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The Influence of Binocular Visual Deprivation on the Development of Visual-Spatial Attention

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This article examines the effects of visual input on the development of attention by comparing normal children to children, all more than 8 years old, who had been treated for bilateral congenital cataracts during infancy. In Experiment 1, patients pushed a button as soon as they detected a target that appeared 100, 400, or 800 msec after a central cue. The cue either validly cued the upcoming location or invalidly cued the wrong location. Patients (n = 16) performed normally at the 100 msec and 400 msec stimulus onset asynchrony (SOA). However, when the cue preceded the target by the 800 msec SOA, patients' reaction times were not affected by the validity of the cue, especially when deprivation had extended past 4 months of age. In Experiment 2, patients indicated which of two shapes appeared in the periphery 400 msec after a central cue, with those shapes surrounded by compatible or incompatible distractors. Patients (n = 15) differed from age-matched controls in (a) being slowed more by incompatible distractors on invalid trials, and (b) tending to show a larger than normal effect of the validity of the cue preceding targets in the upper visual field. Together, these findings suggest that the normal development of attention is influenced by early visual experience.

For many years after birth there are improvements in visual–spatial attention; that is, in the ability to attend to specific locations in space where a stimulus is expected and to shift attention rapidly to another location should a stimulus appear there. In this article, we studied the effect of visual experience on the development of these abilities by comparing children treated for bilateral congenital cataracts to children with a normal visual history. A cataract is an opacity of the lens of the eye. In the patients we selected for study, the cataracts had been dense and central so that they had prevented patterned visual input from reaching the retina. Treatment involved surgical removal of the cataracts followed by fitting the eyes with contact lenses to focus visual input on the retina. At the time of the tests, the patients were at least 8 years old and hence had years to recover from the initial deprivation. The purpose of our studies was to examine whether the initial deprivation, which had lasted from birth until 1.8 to 19.5 months of age, had a permanent effect on the development of visual–spatial attention.

Previous research indicates that early binocular deprivation interferes with the postnatal development of sensory visual functions, including visual acuity (reviewed in Maurer & Lewis, in press), spatial contrast sensitivity (Birch, Stager, Leffler, & Weakley, 1998; Ellemberg, Lewis, Maurer, Liu, & Brent, 1999; Mioche & Perenin, 1986; Tytla, Maurer, Lewis, & Brent, 1988), temporal contrast sensitivity (Ellemberg et al., 1999), stereopsis (Tytla, Lewis, Maurer, & Brent, 1993), and peripheral light sensitivity (Bowering, Maurer, Lewis, & Brent, 1993, 1997). It also affects the control of eye movements, such that patients treated for bilateral congenital cataract often have unsteady fixation, show a spontaneous or latent nystagmus that is predominantly horizontal (Lewis, Maurer, & Brent, 1995), and have asymmetrical optokinetic nystagmus in response to a moving pattern viewed monocularly (reviewed in Maurer & Lewis, 1993; Maurer, Lewis, & Brent, 1989). In contrast, early binocular deprivation has no apparent effect on abilities that are rel-

atively mature at birth, such as color vision and the ability to recognize grossly different shapes (reviewed in Maurer & Lewis, 1993; Maurer, Lewis, & Brent, 1993). For some affected visual functions, the outcome is related to the duration of deprivation from birth, such that the deficits are larger when the initial deprivation lasted longer (reviewed in Maurer & Lewis, 1993). For all affected visual functions, the deficits are largest when the deprivation began at birth. Studies of monkeys indicate that binocular deprivation exerts its effect at the level of the striate cortex and beyond: there are no changes in photoreceptors (Hendrickson & Boothe, 1976) in the electroretinogram (Crawford, Blake, Cool, & von Noorden, 1975), or in the physiological properties of neurons in the lateral geniculate nucleus (Blakemore & Vital-Durand, 1986; Boothe, Dobson, & Teller, 1985). In contrast, striate neurons respond sluggishly, are poorly tuned, have receptive fields that are abnormally large, and are less likely than normal to be driven by both eyes (Crawford et al., 1975; Crawford, Pesch, von Noorden, Harwerth, & Smith, 1991; reviewed in Maurer & Lewis, in press). In the studies reported here, we explored whether binocular deprivation also affects the development of higher visual functions, either because of poor input from the striate cortex or because of effects on extrastriate projections.

We hypothesized that visual experience might be necessary for the normal development of visual–spatial attention because it is immature at birth, with abilities emerging postnatally and then developing slowly into middle childhood. Under most circumstances, young infants have difficulty shifting their eyes, and hence presumably their attention, from the object they are fixating to another object. This "obligatory attention" or "sticky fixation" is most apparent around 1 to 2 months of age (Atkinson, Hood, Wattam-Bell, & Braddick, 1992; Hood, 1995; Hood & Atkinson, 1993; Johnson, 1990, 1995) and then diminishes around 3 to 4 months of age (Atkinson et al., 1992; Hood, 1995; Hood & Atkinson, 1993; Johnson, Posner, & Rothbart, 1991), at the same time as babies first show the ability to anticipate the location of an upcoming target (Canfield & Haith, 1991; Haith, Hazen, & Goodman, 1988; Johnson et al., 1991) and to orient attention covertly, without an eye movement (Johnson et al., 1991; Johnson, Posner, & Rothbart, 1994; Johnson & Tucker, 1996). Even after infancy, it takes many years before children are as good as adults at redirecting attention quickly and at ignoring distracting information. One demonstration comes from studies in which participants are asked to press a button as soon as a target appears in the periphery and are cued with a visual marker (exogenous cue) at the upcoming location or at the wrong location. Both adults and children respond more quickly when the target appears at the cued location, even when they must shift attention covertly because eye movements are not allowed (e.g., Akhtar & Enns, 1989; Enns & Brodeur, 1989). However, all studies using exogenous cues agree that children between the ages of 5 to 9 show a larger validity effect than do adults, presumably because they are less able to shift attention from the incorrectly cued location to the position where the target appears. A

similar developmental pattern is evident when the location of the upcoming target is signaled by a central or endogenous cue that provides information about the likely location of the upcoming target: Children younger than 8 to 10 years of age show a larger validity effect than adults (Brodeur, 1993; Brodeur, Trick, & Enns, 1997; Goldberg, Maurer & Lewis, 2001; Pearson & Lane, 1990).

The slow development of adult-like ability to ignore distractors has been demonstrated using a number of paradigms including Stroop color-word naming, same and different judgments, and speeded classification (e.g., Enns, 1990; Enns & Akhtar, 1989; Enns & Cameron, 1987; Lane & Pearson, 1982; Plude, Enns, & Brodeur, 1994; Ridderinkhof & van der Molen, 1995; Strutt, Anderson, & Well, 1975; Tipper, Bourque, Anderson, & Brehaut, 1989; Well, Lorch, & Anderson, 1980). For example, in a speeded classification task, children between the ages of 4 to 7 (Enns & Akhtar, 1989) and 5 to 9 (Ridderinkhof & van der Molen, 1995) are slowed much more than adults by information incompatible with the current classification. Distractors surrounding a peripheral target increase reaction time at all ages, especially if the target and distractor signal incompatible responses. However, under some conditions they have a larger effect on children's reaction times until at least age 10 (Goldberg et al., 2001) and can be especially problematic for children when the target and distractors appear far from where the child is attending (Akhtar & Enns, 1989).

