Motion Detection Deficits in Infantile Esotropia Without Nystagmus

Josephine Shallo-Hoffmann,* Mary Faldon,* Susan Hague,† Paul Riordan-Eva,*‡ Peter Fells,‡ and Michael Gresty*

Purpose. To investigate whether adults with infantile strabismus but without latent nystagmus have abnormalities of horizontal motion detection.

Methods. Eleven adult subjects with infantile esotropia but without latent nystagmus and 15 control subjects were required to detect the onset of motion and drift direction of a sinusoidal, spatial frequency grating that moved with linearly increasing velocity. The grating was presented monocularly in paracentral vision at an eccentricity of 16.5° with a field size of 18°. The contrast of the grating was just above contrast threshold for visibility.

Results. The mean velocity threshold for detection of motion was raised significantly in the patient group compared with the control group. Nine of the 11 subjects with infantile esotropia demonstrated directional asymmetry for the detection of motion. Thresholds were elevated more often when the grating was moving nasally in the squinting eye and temporally in the nonsquinting eye, and raised thresholds were more prevalent in the squinting eye.

Conclusions. The findings indicate that in infantile esotropia, the presence of motion perception deficits are not always associated with the development of latent nystagmus. The predominance of nasally directed motion deficits in the squinting eye and temporally directed motion deficits in the nonsquinting eye was unexpected and may have been caused by abnormal development of cortical motion processing. Invest Ophthalmol Vis Sci. 1997;38:219-226.

The purpose of this study was to investigate whether adults with infantile esotropia but without nystagmus have an abnormal threshold for motion detection and whether any such abnormality was related to the direction of the stimulus motion (either temporally or nasally to the viewing eye) or the part of the retina stimulated (either nasal or temporal retina).

The pathogenesis of infantile esotropia is unknown. It can occur at birth or can develop before the age of 6 months. It is associated with latent or manifest latent nystagmus in 25% of patients and with dissociated vertical deviation in 43% of patients. Almost all patients with latent nystagmus or dissociated vertical deviation have infantile strabismus. Latent nystagmus is a conjugate jerk nystagmus that first occurs in early infancy and persists throughout life. When both eyes are viewing the nystagmus is minimal (rarely completely absent), whereas with monocular viewing the nystagmus increases in intensity. The slow phases of the nystagmus are directed temporal-to-nasal with respect to the viewing eye.

Latent nystagmus has been hypothesized to be caused by a nasal–temporal velocity bias in the optokinetic system or in the smooth pursuit system, caused by a degradation of cortical motion signals specific to the monocular viewing condition. Asymmetry in motion perception is, therefore, to be considered as a potential primary deficit, leading to the development of latent nystagmus. An alternative view is that anomalous optokinetic nystagmus (OKN) and pursuit responses (either OKN or pursuit) may be caused by abnormal visual motion processing secondary to the heterotropic eye position and lifelong presence of nystagmus. Previous studies involving patients with infantile esotropia and nystagmus attempted to control for the influence of nystagmus.
by taking the slow phase velocity into account when analyzing results. However, it is difficult to control for the confounding influence of the nystagmus during testing because any stimulus motion that is not orthogonal to the nystagmus will influence the response. This study demonstrates that motion detection deficits occur in persons with infantile esotropia without nystagmus.

METHODS

Visual Motion Detection Task

Determination of Threshold Values for Visibility of the Static Stimulus. Naive, dark-adapted subjects sat 106 cm before a screen (236 cm × 145 cm), monocularly fixing a dim white target (3 cm × 2.5 cm) in the straight-ahead direction, with head and chin restrained. The stimulus consisted of a back-projected, sinusoidally modulated, gray scale spatial frequency grating (0.25 cycle/deg, 57 cm in diameter), located 16.5° off axis (13 cm from the fixation target to the border of the grating), either to the right or to the left of the fixation target. In this position, the grating subtended an angle of 18° parfoveally. By varying the location of the grating (either to the right or to the left of the subject), the nasal and temporal parts of the retina of each eye were stimulated. The grating was vignetted with a circular mask to eliminate edge flicker and motion cues derived from stripe width (Fig. 1).

