Monocular and Binocular Optokinetic Nystagmus in Humans With Defective Stereopsis

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In order to investigate the previously suggested relation between cortical binocular function and deficits in monocular optokinetic nystagmus (OKN), monocular and binocular OKN was examined in normal, stereodeficient, and stereoblind observers, using a variety of stimulation velocities and stimulus field sizes. Most stereodeficient and stereoblind observers showed monocular OKN deficits in one or both eyes, which took the form of either a directional asymmetry or a reduction of the response in both directions. The deficits were often more pronounced at higher stimulation velocities and with smaller stimulus field sizes. The severity of the OKN deficit was related to the degree of residual foveal stereopsis, while the type of the deficit seemed to be influenced by the presence and depth of amblyopia. Binocular OKN resembled the best monocular response in subjects with a directional or ocular asymmetry. The results fit well with current animal models of the neural control of OKN. Invest Ophthalmol Vis Sci 27:574–583, 1986

In normal human subjects with good visual acuity in both eyes and normal binocular functions, optokinetic stimulation with a patterned stimulus drifting to the left or to the right evokes a symmetrical nystagmus both with binocular and monocular stimulation. By contrast, amblyopic subjects often show a characteristic asymmetry of monocular optokinetic nystagmus (OKN), where stimulation from nasal-to-temporal through the visual field (NT) results in a poorer response than stimulation from temporal-to-nasal (TN). This asymmetry may be present in the amblyopic eye only or also in the nonamblyopic eye. Behavioral, electrophysiological and anatomical experiments on cats have provided a model of the neural control of OKN which suggests that the deficits seen in amblyopes are due to a disruption of binocular cortical input to subcortical structures involved in the mediation of OKN. In the cat, the major visual input to the subcortical OKN system comes from the pretectal nucleus of the optic tract (NOT). Cells in this nucleus are strictly direction-specific, being stimulated by movement in the ipsilateral direction, and inhibited by movement in the opposite direction. The optokinetic response is thought to be determined by the difference in total activity, excitatory and inhibitory, in the two nuclei. Experimental manipulations interfering with normal binocular experience early in life, such as monocular deprivation or artificial strabismus, are well known to lead to a loss of binocular interaction in the visual cortex, reflected in a behavioral deficit of binocular depth perception. The same conditions have also been shown to result in a loss of the cortically mediated binocularity in the NOT, leading to an OKN impairment particularly of the NT component and at high stimulation velocities. It was therefore suggested that the monocular OKN deficits might be related to the loss of cortical binocularity.

Human amblyopes, whose OKN deficits closely parallel those of monocularly deprived or strabismic cats, indeed often show impairments of stereoscopic depth perception, indicating reduced binocular cortical function. Previous investigations of monocular OKN in amblyopes, however, did not systematically examine the relationship between the observed OKN impairments and reductions of stereopsis, concentrating instead on the amblyopic acuity loss. One study does...
mention OKN asymmetries in nonamblyopic observers with a total loss of stereopsis, but the possible interactions or differential effects of stereodeficiency and the amblyopic acuity loss on OKN have as yet not been studied in detail. In the present study, we therefore investigated binocular and monocular OKN in subjects with varying degrees of stereopsis, most, but not all of whom were also amblyopic. Since OKN deficits have been reported to be more pronounced with small stimulation fields, three different field sizes were used.

**Materials and Methods**

**Subjects**

Six subjects with corrected acuity of 6/6 or better in each eye and normal stereopsis (≤50") served as controls. Six subjects had deficient or very rudimentary stereopsis (the fly in the Titmus test). Five of them were mild, and one a deep amblyope; three were esotropic, three exotropic, with no or mild (up to 1.5 D) anisometropia. Six subjects were stereoblind; three of these were deep and one a moderate amblyope, the other two were nonamblyopic strabismic alternators (one esotrope, one exotrope). Of the amblyopes, two were esotropes, the other two anisometropic exotropes. Clinical details of the experimental subjects are given in Table 1. Informed consent to participate in the experiments was obtained from each subject.

