INFLUENCE OF BINOCULAR DEPRIVATION ON THE DEVELOPMENT OF ATTENTION

By

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ATTENTION OF NORMAL AND VISUALLY DEPRIVED CHILDREN

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I examined the influence of binocular deprivation on the development of visual spatial attention in humans. I began by testing normals, in order to work out procedures appropriate for monocular tests of patients (aged 8 to 20 years) who had been treated for bilateral congenital cataracts during infancy. There were two reaction time tasks (Eriksen 1995; Posner, Nissen, & Ogden, 1978): 1. Detection–Participants pushed a button as soon as they detected a target that was presented in a cued, miscued, or noncued peripheral location and that appeared 100, 400, or 800 msec (SOAs) after a central cue; 2. Discrimination with Distractors–Participants indicated which of two shapes appeared in the periphery 400 msec after a central cue, with those shapes surrounded by compatible or incompatible distractors.

On task 1, I compared 32 eyes of 16 patients (mean duration of deprivation = 5.1 mos; range = 1.8 - 19.5 mos) to three groups of normals (8, 10 years, adult, N = 24 per grp). Covert orienting was adult-like by 8 years of age. Patients performed normally when the cue appeared 100 or 400 msec before the target. Patients with more than 4 months of deprivation, unlike normals and patients with shorter deprivation, responded no more quickly on trials with valid cues than on those with invalid cues 800 mec before the target.

On task 2, I compared 30 eyes of 15 patients (mean duration of deprivation = 4.5 mos; range = 1.8 - 7.6 mos) to 15 age-matched controls, and 24 normal 10-year-olds to 24 adults. On invalid trials, patients were slowed more than age-matched controls by incompatible distractors. As on Task 1 at a 400 msec SOA, patients showed a normal validity effect in the lower visual field. In the upper visual field, there was a larger-than-normal validity effect. As in Task 1, normal 10-year-olds were adult-like in the ability to shift attention covertly; however, they were slowed more than adults by incompatible distractors.

Together these findings indicate that the normal development of attention is sensitive to experience early in life: Deprivation disrupts aspects of the attentional system that are involved in shifting and maintaining attention. The results also add to the evidence that deprivation especially disrupts abilities that are immature at birth and that develop slowly during childhood (Maurer et al., 1989).

These studies on the normal development of attention showed that while covert orienting is developed by 8 years of age, at least under some conditions, the ability to ignore distracting information is not yet fully developed even by 10 years of age. This result suggests that the parietal system, involved in the ability to shift attention covertly in adults, and the frontal system, involved in the ability to ignore distractors, develop at different rates.

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CHAPTER 1

INTRODUCTION

Adults usually shift their attention and move their eyes to the location of a change of stimulation (e.g., a flashing light in the periphery) or to information they wish to process (e.g., the face of one person in a group photograph). To study the attentional component in isolation, participants are asked to shift their attention from one place to another in the visual field without making an eye movement. Shifting attention without eye movements is often referred to as covert orienting. Adults can indeed shift attention to another location without moving their eyes (e.g., shift attention to a child playing while looking at the adult with whom they are speaking). Objects at locations where adults are attending covertly are processed more accurately and quickly than objects at other locations (Posner, Nissen, & Ogden, 1978; Posner, 1980; Posner, Cohen, & Rafal, 1982; Posner, 1988). It is advantageous to be able to attend selectively to events in the environment and to be able to shift attention to important information. For example, a child who can attend efficiently to a teacher's instructions in the classroom may be at an advantage at school. These skills are not present at birth but develop post-natally (reviewed in Ruff & Rothbart, 1996).

In this thesis, I examine the influence of visual deprivation on the development of attention in humans. I compare the covert orienting ability of children who were visually deprived early in life to that of children who had normal visual experience. This research examines issues important to both developmental psychology and cognitive neuropsychology. The study of normal children examines the component processes involved in the normal development of attention, a topic on which there is only a small literature. The exploration of deficits in attention following abnormal experience, combined with evidence from animal models on the neural structures involved, affords us the opportunity to draw inferences about the neural structures that play a key role in attention.

This is the first study to examine the influence of visual deprivation on the development of attention in humans. Previous studies have examined the influence of visual deprivation on sensory visual perception and have studied it by looking at animals visually deprived by eyelid suture (reviewed in Boothe, Dobson, & Teller, 1985) and humans visually deprived by cataracts (reviewed in Maurer & Lewis, 1993). In humans, the effects of visual deprivation from a dense and central cataract are comparable to eyelid suture in animals because, in both types of deprivation, mainly diffuse light reaches the retina. In humans, the cataract is extracted surgically, and the aphakic eye (i.e., without a lens) is fitted with a contact lens to allow approximately normal visual input; this procedure is analogous to opening the eyelid after lid suture. We can then compare subsequent development after deprivation to normal development.

For this thesis, I studied children who were treated for dense and central cataracts that were present in both eyes from birth. Treatment occurred early in life by surgical removal of the cataract and fitting with contact lenses so that the retina would receive approximately normal visual input. Variations in the age at which the treatment occurred allowed us to examine effects of the duration of deprivation on the development of attention. Drs. Maurer, Lewis, and Brent have been following these patients at The Hospital for Sick Children in Toronto and have good documentation on the nature of the cataract, the age of treatment, any subsequent complications, and the development of many aspects of vision (e.g., visual acuity, contrast sensitivity, peripheral vision, color vision).

Organization of Thesis

I began this research with adults (Chapter 2) in order to work out procedures appropriate for testing children. I chose a subset of conditions from Chapter 2 and tested them with normal children to (a) provide a comparison for deprived eyes and to (b) find out more about the nature of normal development (Chapter 3). In Chapter 4, I report tests of children treated for binocular deprivation whose data were compared to those from normals (from Chapter 3) to determine whether experience is important for the normal development of attention. In Chapter 5, I present the rationale for developing a second, more complex task. I then present results for this task from (a) normal 10-year-olds and adults and (b) patients treated for bilateral congenital cataract and age-matched controls. In the last chapter, I summarize the behavioral findings and discuss the developmental and neurophysiological implications of the results. In the rest of this chapter, I will provide a rationale for the hypothesis that experience is important for the normal development of attention.

The Ability to Orient Attention Develops

One reason to suspect that experience may be important for the normal development of attention is that it is immature at birth and develops slowly during childhood (Atkinson, Hood, Wattam-Bell, & Braddick, 1992; Hood, 1995; Hood & Atkinson, 1993; Johnson, Posner, & Rothbart, 1991; Johnson & Tucker, 1996; reviewed in Ruff & Rothbart, 1996). I will briefly discuss this literature here; a more complete summary is provided in Chapter 3.

Under some conditions, prior to four months of age, infants appear to have great difficulty moving their eyes away from a fixation object and toward another object (Atkinson et al., 1992; Hood & Atkinson, 1993; Johnson et al., 1991)–a phenomenon that has been called "sticky fixation." Johnson et al. (1991) attracted infants' fixation to a colorful, rotating shape presented on a central monitor and recorded the direction and latency of babies' orienting to a target-shape 34 degrees to the left or right of center. Two- and 3-month-old babies oriented away from center and toward the peripheral target on fewer than half of the trials within the 8 seconds allowed. However, 4-month-olds oriented toward the peripheral target on almost every trial, usually within one second. The authors conclude that "only the 4-month-old group could easily and consistently disengage their gaze from the central stimulus to orient toward a simultaneously presented peripheral target (page 339)." These findings suggest that under such testing conditions, babies can more easily disengage their gaze around 4 months of age than at younger ages.

The results from Johnson et al. (1991), as well as other developmental studies using eye movements (e.g., Atkinson et al., 1992; Hood & Atkinson, 1993), leave room for two possible interpretations: (1) Young infants have difficulty disengaging attention and/or (2) young infants have difficulty programming eye movements used to measure shifts of attention. To circumvent this problem, researchers have developed procedures to test the ability to shift attention covertly.

A procedure that has been standardized in adults–the Posner Paradigm (Posner et al., 1978)–encourages participants to use a cue to shift attention to a peripheral location without making an eye movement. Participants signal as soon as they detect a target in an expected or unexpected location. Studies of children using this procedure indicate that by 3 - 4 years of age they can shift attention covertly; that is, like adults, children respond more quickly to a target at a correctly cued location than to a target at an incorrectly cued location (Akhtar & Enns, 1989; Brodeur, 1990, 1993; Enns & Brodeur, 1989; Pearson & Lane, 1990). However, the effect of incorrect cueing on reaction time is greater in children than it is in adults. This pattern suggests that children have a problem with one or more component(s) of attention. Although none of these studies has pinpointed the age at which covert attention becomes adult-like, the evidence that young babies have sticky fixation, combined with the evidence that even young children are harmed more than adults by incorrect cueing, suggests that attention may be sensitive to abnormal visual experience during development.

The Role of Experience in Development

One reason to suspect that visual experience might be important for the normal development of attention is that studies in humans, just as in animals, show that early visual deprivation has larger adverse effects on visual abilities that are immature at birth (e.g., symmetrical OKN, stereopsis, visual acuity, contrast sensitivity and peripheral vision) than it does on visual abilities that are relatively mature at birth (e.g., the discrimination of grossly different forms) (Maurer et al., 1989). Because the ability to shift attention develops slowly (reviewed in Ruff & Rothbart, 1996), we would expect that the ability to orient attention might also be affected adversely by early visual deprivation.

The effects of visual deprivation on sensory visual development have been studied prospectively in animals by varying the time during development when the deprivation began and how long it lasted (e.g., Crawford, Harwerth, Smith, & von Noorden, 1993; Harwerth, Crawford, Smith, & Boltz, 1981; Movshon & Van Sluyters, 1981). These variables have also been studied in humans by examining children who had deprivation from birth, or who were deprived of visual input sometime after birth (Maurer & Lewis, 1993), and by comparing children whose deprivation began at the same time but differed in its duration. For some visual functions, deficits are worse following longer than shorter periods of early deprivation (i.e., when the surgery and optical correction are delayed for a cataract present from birth).

The development of visual acuity nicely illustrates the effects of the timing and duration of deprivation. Snellen acuity is worse after relatively long periods of deprivation (more than 5 months) than after shorter periods of deprivation (Maurer et al., 1989). It is also worse in children binocularly deprived from birth than in children who had had normal visual experience until dense and central cataracts developed in both eyes some time after six months of age. "Moreover, there appears to be an interaction between the duration and timing of deprivation, such that the worst acuity was shown by children who had had long periods of deprivation beginning at birth" (Maurer et al., 1989, page 159).

Research from monkeys binocularly deprived from shortly after birth indicates that binocular deprivation during the first year of life dramatically reduces the sensitivity of parietal cells 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). In addition, there is an increase in the percentage of cells activated by minor motor movements, or by feeling or touching objects. This result suggests that when visual input to the parietal cortex is absent early in life, its neural space is taken over by input from another modality. These results are discussed in more detail in Chapter 4.

Several lines of evidence indicate that the parietal cortex plays a major role in attention. Here I provide one example of each type of evidence gathered from neuroimaging in normal adults (e.g., Corbetta, Miezin, Shulman, & Petersen, 1993; Petersen, Corbetta, Miezin, & Shulman, 1994), recording single cell properties in monkeys (e.g., Mountcastle, Lynch, Georgopoulos, Sakata, Acuna, 1975; Steinmetz, & Constantinidis, 1995), and documenting deficits in humans after strokes (e.g., Posner, Walker, Friedrich, & Rafal, 1984). A more complete review is provided in Chapter 4.

Studies of human adults using Positron Emission Tomography (PET) show greater increases in blood flow in the parietal region compared to other cortical regions (e.g., frontal, visual, motor) when participants' attention is covertly shifted to a peripheral location in response to a peripheral or a central cue (Corbetta et al., 1993; Petersen et al., 1994). Single unit recordings from monkeys' posterior parietal cortex indicate an increase in firing relative to spontaneous activity when the monkey maintains visual fixation on a central target while attending covertly to a peripheral target within a cell's receptive field (Steinmetz & Constantinidis, 1995; Steinmetz, Connor, Constantinidis, & McLaughlin, 1994). Tests of monkeys (Petersen & Robinson, 1986) and human adults with unilateral parietal damage (Baynes, Holtzman, & Volpe, 1986; Petersen, Robinson, & Currie, 1989; Posner et al., 1984; Posner, Walker, Friedrich, & Rafal, 1987) show that reaction times on Posner tasks are longer than normal on incorrectly cued trials when attention must be *disengaged* from an attended location and then directed toward the target at an unattended location (Posner et al., 1982; Posner, et al., 1984; Posner, et al., 1987; Posner, 1988, 1990). Specifically, these patients have extremely long reaction times when attention is cued to their *good* visual field (ipsilateral to the parietal lesion) and they are required to shift attention from their good visual field to detect a target that is presented in the *bad* visual field (contralateral to the parietal lesion).

