15 OD 20/60 OS 20/40</td>
<td>12° RXT, 16° RHT</td>
<td>+</td>
<td>OD −½</td>
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<td>+</td>
<td>IVH, hydrocephalus, seizures</td>
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</tr>
<tr>
<td>22/F/19 OD 20/60 OS 20/40</td>
<td>16° RXT, 8° RhT</td>
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<td>Porencephaly cyst, right hemiplegia, seizures</td>
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<tr>
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<td>OD −1</td>
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<tr>
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<td>OD full</td>
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<td>−</td>
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<td></td>
</tr>
<tr>
<td>27/F/13 OD 20/20 OS 20/30</td>
<td>25° RXT, 6° RhT</td>
<td>+</td>
<td>OD −½</td>
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<td>16° RXT</td>
<td>+</td>
<td>OD full</td>
<td>+</td>
<td>−</td>
<td>−</td>
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</table>

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; CP, cerebral palsy; CVL, cortical visual loss; DVD, dissociated vertical deviation (divergence); IVH, intraventricular hemorrhage; LET, left esotropia; LHT, left hypertropia; LT(T), intermittent left hypertropia; LH(T), intermittent left hypertropia; LHT, left hypertropia; LN, latent nystagmus; LXT, left exotropia; LHT, left hypertropia; NT, not tested; PACT, prism and alternate cover test; PVL, periventricular leukomalacia; RFT, right exotropia; RF, reversed fixation test; RHT, right hypertropia; RhT, right hypotropia; Rh(T), intermittent right hypotropia; RHT, intermittent right hypertropia; RXT, right exotropia; RX(T), intermittent right exotropia; VA, visual acuity; Δ, prism diopter; °, near measurement.

*Preferred eye for fixation is shown in boldface.*
Using the reversed fixation test, we discovered that dissociated horizontal deviation lies buried away within a consecutive exotropia in 50% of patients who have a history of infantile esotropia. By providing a clinical tool for distinguishing dissociated horizontal deviation from the secondary effects of asymmetric postoperative medial rectus muscle weakness, the reversed fixation test confirms the existence of dissociated horizontal deviation as an entity sui generis. Furthermore, it expands the clinical spectrum of dissociated horizontal deviation to include patients with consecutive exotropia who show no difference in the measured exodeviation when each eye is used to fixate in primary position.

The reversed fixation test provides unique information about the pathophysiology of dissociated horizontal deviation. In the reversed fixation test, fixation through the neutralizing prism with the eye that exerts greater esotonus will induce an abduction movement of the contralateral eye, while fixation through the prism with the eye that exerts less esotonus will induce an equal adduction movement of the contralateral eye. These associated movements occur because the eye that manifests a greater degree of exotropia when the contralateral eye is fixating must also be the eye that generates greater esotonus when it takes up fixation. When fixating through a prism, the more exodeviated eye will drive the contralateral eye inward past midline, both because of its more exodeviated fixational position and because of the lesser esotonus that it exerts. Conversely, the less exotropic eye has to be the eye that generates less esotonus when it takes up fixation. When fixating through a prism, the less exodeviated eye will fail to drive the contralateral eye inward past midline, both because of its less exodeviated fixational position and because of the lesser esotonus that it exerts. From this analysis, we used our clinical examination to determine whether the preferred eye or the non-preferred eye generates greater esotonus in patients with dissociated horizontal deviation.

Using the reversed fixation test, we found that fixation with the non-preferred eye usually exerts greater esotonus than fixation with the preferred eye. In 10 of 14 patients with dissociated horizontal deviation, the preferred eye showed an abduction saccade in the final step of the reversed fixation test (Table 3). Previous clinical descriptions and electro-oculographic studies of patients with dissociated horizontal deviation have also documented a switch from exotropia to esotropia when fixation is switched to the nonpreferred eye. As discussed subsequently, these results cannot be attributed to the effects of asymmetric addiction, demonstrating that dissociated esotonus exists as a distinct condition. This pathomechanism explains how a patient with dissociated horizontal deviation can display an exodeviation of the nonpreferred eye with a paradoxical esodeviation of the preferred eye. Dissociated horizontal deviation produces the equivalent of a primary deviation during fixation with the preferred eye and a secondary deviation during fixation with the nonpreferred eye. Because dissociated horizontal deviation can be evoked in the absence of any movement of the fixing eye, however, the concept of Hering’s law cannot be extrapolated to this dissociated phenomenon.

