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Better perception of global motion after monocular than after binocular deprivation

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Abstract

We used random-dot kinematograms to compare the effects of early monocular versus early binocular deprivation on the development of the perception of the direction of global motion. Patients had been visually deprived by a cataract in one or both eyes from birth or later after a history of normal visual experience. The discrimination of direction of global motion was significantly impaired after early visual deprivation. Surprisingly, impairments were significantly worse after early binocular deprivation than after early monocular deprivation, and the sensitive period was very short. The unexpectedly good results after monocular deprivation suggest that the higher centers involved in the integration of global motion profit from input to the nondeprived eye. These findings suggest that beyond the primary visual cortex, competitive interactions between the eyes can give way to collaborative interactions that enable a relative sparing of some visual functions after monocular deprivation. © 2002 Published by Elsevier Science Ltd.

Keywords: Visual deprivation; Unilateral congenital cataract; Bilateral congenital cataract; Development; Global motion

1. Introduction

In every species studied, early visual deprivation prevents the development of normal visual function. Humans deprived of normal visual input from birth by dense central cataracts in one or both eyes later show losses in acuity, spatial and temporal contrast sensitivity, stereovision, and sensitivity in the peripheral visual field, with worse outcomes after early monocular deprivation than after early binocular deprivation, unless monocular deprivation was followed by extensive occlusion of the nondeprived eye (Mioche & Perenin, 1986; Tytla, Maurer, Lewis, & Brent, 1988, 1993; Lewis, Maurer, & Brent, 1995; Bowering, Maurer, Lewis, & Brent, 1993; Birch, Stager, Leffler, & Weakley, 1998; Ellemberg, Lewis, Maurer, Liu, & Brent, 1999; Ellemberg, Lewis, Maurer, & Brent, 2000). Similarly, in monkeys, early monocular deprivation causes larger reductions than early binocular deprivation in both spatial and temporal vision (Harwerth, Smith, Boltz, Crawford, & von Noorden, 1983a,b; Harwerth, Smith, Paul, Crawford, & von Noorden, 1991).

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, but also by uneven competition for cortical connections between the deprived and nondeprived eyes (Crawford, de Faber, Harwerth, Smith, & von Noorden, 1989; Maurer & Lewis, 1993, 2001; Elliott, Howarth, & Shadbolt, 1996). This explanation is supported by anatomical studies of the monkey's primary visual cortex that show shrunken and fragmented ocular dominance columns for the deprived eye after monocular lid suture and nearly normal ocular dominance columns for both eyes after binocular lid suture (LeVay, Wiesel, & Hubel, 1980; Crawford, Pesch, von Noorden, Harwerth, & Smith, 1991). However, no studies have compared the

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effects of early monocular versus binocular deprivation on aspects of vision that require cortical processing beyond the primary visual cortex.

In the present study, we measured the effects of early monocular and early binocular deprivation on the development of sensitivity to the direction of global motion, an aspect of vision that requires extrastriate regions of the visual cortex including the middle temporal (MT) extrastriate cortex (Maunsell & Newsome, 1987; Newsome & Pare, 1988). Anatomical and physiological evidence suggest that the pathway subserving motion commences in the primary visual cortex (V1), continues through area MT (Maunsell & Newsome, 1987) and projects dorsally to the medial superior temporal and ventral intraparietal areas (Ungerleider & Desimone, 1986). Computational models of motion perception (Wilson, 1999), supported by psychophysical (Williams & Sekuler, 1984; Smith, Snowden, & Milne, 1994), physiological (Newsome & Pare, 1988), anatomical (Born & Tootell, 1992; Scase, Horsfield, Wilcock, & Karwatowski, 1998), and neuropsychological (Barton, Sharpe, & Raymond, 1995; Vaina, Makris, Kennedy, & Cowey, 1998) data indicate that cells in the primary visual cortex signal the direction of motion in local regions of the visual field and that cells in area MT integrate those signals over both space and time to give rise to the perception of global motion. The majority of neurons in area MT have large receptive fields (Maunsell & Van Essen, 1983a, 1987), and are selective to the direction and speed of motion (Maunsell & Van Essen, 1983b; Albright, 1984), features necessary to extract a global motion signal from local motion cues. Further, lesions to area MT cause losses in the perception of global motion, as measured with random-dot kinematograms (RDKs), while sparing stereovision, acuity, contrast sensitivity, and color vision (Newsome & Pare, 1988; Barton et al., 1995; Schiller, 1996).

We measured sensitivity to global motion with RDKs, which are ideal for testing the perception of global motion (Newsome & Pare, 1988). The display consists of randomly positioned dots moving in random directions except for a percentage of signal dots moving in the same direction. These stimuli ensure that any percept of motion arises from the integration of local motion cues. Because some of our patients had nystagmus (see Table 1), we chose stimulus conditions for which nystagmus produces no deficits in motion perception, namely, vertical motion with a speed greater than $6^\circ\ s^{-1}$ (Shallow-Hoffman, Bronstein, Acheson, Morland, & Gresty, 1998). For comparison, we measured grating acuity, an aspect of spatial vision mediated by the primary visual cortex. To evaluate the effects of early visual deprivation, we measured sensitivity to global motion and grating acuity in 22 patients treated for a congenital cataract in one or both eyes (the congenital group). To delineate the sensitive period for normal development, we included 15 patients who developed dense central cataracts in one or both eyes postnatally after a history of normal visual input (the developmental group). 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 including all information about motion. 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, patients were at least 6 years old and those in the congenital group had at least 5 years of patterned visual input after deprivation ended. Results from patients were compared to those of normal controls tested under the same conditions.

