A Psychophysical Measurement and Analysis of Motion Perception in Normal and Binocularly Deprived Monkeys

Lai Ngor Fu[1,2] *and Ronald G. Boothe*[1,2,3]

Purpose. To measure psychophysically the thresholds for motion detection in the nasal and temporal directions under monocular viewing conditions in monkeys reared under conditions of daily alternating monocular occlusion (AMO). The hypothesis was that motion perception would be asymmetric with more sensitivity for motion in the nasal direction.

Methods. Three monkeys subjected to AMO (AMO monkeys) and three normal monkeys were studied. All were trained with operant conditioning techniques to discriminate coherent from random motion in a random dot display. The percentage of dots in the display that moved either left or right was varied. Thresholds for motion detection of nasally directed and temporally directed stimuli were measured to determine whether the motion perception of AMO monkeys was asymmetric, as predicted.

Results. A two-factor analysis of variance revealed a statistically significant difference between treatment groups (normal versus AMO) and directions (nasal versus temporal) and a significant interaction. The interaction was due to a significant difference between nasal and temporal directions for the AMO group, but no significant difference for the normal group. Planned comparisons were performed based on each animal's best eye (eye most sensitive to nasal motion) and worst eye (eye least sensitive to temporal motion). No significant differences were found between the two groups in the best eyes' responses to the nasal direction, but the worst eyes' responses in the temporal direction were significantly poorer in the AMO group. A neural model that can account for these findings is based on a Hebbian teacher located in the nucleus of the optic tract that strengthens connections of a subpopulation of directionally selective cortical neurons.

Conclusions. AMO rearing results in asymmetric motion perception. Thresholds for detecting nasally directed motion are normal, whereas thresholds for detecting temporally directed motion are deficient. These results demonstrate that motion-processing mechanisms in primates exhibit experience-dependent developmental neural plasticity. The locus of the neural plasticity could be a subpopulation of directionally selective neurons in the striate cortex (V1). (*Invest Ophthalmol Vis Sci.* 2001;42:2547–2553)

From the [1]Department of Psychology, [2]Division of Visual Science, Yerkes Regional Primate Research Center, and the [3]Department of Ophthalmology, Emory University, Atlanta, Georgia.

Supported by National Institutes of Health Grant EY05975 and by Grant RR00165 from the National Center for Research Resources to the Yerkes Regional Primate Research Center.

Submitted for publication November 22, 2000; revised April 12, 2001; accepted May 17, 2001.

Commercial relationships policy: N.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be marked "*advertisement*" in accordance with 18 U.S.C. §1734 solely to indicate this fact.

Corresponding author: Ronald G. Boothe, Division of Visual Science, Yerkes Regional Primate Research Center, Emory University, 954 Gatewood Road NE, Atlanta, GA 30322. boothe@rmy.emory.edu

Visual experience during the early postnatal period plays an important role in development of those parts of the brain that process visual information.[1,2] At birth, human and monkey infants exhibit smooth-pursuit eye movements with stronger responses in the nasal than the temporal direction and asymmetrical monocular optokinetic nystagmus (MOKN) responses characterized by a reduction in the frequency of beats and in amplitude of slow-phase tracking in the temporal direction.[3–9] These neonatal asymmetries have been interpreted as reflecting an immaturity in the form of a domination of subcortical neural systems, particularly the nucleus of the optic tract, during early visual development.[10,11]

Evidence of cortical asymmetry in motion processing comes from measurements of visually evoked potentials to motion (MVEP) elicited by oscillating horizontal stimuli.[12–15] MVEP responses produced by human and monkey infants are dominated by the first harmonic component and show responses that are 180° out of phase in the two eyes during monocular viewing. These results have been interpreted as demonstrating that there is a directional bias in neonates that works in opposite directions in the left and right eyes. The neonatal asymmetrical responses to smooth pursuit, MOKN, and MVEPs become symmetrical by approximately 6 months of age in humans and after a few weeks in monkeys.[3,5,8,12–14] However, it has been reported that animals reared under conditions of visual deprivation and humans with disrupted binocular development from conditions, such as misalignment of the visual axes (strabismus), continue to exhibit motion asymmetries on these measures into adulthood.[10–13,15–23]

Behavioral studies have also revealed that humans with early-onset infantile strabismus exhibit asymmetries in perception of visual motion. These perceptual findings have been interpreted as reflecting motion-processing asymmetries in cortical areas of the brain. However, the results of the perceptual studies have not been entirely consistent. Some studies have reported that patients perceive nasally directed stimuli as moving faster than temporally directed stimuli, when actual velocity is the same in the two directions.[24,25] Results of other studies of subjects with early-onset esotropia have been interpreted as demonstrating a perceived monocular speed bias with a preference for temporal motion.[26] One study in which motion asymmetries were assessed by measuring thresholds for random dot motion in patients with infantile esotropia revealed abnormal motion perception, but only for stimuli presented in the nasal hemifield.[27]

These findings of prolonged motion asymmetries of various forms in visually deprived animals and in humans with infantile esotropia have led to the hypothesis that binocular visual experience during a sensitive period of postnatal development may be crucial to the normal development of motion processing. Studies in humans suggest that the sensitive period associated with asymmetric motion processing may end very early. For example, children who experienced early visual deprivation within the first 6 months of life (early-onset esotropia) displayed MOKN asymmetry, even when the strabismus was corrected, and some amount of binocular fusion finally devel-

oped in these patients.[26,28] It has also been noted that the neonatal sensitive period for development of motion processing appears to differ from the more fully characterized later sensitive period during which binocular deprivation produces amblyopia and disruption of binocular function, but no motion asymmetries.[2]

