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Vision Research 42 (2002) 939–948

Vision Research

www.elsevier.com/locate/visres

Sensitivity to global form in glass patterns after early visual deprivation in humans

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Received 15 August 2001

Abstract

To compare the effects of early monocular versus early binocular deprivation on the perception of global form, we assessed sensitivity to global concentric structure in Glass patterns with varying ratios of paired signal dots to noise dots. Children who had been deprived by dense congenital cataracts in one ($n = 10$) or both ($n = 8$) eyes performed significantly worse than comparably aged children without eye problems. Consistent with previous results on sensitivity to global motion [Vision Research 42 (2002) 169], thresholds in the deprived eyes were significantly *better* after monocular deprivation than after binocular deprivation of comparable duration, even when there had been little patching of the nondeprived eye after monocular deprivation. Together, the results indicate that the competitive interactions between a deprived and nondeprived eye evident in the primary visual cortex can co-occur with complementary interactions in extrastriate cortex that enable a relative sparing of some visual functions after early monocular deprivation.

Keywords: Congenital cataract; Binocular deprivation; Monocular deprivation; Visual development; Global form perception; Glass patterns; Humans

1. Introduction

Numerous studies have shown that many aspects of vision are worse in a deprived eye after early monocular deprivation than after early binocular deprivation. However, extensive occlusion of the nondeprived eye after monocular deprivation can result in vision in the deprived eye as good as that achieved after binocular deprivation. This pattern of results has been observed for the spatial and temporal vision of monkeys deprived by lid suture (Harwerth, Smith, Boltz, Crawford, & van Noorden, 1983a,b; Harwerth, Smith, Paul, Crawford, & von Noorden, 1991), and for many aspects of vision in humans deprived by dense congenital cataracts, including grating acuity, linear letter acuity, spatial contrast sensitivity, temporal contrast sensitivity, peripheral vision, and stereo vision (Birch, Stager, Leffler, & Weakley, 1998; Bowering, Maurer, Lewis, & Brent, 1993; Ellemberg, Lewis, Maurer, Brar, & Brent, 2002; Ellemberg, Lewis, Maurer, & Brent, 2000; Ellemberg, Lewis, Maurer, Liu, & Brent, 1999; Lewis, Maurer, & Brent, 1995; Mioche & Perenin, 1986; Tytla, Lewis, Maurer, & Brent, 1993; Tytla, Maurer, Lewis, & Brent, 1988).

The usual explanation for the greater deficits after-monocular deprivation is that monocular deprivation affects visual development not only by depriving neurons in the primary visual cortex of patterned visual input from the deprived eye, but also by uneven competition for cortical connections between the deprived and nondeprived eyes (Crawford, de Faber, Harwerth, Smith, & van Noorden, 1989; Elliott, Howarth, & Shadbolt, 1996; Maurer & Lewis, 1993, 2001a,b). This

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0042-6989/02/$ - see front matter
PII: S0042-6989(02)00041-X

explanation is supported by physiological studies of the monkey's primary visual cortex showing that the deprived eye drives very few cells in striate cortex after early monocular deprivation, far fewer than after binocular deprivation of the same duration, and those cells have extremely poor sensitivity to spatial frequency and contrast (Blakemore, 1998; Crawford, 1998; Crawford, Pesch, van Noorden, Harwerth, & Smith, 1991; Hubel, Wiesel, & Le Vay, 1977). Suturing the fellow eye at the time that the deprived eye is opened increases the proportion of striate cells that can be driven by the originally deprived eye and improves the functional acuity of that eye (Blakemore, Garey, & Vital-Durand, 1978; Crawford et al., 1989; Harwerth, Smith, Crawford, & von Noorden, 1989; Le Vay, Wiesel, & Hubel, 1980; Swindale, Vital-Durand, & Blakemore, 1981).

Since the classical work of Hubel and Wiesel (1970) and Wiesel and Hubel (1965), it usually has been assumed that a deprived and nondeprived eye interact only via a competitive mechanism that results in a worse outcome after monocular than after binocular deprivation. Recently, we (Ellemberg et al., 2002) discovered a different pattern of interaction, namely one that results in a *better* outcome after monocular than after binocular deprivation. Specifically, we measured both grating acuity and sensitivity to the direction of global motion in the deprived eyes of patients treated for dense congenital cataracts in one or both eyes. The results for grating acuity were as expected: worse acuity after monocular deprivation than after binocular deprivation unless, after monocular deprivation, the nondeprived eye had been patched extensively throughout early childhood. In contrast, sensitivity to the direction of global motion was significantly *better* after monocular than after binocular deprivation of comparable duration, even when there had been little patching of the nondeprived eye after monocular deprivation. In fact, sensitivity was only 1.6 times worse than normal after monocular deprivation but 4.9 times worse than normal after binocular deprivation, and the results after monocular deprivation were unrelated to how much the nondeprived had been patched. Since normal grating acuity depends on the integrity of the geniculo-striate pathway (Blakemore, 1990) and normal sensitivity to the direction of global motion depends on the integrity of extrastriate regions including the middle temporal (MT) cortex (Maunsell & Newsome, 1987; Wilson, 1999), Ellemberg et al. (2002) hypothesized that the competitive interactions between a deprived and nondeprived eye that affect connections in the striate cortex can co-occur with collaborative interactions in higher cortical areas. The purpose of the present study was to assess the generality of that hypothesis by measuring sensitivity to global form, an aspect of vision that involves an extrastriate area different from that involved in global motion.