The adverse effects of binocular deprivation on monkeys' parietal cortex, combined with evidence for the role of the parietal cortex in covert shifts of attention, makes it reasonable to hypothesize that experience may be important for the normal development of attention. In the absence of such experience in humans–because of bilateral congenital cataracts–attention might not develop normally and, following treatment, behavior on attention tasks might be similar to that shown in patients with parietal damage. Later in the thesis, I will consider the possible influence of other structures on the development of attention in normal children and following treatment for congenital cataract (e.g., superior colliculus, Goldberg & Wurtz, 1972a, b; pulvinar, LaBerge, 1995a, b; temporal cortex, Robertson, Lamb, & Knight, 1988; Ungerleider & Mishkin, 1982; occipital cortex, Rafal, 1996; and frontal cortex, Fuster, 1988; Posner & Dehane, 1994; Posner & Petersen, 1990; Posner, 1994).

Summary

The literature suggests that early visual experience might be important for the normal development of attention because (1) attention is slow to develop, (2) attention is disrupted by damage to the parietal cortex, and (3) cells in the parietal cortex develop abnormally in the absence of normal visual input. In the following chapters, I describe the first investigation of the effects of visual deprivation on the development of attention in humans and add to the literature on the normal development of attention during childhood.

CHAPTER 2

COVERT ORIENTING IN NORMAL ADULTS

Posner et al. (1978) developed a research paradigm that is used widely to study the mechanisms involved in visual spatial attention, independent of eye movements. On each trial, the participant fixates a central stimulus and signals as soon as a target is detected in the periphery. One of two types of cue precedes the presentation of the peripheral target: an exogenous cue or an endogenous cue. An exogenous cue is typically a visual marker located in the periphery that draws attention automatically. An endogenous cue provides information about the most likely location of the target and is typically presented centrally rather than at or near a target location. Endogenous cues, unlike exogenous cues, do not automatically draw attention to the cued location; instead they prompt the participant to shift attention voluntarily (Jonides, 1981).

Unlike the reflexive, attention-drawing properties of exogenous cues, endogenous cues impose greater demands on cognitive processing as the participant must decode the spatial location signaled by the endogenous cue (e.g., the direction in which an arrow is pointing). Several studies suggest that endogenous cues are more likely than exogenous cues to reveal deficits in attention and higher cognitive function (Posner et al., 1982, 1984). Exogenous cues are more likely than endogenous cues to reveal deficits in sensory processing and midbrain function (Posner & Petersen, 1990). Because endogenous cues are more likely to reveal deficits in attention and higher cognitive function, in this dissertation I used endogenous cues to examine the effects of early visual deprivation on the normal development of attention.[1]

For endogenous cueing, on *valid* trials (usually 80% of the trials), the cue signals the location of the upcoming target. On *invalid* trials (usually 20% of the trials), the cue is misleading and signals a peripheral location where the target does not appear. Normal adults show a *validity effect:* They respond more quickly on valid trials than on invalid trials. Validity effects may be interpreted as follows: Attention is covertly shifted to the cued position, the expected location of the target. Participants detect targets more slowly at unattended locations than at cued locations because they must first *disengage* attention from the expected (attended) location, *move* it to the correct location, and then *re-engage* attention once it reaches the target's location. Hence, reaction times are faster on valid trials than on invalid trials (Posner et al., 1984; Posner, Inhoff, Friedrich, & Cohen, 1987; Posner, 1988, 1990).

[1] It would be interesting for future studies to examine whether there are additional deficits following early visual deprivation that are revealed by exogenous cues.

In studies of spatial orienting, the result of most interest is typically the absolute magnitude of the validity effect. Because the validity effect is defined as the difference between the reaction time on valid trials and the reaction time on invalid trials, it may be viewed as consisting of two additive components: the *benefit* afforded by a valid cue, and the *cost* afforded by an invalid cue. The magnitude of the validity effect may be calculated as the sum of the *benefit* of correct cueing plus the *cost* of incorrect cueing. Thus, a larger validity effect may be the result of increased benefit, increased cost, or both, and the contribution of each may differ in different types of participants or under different testing conditions.

The inclusion of neutral trials can provide an opportunity to examine the contribution of the *costs* (invalid RT - neutral RT) and *benefits* (neutral RT - valid RT) to the overall size of the validity effect. However, in previous studies there have sometimes been problems in interpreting data from neutral trials. Jonides and Mack (1984) warn that to be able to draw conclusions about *costs* and *benefits*, neutral and informative cues must be identical in every way except for information about the location of the upcoming target. In some previous studies, cues used for neutral and informative conditions were not matched on properties such as physical appearance, potential to alert participants that a trial has started, and on ease of encoding (reviewed in Jonides & Mack, 1984). To minimize these problems I matched the area of the cue on valid and invalid trials to the area of the cue on neutral trials. Nevertheless, because I cannot be sure that neutral trials will provide a good baseline, my main analyses will be a comparison of valid versus invalid trials. Only when there are differences between them will I look at the data from neutral trials to see if I can identify the contribution of costs and benefits to the differences.

I first tested normal adults to ensure that the specific procedures adopted for the purpose of testing children and patients treated for cataract, yielded significant validity effects in adults. Given the findings of studies that were summarized in the introduction to this thesis, I predicted that patients treated for cataract would show a pattern of deficit similar to that of parietal patients, that is, larger-than-normal validity effects. Thus, picking a procedure yielding a validity effect in normal adults was a necessary first step. The manipulation of SOA allowed me both to choose the most promising SOAs for the study of patients, and also to study the time course of covert shifts of attention in normal adults.

The magnitude of the validity effect can be influenced both by whether attentional shifts are initiated by exogenous cues or endogenous cues *and* by the length of the time interval between the onset of a cue and the onset of a target (Stimulus Onset Asynchrony, SOA). When attention shifts are initiated by exogenous cues, the observed validity effect increases as the interval between cue and target increases up to an SOA of about 150 msec, after which the validity effect decreases as the interval increases toward 300 to

400 msec (Müller & Rabbitt, 1989; Müller & Findlay, 1988; Shepherd & Müller, 1989). When attention shifts are initiated by endogenous cues, the observed validity effect increases more slowly with increasing SOA than is true for exogenous cues: approximately 300 - 400 msec is needed for the observed validity effect to reach a peak (Müller & Rabbitt, 1989; Remington & Pierce, 1984; Shepherd & Müller, 1989; Shulman, Remington & McLean, 1979; Sorensen, Martin, & Robinson, 1994). These patterns of data suggest different time courses for covert orienting initiated by exogenous and endogenous cues. However, most of the results with endogenous cueing are difficult to interpret because the studies inadvertently confounded the SOA manipulation with differences that could affect underlying psychological processes that were not intentionally manipulated by the experimenter. The participant's task in a Posner cueing paradigm is to respond as quickly as possible to the onset of the target. Reaction time can be affected both by (1) the validity of the cue and (2) by other factors that allow the participant to anticipate when or if a target will appear. SOA may influence reaction time through changes over time in either of these types of influences.

Studies that include trials without targets "catch trials" and intermixed SOAs (e.g., Remington & Pierce, 1984; Shepherd & Müller, 1989; Shulman et al., 1979) are difficult to interpret because at shorter SOAs the participant's anticipation of the target may be stronger than at longer SOAs. For example, consider a block of 100 trials in which there are 64 valid trials, 16 invalid trials, and 20 catch trials. Suppose that two SOAs are tested and that half of the trials in each of these conditions are presented with a short SOA, and half with a long SOA. At the onset of a given trial, the probability of a valid trial is .64, that of an invalid trial is .16, and that of a catch trial is .20. However, during a trial, once the interval of the shorter SOA has elapsed without the onset of a target, these probabilities shift to .53, .13, and .33, respectively. Hence, the probability that the current trial is a catch trial increases from .20 to .33. Consequently, a participant may be less expectant of a target. A smaller validity effect at the longer SOA may reflect a waning of covert attention to the cued location with time (the usual explanation) or decreased vigilance or preparedness because of the decreased likelihood that a target will appear. In general, in studies with intermixed SOAs and catch trials, the perceived likelihood of a target may decrease as the SOA increases, because the probability that the trial is a catch trial increases (e.g., Remington & Pierce, 1984; Shepherd & Müller, 1989; Shulman et al., 1979).

In studies without catch trials and intermixed SOAs (e.g., Sorensen et al., 1994), the predictive value of the cue may also decrease with increasing SOA. At the shortest SOA, the cue may be 80% valid. At the longest SOA, because there are no catch trials, the participant may be well-prepared to respond to the target because the time interval predicts the occurrence of the target 100% of the time. A smaller validity effect at the longer SOA may reflect a waning of covert attention to the cued location with time or an increased vigilance or preparedness because a target will always appear. Thus, the data from previous studies with endogenous cues and intermixed SOAs are hard to interpret because the differences in the size of the validity effect among SOAs could reflect (a) changes in covert orienting with time since the cue and/or (b) changes in the predictive value of the cue that occur because general preparedness also changes with SOA.

These problems can be avoided by testing each SOA in a separate block. In this case, the participant's anticipation of the target is kept constant across SOA. In this chapter, I examine the time course of covert shifts of attention in normal adults by testing each SOA (50, 100, 200, 400, 800 msec) in a separate block. This procedure eliminates both of the confounds identified above; that is, the predictive value of the cue does not change across SOAs, and the general preparedness of the participant at each SOA should not be affected by "influential companion" effects (see Poulton, 1982). The data gathered with this procedure also provided a basis for picking three SOAs to use in testing normal children (Chapter 3) and patients treated for cataract (Chapter 4).

Prior to this study, I conducted four preliminary studies with normal adults and 8-year-olds to develop a procedure that could be used to measure covert orienting reliably (see Appendix A). Table 1 provides a summary of these studies. In these studies, I explored whether significant validity effects occur when the predictiveness of the cue is manipulated by changing the

Table 1.
Preliminary Studies of Covert Orienting in Normal Participants

	Number of Trials per SOA				Predictiveness of Valid Cue at each SOA	
	Valid	Neutral	Invalid	Catch	SOA	
Study 1: SOAs Intermixed	160	40	40	48	100 msec	66%
24 Adults					800 msec	57%
24 8-year-olds						
Study 2: SOAs Intermixed	160	40	40	0	100 msec	80%
24 Adults					800 msec	100%[2]
Study 3: SOAs Blocked Separately	104	20	20	8	100 msec	80%
24 Adults					800 msec	80%
Study 4: SOAs Blocked Separately	104	20	20	16	100 msec	75%
24 Adults					800 msec	75%

[2] Study 2 did not include catch trials. Since a target always appeared by the 800 msec SOA, the cue would be 100% predictive of the target.

proportion of catch trials and the presentation of the SOAs. Study 1 included catch trials and the SOAs were intermixed and presented in a random order. Study 2 did not include catch trials and the SOAs were intermixed in a random order. In Studies 3 and 4, the SOAs were presented in separate blocks to keep the predictiveness of the cue constant across SOA; the proportion of catch trials was 5% in Study 3 and 10% in Study 4. From these preliminary studies, I developed the final procedure used in this experiment. This procedure included catch trials and presented trials at each SOA in separate blocks, to keep the spatial and temporal predictiveness of the cue constant across SOA.

Method

Participants

Participants were 25 undergraduate students from McMaster University (13 male; Mean age = 21 years, 11 months; range = 18y 10 m - 42y 4m). Participants received course credit for participating in a single experimental session lasting approximately one hour. All of the participants passed a screening examination for normal vision (three additional participants were not included in the final sample because they failed the screening examination).

Vision Screening Examination.

Participants were required not to need glasses or contact lenses and were required to pass a screening examination for normal vision. Participants were screened on tests designed to detect problems that might have interfered with normal visual development, including myopia (nearsightedness), hyperopia (farsightedness), and strabismus (misalignment of the eyes).

