Available online at www.sciencedirect.com
SCIENCE DIRECT®
Vision Research 45 (2005) 2877–2884
Vision Research
www.elsevier.com/locate/visres

Greater losses in sensitivity to second-order local motion than to first-order local motion after early visual deprivation in humans

D. Ellemberg [a,b,*], T.L. Lewis [b,c,d], N. Defina [b], D. Maurer [b,c], H.P. Brent [c,d], J-P. Guillemot [e,f], F. Lepore [e]

[a] Département de Kinésiologie, Université de Montréal, Montréal, Qué., Canada
[b] Department of Psychology, Neuroscience & Behaviour, McMaster University, Hamilton, Ont., L8S 4K1, Canada
[c] Department of Ophthalmology, The Hospital for Sick Children, Toronto, Canada
[d] Department of Ophthalmology and Vision Sciences, University of Toronto, Toronto, Canada
[e] Groupe de Recherche En Neuropsychologie Expérimentale, Université de Montréal, Montréal, Qué., Canada
[f] Université du Québec à Montréal, Montréal, Qué., Canada

Received 2 December 2003; received in revised form 26 May 2004

Abstract

We compared sensitivity to first-order versus second-order local motion in patients treated for dense central congenital cataracts in one or both eyes. Amplitude modulation thresholds were measured for discriminating the direction of motion of luminance-modulated (first-order) and contrast modulated (second-order) horizontal sine-wave gratings. Early visual deprivation, whether monocular or binocular, caused losses in sensitivity to both first- and second-order motion, with greater losses for second-order motion than for first-order motion. These findings are consistent with the hypothesis that the two types of motion are processed by different mechanisms and suggest that those mechanisms are differentially sensitive to early visual input.

Keywords: Congenital cataract; Binocular deprivation; Monocular deprivation; Visual development; Motion perception; First-order motion; Second-order motion

1. Introduction

The perception of motion based on local cues arises when the direction of movement can be determined reliably by looking at only a part of an object. Observers are adept at discriminating the direction of motion either from displacements in luminance (first-order cues) or from displacements in other physical characteristics of an image, such as its texture, that are visible even when there is no change in mean luminance (second-order cues). Computational models (Sperling & Zhong-Lin, 1998; Wilson, Ferrara, & Yo, 1992), supported by psychophysical (Chubb & Sperling, 1988; Legdeway & Smith, 1994), neurophysiological (Marischal & Baker, 1998, 1999; Zhou & Baker, 1993), human imaging (Dumoulin, Baker, Hess, & Evans, 2003; Smith, Greenly, Sing, Kramer, & Hennig, 1998), neuropsychological (Greenlee & Smith, 1997; Vaina & Cowey, 1996; Vaina, Makris, Kennedy, & Cowey, 1998), and visual evoked potential (Ellemberg et al., 2003a) data, suggest that first-order and second-order motion are processed by different neuronal mechanisms. For example, neurons in the cat's striate cortex have different spatial frequency tuning for first-order and second-order gratings (Marischal & Baker, 1998, 1999; Zhou & Baker, 1993).

* Corresponding author. Present address: Université de Montréal, Département de Kinésiologie, 2100 Eduard Montpetit, Montréal, Qué., Canada H3C 1J4 (D. Ellemberg).
E-mail address: dave.ellemberg@umontreal.ca (D. Ellemberg).

0042-6989/$ - see front matter © 2005 Published by Elsevier Ltd.
doi:10.1016/j.visres.2004.11.019

Although 2–3 month old infants can detect local motion defined by either first- or second-order cues (Braddick, Atkinson, & Hood, 1996), our previous study comparing thresholds in 5-year olds and adults showed that sensitivity to the two types of motion develops at different rates during childhood (Ellemberg et al., 2003b). At a slower velocity (1.5° s^{-1}), thresholds for 5-year olds were slightly reduced (they needed 2% and 5% more amplitude modulation than adults to see the direction of first- and second-order motion, respectively). However, at a faster velocity (6° s^{-1}), 5-year olds' thresholds were especially immature for second-order motion (they needed 16% more amplitude modulation than adults), but only slightly reduced for first-order motion (they needed 2% more amplitude modulation than adults). These findings indicate that at five years of age, sensitivity to first-order motion is more mature than sensitivity to second-order motion, at least at the faster velocity. The same seems to be true at 3 months of age (Armstrong, Lewis, Ellemberg, Bhagirath, & Maurer, 2004).