Like global motion, the perception of global form requires the integration of information about local elements into a coherent whole. Local elements can be detected by simple and complex cells in the primary visual cortex, the output of which is then integrated by cells in higher cortical areas with larger receptive fields (reviewed in Wilson, 1999). Computational models of form perception (Wilson, 1999; Wilson & Wilkinson, 1998), supported by psychophysical (Glass & Switkes, 1976; Wilson, Wilkinson, & Asaad, 1997), physiological (Gallant, Braun, & van Essen, 1993; Gallant, Connor, Rakshit, Lewis, & van Essen, 1996; Pasupathy & Connor, 1999), neuropsychological (Gallant, Shoup, & Mazer, 2000), and fMRI (Wilkinson et al., 2000) data are consistent with the hypothesis that extrastriate area V4v in the ventral visual pathway plays a role in the perception of global form. For example, single cell recordings of the monkey have identified a type of cell in area V4v responsive primarily to concentric structure (Gallant et al., 1993, 1996; Pasupathy & Connor, 1999), whereas such cells are very rare in area V2, the area preceding V4 in the ventral visual pathway (Kobatake & Tanaka, 1994). One contributing factor may be the fact that receptive field size increases significantly from area V2 to area V4v, making area V4v better suited to integrate local elements (Gattass, Sousa, & Gross, 1988). However, the evidence for the precise role of area V4v in the analysis of global form is far from conclusive (e.g., Braddick, O'Brien, Wattam-Bell, Atkinson, & Turner, 2000).

Glass (1969) patterns are ideal stimuli for studying sensitivity to structure in global form. Glass (1969) noted that when a pattern of random dots is superimposed over an identical pattern and rotated a critical amount about the central axis, a compelling perception of concentric swirls arises. These patterns, now known as concentric Glass patterns, can be used to assess sensitivity to global form by varying the ratio of paired signal dots to noise dots until the subject can no longer discriminate accurately between the signal pattern and a pattern comprised solely of noise dots.

Little is known in any species about the effect of early pattern deprivation on the later development of the perception of global form. To evaluate the effect in humans, we used concentric Glass patterns to measure sensitivity to global form in 18 patients treated for a congenital cataract in one or both eyes. In all cases, the cataracts were sufficiently large (at least 5 mm in diameter) and sufficiently dense to block all patterned information to the retina. Treatment involved surgical removal of the cataractous lens and replacing it with a contact lens that focused visual input on the retina. At the time of the test, the patients were at least six years old, old enough to perform the tests with Glass patterns so that we could study the effects of early pattern deprivation and the nature of the interactions between the eyes in setting up the neural mechanisms used later to

detect global form. Worse performance after monocular than after binocular deprivation would be indicative of competitive interactions between the eyes whereas better performance after monocular than after binocular deprivation would be indicative of some other form of interaction between the eyes. Results from patients were compared to those of normal controls tested under the same conditions.

2. Methods

2.1. Subjects

2.1.1. Patients

Subjects were eight patients treated for bilateral congenital cataracts (mean age at test = 12.5 y, range = 6.3–20.0 y) and ten patients treated for unilateral congenital cataract (mean age at test = 10.5 y, range = 6.0–20 y). Duration of deprivation ranged from 3.0–8.8 months ($M = 4.6$ months) in bilateral cases and from 1.4–10.4 months ($M = 4.6$ months) in unilateral cases. Inclusion and exclusion criteria for the patients have been described elsewhere (Lewis et al., 1995; Maurer & Lewis, 1993). Briefly, patients were included in the study if they met all of the following criteria: (1) diagnosis of a dense central cataract in one or both eyes on the first eye exam, which was always before six months of age; (2) no other abnormalities in the ocular media or the retina, including no evidence of persistent hyperplastic primary vitreous; (3) no ocular disease such as glaucoma; (4) no neurological abnormalities that might interfere with vision such as hydrocephalus; and (5) regular wear of optical correction after treatment (at least 75% of the waking time). We included patients with common associated abnormalities such as strabismus, nystagmus, microcornea, or short axial length and, in unilateral cases, excluded patients with any abnormalities in the fellow nondeprived eye that were likely to interfere with vision (e.g., developing cataract or more than minimal refractive error). None of the participants were developmentally delayed and all attended regular school programmes. Clinical details of the patients are described in Table 1. The final sample included one deprived eye from each patient (see Table 1). The geometric mean Snellen acuity for the included deprived eyes was 20/73 for bilateral cases and 20/107 for unilateral cases.

All unilateral cases 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 nondeprived 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 five years of age ranged from 1.6 to 5.0 waking hours per day (see Lewis et al., 1995 for details of these calculations).

2.1.2. Normal controls

Results from the eight bilateral cases and ten unilateral cases were compared to those of ten comparably aged normal control subjects tested under the same conditions. To choose the most representative range of ages for the control group, we rank ordered the 18 patients by age and then tested a control subject matching the age of alternate patients on the list. For patients younger than 17, the control subject was within three months of the patients' age at test and recruited mainly from a file of potential volunteers; for the rest of the patients, the control subjects were first year Psychology students aged 18–23 who participated for course credit. All controls reported that they had no history of eye problems and all met our criteria on a visual-screening exam. Specifically, children seven years of age or older had a linear letter acuity at least 20/20 in each eye without optical correction on the Lighthouse Distance Visual Acuity Test chart, worse acuity with a +3 dioptre add (to rule out hypermetropia of greater than 3 dioptres), fusion at near on the Worth four dot test, and stereoacuity of at least 40″ on the Titmus test. The criteria for six-year-olds were the same except we included those with 20/25 acuity in each eye on the Lighthouse chart or 20/20 acuity on the Good–Lite Crowding cards. We replaced the youngest control subject (a six-year-old) because her performance with the two sizes of dot was far more inconsistent than that of any other normal or deprived subject. Specifically, the youngest control subject had thresholds that were 2.4 times higher for the 2-min dots than for the 10-min dots whereas the ratio for the remaining subjects never exceeded 1.3.