**Methods**

Acuity was assessed using standard charts of Snellen optotypes. The Maddox cross was used for determining the angle of eso- or exotropia. Eccentric fixation was assessed using the method of Koppenberg.26 This involves fixation of a small spot against a brightly lit background through an eccentrically rotating pinhole. The eccentric motion causes the retinal blood vessels to become visible, with no vessels in the foveal area, and the perceived position of the fovea can then be related to the fixated spot. Eye dominance was assessed with a modified version of the Mirror–Pola test, in which the subject, wearing polarized glasses with perpendicular axes of polarization, views another person wearing identical glasses, and indicates the relative visibility of the second person’s two eyes. The Randot and the Titmus tests, as well as projected Random dot patterns were used for assessing stereopsis; values given in Table 1 are those obtained with the Randot test, except in cases of rudimentary stereopsis, where only the Titmus fly and/or large-disparity, random-dot patterns could be detected. Subjects who failed to perceive depth in all of these tests were classified as stereoblind.

Eye movements were recorded electro-oculographically with Ag-AgCl electrodes attached to the outer canthi of each eye, and a ground electrode on the forehead between the two eyes. The EOG signals were calibrated regularly (4× with each field size) during testing. Signals were fed into a DC amplifier and recorded on paper as well as on electromagnetic tape. Of the tape recorded signals, 8-sec segments containing representative and/or the most vigorous OKN seen during each stimulation condition after at least 5–10 sec of stimulation were selected for computer analysis. The mean slow phase velocity (velocity of the eye movements in the direction of the stimulus movement) and gains (ratio between eye velocity and stimulus velocity) were calculated, and the number of saccades in the computer-analysed segment counted. Inspection of the recordings showed that slow phase amplitude generally did not exceed 20°, and mostly lay within 10°–15°. Furthermore, the basic eye position did not deviate much from a central position even during full field stimulation, possibly due to the strict instructions given (see below). Control measurements indicated that the EOG measurements were approximately linear within this range. It therefore seems unlikely that velocity gain estimates were influenced significantly by nonlinearities of the EOG measurements at eccentric eye positions.

Three field sizes were used for stimulation of OKN: For full field stimulation, the subjects were seated in the center of a rotating drum (150 cm diameter), the walls of which were covered with a regular black and white checkerboard pattern (checksize 1 cm²) or a random-dot pattern (checksize 1 cm²). For smaller field sizes, a large screen oscilloscope (20 × 30 cm) was used, on which high contrast black and white square wave gratings of 1° stripewidth were drifting either to the left or to the right. Either the whole screen (30° at a viewing distance of 57 cm) or a masked off circular field of 15 cm diameter (15° at 57 cm) were used for stimulation. Stimulation velocities were 6°/sec, 15°/sec, and 30°/sec. To elicit OKN, subjects were repeatedly instructed to stare straight ahead at the moving stimulus, and to keep the pattern visible. For monocular stimulation, subjects covered one eye with their hand. At each stimulation velocity, OKN was first stimulated binocularly in both directions and then monocularly, with TN stimulation before NT stimulation for each eye, and in amblyopes the nonamblyopic eye before the amblyopic eye. Repeat tests of some conditions in several subjects gave very similar results, suggesting that fatigue and/or experience had little effect on the results. The velocity gains of the different monocular stimulation conditions (right/left eye, TN/NT, different velocities and field sizes) were statistically analysed for individual subjects as well as within and across groups by means of analyses of variance.
Table 1. Clinical details of the stereodeficient and stereoblind observers