The contrast of the static grating was adjusted to the point of subjective visibility, for each eye separately, by a staircase procedure in which 1 of 40 contrast levels was presented on every trial. The range of contrast values was selected so that the midpoint would be approximately at the threshold detection level, using an adaptive procedure to find the contrast at which the stimulus was 50% visible. The contrast of the grating was set just above this threshold value. The specific increment above threshold was chosen to ensure low but reliable visibility, although the increment was never less than 0.5% nor greater than 0.9%. The threshold was measured before each of the four test conditions, described below.

Motion Detection Task. Motion detection thresholds were measured under four viewing conditions: left eye, temporal retina; left eye, nasal retina; right eye, nasal retina; and right eye, temporal retina. The motion detection task followed directly after the determination of threshold visibility. The grating was initially stationary and then began to move, accelerating at a constant rate of 0.09°/sec². The subject’s task was to report the drift direction of the grating as soon as the grating first appeared to move. In each of the four conditions, the subject saw six trials with motion in the temporal direction and six trials with motion in the nasal direction. Thus, in a single session, the subject experienced 48 motion detection trials.

Infrared limbal reflection (DC, 250 Hz bandwidth, resolution 0.1°) eye movement recordings were performed on all patients as part of their initial screening and on two patients and two control subjects during the test session to verify our clinical observations that the eyes were free of spontaneous or induced nystagmus and were otherwise stable under the conditions of experimentation.

Task Repetition and Performance. Because subjects were not trained in the task before testing for the detection of motion, it was thought that a finding of directional asymmetry may occur because of the novelty of the task. To investigate whether practice would either improve performance or eliminate asymmetric responses, the task was repeated twice, with a rest pause between test sessions, with two patients and two control subjects.

Subjects

Eleven adult subjects (age range, 19 to 46 years) with a history of childhood esotropia that started before 6
months of age (confirmed by examination of medical records to be infantile esotropia) were recruited to participate in this study. All underwent complete ophthalmologic screening at the time of the test session, including ophthalmoscopic examination for minimal signs of nystagmus or other gaze instability. None had evidence of latent or manifest latent nystagmus as verified by eye movement recordings. Eight had undergone strabismus surgery. Six still had esotropia after surgery (residual esotropia), and in all of them, there was no change in the eye that maintained fixation under binocular viewing conditions (nonalternating). The two patients with a divergent squint after surgery (residual esotropia) and the three unoperated patients had alternating strabismus (either eye fixated under binocular viewing conditions) (Table 1). All patients had normal visual acuity.

Fifteen age-matched healthy volunteers with normal visual acuity and orthotropic ocular alignment participated in this study. All participants underwent comprehensive ophthalmologic screening. It was the first time any of the subjects participated in the detection task, and they were naive to the purposes of the study. All participants were fully informed about the nature of the procedures, which were performed according to the guidelines of the Declaration of Helsinki, and all gave their written consent before the beginning of the experiment.

**Data Analysis**

Data were analyzed by comparing motion detection scores between patient and control groups, within patient differences caused by direction of stimulus motion, and within the residual esotropia patient group comparing data from the normally squinting eye with the nonsquinting eye.

**RESULTS**

**Threshold Values for Patients and Controls**

The mean threshold for the detection of motion, over all test conditions, was raised in the patient group \((n = 11; 0.69° ± 0.30°/second)\) compared with the control group \((n = 15; 0.59° ± 0.30°/second)\); \(F = 20.79 (1,972); P < 0.001.\) Figure 2A shows the mean score for the patient (circles) and the control (squares) groups for each of the four conditions when the grating was moving temporally (T) or nasally (N) to the viewing eye, and Figure 2B compares data for the six patients with residual esotropia with respect to their squinting and nonsquinting eyes for each of the four viewing conditions. Significantly elevated threshold values were found in the squinting eye when the grating moved nasally.