2.2. Apparatus and stimuli

The stimuli were generated by an Apple Macintosh G3 computer on a Sony Triniton Multiscan 200 GS monitor which subtended 35.3° × 26.5° at the viewing distance of 50 cm. Frame rate was 75 Hz and screen resolution was 1024 × 768 pixels. Each stimulus was centred on the monitor and had a diameter of 13.6°. The stimuli were composed of white dots (mean luminance = 81.6 cd/m^2) on a gray background (mean luminance = 35.8 cd/m^2).

"Signal patterns" consisted of concentric Glass patterns, constructed in a manner similar to that described by Wilson and Wilkinson (1998). Briefly, pairs of dots were placed at random within the pattern, but the orientation of the pair was always tangent to a circle centred on the pattern. We constructed patterns with two dot sizes. One set of patterns was made up of square "dots" with 2′ sides, a density of 6%, and a separation of

Table 1
Clinical details of the patients

Patient (age/years)	Refraction[a]		Diagnosis/contact lenses (days)	Snellen acuity[a]	Eye(s) tested	Nystagmus[b]	Additional details
Bilateral congenital							
AaB (8.5)	OD OS	+19.50 +19.00	61/91	20/100 20/100	OD	Latent OU	Secondary membrane surgery at age 7 years
JS (6.4)	OD OS	+27.00 +30.00	61/92	20/80 20/80	OD	Manifest	Microcornea OU; Ocular muscle surgery OU at ages 1.5 and 3 years
JF (8.6)	OD OS	+14.50 +14.50	77/100	20/50 20/40	OS	Manifest	Microcornea OU; Ocular muscle surgery OU at ages 1.6 and 4.8 years
A1B (7.3)	OD OS	+21.00 +25.50	63/106	20/100 20/80	OS	Manifest	Microcornea OU; Secondary membrane surgery at ages 0.7 and 2.4 years
AnL (9.4)	OD OS	+14.75 +12.00	Birth/139	20/70 20/50	OS	Manifest	No other surgery or complications
IW (19.0)	OD OS	+11.75 +12.75	92/151 92/264	20/125 20/30	OD	Manifest	Strabismus surgery for LET/RET at age 6.0 years
AC (20.1)	OD OS	+11.00 +12.50	123/196 123/161	20/50 20/60	OS	Manifest	Secondary membrane surgery at age 9 months. Ocular muscle surgery OU at age 3.7 years
CP (18.2)	OD OS	+10.75 +12.00	143/187	20/80 20/25	OS	Latent OU	Strabismus surgery for LET at age 1.8 years
Unilateral congenital							
BM (7.0)	OD OS	+24.50 Plano	7/43	20/100 20/25	OU	Latent OD	Strabismus surgery for RET at age 1.1 years. Patching: 3.7 h/day
RB (7.0)	OD OS	+22.50 +4.00	21/55	20/50 20/25	OU	None	Ocular muscle surgery OU at age 7.2 years. Patching: 2.8 h/day
EH (8.5)	OD OS	+8.50 −2.50	30/56	20/30 20/30	OD	None	No other surgery or complications. Patching: 5.0 h/day
CK (7.1)	OD OS	−2.00 +13.50	15/67	20/70 20/80	OS	Intermittent manifest Latent OU	Strabismus surgery for LET at age 1.2 years. Patching: 4.7 h/day
CPM (9.4)	OD OS	+17.00 Plano	83/116	20/200 20/20	OD	None	Strabismus surgery for RET at ages 0.7 and 5.2 years. Patching: 4.7 h/day
NF (17.3)	OD OS	+11.50 −1.50	90/124	20/40 20/20	OU	Latent OU	Microcornea OD; Secondary membrane surgery at age 0.3 years. Strabismus surgery for RET at age 2.2 years. Patching 4.6 h/day
VC (6.0)	OD OS	+2.00 +26.75	131/163	20/20 20/100	OU	None	Interocular lens inserted at age 4.2 years. Patching: 3.3 h/day
RR (8.7)	OD OS	+15.00 Plano	155/183	20/160 20/20	OU	None	No other surgery or complications. Patching: 2.4 h/day
AT (14.1)	OD OS	Plano +19.50	152/245	20/20 20/160	OU	Intermittent latent OU	Strabismus surgery for LET at ages 1.0 and 1.7 years. Patching: 3.5 h/day
AM (20.5)	OD OS	+1.50 −4.25	88/313	20/800 20/20	OU	Occasional latent OD	Secondary membrane surgery at 0.7 years. Strabismus surgery for RET at age 1.3 years. Patching: 1.6 h/day

Patients are in order of increased deprivation.
OD = right eye; OS = left eye; OU = each eye; RET = right esotropia; LET = left esotropia.
[a] Measurement closest to the time of the test. Refractions and spherical equivalents.
[b] History of nystagmus since first optical corrections.