The hypothesis that perceptual motion asymmetries are caused by binocular deprivation during a neonatal sensitive period has never been put to a direct test. In studies of human patients, it has not been possible to rule out an alternative hypothesis that motion asymmetries are part of an underlying initial deficit rather than a secondary result of binocular deprivation. In animal studies to date, investigators have examined motion perception only indirectly, through measures of MOKN and MVEP. The purpose of the present study was to use a monkey model to put to a direct test the hypothesis that disruption of binocular stimulation during early infancy is a sufficient condition to produce asymmetries in motion perception. We measured thresholds for motion detection in the nasal and temporal directions under monocular viewing conditions in monkeys reared from birth under conditions of daily alternating monocular occlusion (AMO monkeys). This rearing condition eliminated binocular experience but provided normal experience to each eye to prevent amblyopia. Our prediction was that after AMO rearing, the monkeys would display asymmetric motion perception with better sensitivity in the nasal than the temporal direction.

Materials and Methods

Six rhesus macaque monkeys (*Macaca Mulatta*) raised from birth at the Yerkes Regional Primate Research Center of Emory University were used in the present study. All were between 1 and 3 years old at the time of testing.

Three monkeys (RUK5, RWO5, and RRO5) were reared under conditions of daily AMO. An occluder lens was placed on one eye on the day of birth. The next day the occluder lens was moved to the opposite eye. This daily alternation between the two eyes was continued until the monkeys were 4 to 6 months of age. The other three monkeys (RJG5, RLY5, and REY5) were raised under conditions of normal visual exposure.

All procedures were performed in strict compliance with National Institutes of Health guidelines and the ARVO Statement for the Use of Animals in Ophthalmic and Vision Research, and the protocols were reviewed and approved by the Institutional Animal Care and Use Committee at Emory University.

Visual Stimuli

Random dot displays were generated by computer (Power Macintosh 8100/80AV; Apple Computer, Cupertino, CA) and displayed on a monitor in black-and-white mode (14-in. Macintosh monitor; Apple) A computer graphics program was written in C language for generating the random dot display. The visual stimuli were composed of dynamic random dots that provided a pure motion signal. Stimuli consisted of successive frames of random dot patterns. Each frame was composed of 200 white dots with a density of 16.7 dots per degree squared. It has been demonstrated in psychophysical experiments that motion sensitivity is little affected by density, as long as it remains at or above this value.[29,30]

Each frame was presented for 50 msec. In successive frames, each dot was either displayed at successive positions along a specified direction (motion dots) or replaced by a partner dot at a random location (noise dots). The relative percentage of motion dots that were displayed could vary from 0% to 100% and were referred to as the correlation of the motion display. Each individual motion dot disappeared from the screen after three frames (150 msec) and was replaced by a new dot at a new (randomly determined) location of the screen. The velocity of the motion dots was 1.81 deg/sec. Previous studies have demonstrated that this velocity is near the optimal value at which human or monkey subjects can reliably report the direction of motion.[31]

Psychophysical Procedures

The goal of the psychophysical measurements was to determine the threshold amount of correlation for which the monkey could reliably discriminate coherent motion in the nasal and temporal directions from random motion. Stimuli were displayed on the computer screen 100 cm in front of monkey. The monkey being tested sat either in a primate chair or in a specially designed test cage that had a face mask on one wall through which the monkey could view the display. Two bars were positioned such that the monkey could easily manipulate them. Random dot displays were presented on two windows located side by side on the video monitor. A coherent motion moving either right or left was displayed in one window, while the other displayed random motion. Coherent motion was randomly assigned to either the left or right window on each trial. Monocular viewing was accomplished by inserting an occluder in front of the nontest eye. The monkey reported its judgment of the coherent motion by pulling one of the two bars. For example, the monkey was taught to pull the right bar for either left or right coherent motion whenever it was displayed in the right window of the computer screen.

Monkeys were trained with operant conditioning techniques[32] to perform on a two-alternative forced-choice visual discrimination task during daily training sessions that lasted approximately an hour. They were rewarded with either a small amount of fruit juice or a fruit-flavored food pellet after a correct trial. Incorrect trials resulted in a time-out period of up to 10 seconds signaled by an audio tone. The monkey was allowed to look at the stimuli as long as it wanted before making a decision. The correlation of the motion signal was varied from trial to trial, to establish the monkey's motion threshold.

During initial training, monkeys were taught to distinguish motion dots (100% correlation) in one window from stationary noise dots in the other window under binocular viewing conditions. Once this task had been learned, we gradually introduced motion to the random noise dots by increasing their velocities from 0.0° to 1.81° per second. When this task had been learned to a criterion of at least 80% correctness, the correlation level of the motion dots was gradually reduced. At this point in training, an occluder was inserted in front of the nontest eye. A baseline of performance near threshold was established by the aid of a two-down, one-up staircase method. The correlation of motion dots was reduced by 1% after two consecutive correct trials and increased by 1% after every incorrect response. When performance on this staircase procedure had stabilized, five stimuli with different correlation values were chosen that spanned a range above and below the baseline staircase performance. During actual data collection, these five stimuli were presented repeatedly in random order using the psychophysical method of constant stimuli.