Any abnormal visual input during childhood that affects neural development is likely to cause reduced visual acuity. To detect myopia, participants were asked to read the Snellen eye chart from 6 meters. To pass, participants were required to read the 6/6 line or better, monocularly with each eye. To detect a hyperopia greater than 3 diopters, participants were asked to read the Snellen acuity chart monocularly through a +3 diopter lens. The effect of a +3 diopter lens on normally focused individuals is that it causes light to be focused in front of the retina and hence there is poorer acuity for each eye with the lens compared to without the lens. If there is a hyperopia of +3 diopters or more, then light will be focused closer to the retina with the lens than without it, and acuity will be at least as good with the +3 diopter lens as without it. Thus, to be included in the normal sample, participants were required to show poorer acuity for each eye with the +3 diopter lens than without it.

To detect participants with strabismus or a history of strabismus that interfered with the normal development of binocular vision, I assessed stereoacuity with the Titmus Fly Test and binocular fusion with the Worth Four Dot Test. On the Titmus Fly Test, participants were required to demonstrate stereopsis for the smallest disparity, 40 arc seconds, included in the test. The Worth Four Dot test of binocular fusion is used to determine whether there is fusion of input from the two eyes, whether there is suppression of vision in one eye under binocular conditions, or whether there is double vision. Participants were required to show fusion to be included in the normal sample.

Apparatus and Stimuli

On every trial the computer screen displayed a central fixation stimulus that was a solid black diamond (5.6 degrees high and wide, when viewed at a distance of 36 cm). Figure 1 illustrates that two black outline boxes were also displayed on each trial, one beginning 5 degrees of visual angle above, and one beginning 5 degrees of visual angle below, the central fixation stimulus. The boxes subtended 7.6 degrees of visual angle both horizontally and vertically, and were drawn using lines whose thickness subtended 1.3 degrees of visual angle. Cues appeared inside the solid black diamond. The cues on valid and invalid trials were white triangles with a 2.8 degree base and a 1.4 degree height. The triangles appeared in the upper or lower portion of the solid black diamond and therefore appeared to point up

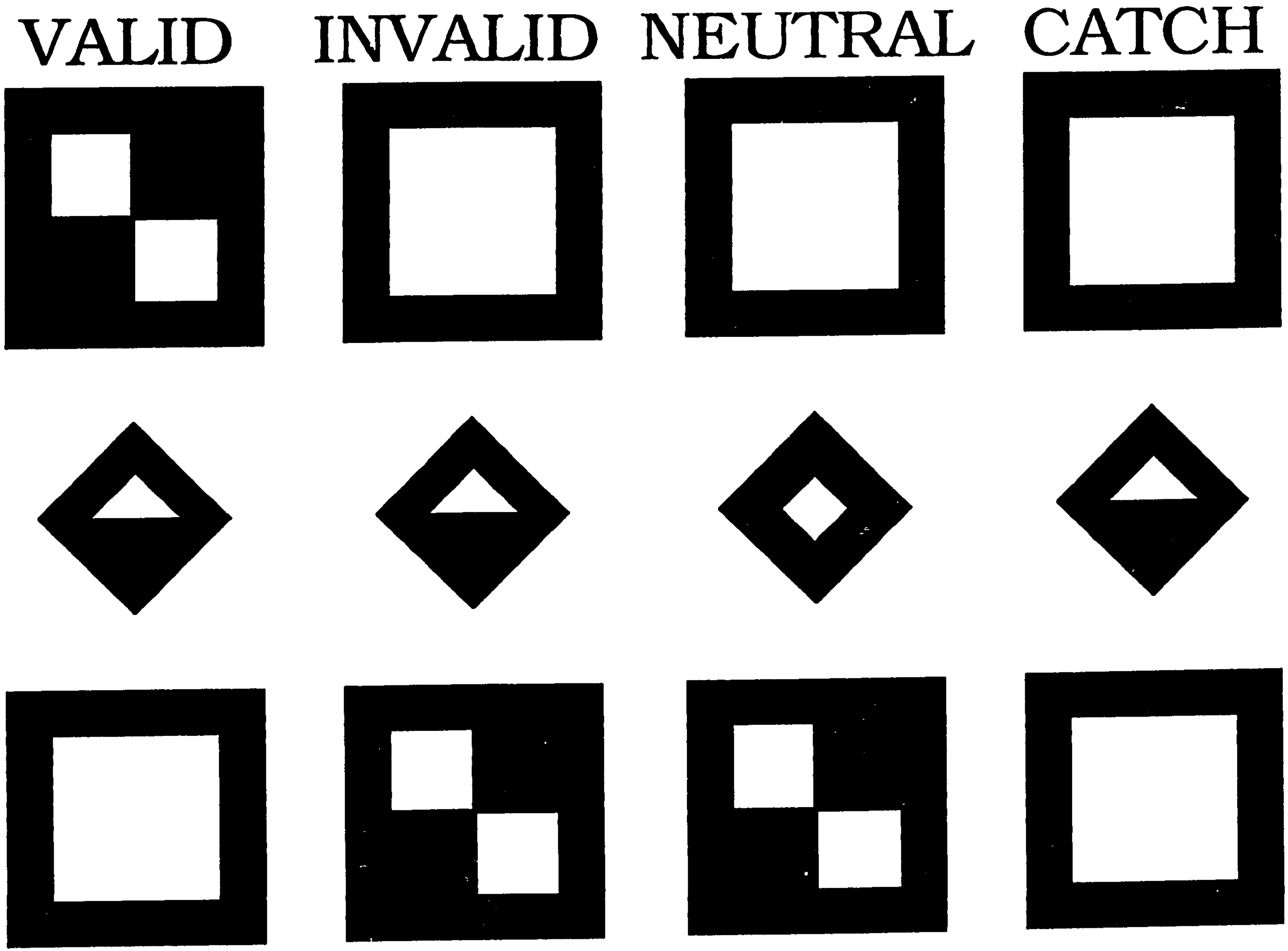

Figure 1. Covert orienting task with endogenous cues. Examples of a Valid, Invalid, Neutral, and Catch trial.

or down. On neutral trials, a white diamond was centered inside the central diamond. The white diamond was 2 degrees wide and high, with the same area as the triangles, 78.8 mm^2. The target was a 2x2 black-and-white checkerboard that filled either of the two peripheral boxes with equal probability. Each black check (9.0 cd/m^2) and each white check (46.7 cd/m^2) was 2.5 degrees wide by 2.5 degrees high.

Stimuli were created using Canvas software and were presented by a Macintosh Powerbook 160 onto an Apple 12-inch monitor (25.3 degrees high and 35.4 degrees wide). SuperLab software, with a 3.96 millisecond time accuracy and a 20 microsecond 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. Reaction time was defined as the time elapsed between onset of the target and the response of the participant. The participant recorded his/her response by pressing a designated button on a keyboard connected to the computer.

Design and Procedure

There were 160 trials at each of five SOAs (50, 100, 200, 400, and 800 msec): 104 valid trials, during which the central cue pointed to the location of the upcoming target; 20 invalid trials, during which the central cue incorrectly signaled the location of the upcoming target; 20 neutral trials, during which a diamond provided no information about the location of the upcoming target; and 16 catch trials during which no target appeared following the cue. These 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 response occurred or until two seconds had elapsed. On valid, invalid, and neutral trials, half of the targets appeared in the upper visual field and half in the lower visual field. Figure 1 shows an example of the stimuli on a valid trial, invalid trial, neutral trial, and a catch trial. To keep the predictive value for a target constant across time intervals I tested each SOA in a separate block. I determined the five-factorial possible sequences for presenting the five SOAs. A sequence was selected, without replacement, for each participant prior to testing. There was the restriction that no more than five participants would begin with a particular SOA.

Participants sat 36 cm from the computer screen with one eye patched with micropore tape. Half of the participants were tested with their left eyes (n = 13) and half with their right eyes (n = 12). Participants' unpatched eyes were aligned level with the central fixation stimulus. The instructions given to each participant are provided in Appendix B. Participants were instructed to maintain fixation on the central fixation stimulus. Participants were told that it was important to practice doing this task as quickly and accurately as possible and to avoid anticipating the appearance of the target, making eye movements toward the location of the target, and responding when no target was presented on a catch trial.

I started each trial by pressing a key when I judged that the participant was fixating on the center of the black diamond. The first stimulus event was the attentional cue. At varying intervals following the onset of the cue the target appeared. Participants were instructed to respond by pressing the letter B on a computer keyboard in front of them as quickly as possible as soon as they detected a 2x2 checkerboard target above or below the central stimulus. At the end of each trial, I coded on the computer whether the participant maintained fixation during the trial or produced an eye movement.[3]

Instructions and Practice

Participants were introduced to the task with a demonstration of the procedure followed by a practice session. Participants were given a practice session of trials at an 800 msec SOA until they completed at least 10 consecutive trials in a row without the occurrence of an eye movement, anticipatory response before the onset of the target, or a false positive response on a catch trial.

Data Analyses

The dependent variable was reaction time, measured as the latency between the appearance of a target and the participant's response. There is a controversy in the literature over the most appropriate measure of central

[3] A control experiment with a volunteer from the laboratory, reported in Appendix F, indicated that I detected 88% of the trials during which the volunteer fixated more than 1 degree off center, and 92% of the trials with an eye movement more than 1 degree off center.

tendency (i.e., mean or median) to use in analyzing reaction times in this type of task. There are often several long reaction times in a sample of reaction time data. Consequently, the distribution of reaction times tends to be positively skewed. Because the median is less sensitive than the mean to extreme scores, the median may provide a less biased estimate of central tendency than the mean. However, Miller (1988) warns about problems in use of the median of positively skewed distributions. In particular, the sample median can overestimate the true median of the distribution, and the more positively skewed the distribution, the greater the overestimate. As long as the median for each condition is biased in the same way, a median bias is inconsequential. However, in some designs, the conditions within an experiment may not be biased in the same way. This can occur when conditions within an experiment are based upon a different number of trials, such as in an endogenous cueing experiment. The median bias gets more extreme as the sample size gets smaller because there is a greater influence of positive skew on the sample median in small samples compared to large samples. This criticism is especially relevant to data from cueing experiments in which there are often four times more valid trials than invalid trials. Miller (1988) showed that in conditions with 8 or 10 trials, the median was overestimated by 7 msec or 5 msec, respectively; in contrast, in a condition with 100 trials it was overestimated by only 1 msec. This information suggests that it is important to make sure that a reported difference in reaction time between invalid and valid conditions is not the result of a median bias.

An alternative to using the median as a measure of central tendency is to use the mean after eliminating outlier observations. One common procedure for rejecting outliers is to reject scores beyond a criterion number of standard deviations from the mean. However, Miller (1991) warns about problems in taking the restricted mean of positively skewed reaction time distributions that differ in sample size. With small samples, there is little bias because only a few observations are likely to be excluded. With large samples (greater than 20 observations), the restricted mean can be biased to underestimate the true mean of the population since more slow reaction times are discarded than fast ones, and the more skewed the distribution, the greater the bias. To deal with this problem, Van Selst and Jolicoeur (1994) developed a *mean outlier elimination procedure* with a *moving criterion* for outlier elimination that is adjusted relative to the sample size. Van Selst and Jolicoeur (1994) demonstrate that to nullify the effect of sample size across the range of theoretical distributions they tested, more observations need to be discarded at smaller sample sizes. Thus, the criterion for outlier elimination was decreased systematically for decreasing sample size. The criterion cutoffs for samples ranging in size from four observations (i.e., 1.45 SDs) to more than 100 observations (i.e., 2.50 SDs) are shown in Table 4 of their paper.

I calculated the mean reaction time for each participant in each condition after outliers were removed (2.99%) using the moving criterion from Table 4 of Van Selst and Jolicoeur (1994). Then I conducted an ANOVA with three within-subject factors: Cue type (Valid, Invalid, Neutral), SOA (50, 100, 200, 400, and 800 msec), and Visual field (upper, lower). Appropriate measures were taken to ensure that assumptions of analysis of variance (e.g., normality of distribution, homogeneity of variance, sphericity) were met. If there was a statistically reliable interaction, analyses of simple effects were conducted to evaluate the source of the interaction. In addition, when there was a significant main effect of Cue type, Tukey post-tests were used to evaluate whether reaction times on valid, invalid, and neutral trials differed from each other. To examine these cueing effects more closely, *validity effects* (invalid - valid RT), *costs* (invalid - neutral RT) and *benefits* (neutral - valid RT) were calculated.

Results

Participants seldom responded on catch trials (M = 5.4%, range 2.5 - 8.5%). Trials on which participants moved their eyes (M = 1.0%, range 0.6 - 1.2%), or responded before the onset of the target (an anticipation) (M = 0.5%, range 0.2 - 0.6%) were excluded from the data analyses.