Thus, visual–spatial attention undergoes protracted development: The ability to disengage and shift attention emerges postnatally and improves for the next 8 to 10 years. The ability to ignore distractors is still not adult-like under some conditions, even at 10 years of age. Because of the slow development of visual–spatial attention, we hypothesized that it might be affected adversely by early visual deprivation, as is the case for other slowly developing visual abilities.

Our second reason for suspecting that early visual input is important for the development of visual–spatial attention is evidence from monkeys that early binocular visual deprivation has an adverse effect on areas of the brain involved in attention. Binocular deprivation beginning shortly after birth dramatically reduces the later sensitivity of cells in the monkey's posterior parietal association cortex (Brodmann's area 7) to visual stimuli, much more than it affects cells in the primary visual cortex (Carlson, Pertovaara, & Tanila, 1987; Hyvarinen & Hyvarinen, 1979, 1982; Hyvarinen, Hyvarinen, & Linnankoski, 1981). Studies of both monkeys and humans indicate that the posterior parietal cortex plays a major role in regulating attention. Cells in that area of the monkey's cortex show increased firing during covert shifts of attention cued by a peripheral exogenous cue (Steinmetz, Connor, Constantinidis, & McLaughlin, 1994; Steinmetz & Constantinidis, 1995). Neuroimaging studies using positron emission tomography and functional magnetic resonance imaging show increased activity in parietal (and frontal) brain regions when human adults use a central endogenous cue to covertly shift attention (Corbetta, 1998; Corbetta, Miezin, Shulman, & Petersen,

1993; Coull, Frith, Buchel, & Nobre, 2000; Nobre, Gitelman, Dias, & Mesulam, 2000; Petersen, Corbetta, Miezin, & Shulman, 1994; Rosen et al., 1999). Moreover, unilateral lesions to the posterior parietal cortex, in both the monkey (Petersen & Robinson, 1986) and the human (Baynes, Holtzman, & Volpe, 1986; Petersen, Robinson, & Currie, 1989; Posner, Walker, Friedrich, & Rafal, 1984, 1987), result in deficits in visual–spatial attention, especially when the participant must disengage attention from a location miscued by an exogenous marker in the good visual field and respond to a target that is presented in the bad visual field (contralateral to the parietal lesion). The one case report of a patient with bilateral parietal lesions also found deficits, specifically in benefiting from exogenous cues directing attention to the left, right, and lower visual fields (Verfaellie, Rapcsak, & Heilman, 1990).

Taken together, it is reasonable to hypothesize that early visual experience might be important for the normal development of visual–spatial attention because (a) it is slow to develop, (b) cells in the parietal cortex develop abnormally in binocularly deprived monkeys, and (c) the parietal cortex plays a major role in controlling visual–spatial attention. However, recent findings about the role of the parietal cortex in controlling attention during infancy make the predictions less straightforward. Studies of exogenously cued shifts of attention following infantile lesions suggest that posterior systems (including the parietal cortex) play a smaller role than in adults, whereas anterior systems (including frontal cortex) play a larger role (e.g., Craft, Schatz, Glauser, Lee, & DeBaun, 1994; Craft, White, Park, & Figel, 1994; Johnson, Tucker, Stiles, & Trauner, 1998). For example, children who had suffered bilateral posterior lesions during early infancy show validity effects of normal magnitude (Craft, White, et al., 1994). Similarly, infants with unilateral posterior lesions perform normally on a spatial cueing task when tested toward the end of the first year of life (Johnson, Tucker, Stiles, & Trauner, 1998). However, spatial cueing effects are abnormal after anterior lesions. When the stimulus onset asynchrony (SOA) is short or the target appears in the right visual field, there is no benefit of prior cueing in infants following left frontal lesions (Johnson et al., 1998) or in children who had suffered bilateral infantile anterior lesions (Craft, Schatz, et al., 1994). When the SOA is long or the target appears in the left visual field, there is an abnormally large validity effect in children who suffered bilateral infantile anterior lesions (Craft, White, et al., 1994). These findings suggest that the neural systems controlling visual–spatial attention are different in infants than in adults. Supporting evidence comes from a study comparing event-related potentials of adults and 6-month-olds who made eye movements toward a peripheral target: unlike the adults, in the infants there was no presaccadic positivity over the parietal cortex, but there was differential activity over the left frontal cortex when the infant had to disengage attention from a central fixation stimulus (Csibra, Tucker, & Johnson, 1998). Together, the evidence suggests that the visual–spatial attention of young infants, compared to adults, is mediated more by frontal pathways and

less by the parietal cortex. After infantile parietal lesions, the frontal cortex appears able to continue to play that role. Thus, we predicted that children who had missed early visual experience because of cataracts might perform abnormally on standard measures of visual–spatial attention, and that the abnormality might reflect abnormal development of the parietal cortex, the frontal cortex, or both. We predicted that the abnormality might be reflected in an unusually large validity effect (as is true generally after adult unilateral parietal lesions and, at some SOAs and in some parts of the visual field, after infantile anterior lesions) or as an unusually small validity effect (as is true after adult bilateral parietal lesions in all but the upper visual field and, at some SOAs and in some parts of the visual field, after infantile anterior lesions). In the General Discussion section, we speculate that the pattern of performance after binocular deprivation most likely reflects abnormal development of a frontoparietal system.

To evaluate the effects of early visual experience on the development of attention, we used standard paradigms from the literature to compare children who had been treated during infancy for bilateral congenital cataracts to normal controls. We used central informative "endogenous cues" because they are more likely than peripheral "exogenous cues" to reveal deficits in attention and higher cognitive functions (Posner, Cohen, & Rafal, 1982; Posner et al., 1984). The cue was either valid, correctly signaling the location of the upcoming target; invalid, cueing the incorrect location; or neutral, providing no information about the location of the upcoming target. In Experiment 1, we probed for possible deficits using a simple reaction time task and large targets so that even a patient with severely impaired acuity would be able to complete the task. We included three time intervals between the cue and target (SOAs) because different mechanisms may be involved in shifting versus maintaining attention (Nakayama & MacKeben, 1989; Posner & Petersen, 1990; Posner et al., 1984) and because the size and type of deficit in patients with cortical damage varies with SOA (Craft, Schatz, et al, 1994, Craft, White, et al., 1994; Johnson et al., 1998; Posner et al., 1984). Because the initial results suggested that patients were relatively normal on this simple task, in Experiment 2 we probed at one SOA using a harder procedure, in which the participant had to indicate which of two targets appeared, and the targets were surrounded by distractors. Together, Experiments 1 and 2 provide the first information on the role of visual experience in the development of visual–spatial attention.