In humans, the lack of normal visual input at birth caused by dense central congenital cataracts in one or both eyes causes losses in virtually every aspect of vision studied. Previous studies have shown that aspects of vision that mature later during normal development are more affected by early visual deprivation than are aspects of vision that mature earlier. For example, acuity and sensitivity at high spatial and low temporal frequencies develop more slowly than do critical flicker fusion frequency and sensitivity at high temporal and low spatial frequencies, both during infancy (Atkinson, Braddick, & Moar, 1977; Banks & Salapatek, 1978, 1981; Regal, 1981) and childhood (Ellemberg, Lewis, Lui, & Maurer, 1999a). It is the slowly developing aspects of spatial and temporal vision that are most affected by deprivation, after either monocular or binocular deprivation caused by dense central cataracts (Ellemberg, Lewis, Maurer, Lui, & Brent, 1999b; Ellemberg, Lewis, Maurer, & Brent, 2000). Because sensitivity to the direction of second-order local motion likely is mediated at early levels of visual processing and because it develops later than sensitivity to the direction of first-order motion, we predicted that sensitivity to the direction of second-order motion is more affected by early visual deprivation. One goal of the present study was to test this hypothesis.

For many aspects of vision, deficits in the deprived eye are greater after early monocular deprivation than after early binocular deprivation, unless monocular deprivation was followed by extensive occlusion of the non-deprived eye. This pattern of deficit has been demonstrated in studies of acuity, spatial and temporal contrast sensitivity, stereovision, and sensitivity in the peripheral visual field (Birch, Stager, Leffler, & Weakley, 1998; Bowering, Maurer, Lewis, & Brent, 1993; Ellemberg et al., 1999b, 2000; Lewis, Maurer, & Brent, 1995; Mioche & Perenin, 1986; Tytla, Maurer, Lewis, & Brent, 1988, 1993). The usual explanation is that monocular deprivation affects visual development not only by depriving neurons in the primary visual cortex of patterned visual input from a deprived eye, but also by uneven competition for cortical connections between the deprived and non-deprived eye (Crawford, de Faber, Harwerth, Smith, & von Noorden, 1989; Elliott, Howarth, & Shadbolt, 1996; Maurer & Lewis, 1993, 2001a, 2001b). However, most previous studies have measured aspects of vision mediated primarily by striate cortex. In contrast, we recently reported a *worse* outcome after early binocular deprivation than after early monocular deprivation of comparable duration for the perception of global motion and of global form (Ellemberg, Lewis, Maurer, Brar, & Brent, 2002; Lewis et al., 2002), aspects of vision that require the spatial and/or temporal pooling of striate cortex sub-unit responses by extrastriate regions of the visual cortex. Based on these findings, we hypothesized that the effects of early deprivation and of uneven competition between the eyes for cortical connections differ for aspects of vision mediated at different cortical levels. A second goal of the present study was to test this hypothesis by comparing our previous results on the effects of monocular versus binocular deprivation on global motion to those obtained here for local motion, an aspect of vision mediated mainly by striate cortex.

We measured thresholds for discriminating direction of motion in patients treated for either bilateral or unilateral congenital cataract using the same paradigm that we had used to study the normal development of sensitivity to first- versus second-order local motion. We compared the results to those of comparably aged subjects with normal vision.

2. Methods

2.1. Subjects

Subjects were nine patients treated for bilateral congenital cataract (mean age at test = 11.8 years, range = 6.4–20.1 years), and 10 patients treated for unilateral congenital cataract (mean age at test = 10.6 years, range = 6.0–20.8 years). For bilateral congenital cases, the duration of deprivation ranged from 43 to 313 days (mean = 138 days); for unilateral cases, it ranged from 91 to 187 days (mean = 129 days). We define the duration of deprivation as the period extending from birth until the age of first optical correction following surgery to remove the cataract. Patients were included in the study if they were diagnosed with a dense central cataract in one or both eyes on the first eye exam and by six months of age and if they met at least one of the fol-

lowing criteria: the eye did not fixate or follow light, no red reflex was visible, and/or the cataract completely blocked the view of the fundus through an undilated pupil. Only patients with no other abnormalities in the ocular media or the retina, including persistent hyperplastic primary vitreous, and no other ocular disease such as glaucoma, were included in the study. Patients with common associated abnormalities such as strabismus, nystagmus, or microcornea were included. Patients who did not wear their optical correction regularly after treatment (at least 75% of the time) were excluded.

All patients treated for unilateral congenital cataract received occlusion therapy as treatment for amblyopia. Occlusion therapy was initiated shortly after the time of the first optical correction and continued through at least five years of age. Depending on the ophthalmologist, patients were instructed to patch the non-deprived eye for times ranging from four waking hours/day to as much as all but 1 h of waking time per day. However, because of variation in compliance, the mean amount of patching from the time of the first optical correction until 5.0 years of age ranged from 1.6 to 5.0 waking hours per day (mean = 3.6 h per day) (see Lewis et al., 1995 for details of these calculations).

2.2. Visually normal controls

For comparison, we tested ten comparably aged control subjects with no history of eye disorders, all of whom met our criteria on a visual screening exam (see Ellemberg et al., 2003b for details).