The results of the ANOVA are shown in Table 2 and the mean reaction times, averaged across participants are displayed in Figure 2.

Table 2.
Time Course of Covert Orienting.

Results of the 3-way ANOVA: SOA, Cue type, Visual Field

Effect	*F*	*df*	*p*
SOA	1.05	4, 96	.38
Cue	**53.69**	**2, 48**	**< .00001**
Field	**6.28**	**1, 24**	**.02**
Cue X SOA	**2.19**	**8, 192**	**.03**
SOA X Field	.38	4, 96	.82
Cue X Field	.45	2, 48	.63
Cue X SOA X Field	1.32	8, 192	.24

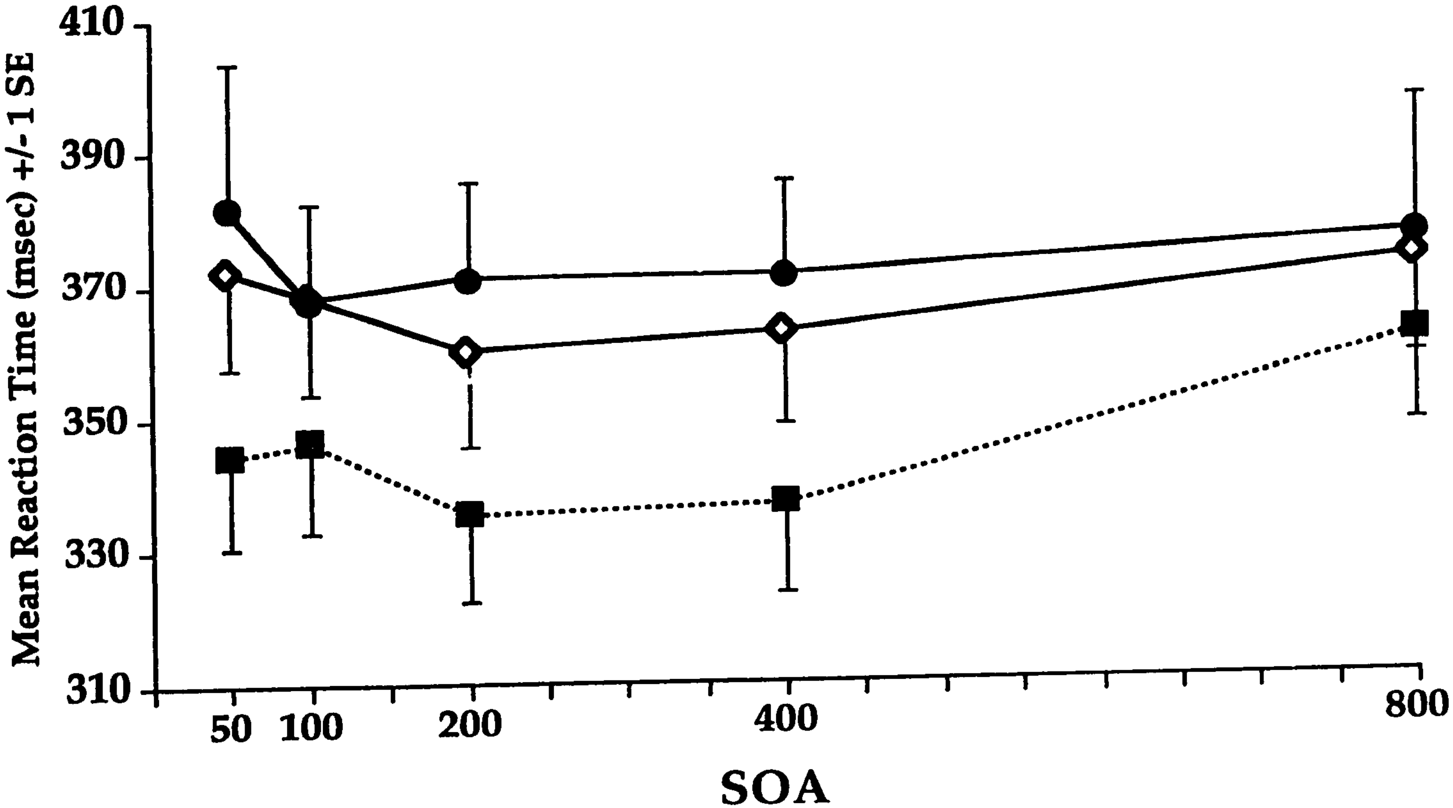

Figure 2. Time course of covert orienting: Reaction times on Valid trials (squares), Invalid trials (circles) and Neutral trials (diamonds) across 5 SOAs.

Effects of Cue and SOA. The size of the cueing effect varied with SOA ($p < .05$). This interaction was examined in more detail with simple effects analyses on each of the cueing conditions. These analyses revealed that reaction time varied with SOA on valid trials ($p < .05$), but not on neutral or invalid trials (both *ps* > .10). On valid trials, reaction times decreased with SOA from 50 - 100 to 200 - 400 msec, then increased between 400 and 800 msec (quadratic trend analysis, $p < .05$).

The main effect of cue condition was also significant. Tukey post-tests revealed that participants responded significantly faster on valid trials than on invalid trials (*Validity effect = 28.2 msec*), and faster on valid trials than on neutral trials (*Benefit = 21.7 msec, ps* < .001), but not significantly faster on neutral trials than on invalid trials (*Cost = 6.4 msec*, $p = .07$).

Effects of Visual Field. Finally, reaction times were faster for targets in the upper visual field (348.6 msec) than in the lower visual field (356.6 msec, $p < .05$). Visual field did not interact with any other factor (*ps* > .10).

Discussion

These results have implications for my three purposes: (1) To examine whether validity effects occur for normal adults despite the modifications to standard cueing procedures, (2) to choose appropriate SOAs to examine the normal development of covert orienting (see Chapter 3) and the effects of visual deprivation on attention in patients treated for cataract (see Chapter 4), and (3) to evaluate the time course of covert shifts of attention in normal adults with the predictive value of the cue kept constant across SOA.

Despite the differences from standard test methods including monocular tests, unusually large cues and targets, and targets along the vertical meridian, the procedure produced a robust validity effect. For subsequent experiments, I chose to use the three SOAs of 100 msec, 400 msec, and 800 msec. I chose to use a 100 msec SOA because it is a relatively short SOA at which adults showed a small validity effect (20.8 msec), a 400 msec SOA because it is the SOA at which there was the largest validity effect (33.8 msec), and an 800 msec SOA because the validity effect appeared to have declined by that point (14.3 msec). I hypothesized that if children treated for cataract are slower than normal children to both engage and disengage attention, they may show no validity effect at a shorter SOA and a larger-than-normal validity effect at a longer SOA.

The results showed that there was no significant cost of invalid cueing at any SOA, although there was a small difference between reaction times on invalid and neutral trials that approached significance. In contrast, there was a large and significant *benefit* of valid cueing at all SOAs. This benefit increased for targets presented after 50 - 100 msec and up to 200 - 400 msec after a valid cue, and then declined thereafter. There was no change in the reaction time on neutral trials with SOA. This result indicates that any additional alerting effect of the cues, independent of whether valid or invalid, did not change with SOA.

My study improved on previous studies involving endogenous cues by matching the area of the neutral cues to the cues on valid and invalid trials, and keeping the predictiveness of the cue constant across the SOAs tested. Nevertheless, my results are consistent with one of the previous studies that also shows that the *benefit* of valid cueing increases up to approximately a 400 msec SOA and then decreases from 400 msec to a 800 msec SOA (Remington & Pierce, 1984). This pattern suggests that changes in the benefit of valid cueing across SOA occur because it takes time to shift the focus of attention, and because attention to a peripheral location is not sustained indefinitely.

On the other hand, my results revealed that there was no significant cost of invalid cueing at any SOA, which is contrary to previous studies that also examined the time course of the *cost* of invalid cueing but that used procedures in which the trials at various SOAs were intermixed. Two such studies found that the cost increased as SOA increased up to approximately 600 msec (Remington & Pierce, 1984; Shepherd & Müller, 1989). As discussed previously, reaction time can be affected both by (1) the validity of the cue and (2) by other factors that allow the participant to anticipate when or if a target will appear. In both of the previous studies (e.g., Remington & Pierce, 1984; Shepherd & Müller, 1989) there were intermixed SOAs and catch trials, and hence the perceived likelihood of a target may have decreased as the SOA increased, because the probability that the trial was a catch trial increased. We do not know how cueing effects interact with preparatory states of the individual, and this in itself makes interpretation of the data from the previous studies difficult to compare to data like those reported in Chapter 2 that do not have this confound.

Participants responded faster to targets in the upper visual field than in the lower visual field. Other studies with normal adults (e.g., Posner, Snyder & Davidson, 1980) have also reported faster reaction times to targets in the upper visual field. In my study, targets may have appeared sooner (by approximately 16 msec) at the top of the monitor display than at the bottom of the display, because the computer screen refreshed starting from the top left corner of the display and ending at the bottom right corner. In Chapter 5, I provide a further discussion about some of the cognitive and neuropsychological differences between the upper and lower visual fields.

In summary, the procedure was effective in producing validity effects and I was able to use the results to select SOAs for testing covert shifts of attention in normal children (see Chapter 3) and in patients following treatment for bilateral congenital cataract (see Chapter 4). Under these testing conditions, with the predictiveness for an endogenous cue kept constant across SOA, there are different effects of SOA on valid and invalid trials: There is no significant *cost* of shifting attention to an incorrect location at any

SOA. In contrast, there is a *benefit* of shifting attention to a correct location that increases up to a 400 msec SOA, and then decreases.

CHAPTER 3

AGE DIFFERENCES IN COVERT ORIENTING IN NORMAL CHILDREN

In this chapter I summarize the literature on the normal development of attention before introducing my own studies with normal 8-year-olds, 10-year-olds, and adults. The general purpose of my studies with normal children was to determine whether there are age differences in performance on the task that I planned to use with patients. These data helped to set age norms and to understand the relationship between normal development and the effects of deprivation.

The Development of Attention During Infancy

In order to understand what was happening during the period when patients were deprived, this chapter begins with a description of five paradigms used with normal infants (disengagement of fixation from a stimulus, anticipatory eye movements, covert orienting, inhibition of return, and spontaneous alternation). For each paradigm, I describe the experimental procedure, developmental changes in performance during infancy, and the hypothesized neural underpinnings of the developmental change. After describing all of the paradigms, I compare the developmental patterns and describe what is implied about the development of neural structures. This review of the literature shows that attention and its neural underpinnings are immature at birth and develop slowly during infancy and childhood.

Disengagement of Fixation from a Stimulus

Even newborns are able to scan the visual field and fixate on salient objects (e.g., Bronson, 1990; Salapatek, 1975). Once they have fixated an object, however, under many circumstances they have difficulty shifting their eyes and hence presumably their attention to another object (but see Goldberg, Maurer, & Lewis, 1997). This "obligatory attention" or "sticky fixation" is most easily seen around 1 month of age (Atkinson et al., 1992; Hood, 1995; Hood & Atkinson, 1993; Johnson, 1990, 1995). For example, Atkinson et al. (1992) and Hood and Atkinson (1993) examined the latency and accuracy of babies' eye movements away from a central stimulus and toward a target in the periphery, under two conditions: (1) A condition during which the central stimulus remained on in the center of the field when a target was presented in the periphery, and (2) a condition during which the central stimulus was turned off when a target was presented in the periphery. Atkinson et al. (Experiment 4, 1992) found that 1-month-olds took significantly longer than 3-month-olds to begin to move their eyes and, that the age difference was greater when the central stimulus remained on than when it was turned off. Similarly, Hood and Atkinson (1993) found that 1.5-month-olds took significantly longer than 3- and 6-month-olds (who did not differ from each other) to move their eyes and that the age difference was greater when the central stimulus remained on than when it was turned off.