<table>
<thead>
<tr>
<th>Subject</th>
<th>Eye*</th>
<th>Refraction</th>
<th>Acuity c.c.</th>
<th>Fixation</th>
<th>Squint</th>
<th>Stereo</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) H.-J. B.</td>
<td>R</td>
<td>-0.75/-0.5 X 45°</td>
<td>6/6.6</td>
<td>central</td>
<td>-2°</td>
<td>100°-500°</td>
<td>not treated</td>
</tr>
<tr>
<td></td>
<td>L*</td>
<td>-</td>
<td>6/15</td>
<td>central, unsteady</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2) B.L.</td>
<td>R</td>
<td>+0.5</td>
<td>6/6.6</td>
<td>central</td>
<td>-1°</td>
<td>800°-3000°</td>
<td>not treated; family history</td>
</tr>
<tr>
<td></td>
<td>L*</td>
<td>+0.75/+0.75 X 90°</td>
<td>6/20</td>
<td>1.5° nasal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3) J. v.H.</td>
<td>R</td>
<td>-</td>
<td>6/6</td>
<td>central</td>
<td>-6°</td>
<td>+ (fly)</td>
<td>not treated</td>
</tr>
<tr>
<td></td>
<td>L*</td>
<td>+3.5</td>
<td>6/20</td>
<td>6° temporal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4) G. B.</td>
<td>R</td>
<td>-2.25</td>
<td>6/5</td>
<td>central</td>
<td>+5°</td>
<td>+ (fly)</td>
<td>occlusion therapy around 5-6 yr (not completed); family history</td>
</tr>
<tr>
<td></td>
<td>L*</td>
<td>-1.25</td>
<td>6/15-6/20</td>
<td>1° nasal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5) M. E.</td>
<td>R*</td>
<td>-2.25</td>
<td>6/10</td>
<td>central</td>
<td>+4°</td>
<td>+ (fly)</td>
<td>not treated</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>-2.75/-1.0 X 100°</td>
<td>6/6.6</td>
<td>central</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6) N. S.</td>
<td>R*</td>
<td>0/-0.5 X 90°</td>
<td>6/50</td>
<td>central, unsteady</td>
<td>+2-+3°</td>
<td>+ (fly)</td>
<td>occlusion therapy at 5 yr</td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>-</td>
<td>6/6</td>
<td>central</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7) R. P.</td>
<td>R*</td>
<td>+3.75/-0.5 X 170°</td>
<td>6/50</td>
<td>5° temporal</td>
<td>-0.5°—1°</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>-0.25</td>
<td>6/5-6/6.6</td>
<td>central</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8) N. K.</td>
<td>R*</td>
<td>-</td>
<td>6/30</td>
<td>central, unsteady</td>
<td>+5°</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>-</td>
<td>6/5</td>
<td>central</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9) M. F.</td>
<td>R</td>
<td>-</td>
<td>6/5</td>
<td>central</td>
<td>+3°</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>L*</td>
<td>+1.0°</td>
<td>4/50</td>
<td>unsteady</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10) L. R.</td>
<td>R*</td>
<td>+4.0</td>
<td>6/50</td>
<td>?</td>
<td>-7.5°</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>-2.0/-0.3 X 105°</td>
<td>6/6.6</td>
<td>central</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11) J. H.</td>
<td>R</td>
<td>-0.5</td>
<td>(6/5)-6/6.6</td>
<td>central</td>
<td>-9°</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>-1.0</td>
<td>6/6.6-6/10</td>
<td>central</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12) V. N.</td>
<td>R</td>
<td>-1.0</td>
<td>6/5</td>
<td>central</td>
<td>+5°</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>L</td>
<td>+0.5/+1.0 X 75°</td>
<td>6/5</td>
<td>1° nasal, 0.4° sup.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Asterisks in the “Eye” column indicate the amblyopic eye. In the “Squint” column, “+” indicates exotropia, “-” esotropia.

**Results**

The control subjects showed symmetrical monocular OKN, i.e., no significant differences between TN and NT stimulation, with all stimulation velocities and field sizes, and both with regard to gain and saccade frequency. Figure 1 shows their mean values of gain and saccade frequency; results from the two eyes have been combined, since there was no difference between the two monocular conditions. As can be seen, OKN gain lay around 90% at all stimulation velocities with full field stimulation, but tended to decrease at the higher velocities with decreasing field size ($P < 0.001$). This effect of field size on OKN gain has been described previously. The effect was very noticeable in some subjects, while others showed hardly any change in OKN with field size. Saccade frequency increased with increasing stimulation velocity, but slightly less so with the smallest field size. Binocular OKN was indistinguishable from monocular OKN (Fig. 1).

The monocular OKN responses of the controls are compared to the mean results of the stereodeficient and stereoblind subjects in Figure 2. As a group, the stereodeficient subjects showed similar TN OKN to the control subjects with all field sizes, but the NT component was slightly reduced ($P = 0.03$, within-group and in comparison to controls). The effects of field size and stimulus velocity were no different from those seen in controls.