Unlike traditional procedures, tests were conducted monocularly because the two eyes of a patient sometimes show different sensory deficits (see Table 1). Targets appeared along the vertical meridian, as opposed to the more customary horizontal meridian, to facilitate steady fixation in patients with nystagmus (repetitive jerky eye movements; see Table 1), which was always horizontal. Fixation was monitored, and trials with eye movements (other than the horizontal eye movements caused by any nystagmus) were eliminated, so that differences in reaction times between conditions reflected covert attentional shifts.

TABLE 1
Clinical Details for the Children Treated for Bilateral Congenital Cataract

Patient	*Refraction (Diopters)*[a]	*Deprivation (Months)*	*Additional Complications*	*Experiment*	*Age*[b] *(Years)*	*Snellen Acuity*[a]
L. M.	OD + 15.50	3.1	Microcornea OD	1	8.2	6/24
	OS + 13.00	3.1	Strabismus surgery for RET at ages 1 and 2			6/15
			Latent nystagmus OU	2	8.9	6/18
						6/15
B. B.	OD + 13.00	5.4	Intermittent manifest nystagmus	1	9.4	6/21
	OS + 14.50	5.4				6/21
A. L.	OD + 16.50	5.4	Latent nystagmus OS	1	9.8	6/15
	OS + 15.50	5.4				6/30
				2	10.4	6/15
						6/30
A. H.	OD + 16.25	2.1	Strabismus surgery for RET/LET at age 1	1	10.9	6/60
	OS + 15.75	2.1	Manifest nystagmus			6/15
			Microcornea OD	2	10.9	6/60
						6/15
J. G.	OD + 20.25	2.7	Microcornea OU	1	10.9	6/18
	OS + 23.75	2.7	Latent nystagmus OU			6/12
				2	11.1	6/18
						6/12
S. S.	OD + 12.75	7.5	Manifest nystagmus	1	11.5	6/18
	OS + 12.00	7.5				6/60
				2	13.1	6/18
						6/60
K. M.	OD + 11.25	2.3	Glaucoma OU diagnosed at age 14	1	11.9	6/6
	OS + 13.25	2.3	Latent nystagmus OS			6/9.5

(*continued*)

TABLE 1 (*Continued*)

Patient	*Refraction (Diopters)*[a]	*Deprivation (Months)*	*Additional Complications*	*Experiment*	*Age*[b] *(Years)*	*Snellen Acuity*[a]
C. B.	OD + 7.00	3.0	Latent nystagmus OS	1	12.1	6/7.5
	OS + 8.50	3.0	Strabismus surgery for LET at age 2			6/60
				2	13.8	6/9
						6/60
J. J.	OD + 14.50	1.8	Latent nystagmus OU	1	12.9	6/12
	OS + 18.50	1.8				6/9
				2	13.4	6/12
						6/9
A. D.	OD + 17.50	3.2	Latent nystagmus OU	1	13.1	6/24
	OS + 15.50	3.2				6/30
				2		6/24
						6/30
C. P.	OD + 9.50	6.1	Latent nystagmus OU	1	14.5	6/9
	OS + 10.75	6.1	Strabismus surgery for LET at age 2			6/15
				2	16.0	6/7.5
						6/15
I. W.	OD + 13.00	5.0	Manifest nystagmus	1	15.1	6/60
	OD + 14.00	8.6	Strabismus surgery for RET at age 3 and			6/7.5
			for RET/LET at age 6	2	15.8	6/30
						6/7.5
G. S.	OD + 25.50	6.1	Manifest nystagmus	2	15.6	6/60
	OS + 24.50	6.1	Microcornea OU			6/60
A. C.	OD + 11.25	6.4	Manifest nystagmus	1	16.2	6/12
	OS + 12.25	5.3	Strabismus surgery for RET/LET at age 4			6/15
				2	16.9	6/12
						6/15

A. R.	OD + 17.00	3.5	Microcornea OU	1	16.8	6/30
	OS + 12.50	3.5	Strabismus surgery for RET ages 1, 2, and 5; for RET/LET at age 7			6/30
			Manifest nystagmus	2	15.8	6/30
						6/30
M. D.	OD + 9.75	4.1	Latent nystagmus OS	1	17.2	6/9
	OS + 9.00	4.1				6/18
				2	17.2	6/9
						6/18
S. G.	OD + 9.75	19.5	Manifest nystagmus	1	20.4	6/18
	OS + 11.25	19.5				6/18

Note. OD = right eye; OS = left eye; RET = right esotropia; OU = both eyes; LET = left esotropia.
[a]Measurement closest to the test. [b]At the time of the test.

EXPERIMENT 1

In Experiment 1, patients and normal controls were tested with a simple reaction time task (Posner, Nissen, & Ogden, 1978) in which they were asked to fixate centrally and to push a button as soon as they detected the target, which appeared in one of two peripheral locations. The target was preceded by a central cue that signaled the likely location of the upcoming target. The measure of covert attentional shifts in this paradigm is called the "validity effect," which is the difference in reaction time between trials during which the cue signaled the correct location ("valid trials") and trials during which the cue signaled the wrong location ("invalid trials"). Patients with unilateral damage to the parietal cortex show abnormally large validity effects because their reactions times are abnormally long on invalid trials. To help with the interpretation of any abnormality, we included catch trials, during which no target was presented following the cue, and neutral trials, during which the central cue had a shape that provided no information about the location of the upcoming target. Patients' data were compared to those of normal 8-year-olds, 10-year-olds, and adults tested with the same procedure for a different study (Goldberg et al., 2001).

The interval between the cue and target (SOA) was varied between blocks of trials. Based on our previous work with normal adults and children (Goldberg, 1998; Goldberg, Maurer, & Lewis, 1996; Goldberg et al., 2001), we used SOAs of 100, 400, and 800 msec to capture the intervals during which the validity effect develops, peaks, and begins to decline. SOA was varied across blocks of trials, but kept constant within each block. When SOAs are intermixed instead, the participant's expectation that a target is about to appear may change between the cue and the time when the target appears. If the procedure includes catch trials, then the longer the interval after the cue, the more likely that the trial is a catch trial and the less likely that a target is about to appear. If there are no catch trials, then as the interval after the cue increases, the more likely it becomes that a target is about to appear, until for the longest SOA, the time interval itself predicts perfectly the appearance of a target. Such changes in expectancy during the course of a trial may differ between patients and normal participants. To avoid any such problem, we kept SOA constant within each block of trials. As a result, we kept constant the probability of a target appearing during the course of each trial.