2. Methods

2.1. Subjects

Subjects were eight patients treated for bilateral congenital cataract (duration of deprivation = 3–8 months; mean = 5.0 months), 14 patients treated for unilateral congenital cataract who had patched their nondeprived eye 2–7 h/day throughout early childhood (duration of deprivation = 1–10 months; mean = 5.0 months), six patients who developed dense cataracts in both eyes between 8 and 57 months of age (mean = 24 months) and endured deprivation lasting 1–6 months (mean = 3.0 months), and nine patients who developed a cataract in one eye between 4 and 177 months of age (mean = 41 months) and endured deprivation lasting 1–5 months (mean = 2.4 months). All patients were at least 6 years old at the time of the tests. Clinical details for each patient are presented in Tables 1 and 2. To be included in the study, the patients had to meet all of the following criteria: dense central cataracts in one or both eyes; no other abnormalities in the ocular media or the retina, including no evidence of persistent hyperplastic primary vitreous, and no ocular disease such as glaucoma. Patients who did not wear their optical correction regularly after treatment (at least 75% of the time) were also excluded. Patients with common associated abnormalities such as strabismus, nystagmus, or microcornea were included.

Patients were included in the unilateral or bilateral congenital groups if they had been diagnosed with a dense central cataract in one or both eyes on the *first* eye exam and by 6 months of age. We assumed that these patients had been deprived from birth because it would be unusual to have dense cataracts develop rapidly between birth and 6 months. Consequently, we defined the duration of deprivation for the congenital groups as the period extending from birth until the age of first optical correction after surgery to remove the cataract (i.e., the

Table 1
Clinical details of patients in the congenital groups (patients are in order of increased deprivation)

Patient (Age/ Years)	Refraction[a]		Diagnosis/contact lenses (days)	Snellen acuity[a]	Nystagmus[b]	Motion thresholds (%)	Grating acuity (min)	Additional details
Bilateral congenital								
AaB (7.5)	OD	+14.75	61/91	20/100	Latent	37	2.3	Secondary membrane surgery at age 7 years
	OS	+16.00		20/100	OU	28	2.5	
JF (6.7)	OD	+22.00	77/100	20/35	Manifest	55	3.3	Microcornea OU; Ocular muscle surgery OU at ages 1.6 and 4.8 years
	OS	+20.00		20/200		30	3.2	
AA (6.7)	OD	+15.50	104/134	20/100	Manifest	26	4.0	Microcornea OU; Strabismus surgery for RET at age 3 years
	OS	+13.50		20/70		19	6.0	
KC (5.0)	OD	+31.00	100/144	20/125	Manifest	63	2.5	Microcornea OU; Strabismus surgery for LET at age 2 years
	OS	+32.00		20/140		75	2.6	
AgL (11.7)	OD	+16.50	61/165	20/50	Latent	48	1.8	No other surgery or complications
	OS	+15.50		20/100	OS	41	2.3	
CP (17.2)	OD	+13.00	143/187	20/50	Latent	28	5.3	Strabismus surgery for LET at age 1.8 years
	OS	+14.50		20/30	OU	27	4.5	
VC (5.3)	OD	+19.00	158/202	20/100	Manifest	43	2.1	Microcornea OU; Strabismus surgery for LET at age 3 years
	OS	+20.00		20/200		64	3.0	
IW (18)	OD	+11.75	92/151	20/70	Manifest	27	6.7	Strabismus surgery for LET/RET at age 6.0 years
	OS	+12.75	92/264	20/50		30	5.2	
Unilateral congenital								
HC (4.1)	OS	+14.50	9/39	20/70	Latent	19	2.4	Strabismus surgery for LET at age 0.7 years. Patching: 5.5 h/day
	OD	plano		20/30	OU	20	1.4	
BM (6.0)	OD	+23.00	7/43	20/40	Latent	19	1.7	Strabismus surgery for LET at age 1.1 years. Patching: 3.7 h/day
	OS	plano		20/20	OD	17	1.2	
MC (5.7)	OS	+14.00	32/55	20/50	Latent	16	2.0	Strabismus surgery for LET at age 1.6 years. Patching: 7.1 h/day
	OD	−1.25		20/25	OU	22	1.1	
EH (6.6)	OD	+10.75	30/56	20/30	None	7	2.3	No other surgery or complications. Patching: 5.0 h/day
	OS	−2.50		20/25		7	1.3	
CK (6.1)	OS	+13.50	15/67	20/60	Manifest	18	2.7	Strabismus surgery for LET at age 1.2 years. Patching: 4.7 h/day
	OD	plano		20/30		15	1.5	
CPM (7.4)	OD	+17.00	83/116	20/200	None	13	2.9	Strabismus surgery for RET at ages 0.7 and 5.2 years. Patching: 4.7 h/day
	OS	plano		20/25		16	1.4	
NF (16.4)	OD	+11.50	90/124	20/50	Latent	10	2.1	Strabismus surgery for RET at age 2.2 years. Patching: 4.6 h/day
	OS	−1.50		20/20	OU	13	1.3	
JW (21.1)	OS	+11.50	136/150	20/30	Manifest	6	1.4	Strabismus surgery for LET at age 13.1 years. Patching: 3.0 h/day
	OD	plano		20/20		7	0.90	
SD (7.9)	OS	+15.50	127/176	20/60	None	15	1.7	Strabismus surgery for LXT at age 3.6 years.
	OD	plano		20/20		18	1.3	
RR (5.8)	OD	+14.25	155/183	20/200	None	14	2.2	No other surgery or complications. Patching: 2.4 h/day
	OS	plano		20/25		18	1.2	