An increase in velocity occurred for the detection of motion in one direction compared with the other in all six patients with residual esotropia, 1 of 2 patients with consecutive exotropia, 2 of 3 patients with unoperated alternating esotropia (patients 1 to 7, 9, and 11) and in 2 of the 15 control subjects. In the patient group, these raised thresholds occurred in 22 of the possible 36 conditions (four conditions per subject); 16 times when the grating moved nasally and six times when it moved temporally to the viewing eye. Data from all the patients are reported in Table 2, and directional asymmetry is illustrated in Figure 3.

No differences pertaining to the area of the retina stimulated were found in the patient group (10 asymmetric responses occurred in the nasal retina and 12 in the temporal retina). Visual inspection and eye movement recordings did not show the presence of optokinetic nystagmus under the specific conditions of paracentral stimulation used during the detection.

**TABLE 1. Clinical Status**

<table>
<thead>
<tr>
<th>Patient Number</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Snellen Acuity Corrected</th>
<th>DVD</th>
<th>Stereopsis (tittnus)</th>
<th>Ocular Alignment*</th>
</tr>
</thead>
<tbody>
<tr>
<td>RET 1</td>
<td>M</td>
<td>30</td>
<td>1.5 1.5</td>
<td>nil</td>
<td>400&quot; Residual eso (r)</td>
<td></td>
</tr>
<tr>
<td>RET 2</td>
<td>F</td>
<td>24</td>
<td>1.2 1.2</td>
<td>nil</td>
<td>140&quot; Residual eso (l)</td>
<td></td>
</tr>
<tr>
<td>RET 3</td>
<td>M</td>
<td>30</td>
<td>1.2 1.2</td>
<td>nil</td>
<td>Residual eso (r)</td>
<td></td>
</tr>
<tr>
<td>RET 4</td>
<td>M</td>
<td>23</td>
<td>1.2 1.2</td>
<td>nil</td>
<td>200&quot; Residual eso (l)</td>
<td></td>
</tr>
<tr>
<td>RET 5</td>
<td>M</td>
<td>24</td>
<td>1.2 1.2</td>
<td>nil</td>
<td>Residual eso (l)</td>
<td></td>
</tr>
<tr>
<td>RET 6</td>
<td>M</td>
<td>25</td>
<td>1.2 1.2</td>
<td>nil</td>
<td>Residual eso (l)</td>
<td></td>
</tr>
<tr>
<td>U ET 7</td>
<td>M</td>
<td>46</td>
<td>1.0 1.0</td>
<td>+</td>
<td>Nil Unoperated eso (alt)</td>
<td></td>
</tr>
<tr>
<td>U ET 8</td>
<td>M</td>
<td>30</td>
<td>1.2 1.0</td>
<td>nil</td>
<td>Unoperated eso (alt)</td>
<td></td>
</tr>
<tr>
<td>U ET 9</td>
<td>M</td>
<td>40</td>
<td>1.0 1.0</td>
<td>nil</td>
<td>Unoperated eso (alt)</td>
<td></td>
</tr>
<tr>
<td>C EX 10</td>
<td>M</td>
<td>28</td>
<td>1.2 1.2</td>
<td>+</td>
<td>Consecutive exo (alt)</td>
<td></td>
</tr>
<tr>
<td>C EX 11</td>
<td>M</td>
<td>19</td>
<td>1.2 1.2</td>
<td>nil</td>
<td>Consecutive exo (alt)</td>
<td></td>
</tr>
<tr>
<td>Con 15</td>
<td>M</td>
<td>19-46</td>
<td>1.0 1.0</td>
<td>nil</td>
<td>40-60&quot; Orthotropia</td>
<td></td>
</tr>
</tbody>
</table>

DVD = dissociated vertical deviation; + = positive; RET = residual esotropia; U ET = unoperated esotropia; C EX = consecutive exotropia; (r) = squinting eye right; (l) = squinting eye left; eso = esotropia; exo = exotropia; alt = alternating; Con = control subjects.