Method

Patients. The final sample consisted of 16 patients treated for bilateral congenital cataract (mean age = 13.2 years on the date of the test, range = 8.2–20.4 years). Table 1 provides clinical details for the patients. Patients were included only if, on the first eye examination, the ophthalmologist determined the cataract inter-

fered seriously with vision because (a) the eye did not fixate or follow a light, (b) the cataract completely blocked the view of the fundus through an undilated pupil, (c) no red light reflex was visible through an undilated pupil, and (d) the cataract looked dense and central. Patients were excluded from the sample if their cataracts were not dense (e.g., dull red reflex visible through the cataract), located only in the periphery of the lens, or smaller than 5 mm. Patients were included only if the cataracts had been identified in both eyes on the first eye exam prior to 6 months of age.

Approximately 1 week after patients had surgery to remove the cataractous lenses, the patients' eyes had been measured for contact lenses, which they began to wear 1 to 2 weeks later. Table 1 shows the duration of deprivation (M = 5.1 months), taken from birth to the age at which the eyes first began wearing contact lenses after surgery. Five patients subsequently switched to wearing glasses. Following treatment, each patient had at least 7 years of patterned visual input before the test of visual–spatial attention.

Patients were excluded from the sample if they had continuing deprivation following surgery because of irregular wear of the optical correction (contact lenses or glasses). Specifically, children who wore the optical correction less than 75% of the waking time during the first 7 years of life or stopped wearing it prior to 7 years of age were excluded. Children were also excluded if there was evidence of significant developmental delay (scores more than 2 *SD* below the normal mean when tested with the Bailey Scale of Infant Development at age 2 or the McCarthy Scale of Children's Abilities at age 5). Developmental delay may be related to deficits in attention independently from the effects of early visual deprivation and may interfere with the ability to perform the task. Patients with additional serious problems that could interfere with vision (e.g., glaucoma, detached retina) were excluded. However, patients with abnormalities commonly associated with congenital cataract such as strabismus (misaligned eyes), horizontal nystagmus (repetitive jerky eye movements in a horizontal direction), or microcornea (eye smaller than normal) were included (see Table 1), and the possible effect of those abnormalities will be considered in the Discussion.

We compared the results from the 16 patients to the results from 16 normal participants spanning the same age range (mean age = 13.0 years, range = 8.2–20.5 years). The comparison participants all had no history of visual or attentional problems, wore no optical correction, and passed a visual screening examination.

Apparatus and stimuli. The sizes of the cues and targets were chosen so as to be visible to patients treated for congenital cataract, who are known to have reduced vision (Bowering, 1992; Ellemberg et al., 1999; Maurer & Lewis, 1993; Tytla, Lewis, Maurer, & Brent, 1991). At the beginning of each trial, patients saw a central fixation stimulus between two square boxes where targets could appear. The central fixation stimulus was a solid black diamond, 5.6º (3.6 cm) high and wide when viewed

from 36 cm. The boxes were 7.6º (4.8 cm) in diameter, formed from lines 1.3º (0.8 cm) thick, and began 5º (3.2 cm) above and below the center of the central fixation stimulus. Targets were 2 × 2 black-and-white checkerboards appearing inside the boxes and were formed from checks 2.5º (16 cm) in diameter. Cues were white shapes appearing inside the central fixation stimulus. Informative cues were solid triangles and were 2.8º (1.8 cm) wide by 1.4º (0.9 cm) high and pointed either up or down. Neutral cues (2º high and wide) were diamonds providing no information about the location of the target and were matched in area to the informative cues (7.9 cm^2). Black stimuli were 9.0 cd/m^2; white stimuli were 46.7 cd/m^2.

Stimuli were presented by a Macintosh Powerbook 160 onto an Apple 12-in. monitor (25.3º high and 35.4º wide). SuperLab software, with a 3.96 msec time accuracy and a 20 microsec timing resolution, controlled the presentation of visual stimuli, the time interval from the onset of a cue to the onset of a target (SOA), and the measurement of reaction time.

Procedure. Patients wore trial frames over their contact lenses or glasses, with lenses of a power designed to focus each eye for the testing distance. Each participant was tested monocularly by having the nontested eye covered by an eye patch made from a double layer of micropore tape (3M; London and Ontario, Canada). The participant sat 36 cm from the screen with the nonpatched eye aligned with the central fixation stimulus, which he or she was instructed to fixate. When the experimenter judged that the participant was fixating on the center of the black diamond, she pressed a key to present a cue. Participants were instructed to maintain central fixation and to respond by pressing the letter B on a computer keyboard in front of them as soon as they detected the target. At the end of each trial, the experimenter pressed one of two buttons to code whether the participant maintained fixation during the trial or produced an eye movement.[1]

Participants were introduced to the task with a demonstration of the procedure. They were then told that it is important to practice doing this task as quickly as possible without making eye movements toward the location of the target or anticipating its appearance. Participants were given practice with trials at a 800 msec SOA until they completed 10 consecutive trials without an eye movement, anticipatory response before the onset of the target, or false positive response on a catch trial. This typically took about 20 trials.

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[1]To evaluate the accuracy of the experimenter's judgments of central fixation, an adult fixated the center of the central stimulus or 1°, 2°, or 3° above or below the center on 35 randomly ordered trials. The experimenter was accurate 100% of the time in judging whether the fixation was central, up, or down, and accurate 88% of the time in judging which of the six locations off center was being fixated. To evaluate the accuracy of the experimenter's judgments of eye movements off center, the adult fixated centrally and then either made no eye movement or moved her eyes 0.5°, 1°, 1.5°, 2°, 2.5°, or 3° above or below center on 65 randomly ordered trials. The experimenter detected all of the eye movements off center and judged their direction and size correctly 92% of the time.

There were 160 trials at each of three SOAs (100, 400, 800 msec): 104 valid trials, during which the central arrow correctly signaled the location of the upcoming target; 20 invalid trials, during which the arrow signaled the incorrect location of the upcoming target; 20 neutral trials, during which the diamond provided no information about the location of the upcoming target; and 16 catch trials, during which no target appeared following the cue. The catch trials provided a measure of false positive responding, that is, the participant's tendency to push the button simply because a target was expected. The cue and target remained on the screen until a keypress occurred or until 2 sec elapsed. Half of the targets appeared in the upper visual field and half in the lower visual field (in a random order).

We counterbalanced the order of testing across eyes. For patients with the same acuity in both eyes as measured by the Sheridan-Gardiner single letter test, we tested the right eye first in half the cases. When the acuity was unequal, we tested the eye with the better acuity first for half the cases and the eye with the worse acuity first for the remaining cases. This was to balance across eyes any benefits from learning or impairments from fatigue. All but one patient completed the testing in one day. That patient, for whom the computer had malfunctioned on the first day, completed the entire protocol on the second day.

The data from patients were compared to those of normal participants (Goldberg et al., 2001) who had been tested in the same way except that they did not wear trial frames with corrective lenses because the natural lens of their eye was able to accommodate for the testing distance. Half of the normal participants had been tested with the right eye, and half with the left eye.