2.3. Apparatus and stimuli

The apparatus and stimuli were identical to those in our previous study of 5-year olds and adults (Ellemberg et al., 2003b). Specifically, the stimuli were generated by a Macintosh G3 computer by means of Pixx 1.55 software, and were displayed on a Sony Triniton Multiscan 200 GS monitor, 32° wide by 25° high when viewed from the testing distance of 50 cm. The monitor had a frame rate of 75 Hz. To increase the likelihood that patients would be able to easily detect the grating, we used a low spatial frequency (1 c deg^{-1}) for which early deprivation causes no or little reduction in contrast sensitivity (Ellemberg et al., 1999b, 2000). The stimuli consisted of horizontal sinusoidal gratings, 10° wide by 10° high when viewed from a distance of 50 cm. All stimuli were made from static two-dimensional random noise, the luminance of which was binary. Each noise element subtended $2 \times 2'$, and was independently assigned with a probability of 50% to be either "light" or "dark". The mean contrast of the noise was set to 0.50. The first-order stimuli were created by adding the carrier to a luminance-modulated sinusoidal grating of 1 c deg^{-1}. This created a sinusoidal modulation of the luminance across the carrier, which appeared like a conventional luminance-modulated sinusoidal grating. The amplitude of the luminance modulation (Michelson contrast) was defined as

$$\text{modulation depth} = (L_{\max} - L_{\min})/(L_{\max} + L_{\min})$$

where $L_{\max}$ and $L_{\min}$ are the maximum and minimum mean luminance averaged over adjacent pairs of noise dots.

The second-order stimuli were created by multiplying the carrier with a luminance-modulated sinusoidal grating of 1 c deg^{-1}. This produced a sinusoidal modulation of the contrast of the carrier. The stimulus consisted of a series of alternating regions of higher and lower noise contrast, each of which had the same mean luminance. The amplitude of the contrast modulation (Michelson contrast) was defined as

$$\text{modulation depth} = (C_{\max} - C_{\min})/(C_{\max} + C_{\min})$$

where $C_{\max}$ and $C_{\min}$ are the maximum and minimum mean local contrast (Michelson) of adjacent pairs of noise.

The space- and time-average luminance of the stimuli and background were maintained at 26 cd m^{-2}. Gamma-correction was verified by means of a Minolta LS-100 photometer. The luminance contrast of the first-order images was linearly related to the voltage of the Z-axis. Using the same procedure as Smith and Ledgeway (1996), we calibrated the second-order images to ensure that gamma-correction was accurate with respect to the characteristics of these stimuli. Specifically, we measured the local luminance values of a stationary and of a slowly drifting second-order stimulus, and adjusted the gamma correction factor to eliminate any differences in luminance between the high and low contrast regions of the envelope. The correction factor was checked regularly throughout the study. Further, small noise dots ($2 \times 2'$) were used so that the second-order stimuli would not contain detectable local luminance cues (Smith & Ledgeway, 1996).

3. Procedure

Informed consent was obtained after the nature of the study was explained to the subjects and, for subjects younger than 17 years of age, to their parents. The experimental protocol was approved by the Committee on the Ethics of Research on Human Subjects, McMaster University and by the Research Ethics Board of The Hospital for Sick Children. The procedure was identical to that of Ellemberg et al. (2003b) except that the deprived eye of each patient was corrected optically for the viewing distance. Each participant was tested monocularly while viewing the stimuli at a distance of

50 cm with the chin on a chin-rest. The eye not being tested was patched with 3M Micropore™ tape.

We tested the worse eye of patients treated for bilateral congenital cataract (as determined by Snellen acuity at the time of test or from clinical history of alignment if acuity was the same in both eyes) and the deprived eye of patients treated for unilateral congenital cataract. As a comparison we tested one eye of each subject in the comparably aged control group.

Each subject was tested on the seven following conditions: first-order gratings moving at 1.5, 6, and 12° s^{-1}; second-order moving gratings moving at 1.5 and 6° s^{-1}; and first- and second-order static gratings. Subjects were tested with first-order motion followed by second-order motion because we found previously that adults and children have lower thresholds when tested in that order (unpublished data). Within each type of stimulus (first-order and second-order), the order of conditions (static condition and the different velocities) was random.

Before testing, each subject was given two demonstration trials (one up and one down) consisting of first-order stimuli moving at 1.5° s^{-1}, and the experimenter told the subject which way the stripes were moving. Then the subject was instructed to fixate the centre of the screen and was told: "You will see a grey box with moving stripes. Your job is to tell me if the stripes are moving up (experimenter points up) or down (experimenter points down)." The experimenter entered the responses by means of the keyboard. The subject was given two complete practice staircases with feedback after each trial: one practice staircase with first-order motion before the test of first-order motion and one with second-order motion before the test of second-order motion. At the end of the practice run, the subject was asked if he/she understood the task and if so, testing began. During the test, no feedback was given but children were praised periodically, regardless of the accuracy of their responses, and were reminded to watch carefully. The experimenter watched the participant's viewing eye continuously to ensure that he/she was looking at the centre of the screen. Thresholds for the discrimination of the direction of motion (up or down) were calculated with a ML-TEST (Maximum Likelihood) staircase procedure (Harvey, 1986), and were defined as the minimum modulation depth necessary to detect the direction of motion accurately.