* At the time of test session.
FIGURE 2. (A) Mean velocity detection threshold values for the patients (circles; n = 11) and control subjects (squares; n = 15) in each of the four viewing conditions. (B) Mean velocity detection threshold values for the six patients with residual esotropia in each of the four viewing conditions for squinting and nonsquinting eyes. Differences occurred only in the squinting eye. Squinting eye, TR: t(10) = 3.25; P < 0.001. Squinting eye, NR: t(10) = 1.71; P < 0.05. Nonsquinting eye, TR: t(10) = 1.27, NS. Nonsquinting eye, NR: t(10) = 1.16. LE TR = left eye, temporal retina; LE NR = left eye, nasal retina; RE NR = right eye, nasal retina; RE TR = right eye, temporal retina. T = grating as it moved temporally to the viewing eye; N = grating as it moved nasally to the viewing eye. Bars = standard deviation. (unfilled icons) Grating as it moved temporally to the viewing eye. (filled icons) Grating as it moved nasally to the viewing eye.

FIGURE 3. (A) Directional asymmetry values for the detection of motion in six patients with residual esotropia presented for the four viewing conditions: 1 = left eye, temporal retina; 2 = left eye, nasal retina; 3 = right eye, nasal retina; 4 = right eye, temporal retina. (unfilled icons) Grating as it moved temporally to the viewing eye. (filled icons) Grating as it moved nasally to the viewing eye. (crosses) Detection values that did not differ significantly with direction of the grating motion. Bars = standard deviation. (B) Directional asymmetry values for the detection of motion from three patients with unoperated esotropia and two patients with consecutive exotropia are presented for the viewing conditions as in A.

Ocular Alignment at the Time of the Test Session

In the six patients with residual esotropia, asymmetric motion detection occurred in 8 of 12 cases when viewing with the squinting eye, compared with 5 of 12 cases when viewing with the nonsquinting eye (12 cases: 6 subjects, 2 stimulus sites per eye). Raised thresholds occurred in the nasal direction 8 of 8 times in the squinting eye (sign test, P < 0.004) compared to 2 of 5 times in the nonsquinting eye (sign test, P = 0.5 NS). Figure 4 shows directional asymmetry data for the six patients with residual esotropia according to eye alignment at the time of the test session, illustrating that significant differences occurred more often in the squinting than in the nonsquinting eye and raised thresholds occurred more often when the grat-
TABLE 2. Mean Threshold Velocity for the Detection of Motion