(*continued on next page*)

Table 1 (*continued*)

Patient (Age/ Years)	Refraction[a]		Diagnosis/contact lenses (days)	Snellen acuity[a]	Nystagmus[b]	Motion thresholds (%)	Grating acuity (min)	Additional details
AT (12.1)	OS	+17.50	152/245	20/200	Latent	19	1.7	Strabismus surgery for LET at age 1.0 and 1.7 years. Patching: 3.5 h/day
	OD	plano		20/20	OU	18	1.3	
TA (16.7)	OD	+17.00	Birth/247	1/200	Latent	11	31.5	Strabismus surgery for RET at 5.0 years & RXT at 13.5 years. Patching: 3.3 h/day
	OS	−1.00		20/30	OS	14	1.4	
AF (17.8)	OD	+19.25	120/250	20/800	None	13	10.3	Strabismus surgery for RET at age 6.2 years. Patching: 2.6 h/day
	OS	plano		20/25		16	1.5	
AM (19.6)	OD	+11.00	88/313	20/400	Latent	14	42.9	Strabismus surgery for RET at age 1.3 years. Patching: 1.6 h/day
	OS	plano		20/20	OD	17	1.9?-	

OD = right eye; OS = left eye;

first time the infant received focused visual input onto the retina). However, we cannot be certain that all of these patients had dense central cataracts at birth and any errors in classification would add noise to the data. We will consider the implications of such errors in Section 4.

Patients were included in the developmental group if they developed a dense central cataract in one or both eyes with evidence that there was a period with no ocular abnormalities before the diagnosis of cataract. We defined the duration of deprivation for the developmental group as the period extending from the onset of a dense central cataract until the age of the first optical correction after surgery to remove the cataract.

In patients treated for unilateral congenital cataract, occlusion therapy was initiated shortly after the time of the first optical correction and continued through at least 5 years of age. Depending on the ophthalmologist, patients were instructed to patch the nondeprived eye for times ranging from 4 waking h/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 7.1 waking hours per day (see Lewis et al., 1995 for details of these calculations).

2.2. Normal controls

For global motion, the results from patients were compared to those from twelve 6-year-olds (±2 months), and 12 adults (mean age = 19.8 years, range = 18–26 years). Thresholds in the two groups of normal subjects did not differ significantly ($p > 0.10$). None of the control participants had a history of eye problems, and all met our criteria on a visual screening exam. Specifically, all had Snellen acuity of at least 20/20 in each eye without optical correction, worse Snellen 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. For grating acuity, the results from patients were compared to those from 14 comparably aged control subjects tested under the same conditions. All controls had no history of eye problems and all met our criteria on a visual screening exam.

2.3. Apparatus and stimuli

For global motion, the stimuli were generated on Apple Macintosh LC475 computer and presented on a two-page display/21gs Radius monochrome monitor, 29° high by 37° wide, with a vertical refresh rate of 75 Hz. The stimuli consisted of limited life-time RDKs. Each frame contained 300 dots, giving a density of 0.75 dots per degree. To compensate for reduced acuity in the patients (see Tables 1 and 2), the black dots were large (30′), and they were presented against a white background subtending 20 by 20 degrees of visual angle. Each dot had a mean luminance of 14 cd m^{-2} whilst the background had a mean luminance of 116 cd m^{-2}. The Michelson contrast between the dots and their background was 78%. Each RDK was composed of 20 frames, each lasting one refresh rate (i.e., 1/75 Hz or 0.013 s) with a resultant speed of 18° s^{-1}. The direction of motion of each dot changed for every frame, after which it was replaced randomly by another direction of motion. On any given trial, a percentage of the dots moved either upwards or downwards, whilst the remaining dots moved in random directions.

Grating acuity was measured with vertical sinusoidal gratings generated on a green phosphor Tektronix 5130 oscilloscope CRT display 13° wide by 10° high. The

Table 2
Clinical details of patients in the developmental groups (patients are in order of age of onset of deprivation)

Patient (Age/Years)	Refraction[a]		Diagnosis/contact lenses (months)	Snellen acuity[a]	Nystagmus[b]	Motion threshold (%)	Grating acuity (min)	Additional details
Bilateral developmental								
VO (7.7)	OD	+13.50	7.7/9.2	20/40	None	7	2.6	No other surgery or
	OS	+17.50		20/40		4	2.0	complications
CW (12.9)	OD	+20.00	12.5/13.5	20/20	None	12	1.4	No other surgery or
	OS	+18.50	9.2/11.7	20/200		11	1.6	complications
ES (7.8)	OD	+18.75	14.0/14.0	20/30	None	9	1.6	No other surgery or
	OS	+18.75		20/40		8	1.5	complications
EH (17.1)	OD	+17.00	20.0/21.4	20/32	None	8	1.5	No other surgery or
	OS	+18.00		20/50		9	1.6	complications
CP (22)	OD	+11.50	39.4/44.4	20/20	None	9	1.4	No other surgery or
	OS	+10.50		20/30		7	1.4	complications
CB (16.2)	OD	+18.50	57/61	20/40	None	10.5	1.2	No other surgery or
	OS	+17.25		20/30		10	0.9	complications
Unilateral developmental								
LK (12.5)	OS	+25.00	3.6/4.6	20/60	None	11.5	2.6	Strabismus surgery for
	OD			20/20		14	1.1	LET at age 2.4 years
AB (11.7)	OD	+14.00	6.7/7.8	20/125	None	10	2.4	Strabismus surgery for
	OS			20/20		10	1.0	RET at age 3.9 years
CY (9.6)	OD	+14.00	7.1/8.2	20/200	Latent	11	2.6	Strabismus surgery for
	OS			20/20	OD	9	1.2	RET at age 1.0 years
AC (6)	OD	+13.00	7.1/8.3	20/30	None	12	1.6	No other surgery or
	OS					10	0.9	complications
JL (9.3)	OS	+18.50	16.0/21.0	20/40	None	14	1.3	No other surgery or
	OD			20/20		8	1.0	complications
MC (9.1)	OS	+16.00	40.8/42.6	20/50	None	9	1.0	No other surgery or
	OD			20/20		9	1.1	complications
SL (9.2)	OS	+17.50	44.4/46	20/40	None	10.5	1.2	No other surgery or
	OD			20/20		11	0.9	complications
MB (15)	OD	+12.00	11 years of age	20/30	None	8	0.9	No other surgery or
	OS					9	1.0	complications
WC (17.8)	OS	+13.00	14 years of age	20/20	None	7	1.0	No other surgery or
	OD					9	1.0?-	complications
							OD = right eye; OS = left eye;	