A closer look at the results showed individual differences which seemed to be related to the etiology of the amblyopia/stereodeficiency. Of the six stereodefi-
cient subjects, the three anisometropic, exotropic amblyopes (subjects 1, 2, 3) showed no significant monocular OKN asymmetry under any stimulation conditions. In two of them (subjects 1, 2), gains and saccade frequency were similar to those seen in the control subjects (Fig. 3). In the other subject (subject 3), who had only very rudimentary stereopsis, both the TN and the NT components were somewhat reduced with small field stimulation, especially at the higher stimulation velocities. Binocular OKN again resembled monocular responses. The three esotropic stereodeficient amblyopes all showed monocular OKN asymmetries, with TN OKN superior to NT OKN \( (P < 0.03) \), either with only the amblyopic eye (subject 4), or with each eye (subjects 5, 6) (Fig. 3). There was a slight tendency for the asymmetry of these subjects to increase with small-field stimulation (see Fig. 3). In subject 5, OKN was reduced more than normally in both stimulus directions with small field stimulation. In the other two subjects, the overall level of OKN, with the exception of the asymmetrical NT components, was comparable to that seen in the controls, both in terms of gain and saccade frequency. Binocular OKN corresponded most closely to the TN components of each eye, particularly with small field stimulation, where the asymmetries tended to be more pronounced. Thus binocular OKN in response to CCW stimulation was very similar to that seen with CCW (=TN) stimulation of the right eye, while CW binocular OKN resembled CW (=TN) OKN of the left eye.

In the stereoblind subjects as a group (Fig. 2), monocular OKN was clearly asymmetrical with all field sizes \( (P < 0.001) \). The mean asymmetry was not sta-

![Fig. 1. Mean gains and saccade frequencies of monocular and binocular OKN in the six normal observers for the three field sizes used. Monocular results for the two eyes of each observer have been combined. TN OKN is indicated by uninterrupted lines, NT OKN by broken lines. Triangles indicate binocular OKN, combined for the two stimulus directions. Error bars give two standard deviations for all the data presented in each graph.](http://iovs.arvojournals.org/pdfaccess.ashx?url=/data/journals/iovs/933128/)

![Fig. 2. Mean monocular OKN gains for the three different field sizes in control (○—○), stereodeficient (Δ—Δ), and stereoblind (○—○) subjects. Open symbols indicate TN OKN, filled symbols NT OKN.](http://iovs.arvojournals.org/pdfaccess.ashx?url=/data/journals/iovs/933128/)
tistically greater than in the stereodeficient subjects, but the degree of significance of the asymmetry within the group, and of the difference from control subjects, was greater than in the stereodeficient group ($P < 0.001$ vs $P = 0.03$ for both comparisons). In addition, the response of the nondominant (or amblyopic) eye was reduced in comparison to the dominant eye ($P < 0.001$). Again, smaller field sizes resulted in a decrease of the optokinetic response, especially at higher velocities; this field effect was significantly greater than in controls or the stereodeficient subjects ($P < 0.001$).

Inspection of the individual results revealed that the means shown in Figure 2 in fact resulted from two separate trends for either a directional asymmetry, most often in both eyes, or a reduced, but not necessarily asymmetrical reaction in the nondominant eye. Individual subjects tended to show one or the other of these response patterns, a tendency which may explain why the mean asymmetry of the stereoblind group was not significantly greater than in the stereodeficient subjects.

Of the four stereoblind amblyopes, one only, the esotropic anisometrope subject 7, showed consistent monocular OKN asymmetries ($P < 0.001$), which were more pronounced in the amblyopic eye. With small field stimulation ($15^\circ$ and $30^\circ$ fields), both the TN and the NT components were slightly reduced at high stimulation velocities in comparison to controls (Fig. 4). A mild esotropic amblyope (subject 8) showed only a slight monocular asymmetry in the amblyopic eye with larger stimulus fields and a symmetrical response with the smallest field size. However, OKN gains in both eyes and both directions were abnormally low and increasingly so with smaller stimulus fields. The response of the amblyopic eye was even poorer than that of the nonamblyopic eye ($P = 0.02$).