Data Analysis

Motion detection thresholds, defined as the correlation that results in 75% correct performance, were estimated statistically using probit analysis.[33,34] For one or both eyes of two monkeys with AMO (RWO5 and RUK5), performance did not exceed 75% correct for any correlation tested for the temporal direction, and for these data sets we defined threshold as 100% correlation.

For the initial analysis, data for the left and right eyes were combined. A two-factor ANOVA was administered to determine whether the mean thresholds for the detection of coherent motion were significantly different between the two groups (AMO monkeys and normal control animals) and the two directions of motion (nasal and temporal). Subsequently, single-factor ANOVAs were used to test direction of motion separately for the AMO and normal control groups. Finally, single-factor ANOVAs were used to perform planned comparisons of the data from the two separate eyes of each animal. There was no a

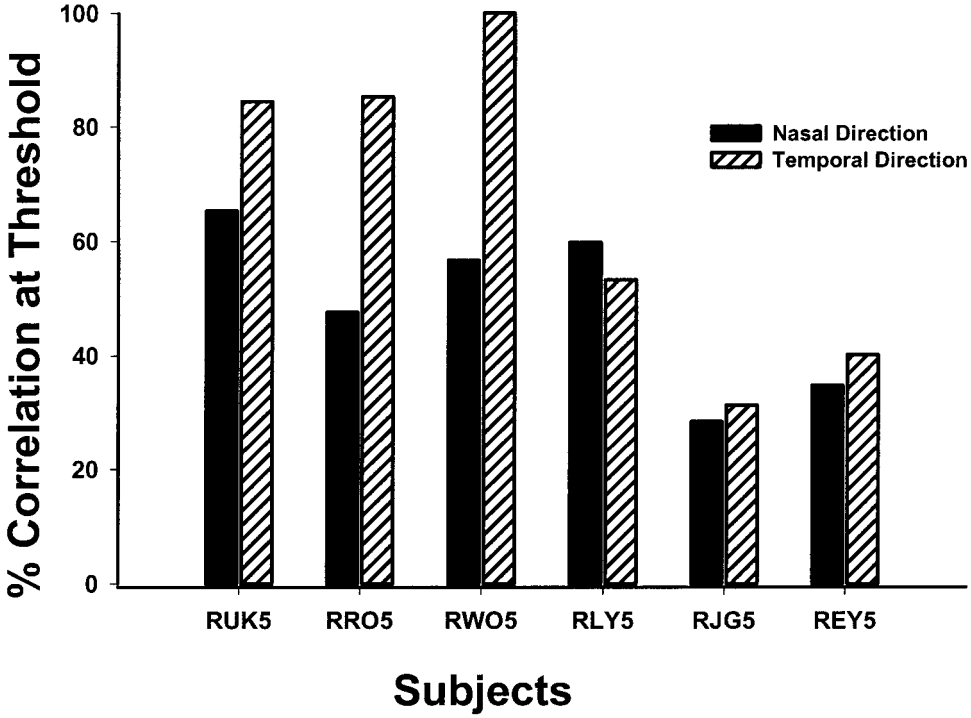

FIGURE 1. Motion detection thresholds in six subjects. Results for the left and right eyes have been combined to show the thresholds for each subject for motion in the nasal and the temporal directions. Examination of these results reveal that the normal control monkeys (JG5, LY5, EY5) showed essentially symmetrical motion perception for nasal and temporal directions, whereas the AMO monkeys (UK5, WO5, RO5) always showed better detection of motion in the nasal direction.

priori basis for predicting which eye should be most affected in each animal. Thus, comparisons were made using "best eye" and "worst eye." The best eye for each animal was defined as the eye having the lowest threshold for nasal motion and the worst eye as the one with the highest threshold for temporal motion.

RESULTS

Motion detection thresholds of each of the six animals for both directions of motion are shown in Figure 1. Qualitative examination of this figure reveals that normal monkeys had similar thresholds for both directions of motion. However, the visually deprived monkeys exhibited differences in thresholds between temporal and nasal directions, with the temporal thresholds being consistently worse.

A two-factor ANOVA revealed a significant effect of both treatment groups ($F_{(1,4)} = 13.56$, $P < 0.05$), direction of motion ($F_{(1,4)} = 17.35$, $P < 0.05$), as well as a significant interaction ($F_{(1,4)} = 16.18$, $P < 0.05$). To help interpret the interaction, a subsequent single-factor, within-subject ANOVA was performed comparing direction of motion separately in the AMO and normal animals. Figure 2 shows the motion thresholds for detecting nasally and temporally directed motion within each group. There was a significant difference between the nasal and temporal directions in the AMO group ($F_{(1,2)} = 20.89$, $P < 0.05$). The normal group did not show any differences between the two directions ($F_{(1,2)} = 0.026$, $P > 0.05$). Thus, the interaction reflects the fact that only the AMO group showed differences in thresholds in the nasal and temporal directions.

Planned comparisons were conducted based on the best eye and worst eye of each animal (defined as described in the Methods section). There were no significant differences in thresholds of the best eyes in the nasal direction between the AMO and normal groups ($F_{(1,4)} = 1.40$, $P > 0.05$; Fig. 3A), but there was a significant difference between the two groups in thresholds of the worst eyes in the temporal direction ($F_{(1,4)} = 43.58$, $P < 0.01$; Fig. 3B).