Other studies confirm that the "sticky fixation" seen around 1 - 2 months diminishes during the first half-year of life (e.g., Atkinson et al., 1992; Hood, 1995; Hood & Atkinson, 1993; Johnson et al., 1991). For example, Johnson et al. (1991) attracted 2-, 3-, and 4-month-old babies' fixation to one of two central displays (either multicolored dots moving into spiral and circular shapes while accompanied by regular bleeps; or a looming box and diamond shapes accompanied by irregular bleeping sounds). After babies fixated a central display, the central stimulus was left on and a peripheral stimulus (a green diamond above a pink rectangle) was presented, beginning 34 degrees to the left or right of center. The direction and latency of babies' first look away from the central display was recorded. Four-month-old babies were significantly more likely (90%) than 2- and 3-month-old babies (35 and 45%, respectively) to look away from center and toward the peripheral target within the 8 seconds allowed. The latency for babies to make an eye movement away from center was faster for 4-month-olds (less than 1 second) than for younger babies. The authors conclude that "only the 4-month-old group could easily and consistently disengage their gaze from the central stimulus to orient toward a simultaneously presented peripheral target (page 339)." Under these testing conditions, babies become able to disengage their gaze quickly around 4 months of age.[4]

Together, the evidence described above indicates that 1-to-2-month-old babies can have difficulty disengaging fixation. Johnson (1990, 1995) attributes this "obligatory attention" or "sticky fixation" around 1 month of age to the development of an inhibitory pathway from the deeper layers of the primary visual cortex through the substantia nigra and basal ganglia to the superior colliculus, which is not yet regulated by input from the parietal cortex and/or frontal cortex (but see Braddick, Atkinson, Hood, Harkness, Jackson, Vargha-Khadem, 1992). The diminution in "sticky fixation" around 3-to-4 months of age (but see Goldberg et al., 1997) has been attributed to the developing influence of the parietal and/or frontal cortices on the superior colliculus (Atkinson et al., 1992; Hood, 1995; Hood & Atkinson, 1993; Johnson, 1990, 1995; Johnson et al., 1991; reviewed in Maurer & Lewis, 1998).

These studies of "obligatory attention" or "sticky fixation" have concerned overt shifts of attention (eye movements) initiated by an exogenous event (a peripheral target). In adults, shifts of attention with eye movements have been dissociated from shifts of attention without eye movements (covert shifts of attention). Even in tasks where adults are restricted to covert orienting, adults show facilitation for anticipated visual

[4] It should be noted that the authors may have found that 4-month-olds, but not 2- or 3-month-olds, could easily disengage their gaze from a central stimulus because they could do so faster than younger babies. Thus, the results are not necessarly due to babies' gaining a new ability mediated by a newly functioning part of the brain (but see Johnson et al., 1991, Table 2).

events: They respond faster to a target that appears in a validly cued location than in an invalidly cued location (see Chapter 2). In the next two sections, I present what is known about anticipatory attention in babies, namely studies of anticipatory eye movements and of spatial cueing. Both sections provide further information on the state of attentional systems during early infancy.

Anticipatory Eye Movements

Anticipatory eye movements are easily documented in babies 3 to 4 months old. Haith, Hazen, and Goodman (1988) showed 3.5-month-old babies pictures in the left or right visual field. Slides were presented either in an alternating sequence with a fixed time interval or in an irregular sequence with three possible time intervals. Babies showed more anticipatory eye movements for slides in the alternating sequence than for slides in the irregular sequence. With the addition of relatively more complex sequences (e.g., left, left, right; left, left, right; left, left, right), Canfield and Haith (1991) examined anticipatory eye movements in 2- and 3-month-old babies. Both 2- and 3-month-old babies shifted fixation in anticipation of the target in an alternating sequence. However, 3-month-olds were faster and more likely to shift fixation correctly to pictures presented in complex sequences compared to 2-month-olds. Thus, babies can easily demonstrate anticipatory looking for complex sequences around 3 months of age but not at younger ages.

Further evidence of anticipatory looking around 3 to 4 months of age is provided by Johnson et al. (1991). Two-, 3-, and 4-month-old babies were shown a central stimulus that predicted the location of a peripheral target. A target was shown to the left or right of center, depending upon which of two central stimuli had preceded it, one second earlier. After 18 training trials, 4-month-old babies made twice as many anticipatory looks (29.2%) as 2- and 3-month-old babies (13.9 and 14.5%, respectively) toward the location predicted by the central stimulus. These findings suggest that by 4 months of age, babies were able to learn the association between the central stimulus and peripheral location, and were able to shift fixation to the expected (or anticipated) location of the target. Johnson and colleagues relate "anticipatory" looking to the development of the frontal cortex, specifically the frontal eye fields (e.g., Johnson, 1990, 1995, 1997; Johnson et al., 1991; also reviewed in Johnson, 1997; Ruff & Rothbart, 1996).

Covert Orienting

Spatial cueing tasks have been designed specially for testing babies' abilities to shift attention covertly (Johnson et al., 1991; Johnson, Posner, & Rothbart, 1994; Johnson & Tucker, 1996). The method involves measuring the influence of a cue (e.g., a peripheral stimulus presented so briefly that it does not elicit an eye movement) on babies' subsequent eye movements to a target presented in a cued location and/or in a different location. The logic underlying these studies is that if babies maintained fixation on the central stimulus while the cue was presented and the cue influences their subsequent response (an eye movement), then the cue was effective in eliciting a covert shift of attention. In studies with adults that measure manual reaction time and that do not permit eye movements, covert orienting is indicated by faster reaction times to targets that appear in a cued location than in an uncued location. The comparable measure in babies is faster eye movements to targets that appear in a cued location than in an uncued location.

There is evidence of covert orienting in 4-month-old babies. Johnson et al. (1994) first trained babies in an experimental group to make an eye movement to the location contralateral to where a cue appeared. During the twelve training trials the procedure was as follows: As soon as babies were fixating a central stimulus, a peripheral cue was presented briefly (100 msec) in a location contralateral to where a target appeared 400 msec later. Babies were then tested with fourteen training trials intermixed with six test trials. On test trials, the target appeared in the same location as the cue after an interval of 100 msec. Evidence of covert orienting toward the cue would be faster eye movements to the target when it appeared unexpectedly in the cued location than when it appeared in the expected location contralateral to the cue. That was the result shown by the 4-month-olds. No such effect was shown in a control group that received cues in both visual fields.

Johnson and Tucker (1996) used the same stimuli as Johnson et al. (1994) to examine facilitory and inhibitory components of attention in babies. According to Posner's theory (e.g., Posner, 1988), exogenous cues at relatively short SOAs result in reaction times that are faster for targets at cued locations than at uncued locations–this effect is called *facilitation*. In contrast, with relatively longer SOAs (e.g., 300 - 1300 msec), reaction times are slower for targets at cued locations than at uncued locations, presumably because attention has moved away from the cued location–this effect is called *inhibition* (this topic is discussed in greater detail in the next section).

To study facilitory and inhibitory effects, Johnson and Tucker (1996) attracted 2-, 4-, and 6-month-old babies' fixation to a central stimulus, then presented a peripheral cue for 100 msec to the left or right of center. The cue and central stimulus were then turned off and 100 msec or 600 msec later, bilateral targets appeared. Facilitation was indicated by looking toward the cued side; inhibition by looking toward the uncued side. Four-month-olds showed facilitation at the 200 msec SOA and inhibition at the 700 msec SOA, while 2-month-olds only showed facilitation at the 200 msec SOA and 6-month-olds only showed inhibition at the 700 msec SOA. The authors suggest that the reason why they failed to easily observe *both* facilitation and inhibition in 2-month-old and 6-month-old babies is that (1) the mechanism underlying the spatial cueing effects develops over the first few months of life and (2) the time course of these effects changes as babies get older.

The development of covert shifts of attention has also been examined with endogenous cues (Johnson et al., 1991). In the study of anticipatory eye movements discussed earlier in this chapter, Johnson et al. (1991) also tested babies' ability to use central cues to shift attention covertly. Babies were taught that there was a contingent relationship between which of two central stimuli was shown and the location (left or right of center) of an upcoming target stimulus (one second later). Babies were then presented with six trials during which targets were shown in both the left and right locations. Johnson et al. (1991) measured whether babies made more eye movements toward the contingent side than toward the other side. If babies used the contingency to covertly shift attention, then they should show a preference for the target on the contingent side. Only 4-month-old babies showed a preference for targets on the contingent side. Two- and 3-month-old babies looked equally often to targets on the two sides. These results suggest that the 4-month-olds might have learned the contingency, shifted attention toward the expected location, and oriented to the target once it appeared there. It should be noted that the results might reflect instead 4-month-olds' learning to program an eye movement toward the upcoming location of the target, but even so, the results at the younger ages imply no covert attention under these conditions because younger babies looked equally often to targets at the two locations.

The ability to shift attention covertly in response to endogenous cues seems to emerge around 4 months of age and has been related to the development of the parietal cortex (Johnson, 1990, 1995, 1997). The involvement of the parietal cortex in covert shifts of attention is suggested also by studies of human adults using PET, single unit recordings from monkey parietal cortex, and tests of monkeys and human adults with unilateral damage to the parietal cortex (reviewed in Chapter 1).

Inhibition of Return

According to Posner's model (e.g., Posner et al., 1987), adults detect targets more slowly at uncued locations than at cued locations because they must first disengage attention from the cued location, move it to the correct location, and then re-engage attention. However, if attention is drawn away from the cued location (e.g., back to center), participants are slower to respond to the target at the cued location relative to other locations. This phenomenon is called *Inhibition of Return* (IOR). IOR may reflect an evolutionarily adaptive mechanism that biases individuals from returning attention to a previously attended location.

A review of the literature on the development of IOR during infancy can help in understanding how another aspect of attention and relevant neural structures were developing during the period when the patients to-be-tested were deprived. Special tasks have been designed to examine IOR in babies. In a typical task, babies are presented with targets in a previously cued location and/or in a novel location--IOR is denoted by a bias to look toward a target in a novel location rather than in a previously attended visual location. Inhibition of return has been studied in babies following covert shifts of attention (Hood, 1993; Johnson & Tucker, 1996) and overt shifts of attention

(Clohessy, Posner, Rothbart, & Vecera, 1991; Harman, Posner, Rothbart & Thomas-Thrapp, 1994; Valenza, Simion, Umilta, 1994).

There is some tentative evidence of inhibition of return in newborn babies following overt shifts of attention (Valenza et al., 1994). When newborns were judged to be fixating centrally on a flashing red bulb, they were presented with a white flashing bulb 30 degrees in the periphery for three seconds. The cue was turned off and the central stimulus was reinstated for two seconds. Then, a flashing white bulb was presented at both peripheral locations for five seconds. The direction and latency of babies' first eye movement away from center was recorded. Newborns made significantly more first eye movements to the uncued location than toward the cued location, and those eye movements were significantly faster. There are, however, some reservations about the evidence for IOR in newborn babies because: (1) babies did not complete one-third (n = 106) of the designated trials because of fussiness; (2) of the remaining trials, about half were lost (138 out of 245) because a) the coders could not agree on the direction of the eye movement, b) babies did not make an eye movement, c) babies never reoriented to center before the two peripheral test stimuli were presented, or d) babies became fussy during the trial; (3) on one-third of the good trials, reaction time was not measured. Thus, the data on eye movements and on latency were missing for most trials.

There is evidence of subsequent developmental increases in IOR following overt shifts of attention. This developmental effect is illustrated in a study by Clohessy et al. (1991). Three-, 4-, 6-, 12-, and 18-month-old babies and adults were seated in front of three computer monitors with their eyes drawn to colored spirals or looming squares on the central screen (Clohessy et al., 1991). When the participant was judged to be fixating centrally, a target was presented on one of the two monitors, 30 degrees to the left or right of the central display. Once the participant made an eye movement toward the cue, it was turned off and then the central stimulus was reinstated until the participant re-fixated the central stimulus. Then a pair of visual stimuli was presented at both locations in the periphery. The trial lasted until the participant looked toward one of the peripheral stimuli, or until five seconds elapsed. Three- and 4-month-old babies looked at the novel location no more frequently than the familiar location. Six-, 12-, and 18-month-old babies (and adults) showed a bias for the novel location, exhibiting inhibition of return. The authors conclude: "Overall, there appears to be a remarkable development in inhibition of return between 3 and 6 months, with much of it occurring between 4 and 6 months. There also appears to be remarkably little change in the percentage of switches from 6 months through adulthood" (page 346). These findings suggest that, under these conditions, inhibition of return is first manifest between 4 and 6 months and becomes adult-like around 6 months of age.

The manifestation of IOR may be influenced by the peripheral location of the target. For example, Harman et al. (1994) demonstrated that IOR can be observed in 3-month-old babies when the stimuli are presented at locations easier for babies to make an eye movement toward (i.e., at 10 degrees rather than 30 degrees in the periphery). Harman et al. (1994) argue that the developmental evidence for inhibition of return at a particular age may be related to the ability to program eye movements to the attended locations.