Data Analyses

The dependent variable was reaction time, measured as the latency between the appearance of a target and the participant's response. Before analyzing the data, we corrected the data for reaction time bias that can inflate the size of a validity effect. Both the mean and median are biased toward long reaction times when the distribution of reaction times is positively skewed (as it usually is) and when the sample of reaction times is small (as is the case for invalid trials; Miller, 1988, 1991). When the sample is larger, as it is in the valid condition, there is less bias. Thus, with positively skewed distributions, estimates of the size of the validity effect (the difference in reaction time between the invalid and valid conditions) based on either the mean or the median are inflated, and would be inflated more in patients than in normal controls if their distributions of reaction times are more variable. Although there are a number of statistical procedures for removing outliers (e.g., Stevens, 1984), Van Selst and Jolicoeur (1994) described a procedure designed specifically for removing the bias from comparisons of conditions with unequal numbers of trials, such as the comparison of invalid and valid trials. Specifically, it uses a moving criterion to remove outliers from the mean, with the criterion adjusted relative to the number of observa-

tions being averaged. For example, for a condition with 50 observations (a typical number for a valid condition), reaction times lying 2.48 *SD* or more away from the mean are eliminated whereas, for a condition with only 10 observations (e.g., an invalid condition), reaction times lying 2.17 *SD* or more away from the mean are eliminated. Van Selst and Jolicoeur showed that this procedure removes the bias from the comparison of the means.

Trials on which participants made eye movements or anticipated the appearance of the target were eliminated before applying the outlier elimination procedure. The percentage of scores eliminated by the moving criterion was 3.30% for patients and 3.11% for normal participants.

To provide the best estimate of the effects of deprivation on the development of attention we averaged the results for the two eyes of each patient in each condition. The data were analyzed with an analysis of variance (ANOVA) that had one between-subject factor, Group (Patient, Normal), and three within-subject factors, SOA (100, 400, 800 msec), Cue (valid, invalid, neutral), and Visual Field (down, up).

Results

Patients made few eye movements or anticipations of the target, and false alarms (responses on catch trials) were no more common than in the normal comparison group (see Table 2).

The ANOVA showed a significant interaction between Group, Cue, and SOA, $F(4, 120) = 2.6$, $p < .05$, and significant main effects of Cue, $F(2, 60) = 15.64$, $p < .001$ and Field, $F(1, 30) = 5.28$, $p < .05$. The main effect of field arose because participants were faster to respond to targets in the upper visual field ($M = 424.9$ msec) than in the lower visual field ($M = 435.5$ msec). None of the other effects or interactions were significant.

Figure 1 shows the mean reaction time on valid, neutral, and invalid trials for patients versus normals at each SOA.

TABLE 2
Mean Percentage of Trials With Eye Movements, With Anticipations, or Responses on Catch Trials in Experiments 1 and 2

	% Eye Movements		*% Anticipations of the Target*		*% Responses on Catch Trials*	
Experiment	*M*	*SE*	*M*	*SE*	*M*	*SE*
1						
Patients	3.1	0.3	0.4	0.1	10.7	1.7
Normal controls	3.3	0.4	0.7	1.0	16.4	2.9
2						
Patients	6.3	0.9				
Age-matched controls	5.2	1.5				

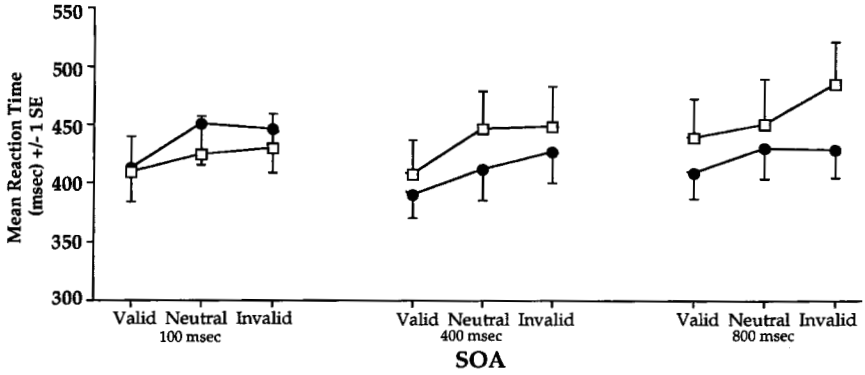

FIGURE 1 Mean reaction time as a function of cue type and stimulus onset asynchrony (SOA) for normals (open squares) and patients (filled circles). Means are connected to facilitate comparisons of the conditions at each SOA.

To analyze the three-way interaction between Group, Cue, and SOA, we conducted separate analyses at each SOA. At the 100 and 400 msec SOAs, there were main effects of cue (ps < .001) and no interaction with Group: overall, participants responded faster on valid trials than on neutral or invalid trials (Tukey posttests, ps < .01), but not significantly faster on neutral trials than on invalid trials (Tukey posttest, $p > .05$). Only at the 800 msec SOA was the Group × Cue interaction marginally significant, $F(2, 60) = 2.52$, $p < .10$. Analyses of simple effects showed that the effect of Cue was significant for normal participants, $F(2, 60) = 8.77$, $p < .001$, and nonsignificant for patients, $F(2, 60) = 2.09$, $p > .10$.

Figure 2 shows the size of the validity effect (mean reaction time on valid trials minus mean reaction time on invalid trials) for each patient at each SOA. The data are plotted as a function of the duration of the deprivation from birth and referenced to the mean validity effect of normal participants at the three SOAs. In Panels A and B (100 and 400 msec SOAs), most dots are above zero (no validity effect) and distribute equally above and below the normal mean. In Panel C (800 msec), the majority of dots fall below normal values and many even fall below zero, especially when deprivation lasted past 4 months of age.

Discussion

Under these conditions, patients who had been binocularly deprived during infancy performed normally at 100 and 400 msec SOAs; at those SOAs, the validity of the cue altered their reaction time normally, even when the deprivation had lasted for more than the first 4 months of life. Those results imply that the ability to use endogenous cues to covertly orient attention can develop even when the first visual in-