Taken together, our findings demonstrate that the two groups differed only in their abilities to detect temporally directed motion. The AMO group performed similar to the normal control animals in detecting nasally directed motion but always displayed higher thresholds for detecting temporally directed motion.

DISCUSSION

In our study, monkeys reared under AMO conditions displayed asymmetric motion perception. AMO animals exhibited essentially normal motion detection for nasally directed motion but deficits in detecting temporally directed motion.

One issue that warrants discussion is whether perceptual asymmetries in the AMO animals might be secondary to asymmetrical eye movements. This concern has been raised regarding reports of motion asymmetries measured in human patients using MVEPs. For example, Kommerell[35] argued that MVEP asymmetries measured in patients with infantile strabismus might be the result of latent nystagmus (LN). However, a previous study in our laboratory documented a motion asymmetric MVEP in AMO-reared monkeys in which all eye movements were eliminated during the recording.[36] In addition, this same study demonstrated that an asymmetric MVEP was present in an AMO monkey in which eye coils were used to demonstrate that LN was not present. Thus, the presence of LN is not a prerequisite for development of motion asymmetry.

Another issue that warrants discussion is that the motion thresholds for our monkeys, normal as well as AMO, were relatively high compared with that reported in the literature. Normal monkeys have been reported in several previous studies to have motion thresholds that are in the range of 5% correlation, a value that is indistinguishable from motion thresholds measured in normal humans.[31] The thresholds in all our monkeys were substantially poorer than this expected value (Fig. 1). The poor thresholds obtained in our studies were probably caused by a seemingly slight difference between our protocols and those used in previous studies. We

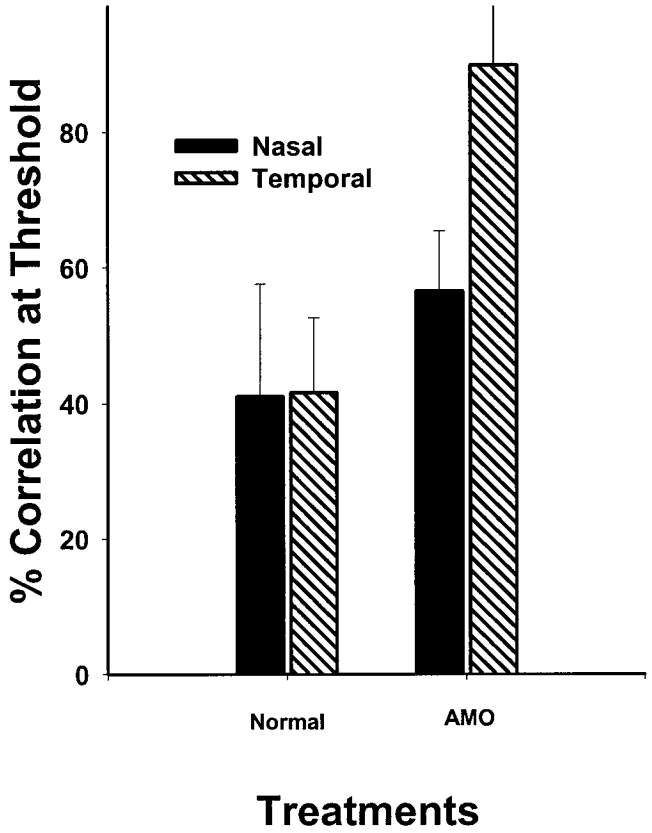

FIGURE 2. Average motion detection thresholds organized by treatment group and direction of motion. There was a significant difference between nasal and temporal directions for the AMO group, but not for the normal control group.

used a display in which two stimuli were presented side by side on a video display monitor. Our monkeys had to choose which side of the display (left or right) had coherent motion, regardless of whether the coherent motion itself was in the left or right direction. In previous studies performed with normal monkeys or humans, only a single stimulus was used that covered the entire screen, and the task was simply to indicate—for example, with a left or right response—whether the coherent motion was in the left or right direction. We made a decision to use the two-window display, because we wanted to use methods that could measure a bias in perception but would not be affected by a potential confounder in the form of a bias for responding left or right. However, our change in methods had the unanticipated effect of substantially increasing the monkeys' thresholds compared with those of previous studies.

There are two possible reasons that thresholds based on a two-screen display might be expected to be poorer than those for a one-screen display. First, an analysis of our stimuli in terms of signal detection theory factors predicts an increase in threshold for the two-screen condition by approximately a factor of 1.5, relative to the one-screen condition.[37] We evaluated this prediction by measuring thresholds in one of the authors (LNF) and obtained thresholds of 4.89% for the one-screen and 9.1% for the two-screen conditions. This result demonstrates that signal detection factors can explain most of the increase for human observers. We also tested one of our AMO monkeys (RUK5) in both conditions and obtained thresholds of 12% and 75.08% for the one- and two-screen conditions, respectively. This result demonstrates that for monkeys there is an additional component that increases thresholds in the two-screen condition, over and above the increase expected based on signal-detection-theory factors.