To determine whether any deficits in patients could be attributed to an inability to see the stimuli, we also used a ML-TEST staircase procedure to measure detection thresholds for static first- and second-order stimuli. On each trial, the subject was asked to identify the orientation of first- and second-order gratings that were displayed either vertically or horizontally. We defined the threshold for each type of stimulus as the minimum modulation depth necessary to detect the orientation of the grating accurately.

4. Results

4.1. Static condition

Fig. 1 shows the mean modulation depth thresholds for the static condition. ANOVAs for each stimulus revealed no difference between the groups (bilateral patients, unilateral patients, controls), both for the first-order condition ($F_2 = 0.55$, $p > 0.05$) and for the second-order condition ($F_2 = 0.76$, $p > 0.05$). These results indicate that the patients were normal at detecting the spatial structure of the first- and second-order patterns.

4.2. Motion condition

Fig. 2 shows the mean thresholds for the motion conditions. For first-order motion, an ANOVA with groups and velocity as factors yielded a significant interaction between group and velocity ($F_4 = 11.46$, $p < 0.0001$), as well as significant main effects of group ($F_2 = 29.52$, $p < 0.01$) and of velocity ($F_2 = 62.38$, $p < 0.01$). The thresholds of unilateral and bilateral patients did not differ significantly at any velocity ($p > 0.10$), and patients had significantly higher thresholds than controls for each velocity ($ps < 0.05$). An analysis of simple effects on the interaction revealed that, for both unilateral cases ($p < 0.001$) and bilateral cases ($p < 0.001$), thresholds decreased with increasing velocity, whereas for

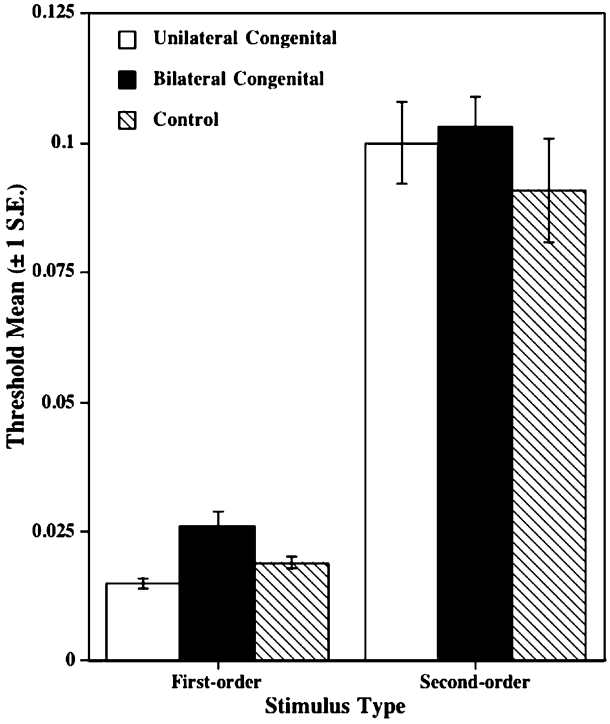

Fig. 1. Mean modulation depth thresholds (±1 standard error) for the discrimination of the orientation of static first- and second-order gratings in unilateral cases (white bars), bilateral cases (black bars), and control subjects (striped bars).

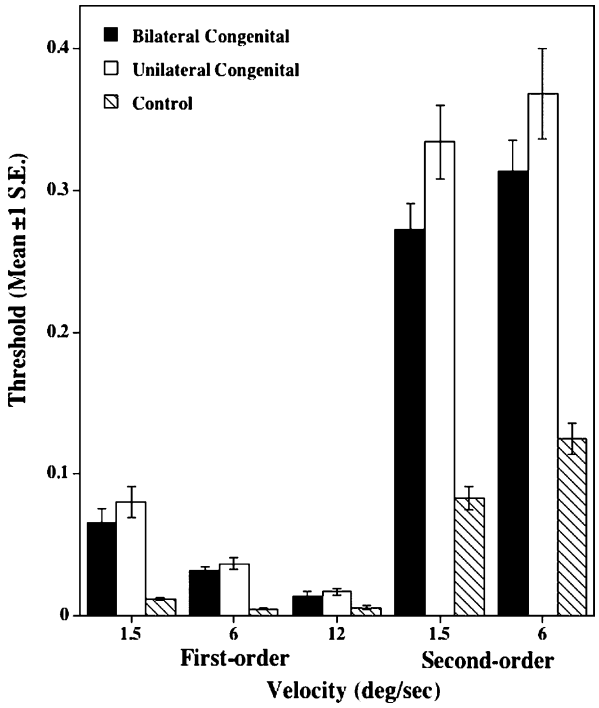

Fig. 2. Mean modulation depth thresholds (±1 standard error) for the discrimination of the direction of first- and second-order gratings in unilateral cases (white bars), bilateral cases (black bars), and control subjects (striped bars). Thresholds represent modulation of luminance for first-order motion at 1.5, 6, and 12° s^{-1}, and modulation of contrast for second-order motion at 1.5 and 6° s^{-1}. The means for second-order motion exclude the two patients who could not see the second-order motion.

controls, thresholds did not change with velocity ($p > 0.10$). This indicates that patients' deficits decreased with increasing velocity.