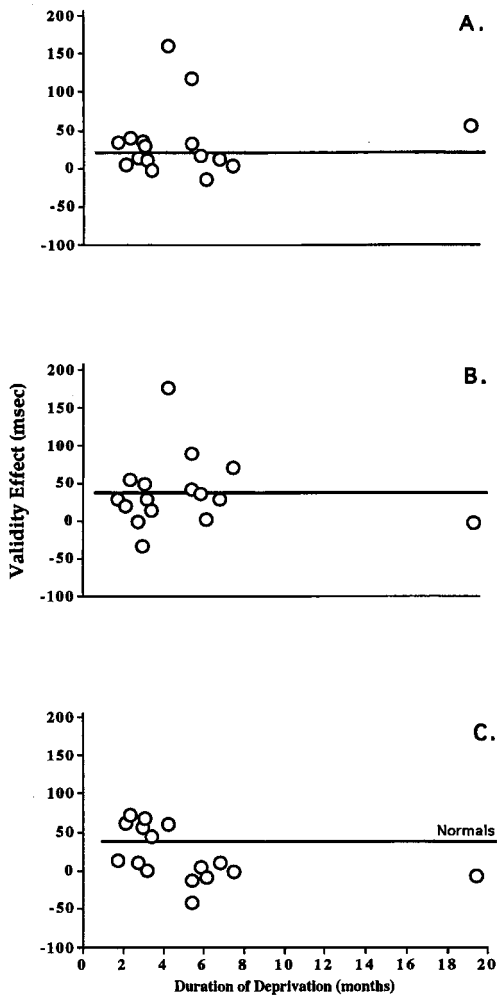

FIGURE 2 Validity effect (invalid–valid reaction time) at the 100 msec stimulus onset asynchrony (SOA) (Panel A), 400 msec SOA (Panel B), and 800 msec SOA (Panel C) for patients as a function of the duration of deprivation (months). The horizontal lines indicate the normal value at each SOA. Each dot represents the result for the mean of the two deprived eyes.

put is delayed past the age at which a number of visual–spatial attentional skills, including the ability to easily disengage attention, first emerge (Atkinson et al., 1992; Canfield & Haith, 1991; Haith et al., 1988; Hood, 1995; Hood & Atkinson, 1993; Johnson, 1990, 1995; Johnson et al., 1991, 1994; Johnson & Tucker, 1996). However, delayed visual input was not adequate to allow the development of normal

performance at the 800 msec SOA. In particular, the validity of the cue presented 800 msec before the target had no effect on patients' reaction times, especially when there had been more than 4 months of deprivation. The abnormality is likely to reflect a problem with attention and not some difficulty in understanding the task because the patients had a low rate of false alarms and performed normally at two SOAs. Instead, binocular deprivation from birth appears to interfere with the development of the ability to maintain attention on a peripheral location, especially if the deprivation lasts past 4 months of age.

The results can be understood in the framework developed by Nakayama and MacKeben (1989) who, on the basis of a series of visual search tasks, distinguished between a sustained and a transient component of attention. Our results suggest that the patients, especially those with longer deprivation, have a deficit in the sustained component of attention. However, the transient component of attention is able to function normally, at least under some conditions. Nakayama and MacKeben argued that the sustained component of attention is mediated at higher cortical levels (e.g., V4, inferotemporal, parietal, and frontal cortex) rather than at lower levels (e.g., superior colliculus, V1). Moreover, a recent functional magnetic resonance imaging study by Coull et al. (2000) found activation of right posterior extrastriate cortex to unexpected targets at short SOAs, but activation of the right frontal areas (including premotor cortex, and ventrolateral and dorsolateral prefrontal cortex) at long SOAs. Thus, early visual deprivation might prevent the development of normal attentional networks involving higher cortical levels, particularly the right prefrontal cortex.

EXPERIMENT 2

In Experiment 1, patients with early binocular deprivation performed normally at the 100 and 400 msec SOAs, but not at the 800 msec SOA. In Experiment 2, we used a more difficult task that might reveal additional difficulties even at the 400 msec SOA. Specifically, we made the task more difficult by requiring discrimination between two target shapes (a circle and a plus sign) and the ignoring of distractors. To better measure any deficits, we recorded accuracy as well as reaction time.

In this task, it is presumed that the participant has to both shift attention before the target appears and then ignore distracting stimuli. On valid trials, the participant can use information provided by a central arrow to shift attention to the location where a target will appear surrounded by distracting stimuli. The participant may then narrow the focus of attention on the location of the target to ignore the distractors and recognize which target appears. The participant's task may be harder on invalid trials, on which attention is shifted to the wrong location, and the participant has to disengage attention, move attention to the location of the target, and focus attention on the target while ignoring the distractors. As described in the

Introduction, the ability to ignore distractors develops slowly during childhood, not reaching adult levels until after age 10 (Enns, 1990; Enns & Akhtar, 1989; Enns & Cameron, 1987; Goldberg et al., in press; Lane & Pearson, 1982; Plude et al., 1994; Ridderinkhof & van der Molen, 1995; Strutt et al., 1975; Tipper et al., 1989; Well et al., 1980).

Method

Participants. The final sample consisted of 15 patients treated for bilateral congenital cataract from the same sample reported in Experiment 1 and 15 age-matched children, all of whom passed a visual screening exam. Detailed characteristics of the patients are provided in Table 1. The patients had a mean duration of deprivation of 4.5 months (range 1.8–7.6 months). At the time of the test, their mean age was 12.8 years (range 8.9 years–17.2 years). Each patient was matched to a child of the same age (± 3 months) with a normal visual history. One additional normal child was excluded because she failed the visual screening exam.

Apparatus and stimuli. The apparatus and cues were the same as in Experiment 1. A plus and a circle were chosen as target shapes because children treated for cataract can easily discriminate these shapes (Maurer et al., 1989). The target was flanked by distractors that were either compatible (the same shape) or incompatible (the other shape) with the response to the target (Eriksen, 1995). Targets were $8^o \times 8^o$ and were centered 9.0^o (5.7 cm) directly above or below the center of the central stimulus. The nearest edge of the shape was 5^o (3.2 cm) from the center of the central stimulus. The distractor shapes were the same size as the targets and were aligned horizontally beginning 1.6^o (1.0 cm) from the nearest edge of the target. The nearest edges of the distractors were 7.4^o (4.7 cm) from the center of the central stimulus.

Procedure. The procedure was the same as in Experiment 1 with the following exceptions. We tested each eye of each patient and each matched control. Participants were asked to discriminate whether a target was a plus sign or a circle by pressing one of two designated buttons on a keyboard. Participants were tested only at a 400 msec SOA and were provided with auditory feedback ("great" or "whoops") regarding the accuracy of their responses. The feedback was auditory so as not to interfere with the processing of visual information.

Participants were tested on a total of 200 trials with one of two targets (plus sign or circle) presented randomly on each trial under a combination of one of three cue conditions (valid, invalid, or neutral) and one of two distractor conditions (compatible distractors or incompatible distractors). On valid trials ($n = 120$), the arrow

pointed to the location of the upcoming target. On invalid trials (n = 40), the arrow pointed to the location where the target would not appear. On neutral trials (n = 40), a diamond matched in area to the arrows provided no information about the target's location. On half of the valid, invalid, and neutral trials (n = 100 trials total), the target was presented with compatible distractors (e.g., circle flanked by circles). On the remaining half of the trials (n = 100 trials total), the target was surrounded by distractors incompatible with the response to the target (e.g., circle flanked by plus signs). On half of the trials the target was a circle, whereas on the other half of the trials the target was a plus. On half of the trials the target appeared in the upper visual field and on the other half of the trials in the lower visual field.