Because dissociated esotonus is made visible only by the reversed fixation test, the fundamental connection between dissociated esotonus and infantile strabismus has largely eluded our clinical surveillance. Our findings indicate that dissociated esotonus, dissociated vertical divergence, and latent nystagmus constitute a triad of dissociated ocular motor responses to unequal visual input. Primary oblique muscle overaction, while driven by similar visuovestibular input, does not behave as a dissociated movement in that it does not change as a function of relative visual input to the 2 eyes. Far from being uncommon, the 50% prevalence of dissociated esotonus in our cohort of patients with consecutive exotropia approximates the 40% to 90% range of estimates for dissociated vertical divergence in infantile esotropia. This prevalence supports the original contention of Raab that these 2 dissociated deviations are part of a single continuous spectrum. The small size of the re-fixation movement (on the order of 2°-10° in this study) elicited by the reversed fixation test does not signify that the amount of esotonus exerted by monocular fixation with either eye is also small. Because the esotonus exerted by 1 eye has been preneutralized during the prism and alternate cover test, it is only the difference in esotonus exerted by the 2 eyes that determines the size of the re-fixation movement in the reversed fixation test.

Wilson has coined the term dissociated strabismus complex to emphasize that the dissociated eye movements that accompany infantile strabismus can be predominantly ver-

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Reversed Fixation Testa</th>
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<tr>
<td>2/F/12 4/M/17 11/F/10 12/F/49 13/M/4 15/M/17 16/F/35 20/F/24 21/F/15 23/M/23 24/F/23 25/F/27 27/F/13 28/F/7</td>
<td>OD 9° abduction saccade OD 6° abduction saccade OD 4° abduction saccade OD abduction saccade OD abduction saccade OD abduction saccade OD abduction saccade OS abduction saccade OS abduction saccade OS abduction saccade OS abduction saccade OS 10° abduction saccade OS No movement OD 8° abduction saccade OS 2° abduction saccade OD NT OS abduction saccade OD 2° abduction saccade OS 4° abduction saccade OD abduction saccade OS 4° abduction saccade OS 4° abduction saccade OS abduction saccade</td>
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</table>

Abbreviations: NT, not tested; a, prism diopter.

Table 3. Clinical Characteristics of Positive Results of Reversed Fixation Test

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tical, horizontal, or torsional. This concept derives direct support from the finding of a positive Bielschowsky phenomenon in both dissociated vertical divergence and dissociated horizontal deviation. In dissociated vertical divergence, placement of darkening filters of increasing density before the fixating eye causes the hyperdeviating eye to descend progressively below midline. Because the darkened eye maintains fixation, this change cannot be caused by a fixation shift, but results strictly from a change in the balance of luminance to the 2 eyes. In their seminal observations, Wilson and McClatchey observed that “the outwardly drifted eye returned to and crossed the midline, to become slightly esotropic as the fixating eye received progressively less luminance.” In light of our findings, one can speculate that this sensorimotor response may reflect 2 overlapping mecha-

Figure 3. Dissociated horizontal deviation. A and B, The patient shows a greater exodeviation when the preferred right eye is used for fixation. C, Small esodeviation of the right eye after the left exodeviation has been prismatically neutralized. D, Small exodeviation of the left eye after the right exodeviation has been prismatically neutralized. The refixation movement in the final step of the reversed fixation test will therefore consist of a small abduction saccade in the preferred right eye and a small adduction saccade in the nonpreferred left eye. E, Photograph obtained when the patient was under nondepolarizing paralyzing anesthesia shows a large bilateral exodeviation (courtesy of Susana Gamio, MD).
nisms. Assuming that the preferred eye was used for fixation during the test to simulate real-world conditions, shifting the balance of luminance in favor of the nonpreferred eye would be expected to induce a progressive esodeviation because the nonpreferred eye evokes relatively greater esotonus. However, it may also be that increasing fixational effort through the dark filter with the preferred eye directly increases esotonus in the same way that inducing fixation with the nonpreferred eye does. We did not test for the Bielschowsky phenomenon as part of our protocol because Graef has reported it to be difficult to elicit and not as sensitive as reversed fixation testing for establishing the diagnosis.

The frequency of unequal exodeviations did not differ significantly in patients with and without dissociated horizontal deviation. For this reason one should not rely on a difference in the measured exodeviation with each eye fixating in primary position to establish the diagnosis of dissociated horizontal deviation. As seen in cases 27 and 28, it is possible to have dissociated horizontal deviation, as demonstrated by reversed fixation testing, even when the horizontal deviation is the same when either eye fixates in primary position. This situation occurs when the nonpreferred eye exhibits a greater medial rectus weakness secondary to the previous strabismus surgery. Because of the resulting adduction lag, greater medial rectus innervation becomes necessary to fixate with the nonpreferred eye in primary position. As an isolated force, this “fixation duress” would produce a larger primary-position exodeviation of the preferred eye. However, this effect is offset by the greater dissociated esotonus that is simultaneously exerted during fixation with the nonpreferred eye, resulting in no net difference in the measured exodeviations when each eye fixates in primary position. Such “masked” cases have not been included in earlier clinical descriptions of this dissociated horizontal deviation.