19′ between members of a pair. Under these conditions, the mean dot spacing overall was 9′, less than half the spacing between members of a pair. This arrangement ensured that the perception of global structure was not based on local cues of dot spacing. The dots were identical in physical size to those used previously by Wilson and Wilkinson (Wilson & Wilkinson, 1998; Wilson et al., 1997) to test normal adults but subtended

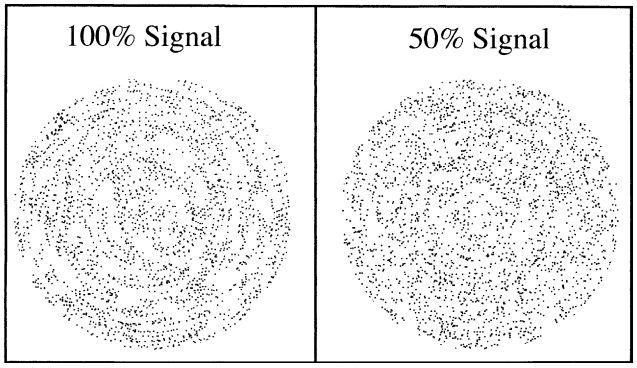

Fig. 1. Examples of concentric Glass patterns with 100% signal (left panel) and 50% signal (right panel). The stimuli were composed of either 2′ dots with a density of 6% or 10′ dots with a density of 1%. For clarity, the patterns are illustrated with black dots on a white ground but the actual stimuli contained white dots on a grey ground.

twice the visual angle because they were viewed from half the distance. To compensate further for reduced acuity in the patients (see Table 1), we constructed a second set of patterns with dots five times larger than those in the first set. The square "dots" in this set had 10′ sides, a density of 1%, and a separation of 93′ between members of a pair. Mean dot spacing was 44′ and again, was less than half the spacing between members of a pair. To measure thresholds for detecting global structure in Glass patterns, the global form was degraded by replacing a percentage of the signal dot pairs with an equal number of randomly spaced noise dots that were the same size and shape as the signal dots. Fig. 1 illustrates examples of Glass patterns with 100% and 50% signal. Signal patterns were compared to "noise patterns" that were created by replacing all of the signal dots with noise dots. Thus, regardless of the percentage of signal dots, each stimulus contained the same total number of dots and the same mean dot density.

2.3. Procedure

The procedures were explained and written consent was obtained from the parents of the children and from the adults who participated. Subjects between 7 and 16 years of age also gave informed assent. 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.

Participants were tested monocularly in a room illuminated only by the computer monitor and were adapted to the lighting conditions prior to the test. We tested one randomly selected eye of patients treated for bilateral congenital cataract (see Table 1), the deprived eye of patients treated for unilateral congenital cataract, and one randomly selected eye of normal subjects. The eye not being tested was patched with 3M Micropore™ tape and the deprived eye of each patient was corrected optically for the viewing distance. Participants were seated 50 cm from the computer screen with their chin in a chin rest. Parents of children sat in the testing room out of their child's sight and were asked to remain silent during testing.

The experimenter began by instructing the subject to fixate the centre of the monitor and saying: "You are going to see a circle filled with dots and it is your job to tell me if the dots look all messy (experimenter moves his/her finger in random directions in front of the computer screen) or if you see swirls (experimenter draws imaginary circles in front of the computer screen)." The experimenter pressed a key to begin a trial and each stimulus remained on the screen for 1500 ms. The experimenter watched the subject to ensure that he/she maintained central fixation, provided regular reminders to do so, and began a trial only when the subject was looking in the middle of the screen. The procedure began with demonstration trials and a practice run.

2.3.1. Demonstration trials

The demonstration consisted of ten trials with concentric patterns interspersed with six trials with noise patterns. Across the ten signal trials, the percent signal was reduced systematically from 60% (the first four signal trials), to 40% (the next two signal trials), to 25% and then 10% (one trial each), with the final two signal trials returning to 60% and 40% signal, respectively. During the first four demonstration trials, the experimenter taught the subject to discriminate signal from noise trials by providing the correct answer followed by a verbal explanation and, for signal trials, tracing the pattern with his/her finger. For the remaining trials, the subject gave verbal responses and received feedback. Subjects were reminded regularly to fixate the centre of the screen and to watch carefully because the game might be getting harder.

2.3.2. Practice run

For the practice run and the subsequent threshold measurements, we used a two-alternative temporal forced-choice procedure combined with the method-of-constant stimuli. One interval contained a stimulus with concentric structure and the other contained a stimulus with randomly positioned noise dots. Dot size was the same as that presented during demonstration trials. The two intervals in a trial each lasted 1500 ms, began with a brief "beep" sound, and had an inter-stimulus interval of 500 ms. To help keep the subject alert, the experimenter often said: "Are the swirls in the first or in the second interval?" with the mention of each interval timed to the presentation of that interval. After each trial, the experimenter entered the subject's response on a keypad by pressing "1" if the subject chose interval 1, or "2" if the subject chose interval 2. A practice run

consisted of 16 trials during which four signal values (60, 40, 25, and 10%) were each presented four times in a random order. Across trials, the signal appeared randomly in interval 1 or 2. The experimenter was aware of the stimulus presented during each interval and, if the subject began making mistakes on "easy" trials, provided feedback. All subjects seemed to understand the task by the end of the practice trials.

2.3.3. Test of thresholds

The procedure for measuring each threshold was identical to that for the practice run except (1) the four signal values were each presented 20 times in a random order and (2) the experimenter was unaware of the stimulus presented during each interval and provided encouragement but no feedback. The percentage of correct responses was plotted as a function of signal value and the data were fit by a Quick (1974) or Weibull (1951) function using a maximum likelihood procedure (Wilson & Wilkinson, 1998). Thresholds were defined as the percent signal necessary to obtain 75% correct responses.

Each subject completed two tests per included eye: one with the smaller dots and one with the larger dots. Half the subjects in each group first completed a test with the smaller dots and half, with the larger dots. The procedure was identical for each dot size except that demonstration trials were omitted after the first test.

2.4. Data analyses

To assess the effects of deprivation on sensitivity to global structure, we conducted a two-way mixed analysis of variance (ANOVA) with a between-subject variable of group with three levels (bilaterally deprived patients, unilaterally deprived patients, normal controls) and a within-subject variable of dot size with two levels (small versus larger dots). The between-subjects factor was further analyzed using Fisher's PLSD test (Howell, 1989).