Data Analyses

Trials with eye movements were excluded from the analyses. Mean reaction times were calculated for trials with correct responses under each condition. As in Experiment 1, outliers in reaction time were eliminated with a moving criterion adjusted relative to the number of trials per condition (Van Selst & Jolicoeur, 1994). The percentage of reaction time data eliminated with the outlier elimination procedure was 2.43% for patients treated for bilateral cataract and 2.44% for the age-matched comparison group.

Analyses were based on the mean of the two eyes from each participant in each condition. We conducted separate ANOVAs on mean reaction time and accuracy with one between-subject factor, Group (patient, age-matched control), and three within-subject factors, Distractor type (compatible, incompatible), Cue type (invalid, neutral, valid), and Visual field (lower, upper).

Results

Patients treated for bilateral congenital cataract were able to perform this relatively complex task as well as age-matched controls, with few eye movements (Table 2) and a mean accuracy of 97.3%, compared to the controls' accuracy of 97.1%. The ANOVA on accuracy indicated that patients did not differ significantly from age-matched controls for any condition. Overall, participants were significantly more accurate on trials with compatible distractors (97.2%) than on trials with incompatible distractors (96.5%) (main effect of distractor type, $F(1, 28) = 4.31$, $p < .05$). None of the other main effects or any of the interactions were significant.

The results from the ANOVA on reaction time indicated there was a significant interaction between Group, Distractor, and Cue, $F(2, 56) = 3.25$, $p < .05$. To examine this three-way interaction, we conducted separate ANOVAs for invalid, neutral, and valid trials. For neutral and valid trials, there were only main effects of

Distractor (because both patients and normal controls were slower on trials with incompatible distractors). The ANOVA on invalid trials indicated a significant Group by Distractor interaction, $F(1, 28) = 4.52$, $p < .05$, and a significant main effect of Distractor, $F(1, 28) = 32.9$, $p < .001$. Figure 3 illustrates that on invalid trials, patients were slowed more than age-matched controls by incompatible distractors (analyses of simple effects, $p < .001$).

There was also a significant Group by Cue by Field interaction, $F(2, 56) = 3.24$, $p < .05$. To examine the source of the interaction, we conducted separate ANOVAs for the upper and lower visual fields using Cue and Group as factors. The ANOVA in the upper visual field indicated a marginally significant Group by Cue interaction, $F(2, 56) = 2.91$, $p = .06$, and a significant main effect of Cue, $F(2, 56) = 14.7$, $p < .0001$. The ANOVA in the lower visual field indicated a significant main effect of Cue, $F(2, 56) = 14.24$, $p < .001$, but no significant Group by Cue interaction, $F(2, 56) = 0.67$, $p > .50$.

Figure 4 illustrates that patients showed a normal validity effect when the target appeared in the lower visual field. In the upper visual field they showed a larger than normal validity effect (analyses of simple effects, and Tukey posttest, ps < .05). The overall pattern illustrated in Figure 4 suggests a deficit in shifting attention toward the upper visual field. In patients, correct cueing to the upper visual field did not decrease reaction time relative to neutral cueing (A vs. B). Incorrect cueing to the lower visual field greatly increased reaction time to recognize a target in the upper visual field (B vs. C). Incorrect cueing to the upper visual field did not increase reaction time (D vs. E). All three of these comparisons suggest a deficit in using the central arrow to guide covert orienting to the upper visual field.

There were no other significant interactions, no main effect of group, and the significant main effects were qualified by the interactions previously described.

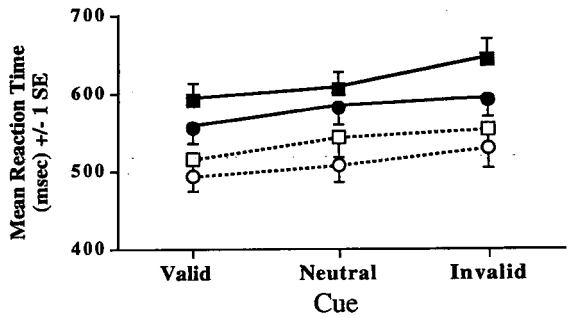

FIGURE 3 Mean reaction time as a function of cue type on trials with incompatible distractors (squares) and compatible distractors (circles) for patients (filled symbols) and age-matched controls (open symbols). Means are connected to facilitate comparisons of the effect of cue for the different conditions.

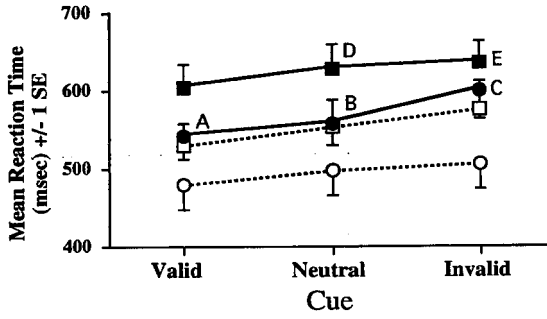

FIGURE 4 Mean reaction time as a function of cue type on trials with the target in the lower visual field (squares) and upper visual field (circles) for patients (filled symbols) and age-matched controls (open symbols). Means are connected to facilitate comparisons of the effect of cue for the different conditions.

Figure 5 shows the relation between the duration of deprivation and the size of the two effects on which the patients differed from the normal comparison group: the size of the validity effect in the upper visual field (top panel) and the effect of incompatible distractors (i.e., the difference in reaction time between trials with incompatible and compatible distractors) on invalid trials (bottom panel). Both graphs indicate larger effects in patients than in the normal comparison group (most circles lie above the horizontal line indicating the normal mean) but no obvious relation to the duration of deprivation.

Discussion

In Experiment 2, incompatible distractors slowed patients' reaction times on valid trials and on neutral trials no more than normal. However, on invalid trials patients were slowed more than normal by incompatible distractors. Interestingly, this pattern of effects is similar to one demonstrated during normal development, with exogenous cues: On valid trials, 5-, 7-, and 9-year-olds are influenced like adults by distractors. However, on invalid trials they are slowed more than adults by incompatible distractors (Akhtar & Enns, 1989). The effect of distractors on invalid trials suggests that deprivation interferes with the slowly developing ability to disengage attention from an endogenously miscued location, move attention to a new location, and then narrow the focus of attention on a target while ignoring distractors.