Two patients (one bilateral, one unilateral) were unable to see second-order motion even at the highest contrast, despite having normal thresholds for the identification of the orientation of a static second-order stimulus. An ANOVA for the remaining subjects yielded a significant main effect of group ($F_2 = 40.35$, $p < 0.01$). Thresholds of unilateral and bilateral patients did not differ significantly ($p > 0.10$), but patients had significantly higher thresholds than controls ($p < 0.001$). There was also a main effect of velocity ($F_2 = 31.52$, $p < 0.01$) because thresholds were lower for the slower velocity (1.5° s^{-1}) than for the faster velocity (6° s^{-1}). There was no significant interaction between group and velocity ($p > 0.10$), a result indicating that the extent of the patients' losses for second-order motion was the same for the slower and the faster velocities (see Fig. 2).

5. Discussion

Sensitivity to the direction of first- and second-order local motion is slightly but significantly reduced after early visual deprivation caused by dense central congenital cataract in one or both eyes. However, sensitivity to second-order motion is more affected by early deprivation than is sensitivity to first-order motion. For first-order motion, deficits increased as velocity decreased, from needing 1% more amplitude modulation than controls at 12° s^{-1} to needing 6% more amplitude modulation at 1.5° s^{-1}. For second-order motion the extent of the losses was the same for the slower (1.5° s^{-1}) and the faster (6° s^{-1}) velocities (patients needed 22% more amplitude modulation than controls).

The different pattern of findings for first-order and second-order motion suggests that they are mediated by separate mechanisms that are differentially affected by early visual deprivation. To investigate this possibility further, we examined whether there was a correlation in the extent of the deficit for the two types of motion. Because each patient did not have an age-matched control (see Section 2), we matched the bilateral patients in the current study to visually normal controls of the same age who had been tested in the same way for another project. The results indicated no correlation in the extent of the deficit for first-order and second-order motion either at the slower ($r = 0.20$, $p > 0.06$) or at the faster velocity ($r = 0.47$, $p > 0.20$). Together, the results are consistent with previous evidence for separate mechanisms for the processing of first-order and second-order motion (e.g., Chubb & Sperling, 1988), and indicate that these different mechanisms are differentially affected by early visual deprivation.

Deficits in contrast sensitivity and pattern processing are unlikely to be responsible for the observed losses in the discrimination of the direction of local motion: we used a low spatial frequency envelope for which such patients have normal contrast sensitivity and found that when the stimuli were static, patients had normal thresholds for the discrimination of the orientation of the first- and second-order stimuli. Associated disorders like horizontal nystagmus and strabismus are also not likely to be responsible for the observed losses in sensitivity. Our stimuli moved vertically in order to prevent any interference from the horizontal nystagmus. Further, nystagmus was more prevalent in the patients treated for bilateral congenital cataract than in those treated for unilateral congenital cataract, but there was no difference in their deficits for local motion. Two bilateral patients had similar deficits whether tested binocularly or monocularly, despite the fact that they had nystagmus only when tested monocularly (i.e., they had a latent nystagmus but no manifest nystagmus). Four of the patients had no history of strabismus; yet their pattern of deficits for both first- and second-order motion was similar to that of the patients with strabismus. Thus, the pattern of losses for first- and second-order motion likely resulted from early pattern deprivation and not from an inability to see the stimuli

or from associated disorders such as strabismus or nystagmus.

Larger deficits for second-order motion than for first-order motion cannot be attributed to the poor visibility of the noise carrier. Overall, as shown in Fig. 1, controls performed no better than patients in discriminating the orientation of second-order static patterns. Specifically, controls needed 5–14% amplitude modulation to discriminate horizontal from vertical second-order patterns, similar to that required by the unilaterally (5–14%) and bilaterally (6–12%) deprived patients. Moreover, the two patients who were unable to see second-order motion even at the highest amplitude modulation could discriminate the orientation of the static second-order gratings and do so as well as controls.