The other two subjects (subjects 9, 10), both deep amblyopes, showed no monocular asymmetry in either eye under any condition; the NT component of the amblyopic eye sometimes even had a slightly higher gain and saccade frequency than the TN component (Figs. 4, 6). In both these subjects, however, responses of the amblyopic eye were clearly lower in both directions than in the nonamblyopic eye, which showed normal OKN ($P < 0.001$); this was particularly evident at higher stimulation velocities and with the smallest field size.

The two nonamblyopic stereoblind subjects (subjects 11, 12), on the other hand, both showed clearly asymmetrical monocular OKN, both in gain and saccade frequency ($P < 0.006$) (Figs. 5, 6). In the exotropic alternator (subject 11), responses in the left eye were slightly poorer than those in the right eye ($P = 0.013$), which may be related to a previous, successfully treated amblyopia in the left eye. Stimulation with the smaller field sizes ($30^\circ$ and $15^\circ$) in this subject resulted in a clear reduction of both TN and NT components, while the asymmetry was not greater than that seen in the full field.

The esotropic alternator (subject 12) showed large consistent monocular asymmetries in both eyes, which tended to be greater at high stimulation velocities, and clearly increased with decreasing field size. The TN component of both eyes was normal under all conditions (Figs. 5, 6).

Binocular OKN in the stereoblind subjects generally corresponded to the better monocular response for each
direction. Thus in subjects with a clear directional asymmetry (subjects 7, 12), binocular OKN resembled the TN component of the two eyes, whereas in subjects with reduced responses of the amblyopic eye (subjects 9, 10), binocular OKN corresponded to the TN and NT components of the nonamblyopic eye (Fig. 7). The stereoblind subject with a mixed response pattern (asymmetrical OKN with reduced reactions of the non-dominant eye) (subject 11) found binocular stimulation very confusing and difficult, and the resulting OKN showed no clear similarity to any of the monocular conditions, although there was a tendency to follow the dominant eye (Fig. 7).

**Discussion**

The present findings lend support to the hypothesis of a relation between cortical binocularity, as assessed by stereopsis, and monocular OKN deficits. The deficits most often took the form of an asymmetrical response with poorer NT-OKN, but impairments of the TN component were also very common, especially at higher stimulation velocities. The TN deficits were usually less severe than the NT impairments, so that a directional asymmetry remained evident. Occasionally, however, the reduction of both components was equally great, in which case the response was directionally symmetrical, yet impaired in overall level in comparison to normal control subjects. Both the incidence and severity of these OKN deficits increased with decreasing stereopsis. While normal, symmetrical OKN was seen in the presence of reduced but still quantifiable stereoacuity, very rudimentary stereopsis was more often associated with OKN deficits, and clear asymmetries and/or bidirectional impairments were seen in the stereoblind subjects. Furthermore, a loss of stereopsis alone, in the absence of amblyopia, was sufficient for the occurrence of clear OKN deficits, confirming a previous report. ²

Despite this close association between stereopsis and monocular OKN deficits, the neural mechanisms involved are not necessarily entirely identical. The loss of binocular vision in amblyopes is generally thought to be due to cortical suppression of visual input from
the amblyopic eye, and sensory deficits of acuity, contrast sensitivity, etc are also restricted to this eye. OKN deficits, on the other hand, are frequently seen also in the nonamblyopic eye and seem to involve an additional suppression, at the subcortical level, of the ipsilateral cortico-pretectal projection from the nonamblyopic eye.9-11 (see below). Nevertheless, since the disruption of both cortical and subcortical binocularity...
is presumably caused by the same environmental factor, ie, incongruous visual input from the two eyes early in life, and since the loss of cortical binocularity accounts for at least part of the subcortical deficit, it is not surprising that the incidence and severity of the ensuing functional deficits show close parallels.