3. Results

All patients could detect the global form when at least 65% of the dots were paired signal dots. Fig. 2 shows the minimum percentage of paired signal dots necessary to detect the global structure in Glass patterns for one deprived eye of bilaterally deprived patients, the deprived eye of unilaterally deprived patients, and one eye of comparably aged normal control subjects. There was a significant difference in performance amongst the three groups (main effect of group, $F_{2,25} = 8.41$, $p = 0.002$). As expected, thresholds were lower in normal control subjects ($M = 25.2\%$) than in either bilaterally ($p < 0.0001$) or unilaterally ($p = 0.02$) deprived patients. However,

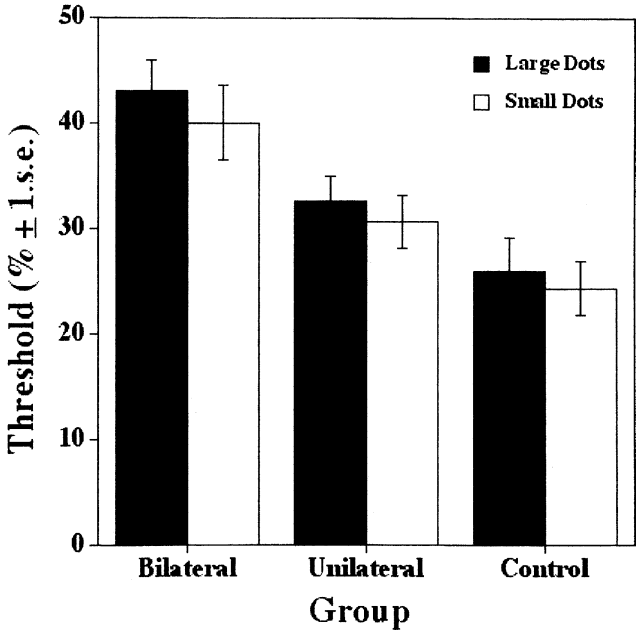

Fig. 2. Mean threshold (± 1 S.E.) representing the minimum percent signal necessary to perceive the global structure in Glass patterns accurately 75% of the time. Data are for one deprived eye of patients treated for bilateral congenital cataract, the deprived eye of patients treated for unilateral congenital cataract, and one eye of comparably aged normal controls. Black bars represent the results for the larger 10 dots and white bars represent the results for the smaller 2 dots.

thresholds in bilateral cases ($M = 41.6\%$) were significantly *worse* than in the deprived eye of unilateral cases ($M = 31.8\%$) ($p = 0.001$) despite the fact that the duration of deprivation ($t_{16} = 0.28$, $p > 0.70$, two-tailed) and log acuity at the time of the test ($t_{16} = 1.09$, $p > 0.20$, two-tailed) were comparable for the two groups of patients. For all three groups, thresholds were significantly higher for patterns with larger dots than for patterns with smaller dots (main effect of dot size, $F_{1,25} = 6.78$, $p = 0.01$), but the differences were small ($M = 2.2\%$) and were comparable across the three groups (nonsignificant interaction between group and pattern, $F_{2,25} = 0.246$, $p > 0.70$).

4. Discussion

Our findings indicate that the absence of patterned visual input to one or both eyes from birth prevents the normal development of sensitivity to global form in the treated eye(s). Moreover, our findings of greater losses after binocular deprivation than after monocular deprivation indicate that normal visual input to one eye from birth is enough to reduce the deleterious effects of deprivation on the deprived eye. This pattern of a better outcome in the deprived eyes after monocular than after binocular deprivation is similar to that reported for the perception of global motion (Ellemberg et al., 2002) but

very different than that reported for spatial and temporal vision (Birch et al., 1998; Bowering et al., 1993; Ellemberg et al., 2002, 2000, 1999; Lewis et al., 1995; Mioche & Perenin, 1986; Tytla et al., 1993, 1988).

Smaller deficits after monocular than after binocular deprivation cannot be explained by differences in acuity between the two groups. Acuity did not differ significantly between the two groups of patients and in fact, as would be expected from the literature, the mean acuity tended to be worse after monocular than after binocular deprivation. (Apparently, the amount of occlusion in our unilateral cases was sufficient to offset any significant differences in acuity between the two groups.) Moreover, although the differences were small, both groups of patients, like normal controls, did significantly better on patterns constructed from small dots than on patterns constructed from larger dots, perhaps because density was six times greater for the small dots than for the larger dots (6% versus 1%, respectively).

The deficits also cannot be attributed to associated disorders such as strabismus, microcornea, and/or shortened axial length. Although strabismus was present in most patients, the incidence and degree of strabismus were no different after binocular than after monocular deprivation and, within the binocularly deprived group, the global form deficits were no greater in the patients who had strabismus than in those who did not. For similar reasons, microcornea and shortened axial length also are unlikely to be responsible for the pattern of deficits. The incidence of these conditions was no different after binocular than after monocular deprivation, and the pattern of deficits was unrelated to their presence.

The incidence of manifest nystagmus (evident when both eyes are open) and of latent nystagmus (evident only when one eye is occluded) was greater after binocular than after monocular deprivation (see Table 1) and thus may have contributed to the larger deficits after binocular deprivation. However, about half the patients in the monocularly deprived group suffered from spontaneous nystagmus during development either because they had a manifest nystagmus (patient CK) or because they received occlusion therapy part of each day and had a latent nystagmus in the deprived eye. Yet their performance was no worse than that of the unilateral cases with no history of manifest or latent nystagmus. To assess the contribution of nystagmus to the deficits in the binocularly deprived group, we selected two patients from that group (CP and AaB) who had only a latent nystagmus and retested them under binocular and monocular viewing conditions. Neither of these patients had experienced nystagmus routinely during development. As shown in Fig. 3, their thresholds were just as poor when tested binocularly (no nystagmus condition) as when tested monocularly (latent nystagmus condition). Thus, it seems that the larger deficits after binocular deprivation compared to those after monocular deprivation result from early binocular pattern deprivation and not from associated conditions such as strabismus, microcornea, shortened axial length, or nystagmus.