IOR following covert shifts of attention has also been documented during infancy (e.g., Hood, 1993; Johnson & Tucker, 1996). It appears to be non-existent at 2 months (Johnson & Tucker, 1996), but has been reported at both 4 months (Johnson & Tucker, 1996) and 6 months (Hood, 1993, Johnson & Tucker, 1996). For example, Johnson and Tucker (1996) examined age differences in inhibition of return following covert shifts of attention in 2- 4- and 6-month-old babies. Cues and targets were presented 29 degrees in the periphery. The results showed that 2-month-olds did not show any significant evidence of IOR at a 200 msec or 700 msec SOA, based upon the direction and latency of their eye movements. Four-month-olds made significantly more eye movements and were faster to respond to the target at the uncued location than at the cued location at a 700 msec SOA. Six-month-olds responded equally to targets at cued and uncued locations at the 200 msec SOA, and showed significantly greater orienting to the uncued location (IOR) at the 700 msec SOA. Six-month-olds did not show significant effects on measures of latency. In a second experiment using a wider range of SOAs, 7-month-olds showed inhibition of return with a 700 msec SOA for both the eye movement and latency measures. These results suggest that the underlying mechanisms involved in inhibition of return following covert shifts of attention develop over the first few months of life.

In conclusion, IOR for covert shifts of attention develops between 2 and 4 months of age, while IOR for overt shifts may be present earlier but nevertheless increases in strength during the first few months of life. The neural structures possibly involved in IOR include the frontal eye fields and the superior colliculus (e.g., Clohessy et al., 1991; also reviewed in Johnson, 1997, Ruff & Rothbart, 1996). The evidence for this assertion comes in part from studies of patients with progressive degeneration of the superior colliculus. These patients have associated deficits in producing vertical saccadic eye movements. In an IOR task, they are just as fast to detect a target in a previously attended location as in a new location along the vertical meridian (Rafal, 1996). They show normal IOR (and eye movements) along the horizontal meridian. The findings suggest that IOR may be related to the ability to program eye movements and complements the evidence for IOR in 3-month-olds only at peripheral locations toward which they can easily make eye movements (Harman et al., 1994). The results from patients also suggest that in adults, IOR does not require an actual eye movement, but rather the ability to prepare an eye movement (Rafal, 1996).

Spontaneous Alternation

Spontaneous alternation of reaching is the reduced tendency to reach toward a spatial location toward which one has reached previously (Vecera, Rothbart & Posner, 1991). Like IOR, it may have an important evolutionary purpose, that is, it is more adaptive for an animal to search for food in a new location than in a previously searched location. Vecera et al. (1991) studied spontaneous alternation of reaching and IOR (with the same methods as Clohessy et al., 1991) in 6- and 18-month-old babies. In the spontaneous alternation procedure babies were allowed to play with a toy at one spatial location and then were presented with two toys identical to the first, one at the same location and one at a novel location. Six-month-old babies reached equally often toward the two locations; they did not show a preference for a toy at a novel location. However, at 6 months they exhibited inhibition of return--they made more eye movements to the stimulus in the novel location. Eighteen-month-old babies showed evidence of both spontaneous alternation and inhibition of return. These data suggest that spontaneous alternation develops later in infancy than inhibition of return and that perhaps different mechanisms cause these two effects. Vecera et al. (1991) relate spontaneous alternation to the functioning of the hippocampus and the anterior attentional system, and IOR to the functioning of the superior colliculus and posterior attentional system.

Summary of Developmental Changes During Infancy

Table 3 summarizes the five attentional abilities discussed in this chapter, the age of emergence, and the hypothesized underlying neural mechanisms (see Johnson, 1997 and Ruff & Rothbart, 1996 for reviews). In summary, there is a lot of development of visual-attentional abilities over the first 3 to 4 months of life that involves many parts of the brain. Hence there is much potential for the absence of visual input during that time to have an effect on development.

The Development of Attention During Childhood

A number of different paradigms (e.g., incidental learning, Stroop, visual search, speeded classification, spatial cueing) have been used to examine the development of attention during childhood. The results show progressive improvements with age in the ability to focus attention (e.g., Enns & Cameron, 1987; Enns & Girgus, 1985; Pick & Frankel, 1974), ignore distracting information (e.g., Akhtar & Enns, 1989; Enns & Brodeur, 1989; Tipper, Bourque, Anderson, & Brehaut, 1989; reviewed in Lane & Pearson, 1982; Ruff & Rothbart, 1996), and to disengage attention (Akhtar & Enns, 1989; Brodeur, 1993; Brodeur, 1995; Enns & Brodeur, 1989; reviewed in Johnson, 1997; Pearson & Lane, 1990).

Table 3.
Summary of Attentional Abilities, Age of Emergence, and Hypothesized Neural Structures Involved.

Attentional Ability	Neural Structure(s)	Age of Emergence
Disengagement of Fixation from a Central Stimulus	Influence on the Superior Colliculus from Frontal and/or Parietal cortices	3 - 4 mos
Anticipatory Eye Movements	Frontal Cortex, especially Frontal Eye Fields	3 - 4 mos
Covert Orienting	Parietal Cortex	4 mos
Inhibition of Return	Frontal Eye Fields Superior Colliculus	birth? 3 - 6 mos
Spontaneous Alternation	Hippocampus Anterior Attention System	Between 6 & 18 mos

In this chapter, I will review studies that examined age differences during childhood in covert shifts of attention initiated by exogenous (Akhtar & Enns, 1989; Brodeur, 1993; Enns & Brodeur, 1989; Pearson & Lane, 1990) and endogenous cues (Brodeur, 1993; Brodeur, 1995; Pearson & Lane, 1990). The procedures used for testing children are similar to those used for testing adults and to those described in Chapter 2 (e.g., Posner et al., 1978). Subjects are asked to signal the detection of a target by making a manual response while always maintaining fixation on the central stimulus. Attention is assessed by differences in reaction time on trials that are validly cued versus invalidly cued. This procedure is different from that used in testing infants, where the dependent measure is the direction and/or latency of an eye movement to a target in a cued or uncued location. It can be argued that the studies of infants measure a different phenomenon than the studies of adults because attention is not studied in isolation of eye movements. Studies in the literature have reported success in measuring covert shifts of attention in children as young as three years of age with procedures that do not involve eye movements (Enns, 1990). With this adult-like method, investigators have examined whether children are able to shift attention covertly as efficiently as adults in response to exogenous cues and endogenous cues. Some procedures have varied the time interval between the onset of a cue and the onset of a target to investigate age differences in the time course of covert orienting.

Covert Orienting Initiated by Exogenous Cues

Several studies indicate that there are age differences in covert shifts of attention initiated by exogenous cues (Akhtar & Enns, 1989; Brodeur, 1993; Enns & Brodeur, 1989; Pearson & Lane, 1990). Despite variations in parameters, all agree that under most conditions the validity effect is larger in children aged 5 to 9 years than in adults.

Two studies have attempted to determine whether the decrease in the size of the validity effect with age is caused by a decrease in cost and/or benefit, or both (Akhtar & Enns, 1989; Enns & Brodeur, 1989). In the first study, Enns and Brodeur (1989) instructed 6-year-old, 8-year-old, and adult participants to maintain fixation on a central fixation point for the duration of each trial. When participants were judged to be fixating centrally, an exogenous cue (a black square) was presented for 50 msec, 2.4 degrees to the left or right of fixation. An arrow appeared to the left or right of fixation 150 msec later. Participants were to push one of two buttons that corresponded to the direction in which the arrow was pointing. On valid trials (80%), the target appeared in the same location as the peripheral cue. On invalid trials, the target appeared in the location opposite the peripheral cue. On neutral trials, the central location and the two peripheral locations were cued and the target was equally likely to appear to the left or right of fixation.

The results showed that the difference between valid and invalid reaction times was larger for 6- and 8-year-olds (who did not differ from each other) than for adults. The difference between invalid and neutral reaction time (*costs*) decreased with age. This suggests that children were slowed more than adults by the misdirection of the cue on invalid trials. There were no measurable benefits of valid cueing at any age. However, the authors suggest that problems with the neutral condition may be responsible for the lack of benefit because the neutral cues were not matched physically to the cues under valid or invalid conditions. Under these conditions, Enns and Brodeur's (1989) findings indicate that validity effects decrease with age; although because of possible problems with the neutral cue, it is not clear whether the change results from decreased benefit of valid cueing or decreased cost of invalid cueing, or both.

Another developmental study of covert shifts of attention initiated by exogenous cues (Akhtar & Enns, 1989) suggests that children have larger validity effects than adults because they have both larger costs from invalid cueing and larger benefits from valid cueing. Akhtar and Enns (1989) asked 5-, 7-, and 9-year-old children and adults to perform a two-alternative forced-choice recognition task. The procedure was similar to that of Enns and Brodeur (1989), except that participants were to press one of two buttons to a square or a cross that appeared 3.5 degrees to the left or right of fixation. As in Enns and Brodeur (1989), the cue was a black square that appeared for 50 msec in one of the two locations at which a target would appear, 150 msec before the target. Unlike Enns and Brodeur (1989), on neutral trials, the cue appeared in both possible target locations in Experiment 1 and at central fixation in Experiment 2. Participants were provided with feedback after each trial regarding the accuracy of their response.

Overall, the difference in reaction time [RT/p(correct)] on valid versus invalid trials was larger for 5- and 7-year-old children than for 9-year-olds, and larger for 9-year-olds than for adults. Five- and 7-year-old children showed a significant *benefit* from valid cueing (as indexed by Neutral - Valid reaction time) but 9-year-olds and adults did not. The *cost* from invalid cueing (as indexed by Invalid - Neutral reaction time) was significant in all three groups of children (five, seven, and nine years) but not in adults. In summary, the findings show that the validity effect decreases with age. The results suggest that under these conditions, children both benefit from valid cueing more than adults and are hurt more by invalid cueing. The results for 9-year-olds suggest that changes in *benefit* may occur earlier in development than changes in *cost*, and hence may depend on the development of different mechanisms. It should be noted that as in Enns and Brodeur's study, the conclusions about cost and benefit depend upon the neutral condition as a baseline for valid and invalid conditions.

Both studies (Akhtar & Enns, 1989; Enns & Brodeur, 1989) showed that the validity effect decreases with age, at least in part because children are hurt more by invalid cueing than are adults. This slowing on invalid trials may occur because children are slower than adults to disengage attention from the incorrectly cued location on invalid trials.

Only a few studies have manipulated the time interval between the onset of a peripheral cue and a target to investigate age differences in the time course of covert shifts of attention initiated by exogenous cues (Brodeur, 1993; Pearson & Lane, 1990; Wainwright-Sharp, 1995). All of these studies are hard to interpret because: (1) The results were collapsed across the type of cue (exogenous or endogenous) despite a significant interaction involving this factor (Pearson & Lane, 1990); (2) adults and children were tested using different SOAs (Brodeur, 1993); or (3) because eye movements were allowed, participants may not have been performing the task with covert attention (Wainwright-Sharp, 1995).

Perhaps because of these methodological problems, the previous studies disagree about the pattern of developmental change in the effects of SOA. For example, Wainwright-Sharp (1995) found no age differences between 6-, 10-, and 14-year-old children and adults in the size of the validity effect at a 100 msec SOA. However, at a 800 msec SOA, 6-year-olds showed significantly larger validity effects than did 10-year-olds, 14-year-olds, and adults, all of whom did not differ from each other. Although Brodeur (1993) made no statistical comparison, inspection of her Figure 3 indicates that the validity effect was larger at all SOAs in 6- and 8-year-olds than in adults. Moreover, the influence of SOA was different for children and adults.

Children aged 6, 8, and 10 years showed validity effects at 133, 250, and 450 msec SOAs. While adults also showed validity effects at the shorter SOAs (150 msec and 200 msec), unlike children, they showed inhibition of return at 400 msec and 800 msec SOAs. In spite of the problems mentioned above, these two studies indicate that age differences in the size of the validity effect may depend on the SOA at which it is tested.

Covert Orienting Initiated by Endogenous Cues

There is only one published study on the development of covert shifts of attention initiated by endogenous cues (Pearson & Lane, 1990). This study shows that the size of the validity effect is larger in 8-year-old children than it is in adults.