<table>
<thead>
<tr>
<th>Patient</th>
<th>LE TR (°/s)</th>
<th>Direction of Asymmetry</th>
<th>LE NR (°/s)</th>
<th>Direction of Asymmetry</th>
<th>RE NR (°/s)</th>
<th>Direction of Asymmetry</th>
<th>RE TR (°/s)</th>
<th>Direction of Asymmetry</th>
</tr>
</thead>
<tbody>
<tr>
<td>R ET 1</td>
<td>1.04 ± 0.12</td>
<td>N*</td>
<td>0.72 ± 0.09</td>
<td>T†</td>
<td>1.04 ± 0.20</td>
<td>N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R ET 2</td>
<td>0.61 ± 0.22</td>
<td>NS</td>
<td>0.93 ± 0.13</td>
<td>N*</td>
<td>0.81 ± 0.10</td>
<td>NS</td>
<td>0.67 ± 0.17</td>
<td>T†</td>
</tr>
<tr>
<td>R ET 3</td>
<td>0.59 ± 0.15</td>
<td>NS</td>
<td>0.50 ± 0.18</td>
<td>N§</td>
<td>0.80 ± 0.15</td>
<td>NS</td>
<td>0.60 ± 0.08</td>
<td>T†</td>
</tr>
<tr>
<td>R ET 4</td>
<td>1.21 ± 0.20</td>
<td>NS</td>
<td>1.10 ± 0.13</td>
<td>NS</td>
<td>1.02 ± 0.24</td>
<td>NS</td>
<td>1.19 ± 0.17</td>
<td>N</td>
</tr>
<tr>
<td>R ET 5</td>
<td>1.12 ± 0.11</td>
<td>NS</td>
<td>1.01 ± 0.37</td>
<td>NS</td>
<td>1.09 ± 0.15</td>
<td>NS</td>
<td>0.90 ± 0.21</td>
<td>N</td>
</tr>
<tr>
<td>R ET 6</td>
<td>1.34 ± 0.36</td>
<td>NS</td>
<td>0.79 ± 0.20</td>
<td>N*</td>
<td>0.62 ± 0.13</td>
<td>NS</td>
<td>0.82 ± 0.13</td>
<td>NS</td>
</tr>
<tr>
<td>R ET 7</td>
<td>0.70 ± 0.20</td>
<td>NS</td>
<td>0.47 ± 0.07</td>
<td>NS</td>
<td>0.76 ± 0.10</td>
<td>NS</td>
<td>0.68 ± 0.15</td>
<td>T†</td>
</tr>
<tr>
<td>R ET 8</td>
<td>0.57 ± 0.23</td>
<td>N*</td>
<td>0.41 ± 0.12</td>
<td>N§</td>
<td>0.27 ± 0.06</td>
<td>T†</td>
<td>0.40 ± 0.10</td>
<td>NS</td>
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<tr>
<td>R ET 9</td>
<td>0.27 ± 0.08</td>
<td>NS</td>
<td>0.28 ± 0.10</td>
<td>NS</td>
<td>0.45 ± 0.11</td>
<td>NS</td>
<td>0.35 ± 0.10</td>
<td>T†</td>
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<tr>
<td>U ET 7</td>
<td>0.46 ± 0.08</td>
<td>NS</td>
<td>0.42 ± 0.11</td>
<td>NS</td>
<td>0.41 ± 0.11</td>
<td>NS</td>
<td>0.50 ± 0.05</td>
<td>N§</td>
</tr>
<tr>
<td>U ET 8</td>
<td>0.25 ± 0.07</td>
<td>NS</td>
<td>0.52 ± 0.09</td>
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<td>0.52 ± 0.11</td>
<td>NS</td>
<td>0.36 ± 0.04</td>
<td>T†</td>
</tr>
<tr>
<td>U ET 9</td>
<td>0.66 ± 0.17</td>
<td>T*</td>
<td>0.92 ± 0.23</td>
<td>T*</td>
<td>1.03 ± 0.18</td>
<td>N§</td>
<td>0.80 ± 0.08</td>
<td>N§</td>
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<tr>
<td>U ET 10</td>
<td>1.06 ± 0.15</td>
<td>NS</td>
<td>1.41 ± 0.16</td>
<td>NS</td>
<td>0.80 ± 0.24</td>
<td>NS</td>
<td>0.66 ± 0.18</td>
<td>NS</td>
</tr>
<tr>
<td>U ET 11</td>
<td>0.74 ± 0.11</td>
<td>NS</td>
<td>1.08 ± 0.25</td>
<td>NS</td>
<td>0.80 ± 0.19</td>
<td>NS</td>
<td>0.91 ± 0.16</td>
<td>NS</td>
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<tr>
<td>C EX 10</td>
<td>0.85 ± 0.15</td>
<td>NS</td>
<td>0.97 ± 0.23</td>
<td>NS</td>
<td>0.93 ± 0.18</td>
<td>NS</td>
<td>0.95 ± 0.18</td>
<td>NS</td>
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<tr>
<td>C EX 11</td>
<td>0.61 ± 0.10</td>
<td>NS</td>
<td>0.46 ± 0.12</td>
<td>NS</td>
<td>0.69 ± 0.17</td>
<td>NS</td>
<td>0.69 ± 0.10</td>
<td>N§</td>
</tr>
<tr>
<td>C EX 12</td>
<td>0.60 ± 0.10</td>
<td>NS</td>
<td>0.54 ± 0.15</td>
<td>NS</td>
<td>0.58 ± 0.19</td>
<td>NS</td>
<td>0.51 ± 0.15</td>
<td>N§</td>
</tr>
<tr>
<td>C EX 13</td>
<td>1.04 ± 0.15</td>
<td>NS</td>
<td>0.57 ± 0.07</td>
<td>NS</td>
<td>0.62 ± 0.15</td>
<td>NS</td>
<td>0.54 ± 0.15</td>
<td>NS</td>
</tr>
<tr>
<td>C EX 14</td>
<td>0.94 ± 0.25</td>
<td>NS</td>
<td>0.68 ± 0.19</td>
<td>NS</td>
<td>0.79 ± 0.12</td>
<td>NS</td>
<td>0.67 ± 0.17</td>
<td>NS</td>
</tr>
<tr>
<td>C EX 15</td>
<td>0.55 ± 0.11</td>
<td>N§</td>
<td>0.52 ± 0.18</td>
<td>N§</td>
<td>0.51 ± 0.14</td>
<td>T†</td>
<td>0.52 ± 0.13</td>
<td>N*</td>
</tr>
<tr>
<td>C EX 16</td>
<td>0.25 ± 0.07</td>
<td>NS</td>
<td>0.36 ± 0.15</td>
<td>NS</td>
<td>0.51 ± 0.11</td>
<td>T†</td>
<td>0.32 ± 0.09</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS = not significant; LE, RE = left and right eye, respectively; N = nasalward; T = temporalward; TR, NR = temporal and nasal retina, respectively. The top and bottom value for each pair represents finding when the grating was moving nasally and temporally respectively.