gratings had a contrast of 52%. The space- and time-average luminances of the test stimuli were 9 cd m^{-2}. Gamma correction was verified by using a Minolta LS-100 photometer. All stimuli were within the range in which contrast was linearly related to the *Z*-axis voltage (i.e., 52%).

2.4. Procedures

The nature of the studies was explained to the subjects and, for younger children, also to their parents. For participants younger than 17 years of age, informed consent was obtained from a parent and informed assent from the participant. Older subjects gave informed consent for themselves. 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.

Each participant was tested monocularly while viewing the stimuli through a 3.5 mm artificial pupil designed to minimize the effects of differences among patients in the shape and size of their pupils. Patients received the appropriate optical correction over their usual contact lens to focus visual input at the testing distance of 50 cm for global motion and 228 cm for grating acuity. Half of the participants in each group

were tested with the left eye first, whilst the remaining half were tested with the right eye first. The eye not being tested was patched with 3M Micropore™ tape.

For tests of sensitivity to the direction of global motion, participants were instructed to fixate a cross at the centre of the screen which disappeared during the presentation of each RDK, and were asked to judge whether the global motion of the dots was upward or downward. Specifically, both adults and children were told: "There will be dots moving either up or down on the screen. At first, all the dots will be moving together but then some of them will start moving in many different directions. Your job is to tell me whether the dots that are moving together are going up or down the screen." Subjects responded verbally and/or by pointing. The experimenter entered the responses into the computer by pressing a key on the keyboard. Before the test, each subject had a demonstration run with feedback, during which they viewed the display with both eyes, and one practice run with each eye. Subsequently, one threshold was measured for each eye. No feedback was given during the test but children were praised periodically 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.

We used a 2-down, 1-up staircase procedure (Levitt, 1971) to measure motion coherence thresholds (i.e., the minimum percentage of dots that had to be moving in the same direction for the subject to detect the overall direction of motion with 71% accuracy). The initial coherence level was 100% and the initial step size was one octave. The step size decreased to a half octave after the first reversal and to a quarter octave at all subsequent reversals. There were eight reversals after the first quarter octave step. The coherence threshold was taken as the mean of the last six reversals.

Grating acuity was measured using the method of limits. Subjects were asked to indicate when the grating first disappeared as spatial frequency was increased from suprathreshold values, or just reappeared as spatial frequency was decreased from subthreshold values. Three ascending and three descending thresholds were measured. Specifically, both adults and children were asked to: "Say 'there' as soon as the stripes *appear* and say 'gone' as soon as the stripes *disappear.*" No feedback was given during the test but children were praised periodically for their good efforts (e.g., "that's great; you're doing a good job"). A practice run was given before the test.

2.5. Data analysis

For global motion, the results from patients were compared to the combined data of the 6-year-olds and adults with normal vision, whose thresholds did not differ significantly ($p > 0.10$). We used a one-way analysis of variance (ANOVA) to compare coherence thresholds for one eye of each of five groups of subjects: the worse eye (as determined from the closest Snellen acuity to the time of test) of patients treated for bilateral congenital cataract, the deprived eye of patients treated for unilateral congenital cataract, the worse eye (as determined from clinical history of alignment and Snellen acuity) of patients treated for bilateral developmental cataract, the deprived eye of patients treated for unilateral congenital cataract, and a randomly selected eye from each of the 24 control subjects. Bilateral cases with equal alignment and acuity histories for the two eyes had one eye assigned randomly to each category. To determine the source of the main effect, we used pairwise post-hoc comparisons. We used *t*-tests with Bonferroni corrections to compare results from the nondeprived eye of unilateral congenital cases to their deprived eye and to the other eye of normal controls, and to compare the results from the deprived eye of unilateral cases and the better eye of bilateral congenital cases.

For grating acuity, the data were analyzed in the same manner as for global motion except that the data for the visually normal group came from 14 comparably age controls. Grating acuities for the congenital groups and some of the normal controls were reanalyzed from Ellemberg et al. (1999, 2000).