Specifically, to examine age differences in validity effects, costs, and/or benefits at different time intervals after an endogenous cue, Pearson and Lane (1990) required 8-year-olds, 12-year-olds, and adults to discriminate a target letter (W or O) that could appear to the left or right of fixation, at either one of two distances (2.8 degrees or 8.2 degrees). At one of five SOAs (83, 150, 200, 267, and 300 msec) before a target, a centrally-located arrow pointed to the side where a target would appear on valid trials (80%) or to the opposite side on invalid trials (20%). On neutral trials (16.7%) the arrow pointed to both sides. The arrow remained in the center of the visual field until the end of the trial. The authors also included trials with exogenous cues where a peripheral cue (an exclamation mark) appeared in the same location as the target on valid trials, in the opposite location in invalid trials, and in both peripheral locations on neutral trials.

As SOA increased from 83 msec to 300 msec, 8-year-olds but not 12-year-olds or adults, showed increasing reaction time following invalid cues (collapsed across trials with exogenous and endogenous cues). With increasing SOA, participants at all ages showed increasing reaction time following valid cues. This suggests developmental changes in the time course of the *cost* of invalid cueing. Overall, collapsed across SOA, and collapsed across endogenous and exogenous cues, the *benefit* of valid cueing and the *cost* of invalid cueing both decreased with increasing age. Additional interactions from the analyses suggested that other factors (such as the effects of endogenous versus exogenous cues) have different influences at different ages but the authors did not pursue the interactions to find out the nature of the differences.

Three other studies investigate the effects of SOA on the validity effect for endogenous cues but there are problems in the interpretations of all three. One study is an unpublished doctoral dissertation in which adults and children were tested using different SOAs (Brodeur, 1993). Two additional studies allowed eye movements (Wainwright-Sharp, 1995) and/or did not provide enough information to critically assess the methods and to interpret the findings (Brodeur, 1995). In the study that allowed eye movements, there was no difference between 6-, 10-, and 14-year-old children and adults in the size of the validity effect at a 100 msec SOA or at a 800 msec SOA; for all ages validity effects were larger at the 800 msec SOA (Wainwright-Sharp, 1995).

In Brodeur's thesis (1993), adults showed validity effects across all SOAs tested (from 150 msec to 800 msec). This result agrees with the literature on endogenous cues and the data I presented for adults in Chapter 2. Like adults, children at all three ages showed validity effects at a short (133 msec) SOA. However, unlike adults, validity effects diminished at longer SOAs (250 and 450 msec). The pattern of results for accuracy mimicked the pattern of results for reaction time. The data on *costs* and *benefits* did not create a sensible pattern across SOA.

In summary, Pearson and Lane (1990) show that the size of the validity effect decreases with age. However, the unpublished studies do not support this finding. Moreover, Pearson and Lane (1990) indicate that there is an interaction with SOA that can be understood by an evaluation of cost and benefit: there was an increasing cost of invalid cueing as SOA increased from 83 msec to 300 msec in 8-year-olds but a flat function in older participants. There was no influence of SOA on age differences in the benefit of valid cueing. Although Pearson and Lane show age differences in the time course of covert orienting, there are at least two problems that limit the conclusions that can be drawn from their findings about endogenous cueing: (1) the data for tasks with endogenous and exogenous cues were combined in the analyses; and (2) the neutral cues were not matched physically to the cues on valid and invalid trials (see Jonides & Mack, 1984).

Summary of Developmental Changes During Childhood

To summarize, previous studies on the development of covert shifts of attention with endogenous (Brodeur, 1993; Pearson & Lane, 1990) and exogenous cues (Akhtar & Enns, 1989; Enns & Brodeur, 1989) show age differences in validity effects. Specifically, children appear to be slowed more than adults by invalid cueing. What is not clear from the previous literature is the contribution of cost and benefit to this developmental change or how it interacts with SOA.

In addition to the problems mentioned above in individual studies, there is also a general limitation in the previous literature: In none of the studies were the data corrected for a reaction time bias (see Chapter 2). A reaction time bias is a problem since it could have inflated reaction times in the invalid condition, with fewer observations than in the valid condition, and more so in groups of children who are likely to have had more long reaction times (i.e., more positive skew). These problems could have led to incorrect conclusions about development in previous studies.

In the studies reported in this chapter, I examined the normal development of the time course of covert orienting initiated by central, endogenous cues in groups of 8-year-old, 10-year-old, and adult participants. I used endogenous cues rather than exogenous cues in order to learn about higher cognitive aspects of attention that are more likely to tap the parietal cortex (see Chapter 2). I tested larger groups of participants (n = 24 per group) than in previous studies using endogenous cues (Brodeur, 1993; Pearson and Lane, 1990), matched neutral and informative cues, and corrected the data for a reaction time bias. Like previous studies, I tested with a relatively short SOA (i.e., 100 msec) and like Brodeur (1993), I included a 400 msec and a 800 msec SOA. I decided to test children with a 100 msec SOA because it is a relatively short SOA at which the adults in Chapter 2 showed a small validity effect, a 400 msec SOA because it is the SOA at which adults showed a large validity effect, and a 800 msec SOA because it is when the validity effect declined in adults. I wanted to add to the small literature on age differences in covert orienting and to establish the normal development of covert orienting on this task, to provide a comparison for the results from patients deprived of normal visual experience (Chapter 4).

I also included two conditions with 10-year-olds in anticipation of later testing involving children treated for cataract. Children treated for binocular deprivation can have greater deficits in one eye compared to the other eye because of a different history of visual deprivation to each eye. To determine that inter-ocular differences in attention in patients are not due to differences that normally exist between the left eye and the right eye, in this chapter, I evaluated both the left and right eyes of normal 10-year-old children. Since the patients were to be tested twice (once for each eye), I also examined the effects of practice by retesting one eye.

Method

Participants

The final sample of participants consisted of groups of 24 8-year-olds (mean age = 8.1 years, range = 7.9 - 8.2 years), 10-year-olds (mean age = 10.0 years, range = 9.8 - 10.3 years), and adults (mean age = 20.2 years, range = 18.8 - 29.6 years) (see top half of Table 4). Half of the participants in each age group were tested with the right eye, and half were tested with the left eye.

To be included in the final sample, each participant had to pass a vision screening examination. Because by age 8 performance should be adult-like on all parts of the screening exam (e.g., acuity, hyperopia, stereopsis, fusion) the inclusion criteria were the same as described in Chapter 2. Participants with any previously noted attentional problems or who were currently taking any medication to regulate behavior were excluded. An additional nine participants were excluded from the final sample because they failed the vision screening examination (four 8-year-olds; two 10-year-olds; two adults) or were taking medication to control seizures (one 8-year-old).

Procedure

The testing procedure was the same as in Chapter 2 with the following exceptions: (1) I obtained parental consent if the participant was a minor. (2) SOAs were restricted to 100, 400, and 800 msec SOAs, presented in separate

Table 4.
Summary Characteristics of the Normal Subjects[1] who Contributed to Each Task Across All Chapters.

Task	# Eyes	# Subjects	Mean Age in years (range)	Chapter
Covert Orienting Task				
Time-Course of Covert Orienting				
Adults (50, 100, 200, 400, 800 msec SOAs)	25	25	21.9 (18.8 - 42.3)	2
Age-Differences in Covert Orienting (100, 400, & 800 msec SOAs)				
8-year-olds	24	24	8.1 (7.9 - 8.2)	3
10-year-olds	24[2]	24	10.0 (9.8 - 10.3)	
Adults	24	24	20.2 (18.8 - 29.6)	
Inter-Ocular Differences in Covert Orienting				
10-year-olds (test of second eye)	24[2]	24	10.0 (9.8 - 10.3)	3
Effects of Re-Testing on Covert Orienting				
10-year-olds	24[2]	24	10.1 (9.8 - 10.3)	3
Filtering Attention Task (400 msec SOA)				
Age-Differences in Filtering Ability				
10-year-olds	24	24	10.0 (9.8 - 10.2)	5
Adults	24	24	20.4 (18.6 - 26.2)	
Filtering Ability of Age-Matched Controls				
Controls for Bi-Short Group	16	8	11.8 (8.8 - 15.7)	5
Controls for Bi-Long Group	16	8	14.4 (10.2 - 17.1)	
Controls for Unilateral Group	7	7	14.9 (8.6 - 25.9)	Appendix C

[1] All subjects had to pass a vision screening examination in order to be included in the normal sample. See Chapter 2.
[2] These are the same group of 10-year-olds.

blocks. (3) I patched each participant's untested eye so that it could remain open under the patch and diffuse light could enter the eye through the patch. To do so, I placed one-half of a plastic petri-dish (4 cm in diameter) over the untested eye, covered it with micropore tape, and secured the edges of the tape onto the face. This method of patching was designed to minimize latent nystagmus in patients treated for cataract when one eye is covered (see Chapter 4). (4) I asked participants to read single letters on the Sheridan Gardiner Test to determine single letter acuity at near (35.6 cm). I used this test to verify that the central cues were visible for participants with normal vision (this chapter) and for patients treated for cataract (Chapter 4). (5) After the covert orienting test, I gave participants post-tests to evaluate: a) how well they understood the meaning of the cues and b) whether they could detect boxes in the periphery where a target could appear. I included these post-tests so that should children (this chapter) or patients (Chapter 4) do poorly on the main task, I would be able to differentiate among possible reasons. To evaluate how well participants understood the meaning of the cues, I presented ten trials on which they were to say whether they thought the cue was a "helper" (the arrow helped them locate the target), a "fooler" (the arrow pointed away from the target), or "no clue" (the arrow pointed in both directions), without making any eye movements. I recorded the number of correct responses and the number of eye movements during the ten trials. To determine how well participants were able to detect boxes in the periphery, they were presented with one or two boxes, located either above or below fixation, on the computer display. Participants were instructed to say whether they saw one or two boxes without making an eye movement. Again, I recorded the number of correct responses and eye movements that occurred during the ten trials.

I conducted additional testing with 10-year-olds in order to examine inter-ocular differences in attentional abilities and the effects of practice on the validity effect. Ten-year-olds were tested with the left eye and with the right eye on the first day (half had the left eye tested first and half had the right eye tested first) and were then re-tested with one eye (either the left eye or the right eye) on a second day. For half of the 10-year-olds, the eye that had been tested first was retested, and for the other half, the eye that had been tested second was retested.

Data Analyses

To examine whether there are age differences in covert orienting in normal participants, I used an ANOVA on mean reaction times with outliers eliminated (Van Selst & Jolicoeur, 1994) for the reasons described in Chapter 2. The percentage of scores eliminated was 3.19% for 8-year-olds, 3.13% for 10-year-olds, and 3.01% for adult participants. The ANOVA had one between-subject factor, Age (8-year-olds, 10-year-olds, adults) and three within subject factors: SOA (100, 400, 800 msec), Cue type (invalid, neutral, valid), and Visual field (upper, lower).

To examine differences between right and left eyes of normal 10-year-old participants, I conducted an ANOVA on the corrected mean reaction times (Van Selst and Jolicoeur, 1994; 3.12% of scores eliminated). The ANOVA had four within subject factors: Eye (right or left), SOA, Cue type, and Visual field.

To examine the effects of practice in normal 10-year-old participants, I conducted an ANOVA on corrected mean reaction times (Van Selst and Jolicoeur, 1994; 3.10% of scores eliminated) with four within subject factors: Session (first or repeat), SOA, Cue type, and Visual field.

Results

Preliminary analyses

Table 5 shows the mean percentage of eye movements, responses on catch trials, and anticipations of the target for 8-year-olds, 10-year-olds, and adults. Adults made fewer eye movements and responded on fewer catch trials than 8-year-olds or 10-year-olds, who did not differ significantly from each other (Separate ANOVAs on Eye Movements and Catch Trials, Main effects of age, Tukey post-tests, $ps < .05$). Participants rarely anticipated the appearance of the target.

Table 5.
Mean Percentage (Standard Error) of Trials with Eye Movements, Responses on Catch Trials, and Anticipations of the Target for 8-year-old, 10-year-old and Adult Participants.

