Effect of Monocular Visual Loss upon Stability of Gaze


Using the eye-coil/magnetic field method, we measured horizontal and vertical movements of both eyes in four patients with monocular loss of vision while they attempted steady, binocular fixation of a visual target. We also measured gaze stability in two normal subjects while they fixed upon a target monocularly, and in one patient with congenital, bilateral blindness. In the patients with monocular visual loss, gaze instability was greater in the blind eye, both vertically and horizontally, compared either with their seeing eye or with nonviewing eyes of control subjects. Gaze instability due to monocular blindness resulted from: (1) low-frequency, low-amplitude, bidirectional drifts that were more prominent vertically; and (2) unidirectional drifts, with nystagmus, that were more prominent in the horizontal plane. Gaze-evoked nystagmus, however, was not a feature of monocular blindness.

Thus, the gaze instability of monocular blindness may reflect disruption of: (1) a monocular visual stabilization system; (2) fusional vergence mechanisms; or (3) both. In contrast, bilateral congenital blindness led to nystagmus with horizontal and vertical components and a wandering null point, indicative of an abnormal neural integrator.

Materials and Methods

Patients with monocular visual loss and clinically evident vertical gaze instability were selected for study by one of us (RLT). Clinical data from the four patients and two normal subjects (one naive, one experienced) are summarized in Table 1. In patients 3 and 4, vision was modestly reduced in the "good eye" due to recent optic neuritis and congenital optic nerve hypoplasia, respectively. After obtaining informed consent, movements of both eyes were recorded simultaneously using a magnetic search coil system (CNC Engineering, Seattle, WA). The horizontal system used a rotating vector and the vertical system an alternating vector. The electronic noise of the system was <3.0 minutes of arc and, after differentiation, 0.5 deg/sec. Scleral search coils were calibrated on a protractor device prior to placing them on patients' and subjects' eyes. During the recording session, it was not possible to stabilize patients' heads on a bite board; both normal subjects and patients braced their heads against a firm support. Patients and subjects fixed upon a projected laser spot subtending 9 minutes of arc on a tangent screen at 1.3 m, for several 30 sec epochs; low, background lighting was present. Subjects and patients were encouraged to fixate the target and not to blink throughout the test. In addition, patients' eye movements were recorded while the "good eyes" were occluded and they

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attempted to look at the remembered target location. Control subjects first fixated monocularly with the right eye and then with the left eye; their eye movements were also recorded in darkness while they attempted to look at the remembered target location. The bilaterally blind patient was asked to imagine a target straight ahead. In addition to fixation behavior, we measured saccades to targets located 10° from the fixation light, both horizontally and vertically.

Data were stored on magnetic tape and displayed on a pen recorder (system bandwidth 0–100 Hz). Data were digitized, off-line, at 100 Hz after filtering at 0 to 40 Hz and analyzed using an interactive program. Since neither eye position nor eye velocity was normally distributed, due to disruptions of fixation by drifts and saccades, we calculated both standard deviation (SD) and interquartile range as separate measures of the variation of the data. Both gave similar results, so we present just SD values. We discarded samples contaminated by blinks and then made measurements of SD of gaze and gaze velocity, based on a minimum of 1000 consecutive data samples (corresponding to 10 sec). In addition we conducted a 2048 point fast Fourier transform on eye velocity data.

In addition to measurement of fixation behavior, we compared horizontal and vertical saccades in patients 1 and 2, who had normal vision in one eye. We measured saccadic pulse gain (magnitude of saccadic pulse/magnitude of target displacement) from analog records that synchronously displayed eye position and velocity. The movement due to the saccadic pulse was defined from the point where eye velocity first exceeded 40°/sec to where it first fell to below 40 deg/sec. We were unable to reliably measure saccadic step gain because of the continuous drifts that occurred in the eyes with poorer vision.

Results

Measurements of horizontal and vertical gaze stability during attempted fixation are summarized in Table 2. Steady fixation was variably disrupted by slow drifts of the eyes or by small saccades. Slow, low-frequency drifts of the eyes may cause appreciable increases in SD of gaze position but have little effect on SD of gaze velocity. Conversely, small, to-and-fro saccades may have little effect on SD of gaze position but cause a considerable increase in SD of gaze velocity. Thus, SD of both gaze position and velocity are presented in Table 2. Differences between the two eyes indicate disconjugacies.