3. Results

Fig. 1 presents the coherence thresholds for sensitivity to the direction of global motion for each subject in each of the four patient groups. Circles represent the data

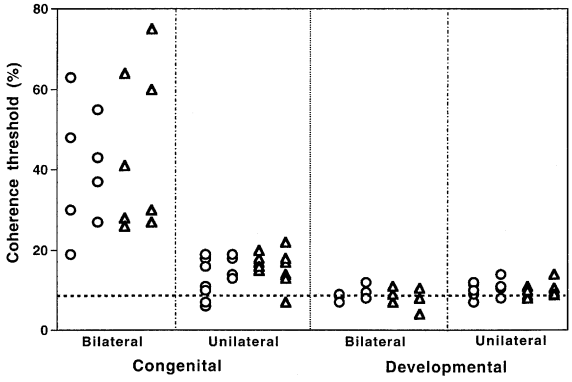

Fig. 1. Global motion coherence thresholds for each subject in each of the four patient groups. Circles represent the data from the better eyes of bilateral cases (determined from clinical history of alignment and Snellen acuity) and the nondeprived eyes of unilateral cases. Triangles represent the data from the worse eyes of bilateral cases and the deprived eyes of unilateral cases. Bilateral cases with equal alignment and acuity histories for the two eyes had one eye assigned randomly to each category. The dashed line represents the mean of 24 control subjects with normal vision.

from the better eyes of bilateral cases and the nondeprived eyes of unilateral cases. Triangles represent the data from the worse eyes of bilateral cases and the deprived eyes of unilateral cases. The dashed line represents the mean of 24 normal subjects. Bilateral and unilateral congenital cases performed significantly worse than normals (main effect of group, $F_{4,54} = 44.40$, $p < 0.001$; post-hoc comparisons, $ps < 0.001$). However, pairwise comparisons indicate that, among patients treated for congenital cataract, patients who had been treated for unilateral cataract had significantly *better* thresholds in the deprived eye than patients treated for bilateral cataract had in either eye ($ps < 0.01$), even when there had been little patching of the nondeprived eye. The thresholds of the deprived eye of the unilateral cases were worse than normal by a factor of 1.6 as compared to a factor of about 4.9 for the bilateral cases. In unilateral cases, the threshold for the nondeprived eye was also significantly worse than normal ($p < 0.01$) and no better than that for the deprived eye ($p > 0.10$). Every patient in the bilateral and unilateral developmental group performed within normal limits ($ps > 0.10$), even in the seven cases whose deprivation began between 4 and 8 months of age.

Fig. 2 presents the grating acuity for each subject in each of the four patient groups. Each patient group performed significantly worse than the control subjects (main effect of group, $F_{4,49} = 38.84$, $p < 0.001$; post-hoc comparisons, $ps < 0.001$). Grating acuity in the deprived eye(s) of every patient treated for congenital cataract was at least 6 times worse than that of the visually normal control subjects. There was no significant difference between the deprived eyes of patients in the congenital groups who had been treated for unilateral versus bilateral cataract ($ps > 0.10$). The worst outcomes were in the three patients (TA, AM, & AF) who had suffered from the most uneven competition because of the longest monocular deprivation (>8 months) and the least patching of the nondeprived eye (2–3 h/day) throughout early childhood (see Fig. 2). In unilateral cases, grating acuity for the nondeprived eye was significantly better than that for the deprived eye ($p < 0.01$). Patients in the bilateral ($p > 0.001$) and unilateral ($p > 0.001$) developmental groups were also significantly worse than the comparably aged control group. However, as shown in Fig. 2, patients treated for developmental cataracts had better acuity overall than those treated for congenital cataract. Every patient with onset of deprivation before 5 years of age had abnormal grating acuity whereas patients with later onset (>11 years) had normal grating acuity. These results indicate that the normal development of grating acuity depends on visual input until at least 5 years of age.

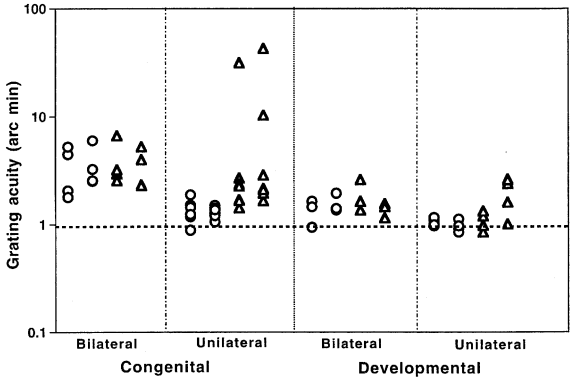

Fig. 2. Grating acuity for each subject in each of the four patient groups. The dashed line represents the mean of 14 comparably aged subjects with normal vision. Other details as in Fig. 1.

4. Discussion

The findings indicate that the absence of patterned and motion information to both eyes from birth prevents the normal development of sensitivity to global motion in either eye. However, normal visual input to one eye from birth is enough to allow the development of nearly normal sensitivity in both eyes. These findings are surprising in that they are the first to document greater losses in any aspect of vision after binocular deprivation than after monocular deprivation. As noted in the Introduction, it is well established that both binocular and monocular deprivation during early infancy result in severe deficits in several aspects of vision, including acuity, spatial and temporal contrast sensitivity, stereovision, and vision in the peripheral visual field (Tytla et al., 1993; Lewis et al., 1995; Bowering, Maurer, Lewis, & Brent, 1997; Ellemberg et al., 1999), with larger reductions after monocular than binocular deprivation unless there was extensive patching of the nondeprived eye (Tytla et al., 1988; Maurer & Lewis, 1993; Lewis et al., 1995; Birch et al., 1998). Measurements of grating acuity in our patients are consistent with these results: grating acuity was abnormal in the deprived eye(s) of every patient treated for congenital cataract, with worse outcomes in the three monocularly deprived patients (TA, AM, & AF) who had suffered the most from uneven competition because of the longest monocular deprivation (>8 months) and had received the least patching of the nondeprived eye (2–3 h/day) throughout early childhood.