Conversely, 3 patients with dissociated horizontal deviation (cases 4, 23, and 24) showed asymmetric exodeviations with each eye fixating in primary position on the basis of asymmetric adduction. In these patients, the “fixation duress” caused by diminished adduction in the nonpreferred eye seemed to override the effects of dissociated esotonus, resulting in a larger exodeviation when the nonpreferred eye was used for fixation. These examples illustrate how asymmetry of exodeviation in the primary position is neither necessary nor sufficient to establish the diagnosis of dissociated horizontal deviation, making the horizontal reversed fixation test the critical diagnostic test for this condition.

Regardless of the presence or absence of dissociated horizontal deviation, we found that 7 of the 9 patients (cases 1, 4, 6, 20, 23, 24, and 27) with asymmetric adduction preferred fixation with the eye that showed stronger adduction. Because a greater adduction lag in the nonpreferred eye generates a larger exotropia of the preferred eye, while dissociated horizontal deviation is usually associated with a greater exotropia of the nonpreferred eye, it follows that the effects of asymmetric adduction must be distinct from those of dissociated horizontal deviation.

A corollary clinical sign of dissociated horizontal deviation was the frequent discrepancy between the cosmetic and the measured exodeviation. It was common to find patients with dissociated horizontal deviation who intermittently manifested a large bilateral exodeviation during periods of visual inattention (Figure 5), while displaying measurements within the monofixation range during prism and alternate cover testing. In our experience, the finding of a simultaneous bilateral exotropia is a distinguishing clinical sign of dissociated horizontal deviation (Figure 5). For reasons that are unclear, we have not observed this sign in patients with other forms of intermittent exotropia.
Previous investigations of patients with infantile esotropia have found latent nystagmus in 25% to 52% and dissociated vertical divergence in 40% to 90% of cases. It is not known how these observed frequencies vary with age, previous surgical realignment, or the development of consecutive exotropia. The present study found latent nystagmus in 7 of 28 patients (25%) and dissociated vertical divergence in 19 of 28 patients (68%) with consecutive exotropia. In our patients with consecutive exotropia, the prevalence of dissociated horizontal deviation fell between that of latent nystagmus and dissociated vertical divergence. Dissociated horizontal deviation correlated significantly with dissociated vertical divergence but not with latent nystagmus, reinforcing the prevailing notion that dissociated horizontal deviation is a variable component of dissociated vertical divergence.

Using scleral search coil recordings, Guyton et al documented the coexistence of dissociated esotonus in patients with dissociated vertical divergence and postulated that dissociated vertical divergence existed solely for the purpose of damping latent nystagmus. In light of our findings, it now seems probable that any horizontal damping of latent nystagmus is an epiphenomenon of the dissociated esotonus that characterizes dissociated horizontal deviation, rather than the result of any elaborate compensatory adaptation to improve visual acuity in latent nystagmus. Our findings support Graf's interpretation that the secondary effect of dissociated horizontal deviation can be to diminish the horizontal component of latent nystagmus. This secondary damping may have contributed to the noncorrelation between dissociated horizontal deviation and latent nystagmus in our study.

The evolutionary underpinnings of dissociated horizontal deviation seem to overlap those of dissociated vertical divergence and latent nystagmus. The relative roles of luminance vs optokinetic input in generating dissociated esotonus are still undetermined, although there is some evidence that both forms of peripheral visual input play a role in its pathogenesis. Dissociated vertical divergence has characteristics of the dorsal light reflex in lateral-eyed animals. This subcortical visual reflex uses relative luminance input to the 2 eyes to maintain vertical orientation. In the upright position, dorsal light from the sky above provides equal luminance to the 2 eyes. Unequal luminance to the 2 eyes is therefore interpreted as body tilt (with the eye receiving greater luminance input misdirected toward the sky), necessitating a postural readjustment and a vertical divergence of the eyes. The striking concordance between dissociated horizontal deviation and dissociated vertical divergence in this study, together with previous observations that both dissociated deviations display the Bielschowsky phenomenon, suggests that these peripheral luminance reflexes can function independently of central fixational mechanisms.

Lateral-eyed animals also display a monocular nasotemporal asymmetry to optokinetic targets, wherein nasally moving rotations of the visual world generate much stronger optokinetic responses than temporally moving rotations. During a head turn, the eye perceiving a nasally stimulated eye misdirected toward the sky), necessitating a postural readjustment and a vertical divergence of the eyes. The striking concordance between dissociated horizontal deviation and dissociated vertical divergence in this study, together with previous observations that both dissociated deviations display the Bielschowsky phenomenon, suggests that these peripheral luminance reflexes can function independently of central fixational mechanisms.

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