We speculate that the second factor in the difference between thresholds with the two displays is that the two-screen condition presents a higher cognitive demand. To perform

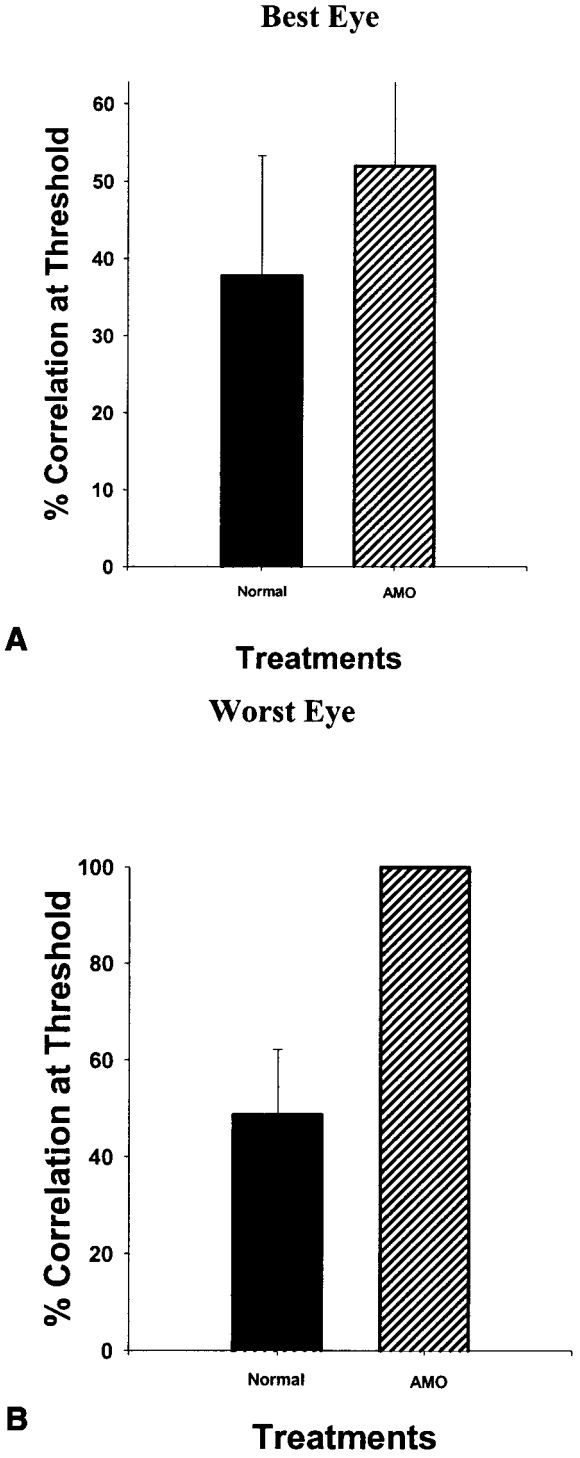

FIGURE 3. (A) A plot of the results from the single eye of each animal that exhibited best performance in the nasal direction. This comparison revealed no significant difference between the AMO and the normal monkeys. (B) A plot of the results for the single eye of each animal that exhibited worst performance in the temporal direction. This comparison reveled a significant difference between the AMO and the normal groups.

correctly on the two-screen task, left versus right position on the screen must be dissociated from left versus right motion, a cognitive task that can be accomplished with only moderate effort by a human but poses a high degree of difficulty for a monkey. Thus, our proposed explanation for the somewhat higher than expected thresholds for our monkeys is based on a combination of signal detection and cognitive factors associated with the two-screen visual display. It is possible that given sufficient training, our monkeys' performances on the task would have eventually become asymptotic at lower thresholds than those measured in our study. We cannot address this question, because the monkeys are not available for further testing. However, the absolute thresholds for our stimuli have no direct bearing on our primary findings, because they are based on relative comparisons between thresholds for nasal and temporal motion as assessed with identical methods.

Measurements of MVEPs have localized an asymmetrical cortical response to motion in AMO monkeys in the striate cortex (V1).[36] We have developed a model based on alterations in specific cortical neural subsystems located in V1 that can, in principle, also account for the perceptual motion asymmetries in animals with AMO. Our model is an elaboration of the model originally developed by Hoffmann and elaborated by him and others to account for the nasal bias in MOKN after neonatal visual deprivation.[10,11,38–44] The Hoffmann model involves cortical connections with neurons in the nucleus of optic tract (NOT) that exhibit ipsiversive directional tuning. Each NOT receives visual information through a direct pathway from the contralateral eye and from indirect pathways from the cortex that carry signals from both eyes. Hoffmann's model is based on the fact that the indirect cortical pathways that are necessary to drive temporal responses are susceptible to neonatal visual deprivation.

The Hoffmann model is sufficient to account for the motion asymmetries in MOKN responses. However, it does not lead to any specific predictions about perceptual motion asymmetries, and furthermore it turns out to be surprisingly difficult to extend the model in a manner that is consistent with the known facts about MOKN, MVEP, and perceptual asymmetries. We present in the present study an elaboration of the Hoffmann model that is based on changes in specific subpopulations of directionally selective V1 neurons. The proposed neural pathways are illustrated in schematic form in Figure 4. The functional connections made by the left eye are shown in Figure 4A and those with the right eye in Figure 4B. In a normal animal, the connections from the two eyes combine to form binocular neurons. For example, *c1* (Fig. 4A) and *i1* (Fig. 4B) combine to form a single binocular neuron that projects from layer 5 of V1 to the NOT. In an animal with AMO, binocular neurons in V1 are lost, creating the separate subpopulations of neurons shown in Figures 4A and 4B.