As noted earlier in the methods section, we cannot be certain that all patients in the congenital groups had dense central cataracts at birth. Thus it is possible that a patient we placed in a congenital group belonged instead in the developmental group. Any such errors in classification would add noise to the data. However, the error is as likely to have occurred in unilateral as in bilateral cases and hence cannot explain the marked difference in motion thresholds between congenital and

developmental cases after bilateral deprivation but not after unilateral deprivation. Further, the fact that every patient in the bilateral congenital group had a large deficit, whilst every patient in the developmental groups performed normally even when the onset of deprivation was as early as 3.7 months of age, suggests that the deprivation for the bilateral congenital group began at, or near, birth.

The losses in the perception of global motion in the bilateral congenital patients are likely to arise from early visual deprivation and not from problems that are associated with treatment for cataracts, such as aphakia, strabismus, microcornea, and shortened axial length. The normal performance of the patients in the developmental group, even when the onset of deprivation occurred during infancy, indicates that aphakia *per se* did not contribute to the losses measured in patients treated for bilateral congenital cataract. The incidence and degree of strabismus were no different after binocular than after monocular deprivation and, within the binocularly deprived group, the deficits were no greater in the patients who had strabismus than in those who did not ($n = 2$, see Table 1). 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. Moreover, each eye in our sample was tested with an optical correction that focused input on the retina and hence compensated for any abnormality in axial length or corneal curvature at the time of the test.

The incidence of nystagmus was greater after binocular than after monocular deprivation (see Table 1), and could possibly be responsible for the unexpectedly large deficits in children treated for bilateral congenital cataract. The adverse effects of nystagmus during visual development have been documented by Shallow-Hoffman et al. (1998). They found that patients with horizontal congenital nystagmus have marked abnormalities in the detection of horizontal motion but not in the detection of vertical motion. Such patients also have marked abnormalities in speed discrimination for vertical motion when velocity is slower than $6^\circ\ s^{-1}$. Even when nystagmus was absent because of a prolonged neutral zone, performance remained abnormal. Therefore, the authors concluded that the observed deficits are caused not by the nystagmus present during the test, but by the history of nystagmus, which may have led to an adaptive mechanism to avoid oscillopsia.

The unexpectedly large deficits in our patients treated for bilateral congenital cataract are likely not attributable to a history of nystagmus: our stimuli contained vertical motion drifting at a speed of $18^\circ\ s^{-1}$, conditions under which there are no deficits in motion perception after congenital nystagmus (Shallow-Hoffman et al., 1998). Moreover, in the bilateral congenital group, the deficits were just as great in the two patients (AaB and CP) who did not experience nystagmus during development as in the six patients who did. AaB and CP did not experience nystagmus during development because their nystagmus was only latent and they did not receive any occlusion therapy. The irrelevance of nystagmus during development to our results is also apparent in the results from the unilateral congenital group. Eight of the patients in this group experienced nystagmus during development, either because of a manifest nystagmus (JW and CK) or because of a latent nystagmus while the good eye was patched (see Table 1). Yet the deficits of these eight patients were no worse than those of the five unilateral cases who had no history of manifest nystagmus or of latent nystagmus in either eye. Together, the results indicate that, under the present testing conditions, the unexpectedly large deficits in patients treated for bilateral congenital cataract cannot be attributed to the effects of prolonged nystagmus during development.

To assess any contribution of nystagmus at the time of our monocular testing, we retested two patients treated for bilateral congenital cataract who had a latent nystagmus but no manifest nystagmus (AaB and CP). We tested these two patients without the artificial pupil both binocularly (no nystagmus condition) and monocularly (nystagmus condition). As shown in Fig. 3, their deficits were just as large when tested binocularly without the artificial pupil as when tested binocularly or monocularly with the artificial pupil. Thus it is also unlikely that nystagmus at the time of the test contributed to the pattern of results.

Neurons in the striate cortex of binocularly and monocularly deprived monkeys respond more sluggishly than normal and have abnormal spatial frequency-tuning (Blakemore & Vital-Durand, 1983; Blakemore, 1990), with more substantial reductions in the number of striate neurons that respond to stimulation of the deprived eye of monocularly deprived monkeys than to the deprived eyes of binocularly deprived monkeys (LeVay et al., 1980; Horton & Hocking, 1998; Crawford et al., 1991). The finding from the present study that binocular deprivation results in a more profound deficit in the perception of the direction of global motion than does monocular deprivation, despite the fact that monocular deprivation causes more severe abnormalities in striate cortex neurons, has two important implications. First, deficits in global motion after early binocular deprivation likely result from damage to directionally selective neurons outside the primary visual cortex. Second, input from one eye at birth permits a relative sparing of visual function mediated by those neurons. The substantial deficits after early binocular but not after early monocular deprivation suggest that, compared to the striate cortex, extrastriate area MT is affected less by uneven competition between the eyes.