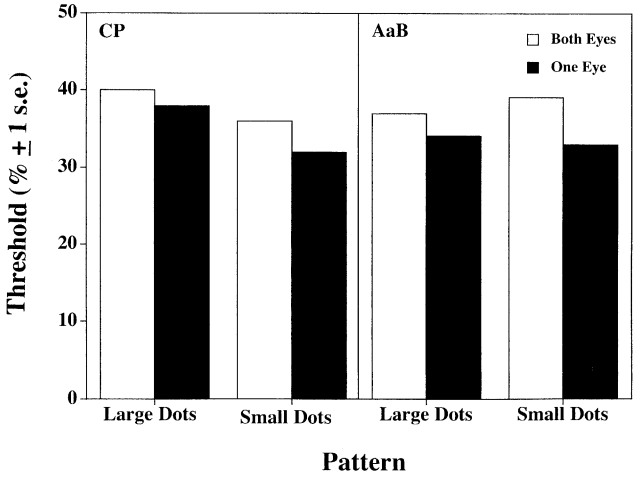

Fig. 3. Thresholds for the larger and smaller dots for two binocularly deprived patients with latent nystagmus (CP and AaB) tested with both eyes open (no nystagmus condition) and with only one eye open (latent nystagmus condition). Thresholds were equally poor under the two conditions.

Although no previous studies have measured thresholds for the detection of global form after early pattern deprivation, there have been assessments of shape discrimination. The only two such studies in humans come from our laboratory (Geldart, 2000; Maurer, Lewis, & Brent, 1989) and both, unlike the present study, found no deficit in the perception of overall shape after deprivation. Geldart (2000) tested only patients treated for bilateral congenital cataract with hierarchical shapes: a circle and a square formed from small broadband elements that were either congruent, or incongruent, with the overall shape of the pattern (e.g., a circle formed from small circles versus a circle formed from small squares). Patients performed normally on a task that required them to ignore the shape of the small elements and to indicate, for each pair of stimuli, whether the overall shape was the same or different. Maurer et al. (1989) tested the ability of visually deprived children to match exemplars to one of four test shapes (triangle, circle, cross, and "U"). Both binocularly and monocularly deprived patients performed normally on the task, even when the exemplars differed from the test shapes in size, luminance, contour, and/or the presence of masking lines and even when deprivation had lasted from birth up to 22 months of age. However, the tasks may not have been sufficiently sensitive to identify deficits. All of the patients in the present study could detect the global form with at least 65% signal dots and, had we not measured thresholds, we would have reached the erroneous conclusion that their ability to perceive global form in Glass patterns was normal.

As discussed in the Introduction, the perception of concentric structure in Glass patterns requires extrastriate ventral visual areas, likely including area V4v. Monocular deprivation may be less disruptive than binocular deprivation for aspects of vision involving area V4v because of converging input from striate and extrastriate pathways onto binocular V4 cells with large receptive fields (4–7 times larger than those in V1) (Desimone & Schein, 1987; Gattass et al., 1988). During early monocular deprivation, the initial development of extrastriate cells in the ventral pathway may be driven by input from the nondeprived eye and after treatment, many of those cells may respond to either eye. Input from the previously deprived eye could reach the extrastriate ventral pathway either via spared cells in the primary visual cortex or via extrageniculate pathways bypassing the primary visual cortex (Rodman, Albright, & Gross, 1990), which may play a more important role after early deprivation than they do after normal development (Azzopardi, Fallah, Gross, & Rodman, 1998; Zablocka, Zernicki, & Kosmal, 1976; Zablocka, Zernicki, & Kosmal, 1980). However, better outcomes after monocular than after binocular deprivation may not be generalizable to aspects of vision mediated by other parts of the ventral stream. Specifically, early monocular deprivation caused by a left congenital cataract results in marked deficits in the perception of facial identity based on configural cues, deficits that are evident even with binocular testing and that are at least as large as those after early binocular deprivation (Le Grand, Mondloch, Maurer, & Brent, 2001a,b).

Poorer sensitivity in a deprived eye after binocular than after monocular deprivation implies that extrastriate areas involved in the perception of global form are not affected by competitive interactions between the eyes for cortical connections of the type well-documented for the primary visual cortex. This conclusion is the same as that first reached by Ellemberg et al. (2002) for area MT based on poorer sensitivity to the perception of the direction of global motion after early binocular deprivation than after early monocular deprivation. Thus, the present results provide additional evidence for the existence of another mechanism by which the eyes can interact, namely complementary interactions. These complementary interactions appear to operate in at least two extrastriate areas that integrate signals from V1, namely areas that integrate form signals in the ventral pathway and those that integrate motion signals in the dorsal pathway. In those extrastriate areas, they appear to replace the competitive interactions evident in primary visual cortex.

Although both the present study and the previous study on the perception of global motion (Ellemberg et al., 2002) found worse outcomes in the deprived eye after binocular than after monocular deprivation, we are aware of two differences in the pattern of results: the magnitude of the difference between binocular and monocular cases and the performance of the nondeprived eye. Specifically, after binocular deprivation, thresholds were only 1.6 times worse than normal for global form but 4.9 times worse than normal for global motion. The relatively small deficits in the perception of global form cannot be attributed to a ceiling effect because all patients had thresholds that were below 65%. Thus, these findings suggest that the deleterious effects of early pattern deprivation are greater for the parts of the dorsal pathway involved in sensitivity to global motion than for the parts of the ventral pathway involved in sensitivity to global form. This pattern is consistent with previous suggestions of greater vulnerability in the dorsal than in the ventral streams after neurological impairment caused by focal brain lesions or Williams' syndrome (Atkinson et al., 1999).