† P < 0.001.
* P < 0.005.
± P < 0.01.
|| P < 0.03.
§ P < 0.05.

ing moved nasally in the squinting eye and temporally in the nonsquinting eye.

Task Repetition and Performance

When the task was repeated, directional asymmetry for the detection of motion was no longer found for the two control subjects, and practice improved the velocity necessary to detect motion by a mean decrease of 0.12°/second (t = 4.29df=46, P < 0.0001) for control subject 1 and 0.09°/second (t = 4.09df=46, P < 0.0001) for control subject 2 (Fig. 5).

Data from patient ET4 showed a decrease of 0.25°/second (t = 4.55df=46, P < 0.0001) to detect motion, and data for patient ET2 showed an increase of 0.13°/second for threshold values. However, practice did not eliminate directional asymmetry in the patients. Values obtained for the two patients are presented in Figure 4, and are labeled 2nd and 3rd repetitions.

DISCUSSION

Detection of Motion at Threshold

The mean threshold value for the detection of motion was raised in the patient group compared with the control group. This finding was unexpected because the patients did not have nystagmus, and it implies that although nystagmus may raise motion detection thresholds, it is not the sole cause of visual motion processing abnormalities.

Directional Asymmetry Values (Nasal Versus Temporal) for Motion Detection

Nine of 11 subjects with infantile esotropia showed significant directional asymmetry values for the detection of velocity, and the asymmetries occurred equally as often when the nasal or temporal retina was stimulated. The six patients with residual esotropia had asymmetrical responses, and thresholds were raised more often when the stimulus was moving nasally to the squinting eye and temporally to the nonsquinting eye, with a prevalence for asymmetry occurring in the squinting eye.

In this study, raised thresholds and directional asymmetries established that patients with infantile strabismus and normal visual acuity showed differences from the control group for the detection of visual motion, which can be attributed neither to nystagmus nor to visual deprivation (amblyopia). It has been proposed that strabismic eye movement may re-
reflect an incomplete visual development during early infancy, and it has been shown that normal infants and adults with strabismus display more vigorous horizontal OKN in the nasal direction. Good stereopsis also has been shown not to be sufficient for the development of symmetric OKN in infants with infantile esotropia, indicating that the timing of onset of esotropia may be a critical factor. Recently, Epelbaum and Teller also postulated that timing may influence OKN, reporting “reversed” OKN asymmetry—that is, diminished responses occurred in the nasal direction with isoluminant stimuli in normal infants. These authors suggested that cortical, uncrossed, chromatic pathways may mature earlier than cortical, crossed, chromatic pathways during early visual development, and it would be in these cortical pathways that an imbalance occurs, causing the asymmetric OKN responses in the infant. Croganale and Schor investigated the possibility that adults with a history of infantile esotropia may show similar OKN asymmetry with isoluminant stimuli. Patients in their study did not have nystagmus, and the nonsquinting eyes were tested. Contrary to the findings of Epelbaum and Teller, Croganale and Schor found that OKN was diminished in the temporal direction with luminance and isoluminance targets (a finding that is consistent with the data presented here for the nonsquinting eye; see Fig. 4, bottom), and they concluded that one possibility for the differences in findings may imply that OKN asymmetry in strabismus does not indicate maldevelopment or incomplete visual development.