Our psychophysical linking hypothesis is that the strength of connections made by each eye with neurons in V1 that respond to the left and right directions determine whether the psychophysical response to motion is symmetrical or asymmetrical. Motion responses elicited from one eye are expected to be symmetrical if there are strong connections formed with directionally selective units that respond to both the left and right directions of motion. If the strength of the connections for directionally selective units responding in one direction is stronger than for the other direction, there will be a corresponding asymmetry in the response to motion.

The challenge in constructing a model is to selectively eliminate subunits depicted in Figure 4 in a manner that can account for the psychophysical results of the present study and remain consistent with known facts regarding asymmetries in MOKN and MVEP. One difficulty in achieving this goal is that the direction of the cortical asymmetry in V1, as assessed with the MVEP, changes direction across eyes but not across hemispheres. Most simple modifications to the original Hoffmann model fail to achieve this condition. One scheme that achieves the desired goal is based on a supervised learning strategy where a Hebbian teacher resides in each NOT and gates the strength of connections in each functional circuit according to whether neural activity is correlated with activity in the neurons in the NOT to which the circuit projects. The circuits that are expected to be strengthened by this rule in animals with AMO are depicted by thick, bold lines in Figure 4A. The neurons in these functional circuits are each involved in processing right motion from the left eye and are expected to be

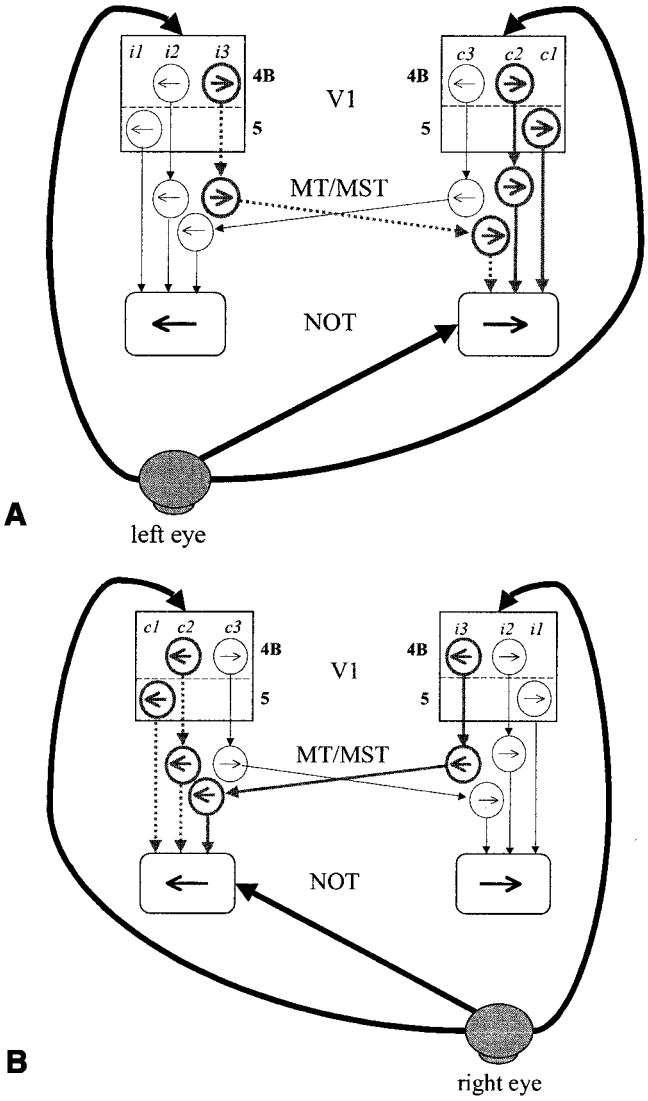

Figure 4. The connectivity of functional circuits between the eyes, cortical areas MT and MST, and the NOTs located in the left and right midbrain. Connections formed by (**A**) the left eye and (**B**) the right eye. Inputs derived from each eye form functional connections with six classes of directionally selective cells in V1, three in the contralateral hemisphere (*c1*, *c2*, and *c3*) and three in the ipsilateral (*i1*, *i2*, and *i3*). One of these types is located in layer 5 and projects directly to the NOT. The others are located in layer 4B and project first to the extrastriate areas, MT and MST, and then secondarily to the NOT. Some of the projections from MST to NOT cross the midline through the corpus callosum and represent the ipsilateral field. The *bold lines* and *symbols* depict functional circuits that may be altered by AMO rearing to cause the functional deficits that have been measured in these animals.

strengthened during left-eye viewing because they are active at the same time as neurons in the right NOT. The mirror-image neurons are expected to be strengthened during right-eye viewing as illustrated by the thick, bold lines in Figure 4B. In general, the cortical neurons that will be strengthened by this mechanism are those that support nasal motion for each eye. This elaborated Hoffmann model is sufficient to account for our results and remains consistent with previous findings of MOKN and MVEP asymmetries.

Previous studies in which investigators have looked for an overall bias in directional selectivity of single units in newborn monkey cortical area V1[45] or in the middle temporal (MT) area of monkeys raised with experimental strabismus[46] have not found evidence of asymmetry. However, our model does not predict that the entire population of directionally selective neurons in V1 or in the MT/medial superior temporal (MST) should be biased, but only that a bias should exist for the specific subpopulations of neurons participating in functional circuits projecting to the NOT.