There are also differences between studies in the findings for the nondeprived eye of children treated for unilateral congenital cataract. For global motion, the reduction in sensitivity after monocular deprivation was comparable for the deprived and nondeprived eyes (about 1.6 times worse than normal; Ellemberg et al., 2002). That pattern could be explained by extrastriate cells that are binocular and insensitive to eye of origin. For global form, this explanation is inadequate. We have data on only seven nondeprived eyes from the unilateral cases but, even within this small sample, thresholds in the nondeprived eye (mean for small elements = 24.86%) were significantly better than those in the deprived eye (mean for small elements = 30.43%), regardless of element size (main effect of group, $F_{1,6} = 7.17$, $p < 0.05$). Although the means for the nondeprived eyes appear normal, we do not have enough cases to rule out the possibility of a small abnormality.

5. Conclusions

The results indicate that the development of the structures involved in the processing of global form is compromised by early pattern deprivation caused by congenital cataract. Our findings of a better outcome after monocular than after binocular deprivation in the processing of global form, together with a similar pattern of findings for the processing of global motion (Ellemberg et al., 2002), provide compelling evidence that the competitive interactions between a deprived and nondeprived eye evident in primary visual cortex cooccur with complementary interactions in at least some extrastriate areas involved in the spatial and temporal pooling of sub-unit responses. These complementary interactions enable a relative sparing of some visual functions after early monocular deprivation.

Acknowledgements

We thank The Hospital for Sick Children in Toronto for providing space for the tests of patients and the children and their families for participation. This research was supported by the Canadian Institutes of Health Research (grant MOP 36430) to DM and by the E.A. Baker award (MRC) to DE. Some of these data were presented at the annual meeting of the Association for Research in Vision and Ophthalmology, Ft. Lauderdale, May 2001.

References

Atkinson, J., Braddick, O., Lin, M. H., Curran, W., Guzzetta, A., & Cioni, G. (1999). Form and motion coherence: Is there a dorsal stream vulnerability in development? *Investigative Ophthalmology and Visual Science, 40*, S395.

Azzopardi, P., Fallah, M., Gross, C. G., & Rodman, H. R. (1998). Responses of neurons in visual areas MT and MST after lesions of striate cortex in macaque monkeys. *Society of Neuroscience Abstracts, 24*, 648.

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

Blakemore, C. (1998). The sensitive periods of the monkey visual cortex. In G. Lennerstrand, G. K. Noorden, & C. C. Campos (Eds.), *Strabismus and Amblyopia: Experimental basis for advances in clinical management* (pp. 219–234). New York: Plenum.

Blakemore, C. (1990). Maturation of mechanisms for efficient spatial vision. In C. Blakemore (Ed.), *Vision: Coding and efficiency* (pp. 254–266). Cambridge University Press: Cambridge.

Blakemore, C., Garey, L., & Vital-Durand, F. (1978). The physiological effects of monocular deprivation and their reversal in the monkey's cortex. *Journal of Physiology, 380*, 453–491.

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 and Visual Science, 34*, 3501–3509.

Braddick, O. J., O'Brien, J. M. D., Wattam-Bell, J., Atkinson, J., & Turner, R. (2000). Form and motion coherence activate independent, but not dorsal/ventral segregated, networks in the human brain. *Current Biology, 10*, 731–734.

Crawford, M. L. J. (1998). Electrophysiology of cortical neurons under different conditions of visual deprivation. In G. Lennerstrand, G. K. von Noorden, & C. C. Campos (Eds.), *Strabismus and Amblyopia: Experimental basis for advances in clinical management* (pp. 150–154). New York: Plenum.

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

Crawford, M. L. J., Pesch, T. W., van Noorden, G. K., Harwerth, R. S., & Smith, E. I. III (1991). Bilateral form deprivation in monkeys: Electrophysiologic and anatomic consequences. *Investigative Ophthalmology and Visual Science, 32*, 2328–2336.

Desimone, R., & Schein, S. J. (1987). Visual properties of neurons in area V4 of the macaque: Sensitivity to stimulus form. *Journal of Neurophysiology, 57*, 835–868.

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 infancy on the later development of spatial and temporal vision. *Vision Research, 40*, 3283–3295.

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

Elliott, T., Howarth, C. I., & Shadbolt, N. R. (1996). Axonal processes and neural and neural plasticity. I: Ocular dominance columns. *Cerebral Cortex, 6*, 781–788.

Gallant, J. L., Braun, J., & van Essen, D. C. (1993). Selectivity for polar, hyperbolic, and Cartesian gratings in macaque visual cortex. *Science, 259*, 100–103.

Gallant, J. L., Connor, C. E., Rakshit, S., Lewis, J. W., & van Essen, D. C. (1996). Neural responses to polar, hyperbolic, and Cartesian gratings in area V4 of the macaque monkey. *Journal of Neurophysiology, 76*, 2718–2739.

Gallant, J. L., Shoup, R. E., & Mazer, J. A. (2000). A human extrastriate area functionally homologous to Macaque V4. *Neuron, 27*, 227–235.

Gattass, R., Sousa, A. P. B., & Gross, C. G. (1988). Visuotopic organization and extent of V3 and V4 of the macaque. *Journal of Neuroscience, 8*, 1831–1845.

Geldart, S. (2000). Experiential influences on the development of face perception. Ph.D. Thesis, McMaster University, Hamilton, Canada.

Glass, L. (1969). Moiré effect from random dots. *Nature, 223*, 578–580.