Apart from the effect of reduced binocular functions, the findings also indicate that the cause and depth of amblyopia may play a role in the kind of OKN deficits seen. Two of the three exotropic anisometropic amblyopes with reduced stereopsis showed normal OKN, while the stereodeficient esotropes had clear deficits. The two stereoblind subjects with a very deep amblyopia showed OKN deficits affecting both TN and NT components only in the amblyopic eye, while in the milder amblyopes, as well as the previously amblyopic alternator, asymmetries as well as reductions of both components were very often evident in both eyes. This suggests that the degree of suppression of the amblyopic eye may have an influence on whether OKN deficits are restricted to one, deeply suppressed eye, or seen in both eyes. Finally, in the esotropic alternator with no history of amblyopia (subject 12), the OKN deficit was restricted to the NT component of both eyes, with normal TN OKN. In view of the small number of subjects and the variable combinations of acuity loss and stereoscopic deficits, more data are of course needed to substantiate these suggestions about the interaction of amblyopia and stereodeficiency on monocular OKN, but the findings do suggest that an orderly relationship may exist.

The present results fit well with the model of neural mechanisms of OKN developed from the animal experiments. To our knowledge, OKN has not been investigated in cats with artificially induced anisometropia or strabismic amblyopia. However, the results of the esotropic stereodeficient subjects, and the stereoblind amblyopes, can be modelled satisfactorily with the findings described in monocularly deprived cats. These animals usually show impairments
of NT–OKN in both eyes, due to the loss of ipsilateral cortical influence on the NOT. The TN component of the deprived eye may also be reduced, especially at higher stimulation velocities, which require cortical mediation; a slight effect on TN–OKN of the nondeprived eye might be explained by the lack of inhibitory responses via the ipsilateral fibres. This is analogous to the results in the mild strabismic amblyopes. Less frequently, monocular deprivation in cats results in a suppression of cortical influence only from the deprived eye, with both contralateral and ipsilateral cortical projections from the nondeprived eye intact. 10 In this case, an OKN deficit is seen only in the deprived eye, similar to our findings in the two deep amblyopes (subjects 9 and 10). The deep suppression of the amblyopic eye, indicated by the profound acuity loss, may favor this second type of OKN deficit.

Cats rendered exotropic, but not amblyopic, by early surgery, have been reported to show a reduction of both TN and NT components in both eyes. 5 Such a deficit was indeed seen in our exotropic alternator (subject 11), who, however, also had a history of amblyopia; a similar deficit was also seen in the mildly amblyopic esotrope (subject 8). By contrast, the non-amblyopic esotropic alternator (subject 12) showed a reduction only of the NT component, with normal TN OKN. No experimental findings on nonamblyopic esotropic cats are known to us.

In normal as well as monocularly deprived and strabismic cats, binocular OKN has been reported to be better or equal to monocular TN responses (even normal cats show slightly asymmetrical monocular OKN with a preference for TN stimulation 9,10). Neither our control subjects nor the stereo-deficient or stereoblind subjects ever showed any evidence of binocular “summation.” In those subjects with monocular OKN deficits, binocular OKN was usually very similar to the better monocular responses in each direction. Thus for subjects with a clear naso-temporal asymmetry, binocular OKN resembled the TN components of the two eyes, while in the two subjects with consistently poorer responses in the amblyopic eye (subjects 9, 10), binocular OKN was more like the TN and NT components of the nonamblyopic eye. Similar findings were mentioned in a previous publication. 2

A restriction of the field size used for eliciting OKN has been reported to result in increased asymmetries of monocular OKN in amblyopes. 2, 3 This was seen very clearly in some of our subjects (eg, subject 12), but the effect was not entirely consistent and varied considerably across subjects. It has been suggested that the greater deficits with small field stimulation are related to the fact that binocular suppression is usually greater in the central visual field. 2, 11 Indeed, it has recently been shown that subjects with no foveal stereopsis, who would normally be classified as stereoblind, may have binocular interactions, including the perception of motion in depth and binocular summation of visual acuity, in the peripheral visual field. 31,32 The relation between the extent and location of such peripheral binocular functions and the variability of the field size effect in monocular OKN deficits, which we observed here, is examined in the accompanying paper.

In conclusion, the present study provided further evidence for the relation between cortical binocular function and monocular OKN deficits. The severity of the stereopsis reduction was predictive of the occurrence and severity of OKN deficits, while the presence, cause and depth of amblyopia seemed related to the type of deficit seen. The results agree well with the model of neural control of OKN derived from animal experiments.

Key words: optokinetic nystagmus, stereopsis, amblyopia, cortical binocularity

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