There is currently some uncertainty about whether the various forms of motion and oculomotor asymmetry that have been measured after neonatal visual deprivation reflect a single mechanism or a number of independent mechanisms. Early reports of MOKN, MVEP, and psychophysical biases after neonatal visual deprivation tended to consider them to be reflections of a common neurologic deficit (e.g., Tychsen[47]). However, in recent studies in humans with congenital strabismus, investigators have discovered that the various forms of asymmetry do not always correlate in individual subjects.[35,48,49] The present study has established a monkey model that can be used to study some of these questions in a systematic manner. Further studies with this animal model have the potential to establish the necessary and sufficient conditions in which neonatal deprivation can lead to the various forms of motion asymmetry and the extent to which the mechanisms underlying these asymmetries are correlated with one another or independent.

Acknowledgments

The authors thank Alcides Fernandes, MD, for performing ophthalmic examinations on the animals and the veterinary and animal care staff at the Yerkes Primate Center for expert assistance in rearing and caring for the monkeys.

References

1. Boothe RG, Dobson V, Teller DY. Postnatal development of vision in human and nonhuman primates. *Annu Rev Neurosci.* 1985;8:495–545.
2. Boothe RG, Fulton AB. Amblyopia. In: Albert DM, Jakobiec FA, eds. *Principles and Practice of Ophthalmology*. Vol. 5. Philadelphia: WB Saunders; 2000:4340–4358.
3. Atkinson J. Development of optokinetic nystagmus in the human infant and monkey infant: an analogue to development in kittens. In: Freeman RD, ed. *Developmental Neurobiology of Vision*. New York: Plenum Press; 1979:277–287.
4. Atkinson J, Braddick O. Development of optokinetic nystagmus in infants: an indicator of cortical binocularity? In: Fisher DF, Monty RA, Senders JW, eds. *Cognition and Vision Perception*. Hillsdale, NJ: Erlbaum; 1981:53–64.
5. Naegel JT, Held R. The postnatal development of monocular optokinetic nystagmus in infants. *Vision Res.* 1982;22:341–346.
6. Roy MS, Lachapelle P, Lepore F. Maturation of the optokinetic nystagmus as a function of the speed of stimulation in fullterm and preterm infants. *Clin Vis Sci.* 1989;4:357–366.
7. Lewis TL, Maurer D, Van Schaik CS. Monocular OKN acuity is asymmetrical in normal 3-month olds [ARVO Abstract]. *Invest Ophthalmol Vis Sci*. 1990;31(4):**S**7. Abstract nr 31-3.
8. Lewis TL, Maurer D, Smith RJ, Haslip JK. The development of symmetrical optokinetic nystagmus during infancy. *Clin Vis Sci.* 1992;7:211–218.
9. Teller DY, Succop A, Mar C. Infant eye-movement asymmetries: stationary counterphase gratings elicit temporal-to-nasal optokinetic nystagmus in 2-month-old infants under monocular test conditions. *Vision Res.* 1993;33:1859–1864.
10. Van Hof-van Duin J. Direction preference of optokinetic responses in monocularly tested normal kittens and light deprived cats. *Arch Ital Biol.* 1978;116:471–477.
11. Hoffmann KP. Optokinetic nystagmus and single-cell responses in the nucleus tractus opticus after early monocular deprivation in the cat. In: Freeman RD, ed. *Developmental Neurobiology of Vision*. New York: Plenum Press: 1979:63–72.
12. Norcia AM, Garcia H, Humphrey R, Holmes A, Hamer RD, Orel-Bixler D. Anomalous motion VEPs in infants and in infantile esotropia. *Invest Ophthalmol Vis Sci.* 1991;32:436–439.
13. Brown RJ. *Deprivation of Binocular Vision and Consequences on Motion Perception* [Master's thesis]. Atlanta, GA: Emory University Graduate School; 1995.
14. Brown RJ, Wilson JR, Norcia AM, Boothe RG. Development of directional motion symmetry in the monocular visually evoked potential of infant monkeys. *Vision Res.* 1998;38:1253–1263.
15. Norcia AM, Hamer RD, Jampolsky A, Orel-Bixler D. Plasticity of human motion processing mechanisms following surgery for infantile esotropia. *Vision Res.* 1995;35:3279–3296.
16. Kommerell G, Ullrich D, Gilles U, Bach M. Asymmetry of motion VEP in infantile strabismus and in central vestibular nystagmus. *Doc Ophthalmol.* 1995;89:373–381.
17. Van Hof-van Duin J. Early and permanent effects of monocular deprivation on pattern discrimination and visuomotor behavior in cats. *Brain Res.* 1976;111:261–276.
18. Cynader M, Harris L. Eye movements in strabismic cats. *Nature.* 1980;286:64–65.
19. Schor C, Levi D. Disturbances of small-field horizontal and vertical optokinetic nystagmus in amblyopia. *Invest Ophthalmol Vis Sci.* 1980;19:668–683.
20. Malach R, Strong N, Van Sluyters R. Analysis of monocular optokinetic nystagmus in normal and visually deprived kittens. *Brain Res.* 1981;210:367–372.
21. Malach R, Strong N, Van Sluyters R. Horizontal optokinetic nystagmus in the cat: effects of long-term monocular deprivation. *Brain Res.* 1984;315:193–205.
22. Van Hof-van Duin J, Mohn G. Optokinetic and spontaneous nystagmus in children with neurological disorders. *Behav Brain Res.* 1983;10:163–175.
23. Lewis TL, Maurer D, Brent HP. Optokinetic nystagmus in normal and visually deprived children: Implications for cortical development. *Can J Psychol.* 1989;43:121–140.
24. Tychsen L, Lisberger SG. Visual motion processing for the initiation of smooth-pursuit eye movements in humans. *J Neurophysiol.* 1986;56:953–968.
25. Tychsen L, Lisberger SG. Maldevelopment of visual motion processing in humans who had strabismus with onset in infancy. *J Neurosci*. 1986;6:2495–2508.
26. Schor CM, Fusaro RE, Wilson N, McKee SP. Prediction of early onset esotropia from components of the infantile squint syndrome. *Invest Ophthalmol Vis Sci.* 1997;38:719–740.
27. Fawcett S, Raymond JE, Astle WF, Skov CM. Anomalies of motion perception in infantile esotropia. *Invest Ophthalmol Vis Sci.* 1998;39:724–735.
28. Westall CA, Eizenman M, Kraft SP, Panton CM, Chatterjee S, Sigesmund D. Cortical binocularity and monocular optokinetic asymmetry in early onset esotropia. *Invest Ophthalmol Vis Sci.* 1998;39:1352–1360.
29. Downing CJ, Movshon JA. Spatial and temporal summation in the detection of motion in stochastic random dot displays [ARVO Abstract]. *Invest Ophthalmol Vis Sci* 1989;30:72. Abstract nr 5.
30. Newsome WT, Pare EB. A selective impairment of motion perception following lesions of the middle temporal visual area (MT). *J Neurosci.* 1988;8:2201–2211.
31. Movshon JA, Newsome WT. Neural foundations of visual motion perception. *Curr Dir Psychol Sci.* 1992;1:35–39.