Glass, L., & Switkes, E. (1976). Pattern recognition in humans. *Perception, 5*, 67–72.

Harwerth, R. S., Smith, E. L., Boltz, M. L. J., Crawford, M. L. J., & van Noorden, G. K. (1983a). Behavioral studies on the effect of abnormal early visual experience in monkeys: Spatial modulation sensitivity. *Vision Research, 23*, 1501–1510.

Harwerth, R. S., Smith, E. L., Boltz, M. L. J., Crawford, M. L. J., & von Noorden, G. K. (1983b). Behavioral studies on the effect of abnormal early visual experience in monkeys: Temporal modulation sensitivity. *Vision Research, 23*, 1511–1517.

Harwerth, R. S., Smith, E. L., Crawford, M. L. J., & von Noorden, G. K. (1989). The effects of reverse monocular deprivation in monkeys. I. Psychophysical experiments. *Experimental Brain Research, 74*, 327–337.

Harwerth, R. S., Smith, E. L., Paul, A. D., Crawford, M. L. J., & von Noorden, G. K. (1991). Functional effects of bilateral form deprivation in monkeys. *Investigative Ophthalmology and Visual Science, 32*, 2311–2327.

Howell, D. C. (1989). *Fundamental statistics for the behavioral sciences* (2nd ed.). Boston, MA: PWS-Kent, pp. 236–240.

Hubel, D. H., & Wiesel, T. N. (1970). The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *Journal of Physiology, 206*, 419–436.

Hubel, D. H., Wiesel, T. N., & Le Vay, S. (1977). Plasticity of ocular dominance columns in monkey striate cortex. *Philosophical Transactions of the Royal Society of London [B], 278*, 377–409.

Kobatake, E., & Tanaka, K. (1994). Neuronal selectivities to complex object features in the ventral visual pathway of the macaque cerebral cortex. *Journal of Neurophysiology, 71*, 856–867.

Le Grand, R., Mondloch, C. J., Maurer, D., & Brent, H. P. (2001a). Early visual experience and face processing. *Nature, 410*, 890, Correction: 2001, 412, p. 786.

Le Grand, R., Mondloch, C. J., Maurer, D., & Brent, H. P. (2001b). Early monocular deprivation affects the development of face processing for both eyes. Paper presented at the biennial meeting of the Society for Research in Child Development, Minneapolis, MN.

Le Vay, S., Wiesel, T. N., & Hubel, D. H. (1980). The development of ocular dominance columns in normal and visually deprived monkeys. *Journal of Comparative Neurology, 191*, 1–51.

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

Maunsell, J. H. R., & Newsome, W. T. (1987). Visual processing in monkey extrastriate cortex. *Annual Review of Neuroscience, 10*, 363–401.

Maurer, M. L. J., & 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.

Maurer, M. L. J., & 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, M. L. J., Lewis, T. L., & Brent, H. P. (1989). The effects of deprivation on human visual development: Studies of children treated for cataracts. In F. J. Morrison, C. E. Lord, & D. P. Keating (Eds.), *Applied developmental psychology, Vol. 3, Psychological development in infancy* (pp. 139–227). San Diego, CA: Aademic Press.

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

Pasupathy, A., & Connor, C. E. (1999). Responses to contour features in macaque area V4. *Journal of Neurophysiology, 82*, 2490–2502.

Quick, R. F. (1974). A vector-magnitude model of contrast detection. *Kybernetik, 16*, 1299–1302.

Rodman, H. R., Albright, T. D., & Gross, C. G. J. (1990). Afferent basis of visual response properties in area MT of the macaque. II. Effects of striate superior colliculus removal. *Journal of Neuroscience, 10*, 1154–1164.

Swindale, N. V., Vital-Durand, F., & Blakemore, C. (1981). Recovery from monocular deprivation in the monkey. III. Reversal of anatomical effects in the visual cortex. *Proceedings of the Royal Society of London [B], 213*, 435–450.

Tytla, M. E., Lewis, T. L., Maurer, D., & Brent, H. P. (1993). Stereopsis after congenital cataract deprivation in the monkey. III. Reversal of anatomical effects in the visual cortex. *Investigative Ophthalmology and 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.

Weibull, W. A. (1951). A statistical distribution function of wide applicability. *Journal of Applied Mechanics, 18*, 292–297.

Wiesel, T. N., & Hubel, D. H. (1965). Comparison of the effects of unilateral and bilateral eye closure on cortical unit responses in kittens. *Journal of Neurophysiology, 28*, 1029–1040.

Wilkinson, F., James, T. W., Wilson, H. R., Gati, J. S., Menon, R. S., & Goodale, M. A. (2000). An fMRI study of the selective activation of human extrastriate form vision areas by radial and concentric gratings. *Current Biology, 10*, 1455–1458.

Wilson, H.R. (1999). Non-fourier cortical processes. In Ulinski et al., (Eds.), Cerebral cortex, *Vol. 13* (pp. 445–477). New York: Plenum Publishers.

Wilson, H. R., & Wilkinson, F. (1998). Detection of global structure in Glass patterns. Implications for form vision. *Vision Research, 38*, 2933–2947.

Wilson, H. R., Wilkinson, F., & Asaad, W. (1997). Rapid communication: Concentric orientation summation in human form vision. *Vision Research, 37*, 2325–2330.

Zablocka, T., Zernicki, B., & Kosmal, A. (1976). Visual cortex role in object discrimination in cats deprived of pattern vision form birth. *Acta Neurobiologiae Experimentalis, 36*, 157–168.

Zablocka, T., Zernicki, B., & Kosmal, A. (1980). Loss of object discrimination after ablation of the superior colliculus-pretectum in binocularly deprived cats. *Behavioral Brain Research, 1*, 521–531.