Also in Experiment 2, regardless of the duration of deprivation, patients appeared to have difficulty shifting attention covertly toward the upper visual field. This suggests that the normal development of the ability to shift attention to specific visual–spatial locations is influenced by experience early in life. In the Gen-

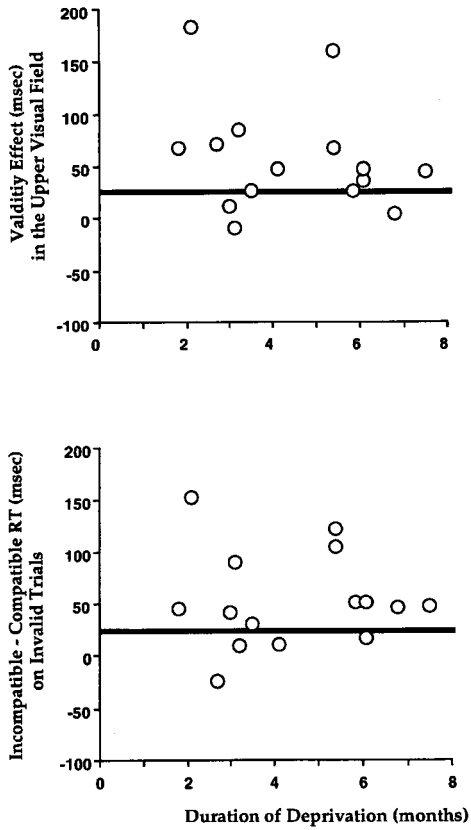

FIGURE 5 The top graph illustrates for patients the size of the validity effect for targets in the upper visual field plotted as a function of the duration of deprivation. The bottom graph illustrates the effect of distractors on invalid trials, plotted as a function of the duration of deprivation. The effect of distractors was measured as the difference in reaction time between trials with incompatible distractors and compatible distractors. The horizontal lines indicate the normal value in each graph. Each dot represents the result for the mean of the two deprived eyes.

eral Discussion section, we consider what these findings suggest about the neuropsychological effects of early binocular visual deprivation on the development of the pathways controlling visual–spatial attention.

GENERAL DISCUSSION

In Experiment 1, patients performed normally when the cue preceded the target by 100 or 400 msec. More interesting is the finding that patients, especially those with

more than 4 months of deprivation, responded abnormally when the cue preceded the target by 800 msec; reaction times were the same whether the cue was valid or invalid. In Experiment 2, patients differed from age-matched controls in (a) being slowed more by incompatible distractors on invalid trials, and (b) tending to show a larger than normal effect of the validity of the cue preceding targets in the upper visual field. Together, these findings suggest that the normal development of attention is influenced by experience early in life.

The deficits are unlikely to arise from sensory or motor deficits which are prevalent in children treated for bilateral congenital cataracts. Their poor acuity and often unsteady eye movements did not interfere with good performance under some conditions. Also, their deficits in either experiment were not correlated with acuity. Specifically, in Experiment 1, deficits at the 800 msec SOA were related to the duration of deprivation and not to acuity, $r(16) = -.29$, $p > .10$. In Experiment 2, there was no significant correlation between acuity and the size of the validity effect in the upper visual field, $r(15) = .02$, $p > .10$, or the slowing by incompatible distractors on invalid trials, $r(15) = .06$, $p > .10$. Rather than being artifacts of sensorimotor deficits, the deficits we found in these experiments appear to arise from abnormalities in visual–spatial attention caused by early binocular deprivation.

The results from Experiment 1 were different from what we would predict if the deficits following early visual deprivation were similar to that following unilateral parietal damage: At the 800 msec SOA, patients in Experiment 1 showed no validity effect, whereas adult (unilateral) parietal patients typically show an abnormally large validity effect. (There have been no studies of endogenous cueing following adult bilateral damage to parietal cortex.) Here we speculate that infantile damage to the parietal cortex from either visual deprivation or brain injury does not have the same consequences as does damage in adulthood. Studies of language have documented that brain injury early in life to Broca's or Wernike's areas does not have the same consequences as injury to the same areas in adulthood (e.g., Bates, Thal, & Janowsky, 1993; Thal et al., 1991). Studies of attention following infantile injury to various parts of the brain suggest that posterior systems (including the parietal cortex) play a smaller role than in adults, whereas anterior systems (including frontal cortex) play a larger role (e.g., Craft, Schatz, et al., 1994; Craft, White, et al., 1994; Johnson et al., 1998). Supporting evidence from studies measuring event-related potentials suggests that the attention of young infants, compared to adults, is mediated more by the frontal cortex and less by the parietal cortex (Csibra et al., 1998). After infantile parietal lesions, the frontal cortex appears able to continue to play that role. If this argument about the neural mediation of attention during infancy is correct, then binocular deprivation might exert its effect by causing impairment to frontal brain regions, the input to them from parietal cortex (Petrides & Pandya, 1999), and the ability of the brain to reorganize the mediation of attention after infancy.

Impairments in ignoring distractors in Experiment 2 are consistent with this hypothesis that early deprivation might affect the development of attention, not

just through effects on the visual responsiveness of parietal neurons, but also through effects on frontal systems and the interconnections between frontal and parietal systems (Petrides and Pandya, 1999). Studies of adults with frontal lesions indicate that the frontal cortex is involved in focusing and sustaining attention, in ignoring distracting information, and in inhibiting prepotent responses (Denckla, 1996; Duncan, 1986; Godefroy, Lhullier, & Rousseaux, 1996; Husain & Kennard, 1997; Richer & Lepage, 1996; Rueckert & Grafman, 1996). Behavioral, anatomical, and positron emission tomography studies indicate that the frontal cortex, although functioning from an early age (Bell & Fox, 1994; Diamond, 1985; Diamond & Goldman-Rakic, 1989), has a more protracted developmental period than the parietal, occipital, and temporal cortices (Chugani, 1994), lasting until early adolescence (Chugani, 1994; Huttenlocher, 1994; Huttenlocher & Dabholkar, 1997; Levin et al., 1991). As would be expected, we found in a separate study that normal 10-year-olds are not adult-like in ignoring distractors on this task (Goldberg et al., 2001). The slow development of the frontal cortex makes it especially vulnerable to disruption from early deprivation.

Patients' tendency to ignore the upper visual field in Experiment 2 is also what would be predicted from frontal damage. Previc's (1990) comprehensive summary indicated that lesions to the parietal cortex of monkeys lead to deficits in peripersonal space—the lower visual field where, like monkeys, we reach for objects—whereas lesions to the prefrontal cortex lead to deficits of the upper visual field, or extrapersonal space. There is converging evidence for the role of the parietal cortex in attending to the lower visual field from a case study of a patient with bilateral parietal damage tested with exogenous cues: Although she performed normally in the upper visual field, she was unable to benefit from a cue in the lower visual field and hence, unlike the patients we tested, had no validity effect for targets appearing in the lower visual field (Verfaellie et al., 1990).

These are the first studies to examine the role of experience in the normal development of attention. The findings show that deprivation, especially when it lasts past 4 months of age, disrupts the ability to maintain attention. Binocular deprivation of any duration was associated with impairments in the abilities to shift attention covertly toward the upper visual field and to ignore distracting information. Together these findings indicate that the normal development of attention is sensitive to experience early in life and add to the evidence that deprivation especially disrupts aspects of vision that are immature at birth and that develop slowly during childhood (Maurer et al., 1989). We speculate from the pattern of deficits that the frontal cortex, probably including its interconnections with the parietal cortex, is able to develop normally during childhood only if it received visual input during early development. Presumably that input sets up the cortical architecture that can be modified by subsequent experience.

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