32. Boothe RB. Development of spatial vision in infant macaque monkeys under conditions of normal and abnormal visual experience. In: Aslin RN, Jeffrey R, Alberts JR, Petersen MR, eds. *Development of Perception: Psychobiological Perspectives.* New York: Academic Press; 1981:217–242.
33. Finney DJ. *Probit Analysis.* Cambridge, UK: Cambridge University Press; 1971.
34. McKee SP, Klein SA, Teller DY. Statistical properties of forced-choice psychometric functions: implications of probit analysis. *Percept Psychophys.* 1985;37:286–298.
35. Kommerell G. The relationship between infantile strabismus and latent nystagmus. *Eye.* 1996;10:274–81.
36. Wilson JR, Noyd WW, Aiyer AD, Norcia AM, Mustari MJ, Boothe RG. Asymmetric responses in cortical visually evoked potentials to motion are not derived from eye movements. *Invest Ophthalmol Vis Sci.* 1999;40:2435–2499.
37. Fu LN. *A Psychophysical Measurement and Analysis of Motion Perception in Normal and Binocularly Deprived Monkeys* [Master's thesis]. Atlanta, GA: Emory University Graduate School; 2000.
38. Hoffmann KP. Cortical versus subcortical contributions to the optokinetic reflex in the cat. In: Lennerstrand G, Zee S, Keller EL, eds. *Functional Basis of Ocular Motility Disorders* Oxford: Pergamon Press: 1982;303–310.
39. Hoffmann KP. The influence of visual experience on the ontogeny of the optokinetic reflex in mammals. In: Rauschecker JP, Marler P, eds. *Imprinting and Cortical Plasticity.* New York: Wiley; 1987.
40. Hoffmann KP. Visual inputs relevant for the optokinetic nystagmus in mammals. *Prog Brain Res.* 1986;64:75–84.
41. Hoffmann KP. Control of the optokinetic reflex by the nucleus of the optic tract in primates. *Prog Brain Res.* 1989;80:173–182.
42. Kato I, Harada K, Hasegawa T, Ikarashi T. Role of the nucleus of the optic tract of monkeys in optokinetic nystagmus and optokinetic after-nystagmus. *Brain Res.* 1988;474:16–26.
43. Cohen B, Reisine H, Yokota JI, Raphan T. The nucleus of the optic tract: its functions in gaze stabilization and control of visual vestibular interaction. *Ann NY Acad Sci.* 1993;656:277–296.
44. Fuchs AF, Mustari MJ. The optokinetic response in primates and its possible neuronal substrate. In: Miles SA, Wallman J, eds. *Visual Motion and Its Role in the Stabilization of Gaze.* New York: Elsevier; 1993;343–369.
45. Hatta S, Kumagami T, Qian J, Thornton M, Smith EL, Chino YM. Nasotemporal directional bias of V1 neurons in young infant monkeys. *Invest Ophthalmol Vis Sci.* 1998;39:2259–2267.
46. Kiorpes L, Kiper DC, O'Keefe LP, Cavanaugh JR, Movshon JA. Neuronal correlates of amblyopia in the visual cortex of macaque monkeys with experimental strabismus and anisometropia. *J Neurosci.* 1998;18:6411–6424.
47. Tychsen L. Motion sensitivity and the origins of infantile strabismus. In: Simmons K, ed. *Early Visual Development, Normal and Abnormal.* New York: Oxford; 1993:364–390.
48. Brosnahan D, Norcia AM, Schor CM, Taylor DG. OKN, perceptual and VEP direction biases in strabismus. *Vision Res.* 1998;38:2833–2840.
49. Birch EE, Fawcett S, Stager D. Co-development of VEP motion response and binocular vision in normal infants and infantile esotropes. *Invest Ophthalmol Vis Sci.* 2000;41:1719–1723.