Findings in Normal Subjects

The two normal subjects showed greater variability of horizontal gaze in the eye under cover than in the fixating eye. There was no difference, however, between the two eyes in terms of vertical gaze or of horizontal or vertical gaze velocity. Overall, the stability of gaze in these normal subjects was similar to that reported by Skavenski and colleagues in normal subjects who were attempting to hold their heads as still as possible.12

Findings in Patients with Monocular Blindness

The stability of gaze in the eyes with better vision was similar to or mildly impaired compared with the viewing eye of control subjects. In patients 1 and 2, who had 20/20 visual acuity in their better eye, SD of gaze position was less than 0.5°, and SD of gaze velocity was less than 2°/sec, except for patient 2 in the horizontal plane, where frequent, small, to-and-fro saccades ("square-wave jerks") disrupted fixation. Square-wave jerks are a normal finding in some elderly subjects.13 Patient 3, despite visual acuity of 20/30, had gaze that was almost as stable as the normal subjects. Patient 4, with longstanding visual acuity of 20/60, had the least stable gaze; in the horizontal plane this was due to manifest latent nystagmus.

In the eyes with poorer vision, gaze stability was disrupted in both the vertical and horizontal planes.
Tabl 2. Measurements of fixation stability

<table>
<thead>
<tr>
<th>Subject</th>
<th>Horizontal position. SD (deg)</th>
<th>Vertical position. SD (deg)</th>
<th>Horizontal velocity. SD (deg/sec)</th>
<th>Vertical velocity. SD (deg/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BE</td>
<td>PE</td>
<td>BE</td>
<td>PE</td>
</tr>
<tr>
<td>P1</td>
<td>0.10</td>
<td>1.12</td>
<td>0.29</td>
<td>2.36</td>
</tr>
<tr>
<td>P2</td>
<td>0.40</td>
<td>0.60</td>
<td>0.31</td>
<td>7.15</td>
</tr>
<tr>
<td>P3</td>
<td>0.09</td>
<td>0.50</td>
<td>0.16</td>
<td>1.83</td>
</tr>
<tr>
<td>P4</td>
<td>0.22</td>
<td>1.09</td>
<td>0.68</td>
<td>1.11</td>
</tr>
<tr>
<td>P5</td>
<td>5.20</td>
<td>6.50</td>
<td>12.70</td>
<td>12.87</td>
</tr>
</tbody>
</table>

Subject BE = Better eye; PE = Poorer eye; SD = Standard deviation.

P = Patient; Ss = pooled subject data; DB = Down-beating; UB = Up-beating; N = Neutral.

Discussion

Using the magnetic search coil method, we have confirmed that long-term deprivation of vision in one eye is associated with disconjugate nystagmus, particularly in the vertical plane. In patients with bilateral visual loss, continuous nystagmus was present. In patients 1 and 2, the coefficient of disconjugacy tended to be larger in the eye with better vision. Patients 3 showed constancy for vertical movements, saccadic pulse gain in the horizontal plane was larger in the eye with better vision (Table 3). For horizontal movements, saccadic pulse gain in the vertical plane was smaller in the eye with better vision. The predominant frequency of horizontal eye movements ranged from 0.20 to 3.96 Hz.

Findings in a Patient with Bilateral Visual Loss

In patient 5, who was bilaterally blind from birth, horizontal nystagmus was present (Fig. 2). In addition, during vertical saccades, an obligate horizontal component with post-saccadic drift occurred in the horizontal plane in the poorer eye (Fig. 1C). When the better eye was covered, patients 1, 2, and 3 showed no change in the direction of saccades, and patients 2 and 4 showed a decrease in the direction of saccades. The predominant frequency of horizontal eye movements ranged from 0.17 to 0.51 Hz.

Comparison of saccadic pulse gain of the two eyes in patients 1 and 2 showed constancy for vertical movements, while horizontal movements showed disconjugacy. The predominant frequency of horizontal eye movements ranged from 0.20 to 3.96 Hz.