Studies that directly investigated motion perception by measuring pursuit responses in subjects with strabismus showed that nasally directed target motion evoked more vigorous responses in adults with strabismus and nystagmus. Because the cortical motion pathways provide visual input for pursuit eye movement, these findings were interpreted as supporting the proposal that the anomalies reflect maldevelopment in these pathways. However, it is difficult to judge the precise influence of latent nystagmus biasing asymmetric responses in these studies because of the waveform characteristics of the nystagmus.

Recently, we investigated the influence of latent nystagmus in patients with infantile strabismus using the same task described here, and the results showed a reduced sensitivity to temporally directed motion. When nystagmus intensity was increased through pro-

FIGURE 4. Directional asymmetry for the detection of motion in six patients with residual esotropia with respect to ocular alignment at the time of the test session. Directional asymmetry occurred more often when the squinting eye was tested (top) with elevated thresholds when the grating moved nasally (filled icons) to the eye. When differences were found in the nonsquinting eye, raised thresholds occurred more often when the grating moved temporally (unfilled icons). TR = temporal retina; NR = nasal retina; T = grating as it moved temporally to the viewing eye; N = grating as it moved nasally to the viewing eye. Bars = standard deviation. 2 and 3 = data from the second and third repetition of the task performed by patients ET2 and ET4.
FIGURE 5. Directional asymmetry that occurred during the first test session for the detection of motion in two control subjects. Data from the second and third test sessions show the absence of directional asymmetry and the decrease in velocity caused by task repetition. C1 = control subject 1; C2 = control subject 2; 1 = first repetition of the task; 2 = second repetition of the task. (unfilled icons) Grating as it moved temporally to the viewing eye. (filled icons) Grating as it moved nasally to the viewing eye. (crosses) Detection values that did not differ significantly with direction of the grating motion. Bars = standard deviation.

Longed monocular occlusion, its influence affected temporally directed motion, even when the viewing eye was held in addiction to damp nystagmus completely during the testing session. However, the influence of latent nystagmus could not explain all the directional asymmetry found. Similarly, Kommerell and colleagues reported asymmetry of motion visual-evoked potentials (VEPs), correlating the strength of latent nystagmus and OKN asymmetry with VEP asymmetry in patients with infantile esotropia. No significant correlation could be found between the strength of latent nystagmus, or velocity of the slow phase of OKN, and VEP asymmetry. Therefore, they concluded that latent nystagmus could not explain all the VEP asymmetries found in these patients with infantile strabismus. Thus, a consensus is emerging that there is a disorder in motion perception in strabismus regardless of the presence of nystagmus.

Task repetition indicated that although practice may decrease detection thresholds, it could account for neither the significant differences in threshold detection values between patient and control groups nor the consistent finding of directional asymmetry in repetitions of the task that occurred only in the patient group. As expected, these findings show that practice can improve performance but cannot account for or eliminate the finding of directional asymmetry in the patient group.

In summary, abnormal motion detection can occur in infantile esotropia in the absence of nystagmus. The threshold for the detection of motion was elevated, and directional asymmetries were observed that cannot be explained by amblyopia, dissociated vertical deviation, or nystagmus. The predominance of nasally directed motion detection deficits was unexpected and was discussed as evidence for theories that the deficits are caused by maldevelopment of cortical motion processing.

Key Words

infantile esotropia, infantile strabismus, motion detection, motion perception, strabismus

References