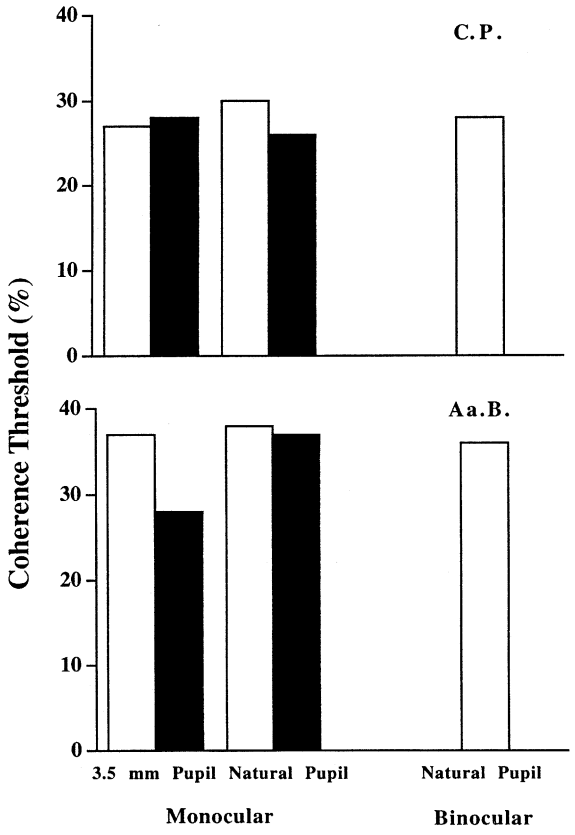

Fig. 3. Global motion coherence thresholds for two binocularly deprived patients with latent nystagmus tested monocularly with and without an artificial pupil and binocularly without an artificial pupil. White bars represent the data from the right eye and shaded bars represent the data from the left eye. AaB had no history of strabismus, equal acuity in the two eyes, and a latent nystagmus in each eye. CP had no history of strabismus, better acuity in the right eye than in the left, and a latent nystagmus in the left eye. Neither patient had nystagmus when viewing binocularly.

Monocular deprivation may be less disruptive for aspects of vision mediated by area MT because of converging input from striate and extrastriate pathways onto binocular MT cells with large receptive fields (Maunsell & Van Essen, 1983a, 1987). During early monocular deprivation, the initial development of MT cells may be driven by input from the nondeprived eye and after treatment, those cells may respond to either eye. This interpretation is consistent with our finding that sensitivity to global motion is reduced slightly and equally for both the deprived and nondeprived eyes. There are two routes by which input from the previously deprived eye could reach area MT: (a) via cells in the primary visual cortex sensitive to low spatial frequencies that are spared after early monocular deprivation (Ellemberg et al., 1999, 2000) and that would be activated by the low spatial frequencies contained in our moving dots or (b) via the pulvinar and/or other extrageniculate pathways bypassing the primary visual cortex (Rodman, Albright, & Gross, 1990), which may play a more important role after early deprivation (Zablocka, Zernicki, & Kosmal, 1976, 1980) than they do after normal development (Azzopardi, Fallah, Gross, & Rodman, 1998). Our hypothesis that the previously deprived eye is able to drive binocular MT cells that were tuned to the direction of motion by input from the nondeprived eye is supported by recent findings from strabismic amblyopes: they show essentially no inter-ocular transfer of motion aftereffects for stimuli tapping the primary visual cortex but nearly normal inter-ocular transfer for global motion, which taps area MT (McColl & Mitchell, 1998).

We also found that the sensitive period for the normal development of the discrimination of the direction of global motion was very short. Patients who received normal patterned input before the visual deprivation in one or both eyes, even for as little as 4–8 months, had normal coherence thresholds for the direction of global motion. In contrast, our results indicate that the normal development of grating acuity depends on visual input until at least 5 years of age, and the sensitive period for peripheral light sensitivity is known to extend into adolescence (Bowering et al., 1993). Electrophysiological studies in kittens who had incurred various forms of abnormal visual input have also shown shorter sensitive periods for direction selectivity than for orientation selectivity or binocularity (Daw & Wyatt, 1976; Berman & Daw, 1977; Daw, Berman, & Ariel, 1978). These findings support the hypothesis that the period of malleability for different visual properties to which neurons become tuned during postnatal development is asynchronous (Rauschecker, 1991; Rauschecker & Marler, 1987).

Prior to the surgical removal of the dense central cataract, our patients had been deprived not only of pattern input, but also of motion signals. We cannot determine the extent to which the pattern versus motion deprivation contributed to the losses in the congenital cases. The effects of motion deprivation alone on the perception of motion have been studied in cats that were reared in an environment illuminated stroboscopically at 8 Hz (Pasternak, Schumer, Gizzi, & Movshon, 1985). This experimental condition preserves patterned input but eliminates motion signals (both signals associated with self-produced motion and motion in the environment) on the retina of both eyes. Such rearing causes marked reductions in the ability to discriminate the direction of motion of gratings, and causes a dramatic reduction in directionally-selective neurons in both the striate cortex (Cynader & Chernenko, 1976; Kennedy & Orban, 1983; Cremieux, Orban, Duysens, & Amblard, 1987) and in the lateral suprasylvian cortex—an area analogous to area MT in human and nonhuman primates (Spear, Tong, McCall, & Pasternak, 1985). Together these findings suggest that, at least in cats, reductions in the number of directionally selective

neurons after motion deprivation cause deficits in the visual discrimination of the direction of motion.

5. Conclusion

We provide the first comparison of monocular versus binocular deprivation on sensitivity to global motion which, unlike spatial vision, depends on neurons beyond the primary visual cortex. Surprisingly, sensitivity to global motion was *better* after monocular than after binocular deprivation and the sensitive period was very short. Thus beyond the primary visual cortex, competitive interactions between the eyes can give way to collaborative interactions that enable a relative sparing of visual function after monocular deprivation.

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References

Albright, T. D. (1984). Direction and orientation selectivity of neurons in visual area MT of the macaque. *Journal of Neurophysiology*, *52*, 1106–1130.

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 for Neuroscience, Abstracts*, *24*, 648.

Barton, J. J. S., Sharpe, J. A., & Raymond, J. E. (1995). Retinotopic and direction defects in motion discrimination in human with cerebral lesions. *Annual Review of Neurology*, *37*, 665–675.

Berman, N., & Daw, N. W. (1977). Comparison of the critical periods for monocular and directional deprivation in cats. *Journal of Physiology*, *265*, 249–259.