When the better eye was covered, patients 2 and 4 showed a decrease in the direction of saccades. The predominant frequency of horizontal eye movements ranged from 0.17 to 0.51 Hz.

In conclusion, long-term deprivation of vision in one eye results in disconjugate nystagmus, particularly in the vertical plane, and the predominant frequency of horizontal eye movements is lower than in normal subjects. The predominant frequency of horizontal eye movements ranged from 0.17 to 0.51 Hz.

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eye causes instability of gaze in that eye. This instability was present in both vertical and horizontal planes, but was greater in the former. In the horizontal plane, horizontal drifts tended to be more unidirectional with consequent nystagmus. In the vertical plane, gaze was disrupted by low-frequency, low-amplitude drifts that generally caused a larger variation in eye position during the test periods than in the horizontal plane. Although it was not possible to rigidly immobilize the heads of our patients on bite-boards, it seems unlikely that small head movements could have caused this disconjugate instability of gaze.

What could be the pathogenesis of these ocular movements that disrupt steady gaze following monocular visual loss? First, the instability of gaze could not be attributed to an abnormality of the "common neural integrator" or gaze-holding network since: (1) it occurred when the eye was close to primary position; and (2) gaze-evoked nystagmus was not a characteristic finding (in contrast to bilateral blindness). The second possibility is that the gaze instability in monocular blindness was caused by fluctuations in the yoking mechanism. However, although the yoking mechanism was quite abnormal, as evidenced by disconjugate saccades, the pattern of disconjugacy was fairly constant (as evidenced by the low coefficient of variation of saccadic pulses). Experimental monocular patching, in monkeys, also leads to a consistent pulse-step mismatch. A third possibility is that the adventitious eye movements that develop with monocular blindness represent an abnormality of a system that normally moves that eye in response to monocular stimulation, such as an abnormal vergence system. The more prominent vertical drifts could be attributed to the different properties of vertical vergence movements. Another possibility is a putative monocular stabilization system. Miles and colleagues have recently described, in rhesus monkeys, a short-latency visual following response that is most vigorous in the wake of a saccade and so serves to stabilize gaze.

Table 3. Ratio of saccadic pulses in patients 1 and 2

<table>
<thead>
<tr>
<th>Horizontal</th>
<th>Vertical</th>
</tr>
</thead>
<tbody>
<tr>
<td>To right</td>
<td>To left</td>
</tr>
<tr>
<td>Patient 1</td>
<td>0.80 ± 0.07</td>
</tr>
<tr>
<td>Patient 2</td>
<td>1.21 ± 0.15</td>
</tr>
</tbody>
</table>

* Expressed as ratio of right eye pulse/left eye pulse. Both patients had impaired vision in their left eye. Values given are mean ± 1 standard deviation.
Fig. 2. Horizontal and vertical movements of both eyes of patient 5 who was bilaterally blind since birth. Note that, in the horizontal plane, nystagmus changes direction and there is a "wandering null point." RHP: horizontal gaze position of right eye; RHV: horizontal gaze velocity of right eye; LHP: horizontal gaze position of left eye; LHV: horizontal gaze velocity of left eye; RVP: vertical gaze position of right eye; RVV: vertical gaze velocity of right eye; LVP: vertical gaze position of left eye; LVV: vertical gaze velocity of left eye. Upward pen deflections indicate rightward or upward gaze movements.

to prevent post-saccadic drift. When saccade-like movements of the visual scene, were applied monocularly as conditioning stimuli, the response was enhanced but with minimal interocular transfer. The latter finding suggests that this ocular stabilization system depends upon connections early in the visual pathways before inputs from the two eyes converge. Abnormalities of vergence and monocular stabilization are not mutually exclusive and might both contribute to the instability of gaze with monocular blindness.

In patients with bilateral blindness the much greater instability of gaze is due to a prominent nystagmus. The nystagmus probably results from an abnormal neural integrator that variably shows leaky and unstable properties. Such an abnormality may represent deprivation of visual inputs to the cerebellum, which is known to be important for normal neural integration of ocular motor signals.

Key words: visual fixation, blindness, scleral induction coil, saccades

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References