The results from the present study for local motion agree with previous studies of acuity, spatial and temporal contrast sensitivity, stereovision, and sensitivity in the peripheral visual field, for which sensitivity is impaired at least as much after monocular deprivation followed by aggressive patching of the non-deprived eye as after binocular deprivation (Birch et al., 1998; Bowering et al., 1993; Ellemberg et al., 1999b, 2000; Lewis et al., 1995; Mioche & Perenin, 1986; Tytla et al., 1988, Tytla, Lewis, Maurer, & Brent, 1993). In contrast, patients who had been treated for unilateral cataract have significantly better thresholds for global motion and global form than patients treated for bilateral cataract (Ellemberg et al., 2002; Lewis et al., 2002). After monocular deprivation, thresholds for global motion were worse than normal by a factor of only 1.6 whereas after binocular deprivation of comparable duration, thresholds for global motion were worse than normal by a factor of 4.9. Unlike local motion, acuity, and contrast sensitivity (which are processed mainly at the level of the striate cortex), the perception of global motion and global form require specialized processing in extrastriate regions of the visual cortex (viz., area MT and V4, respectively). The present findings add additional support for the hypothesis that the relative sparing after monocular deprivation for global form and global motion does not generalize to aspects of vision like sensitivity to local motion which are mediated at lower cortical levels.

Our findings are consistent with the hypothesis that, among abilities mediated at lower cortical levels, early deprivation has larger effects for aspects of vision that are relatively slow to mature (Maurer & Lewis, 1993). Sensitivity to first-order motion, which matures more rapidly during infancy (Armstrong et al., 2004) and early childhood (Ellemberg et al., 2003b), is less affected by deprivation than is sensitivity to second-order motion, which matures more slowly. Even within first-order motion, the velocities that are most affected by deprivation are the ones for which sensitivity is the slowest to mature. Specifically, the losses in the discrimination of the direction of motion are greater at slower than at faster velocities, and previous studies have shown that during infancy, sensitivity to slower velocities matures more slowly than does sensitivity to faster velocities (Aslin & Shea, 1990; Bertenthal & Bradbury, 1992; Freedland & Dannemiller, 1987; Kaufmann, 1995; Roessler & Dannemiller, 1997; Volkmann & Dobson, 1976 but see Ellemberg et al., 2003b). This is similar to our findings for temporal contrast sensitivity: losses in temporal contrast sensitivity are greater at lower than at higher temporal frequencies, and sensitivity to the lower temporal frequencies is the last to mature (Ellemberg et al., 1999a, 1999b).

In the patients we selected for study, dense central cataracts blocked all patterned information from reaching the retina, as well as all information about motion. Therefore, we are unable to determine the extent to which pattern deprivation and motion deprivation each contributed to the losses in our patients. Cats deprived of motion but not of pattern vision, because they were raised in an environment illuminated stroboscopically at 8 Hz, show a dramatic reduction in directionally-selective neurons and a 10-fold increase in contrast threshold for discriminating direction of motion (Cremieux, Orban, Duysens, & Amblard, 1987; Cynader & Chernenko, 1976; Kennedy & Orban, 1983; Pasternak & Leinen, 1986; Pasternak, Schumer, Gizzi, & Movshon, 1985). Moreover, motion deprivation in cats has a greater effect on the detection of slower velocities than on the detection of faster velocities (Cremieux et al., 1987; Pasternak, Merigan, & Movshon, 1981), similar to our results for first-order motion.

Pasternak et al. (1981) provide evidence for considerable recovery in their strobe-reared cats. After 30 weeks of normal input, minimum detectable velocity improved from 15 times to 2 times normal and the number of directionally selective cells increased. Because our patients were tested at least five years after deprivation ended, it is possible that they were able to recover from initially larger deficits and that our measurements represent the residual permanent deficit.

In summary, we found that early visual deprivation caused by dense central cataracts causes losses in the perception of both first- and second-order local motion. Therefore, early visual input to each eye appears to be necessary to set up the neural architecture that will later be fine tuned to detect signals for the discrimination of the direction of local motion. The pattern of deficits appears to be related to the normal pattern of development. Losses for second-order motion were greater than those for first-order motion, and sensitivity to second-order motion develops more slowly. Similarly, for first-order motion, losses were greater at the slower velocities than at the faster velocities, and sensitivity to slower velocities develops more slowly during infancy. These findings are consistent with the hypothesis that the two types of motion are processed, at least in part,

by different mechanisms. Moreover, our findings indicate that these different mechanisms are differentially affected by deprivation.

Acknowledgments

We thank The Hospital for Sick Children in Toronto for providing space for the tests. We also thank the patients, the children and their families for participation. This research was supported by a Canadian Institutes of Health Research grant (MOP 36430) to Daphne Maurer, a Canada Research Chair to Franco Lepore, an NSERC grant to Franco Lepore and to Jean-Paul Guillemot, and a FCAR team grant to Franco Lepore and Jean-Paul Guillemot. Dave Ellemberg was supported by the E.A. Baker award (MRC).

References

Armstrong, V. L., Lewis, T. L., Ellemberg, D., Bhagirath, V., & Maurer, D. (2004). Comparison of sensitivity to first and second order information in infants, children, and adults [Abstract]. *Journal of Vision, 4*(8), 31, Available from: http://journalofvision.org/4/8/31/, doi:10.1167/4.8.31.