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

Blakemore, C. (1990). Vision: Coding and efficiency. In C. Blakemore (Ed.), *Maturation of Mechanisms for Efficient Spatial Vision*. Cambridge: Cambridge University Press.

Blakemore, C., & Vital-Duran, F. (1983). Visual deprivation prevents the postnatal maturation of spatial contrast sensitivity neurons of the monkey's striate cortex. *Journal of Physiology (Lond.)*, *345*, 40P.

Born, R. T., & Tootell, B. H. (1992). Segregation of global and local motion processing in primate middle temporal visual area. *Nature*, *357*, 497–499.

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.

Bowering, E. R., Maurer, D., Lewis, T. L., & Brent, H. P. (1997). Constriction of the visual field of children after early visual deprivation. *Journal of Pediatric Ophthalmology and Strabismus*, *34*, 347–356.

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.

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

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 selective selectivity in the visual cortex of the cat. *Science*, *193*, 504–505.

Daw, N. W., Berman, N., & Ariel, M. (1978). Interaction of critical periods in the visual cortex of kittens. *Science*, *199*, 565–567.

Daw, N. W., & Wyatt, H. J. (1976). Kittens reared in a unidirectional environment: evidence for a critical period. *Journal of Physiology*, *257*, 155–170.

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 plasticity. I. Ocular dominance columns. *Cerebral Cortex*, *6*, 781.

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.

Harwerth, R. S., Smith, E. L., Boltz, M. L. J., Crawford, M. L. J., & von 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.

Horton, J. C., & Hocking, D. R. (1998). Monocular core zones and binocular border strips in primate striate cortex revealed by the contrasting of enucleation, eyelid suture, and retinal laser lesions on cytochrome oxidase activity. *Journal of Neuroscience*, *18*, 5433–5455.

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

LeVay, 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.

Levitt, H. J. (1971). Transformed up–down methods in psychophysics. *Acoustical Society of America*, *49*, 467–477.

Lewis, T. L., Maurer, D., & Brent, H. P. (1995). 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.

Maunsell, J. H. R., & Van Essen, D. C. (1987). Topographic organization of the middle temporal visual area in the macaque monkey: representation biases and the relationship to callosal

connections and myeloarchitectonic boundaries. *Journal of Comparative Neurology*, *266*, 535–555.

Maunsell, J. H. R., & Van Essen, D. C. (1983a). Functional properties of neurons in middle temporal visual area of the macaque monkey. II. Binocular interactions and sensitivity to binocular disparity. *Journal of Neurophysiology*, *49*, 1148–1167.

Maunsell, J. H. R., & Van Essen, D. C. (1983b). Functional properties of neurons in middle temporal visual area of the macaque monkey. I. Selectivity for stimulus direction, speed and orientation. *Journal of Neurophysiology*, *49*, 1127–1147.

Maurer, D., & Lewis, T. L. (2001). Visual acuity and spatial contrast sensitivity: Normal development and underlying mechanisms. In C. A. Nelson, & M. Luciana (Eds.), *The Handbook of Developmental Cognitive Neuroscience*. Boston, MA: MIT Press.

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.

McColl, S. L., & Mitchell, D. E. (1998). Stereodeficient subjects show substantial differences in interocular transfer of two motion adaptation aftereffects. *Vision Research*, *38*, 1889–1900.

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

Newsome, W. T., & Pare, E. B. (1988). A selective impairment of motion perception following lesions of the middle temporal visual area. *Journal of Neuroscience*, *8*, 2201–2211.

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.

Rauschecker, J. P. (1991). Mechanisms of visual plasticity: Hebb synapses, NMDA receptors and beyond. *Physiological Reviews*, *71*, 587–615.

Rauschecker, J. P., & Marler, P. (1987). Cortical plasticity and imprinting: behavioral and physiological contrasts and parallels. In J. P. Rauschecker, & P. Marler (Eds.), *Imprinting and Cortical Plasticity Comparative Aspects of Sensitive Periods* (pp. 349–366). New York: Wiley.

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.

Scase, M. O., Horsfield, M. A., Wilcock, D. J., & Karwatowski, W. S. S. (1998). Brain activation by random dot kinematograms of varying coherence: A fMRI study. *Investigative Ophthalmology and Visual Science*, *39*(Supplement), S1130.

Shallow-Hoffman, J. A., Bronstein, A. M., Acheson, J., Morland, A. B., & Gresty, M. A. (1998). Vertical and horizontal motion perception in congenital nystagmus. *Neuro-ophthalmology*, *19*, 171–183.

Schiller, P. H. (1996). On the specificity of neurons and visual areas. *Behavioural Brain Research*, *76*, 21–35.

Smith, A. T., Snowden, R. J., & Milne, A. B. (1994). Is global motion really based on spatial integration of local motion signals? *Vision Research*, *34*, 2425–2430.

Spear, P. D., Tong, L., McCall, M. A., & Pasternak, T. (1985). Developmentally induced loss of direction selective neurons in cat lateral suprasylvian cortex. *Developmental Brain Research*, *20*, 281–285.

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.

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

Ungerleider, L. G., & Desimone, R. (1986). Cortical connections of visual area MT in the macaque. *Journal of Comparative Neurology*, *248*, 190–222.

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

Williams, D. W., & Sekuler, R. (1984). Coherent global motion percepts from stochastic local motions. *Vision Research*, *24*, 55–62.

Wilson, H. R. (1999). Non-Fourier cortical processing in texture, form, and motion perception. *Cerebral Cortex*, *13*, 445–447.

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.