Aslin, R. N., & Shea, S. L. (1990). Velocity thresholds in human infants: implications for the perception of motion. *Developmental Psychology, 26*, 589–598.

Atkinson, J., Braddick, O., & Moar, K. (1977). Contrast sensitivity of the human infant for moving and static patterns. *Vision Research, 17*, 1045–1047.

Banks, M. S., & Salapatek, P. (1978). Acuity and contrast sensitivity in 1-, 2-, and 3 month-old human infants. *Investigative Ophthalmology and Visual Science, 17*, 361–365.

Banks, M. S., & Salapatek, P. (1981). Infant pattern vision: a new approach based on the contrast sensitivity function. *Journal of Experimental Child Psychology, 31*, 1–45.

Bertenthal, B. I., & Bradbury, A. (1992). Infants' detection of shearing motion in random-dot displays. *Developmental Psychology, 28*, 1056–1066.

Birch, E. E., Stager, D., Leffler, J., & Weakley, D. (1998). Early treatment of congenital unilateral cataract minimizes unequal competition. *Investigative Ophthalmology & Visual Sciences, 39*, 1560–1566.

Bowering, E. R., Maurer, D., Lewis, T. L., & Brent, H. P. (1993). Sensitivity in the nasal and temporal hemifields in children treated for cataract. *Investigative Ophthalmology & Visual Science, 34*, 3501–3509.

Braddick, O., Atkinson, J., & Hood, B. (1996). Striate cortex, extrastriate cortex, and colliculus: some new approaches. In: F. Vital-Durand, O. Braddick, J. Atkinson (Eds.), *Infant vision* (pp. 203–220). Oxford.

Chubb, C., & Sperling, G. (1988). Drift-balanced random stimuli: a general basis for studying non-Fourier motion perception. *Journal of the Optical Society of America, A, 5*, 1986–2007.

Crawford, M. J. L., de Faber, J.-T., Harwerth, R. S., Smith, E. L., & von Noorden, G. K. (1989). The effects of reverse monocular deprivation in monkeys II. Electrophysiological and anatomical studies. *Experimental Brain Research, 74*, 338–347.

Cremieux, J., Orban, G. A., Duysens, J., & Amblard, B. (1987). Response properties of area 17 neurons in cats reared in stroboscopic illumination. *Journal of Neurophysiology, 57*, 1511–1535.

Cynader, M., & Chernenko, G. (1976). Abolition of direction selectivity in the visual cortex of the cat. *Science, 193*, 504–505.

Dumoulin, S. O., Baker, C. L., Jr., Hess, R. F., & Evans, A. C. (2003). Cortical specialization for processing first- and second-order motion. *Cerebral Cortex, 13*, 1375–1385.

Ellemberg, D., Lavoie, K., Lewis, T. L., Maurer, D., Lepore, F., & Guillemot, J.-P. (2003a). Longer VEP latencies and slower reaction times to the onset of second order motion than to the onset of first-order motion. *Vision Research, 43*, 651–658.

Ellemberg, D., Lewis, T. L., Medhji, K., Maurer, D., Guillemot, J.-P., & Lepore, F. (2003b). Comparison of sensitivity to first- and second-order local motion in 5-year-olds and adults. *Spatial Vision, 16*, 419–428.

Ellemberg, D., Lewis, T. L., Lui, C. H., & Maurer, D. (1999a). Development of spatial and temporal vision during childhood. *Vision Research, 39*, 2325–2333.

Ellemberg, D., Lewis, T. L., Maurer, D., Lui, C. H., & Brent, H. P. (1999b). Spatial and temporal vision in patients treated for bilateral congenital cataracts. *Vision Research, 39*, 3480–3489.

Ellemberg, D., Lewis, T. L., Maurer, D., Brar, S., & Brent, H. P. (2002). Better perception of global motion after monocular than after binocular deprivation. *Vision Research, 42*, 169–179.

Ellemberg, D., Lewis, T. L., Maurer, D., & Brent, H. P. (2000). Influence of monocular deprivation during childhood on the later development of spatial and temporal vision. *Vision Research, 40*, 3283–3295.

Elliott, T., Howarth, C. I., & Shadbolt, N. R. (1996). Axonal processes and neural plasticity I: ocular dominance columns. *Cerebral Cortex, 6*, 781.

Freedland, R. L., & Dannemiller, J. L. (1987). Detection of stimulus motion in 5-month old infants. *Journal of Experimental Psychology: Human Perception and Performance, 13*, 566–576.

Greenlee, M. W., & Smith, A. T. (1997). Detection and discrimination of first- and second-order motion in patients with unilateral brain damage. *Journal of Neuroscience, 17*, 804–808.

Harvey, L. O. (1986). Efficient estimation of sensory thresholds. *Behavior Research Methods, Instruments, & Computers, 18*, 623–632.

Kaufmann, F. (1995). Development of motion perception in early infancy. *European Journal of Pediatrics, 154*, S48–S53.

Kennedy, H., & Orban, G. A. (1983). Response properties of visual cortical neurons in cats reared in stroboscopic illumination. *Journal of Neurophysiology, 49*, 686–704.

Legdeway, T., & Smith, A. T. (1994). Evidence for separate motion-detecting mechanisms for first and second order motion in human vision. *Vision Research, 34*, 2727–2740.

Lewis, T. L., Ellemberg, D., Maurer, D., Wilkinson, F., Wilson, H. R., Dirks, M., & Brent, H. P. (2002). Sensitivity to global form in glass patterns after early visual deprivation in humans. *Vision Research, 42*, 939–948.

Lewis, T. L., Maurer, D., & Brent, H. P. (1995). Development of grating acuity in children treated for unilateral or bilateral congenital cataract. *Investigative Ophthalmology & Visual Science, 36*, 2080–2095.

Marischal, I., & Baker, C. L. (1998). Temporal and spatial responses to second-order stimuli in cat area 18. *Journal of Neurophysiology, 80*, 2811–2873.

Marischal, I., & Baker, C. L. (1999). Cortical processing of second-order motion. *Visual Neuroscience, 16*, 527–540.

Maurer, D., & Lewis, T. L. (2001a). Visual acuity and spatial contrast sensitivity: normal development and underlying mechanisms. In C. A. Nelson & M. Luciana (Eds.), *The handbook of developmental cognitive neuroscience* (pp. 237–251). Boston, MA: MIT Press.

Maurer, D., & Lewis, T. L. (2001b). Visual acuity: the role of visual input in inducing postnatal change. *Clinical Neuroscience Research, 1*, 239–247.

Maurer, D., & Lewis, T. L. (1993). Visual outcomes after infantile cataract. In K. Simons (Ed.), *Early visual development: normal and abnormal* (pp. 454–484). Oxford: Oxford University Press.

Mioche, L., & Perenin, M. (1986). Central and peripheral residual vision in humans with bilateral deprivation amblyopia. *Experimental Brain Research, 62*, 259–272.

Pasternak, T., & Leinen, L. J. (1986). Pattern and motion vision in cats with selective loss of cortical direction selectivity. *The Journal of Neuroscience, 6*, 938–945.

Pasternak, T., Merigan, W. H., & Movshon, J. A. (1981). Motion mechanisms in strobe-reared cats: psychophysical and electrophysical measures. *Acta Psychologica, 48*, 321–332.

Pasternak, T., Schumer, R. A., Gizzi, M. S., & Movshon, J. A. (1985). Abolition of visual cortical direction selectivity affects visual behavior in cats. *Experimental Brain Research, 61*, 214–217.

Regal, D. M. (1981). Development of critical flicker frequency in human infants. *Vision Research, 21*, 549–555.

Roessler, J. S., & Dannemiller, J. L. (1997). Changes in human infants' sensitivity spacing to slow displacements over the first 6 months. *Vision Research, 37*, 417–423.

Smith, A. T., Greenly, M. W., Sing, K. D., Kramer, F. M., & Hennig, J. (1998). The processing of first- and second-order motion in human visual cortex assessed by functional magnetic resonance imaging (fMRI). *The Journal of Neuroscience, 18*, 3816–3830.

Smith, A. T., & Ledgeway, T. (1996). Separate detection of moving luminance and contrast modulations: fact or artifact? *Vision Research, 37*, 45–62.

Sperling, G., & Zhong-Lin, L. (1998). A systems analysis of visual motion perception. In T. Watanabe (Ed.), *High-level motion processing: computational, neurobiological, and psychophysical perspectives* (pp. 153–183). MIT Press.

Tytla, M. E., Lewis, T. L., Maurer, D., & Brent, H. P. (1993). Stereopsis after congenital cataract. *Investigative Ophthalmology & Visual Science, 34*, 1767–1773.

Tytla, M. E., Maurer, D., Lewis, T. L., & Brent, H. P. (1988). Contrast sensitivity in children treated for congenital cataract. *Clinical Vision Sciences, 2*, 251–264.

Vaina, L. M., & Cowey, A. (1996). Impairment of the perception of second-order motion but not first-order motion in a patient with unilateral focal brain damage. *Proceedings of the Royal Society, London, 263*, 1225–1232.

Vaina, L. M., Makris, N., Kennedy, D., & Cowey, A. (1998). The selective impairment of the perception of first-order motion by unilateral cortical brain damage. *Visual Neuroscience, 15*, 333–348.

Volkmann, F. C., & Dobson, M. V. (1976). Infant responses of ocular fixation to moving visual stimuli. *Journal of Experimental Child Psychology, 22*, 86–99.

Wilson, H. R., Ferrara, V. P., & Yo, C. (1992). A psychophysically motivated model for two-dimensional motion perception. *Visual Neuroscience, 9*, 79–97.

Zhou, Y.-X., & Baker, C. L. (1993). A processing stream in mammalian visual cortex neurons for non-Fourier responses. *Science, 261*, 98–101.