	Eye Movements	
Normal 8-year-olds	4.6%	(0.5%)
Normal 10-year-olds	4.4%	(0.5%)
Normal Adults	1.0%	(0.3%)

	Responses on Catch Trials	
Normal 8-year-olds	15.1%	(2.4%)
Normal 10-year-olds	21.1%	(2.5%)
Normal Adults	5.4%	(1.4%)

	Anticipations of the Target	
Normal 8-year-olds	0.6%	(0.2%)
Normal 10-year-olds	0.7%	(0.2%)
Normal Adults	0.5%	(0.2%)

Main Analyses

Effects of Age and Its Interactions. Table 6 shows the results from the ANOVA. There was a main effect of age. Adults responded faster than 8-year-olds and 10-year-olds (Tukey post-tests, *ps* $< .05$), who did not differ from each other (Tukey post-tests, *ps* $> .05$). However, there were no age differences in the effect of the cues (non-significant interaction between Cue and Age) nor in the way cues interacted with SOA (non-significant interaction between Age, SOA, and Cue). Figure 3 illustrates the similar effects of cue informativeness at each age. Figure 4 illustrates for participants in each age group, the mean reaction times on valid, invalid, and neutral trials at each SOA.

Age Interacted with Visual Field. Reaction times were faster for targets in the upper visual field than the lower visual field in 8-year-olds, but not in older participants (Analyses of simple effects, *ps* $< .05$). There was a trend for an Age by Cue by Visual field interaction. This trend is illustrated in Figure 5. The figure shows that 8- and 10-year-olds tended to show larger validity effects in the lower visual field than in the upper visual field and adults tended to show larger validity effects in the upper visual field than in the lower visual field. Age did not interact with Visual field and SOA, nor with Visual field, SOA, and Cue.

Table 6.
Normal Development of Covert Orienting

Results of the 4-way ANOVA: Age group, SOA, Cue type, Visual Field.

Effect	*F*	*df*	*p*
Age group	**19.16**	**2,69**	**< .000001**
SOA	**6.33**	**2,138**	**< .01**
Cue type	**45.97**	**2,138**	**< .000001**
Visual Field	**15.79**	**1,69**	**< .001**
Age group X SOA	.43	4,138	.78
Age group X Cue type	1.21	4,138	.31
SOA X Cue type	**3.69**	**4,276**	**< .01**
Age group X Visual Field	**4.52**	**2,69**	**< .05**
SOA X Visual Field	1.25	2,138	.29
Cue type X Visual Field	1.32	2,138	.27
Age group X SOA X Cue type	.91	8,276	.51
Age group X SOA X Visual Field	1.63	4,138	.17
Age group X Cue type X Visual Field	2.31	4,138	.07
SOA X Cue type X Visual Field	.30	4,276	.88
Age group X SOA X Cue type X Field	1.40	8,276	.20

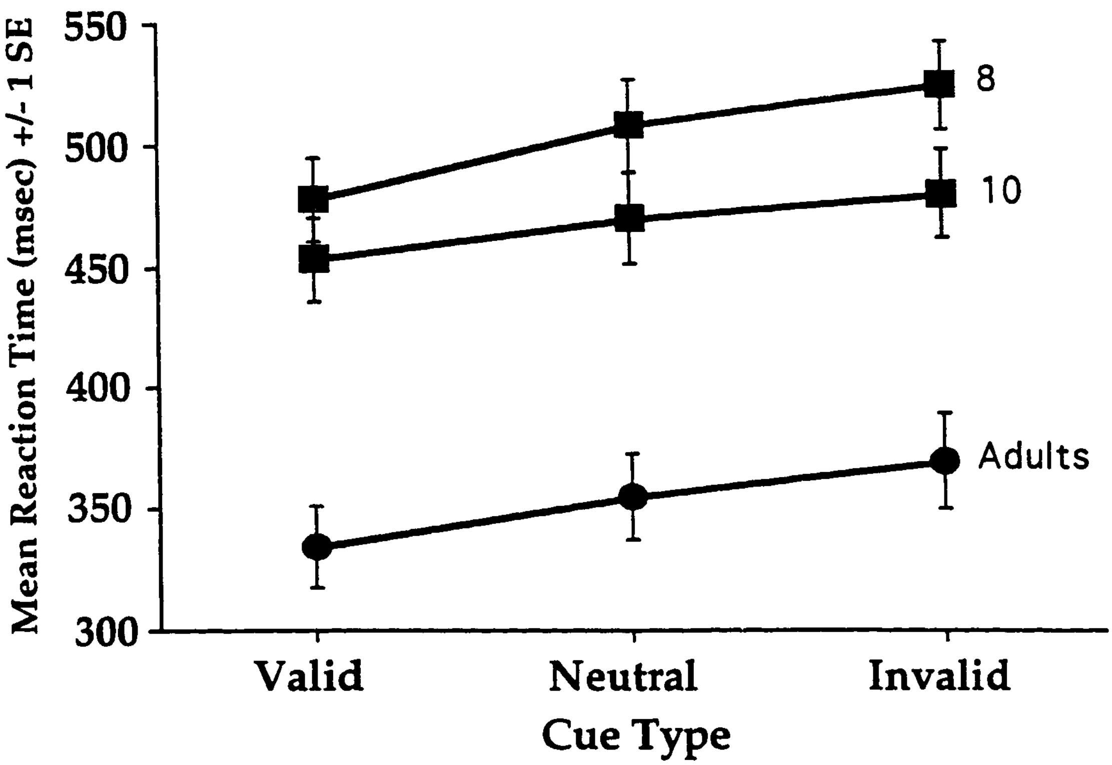

Figure 3. Mean reaction time as a function of cue type for 8-year-olds (filled squares), 10-year-olds (open squares), and adults (filled circles). Means are connected to facilitate comparisons of the effect of cue at different ages.

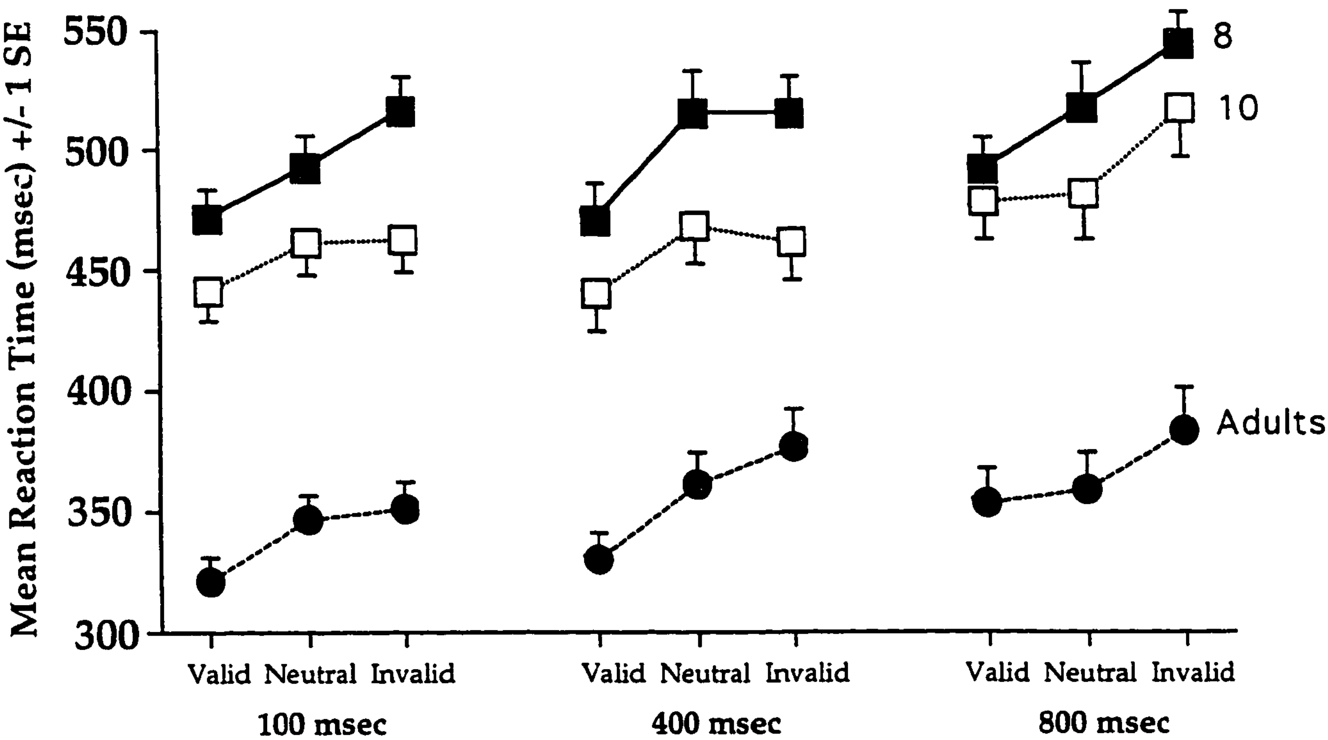

Figure 4. Mean reaction time as a function of cue type and SOA for 8-year-olds (filled squares), 10-year-olds (open squares), and adults (filled circles). Means are connected to facilitate comparisons of the effect of cue at different ages.

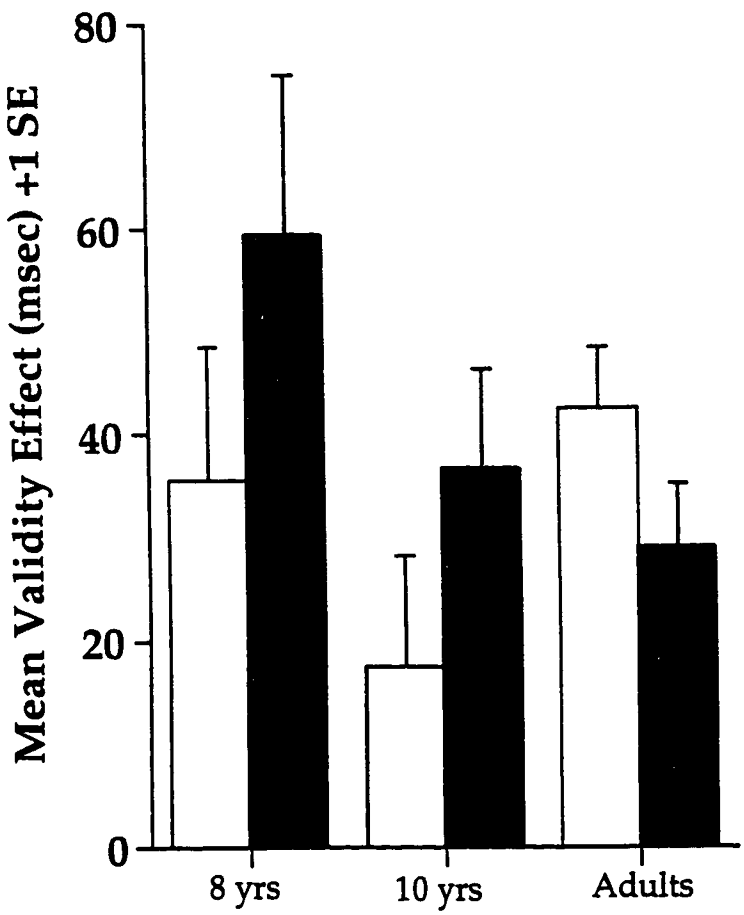

Figure 5. Mean validity effect (Invalid - Valid RT) in the upper visual field (white bars) and in the lower visual field (black bars) for each age group of subjects.

Effects of Cue. There was a main effect of Cue. Reaction times were significantly slower on invalid trials than on neutral trials (Tukey post-test, $p < .001$, *cost* = 14.0 msec) and valid trials (Tukey post-test, $p < .001$, *validity effect* = 36.8 msec). Reaction times were significantly faster on valid trials than on neutral trials (Tukey post-test, $p < .001$, *benefit* = 22.7 msec). Cue interacted with SOA. Figure 6 shows that at the 100 msec and 400 msec SOAs, reaction times were significantly faster on valid trials than on neutral trials and invalid trials; reaction times on neutral and invalid trials did not differ significantly from each other. At the 800 msec SOA the pattern was different: Reaction times on valid and neutral trials did not differ significantly from each other but reaction times were significantly slower on invalid trials than on neutral trials and valid trials (analyses of simple effects and Tukey post-tests, $ps < .05$). Figure 7 shows these data as a function of SOA collapsed into cost (Invalid - Neutral RT) and benefit (Neutral - Valid RT). The benefit of valid cueing was greater at the 100 and 400 msec SOAs than at the 800 msec SOA, while the cost of invalid cueing was greater at 800 msec. Cue did not interact with Visual field nor with Visual field and SOA.

Effect of SOA. There was a main effect of SOA. Reaction times were faster at the 100 msec and 400 msec SOAs than at the 800 msec SOA (Tukey post-tests, $ps < .01$). SOA did not interact with Visual field.

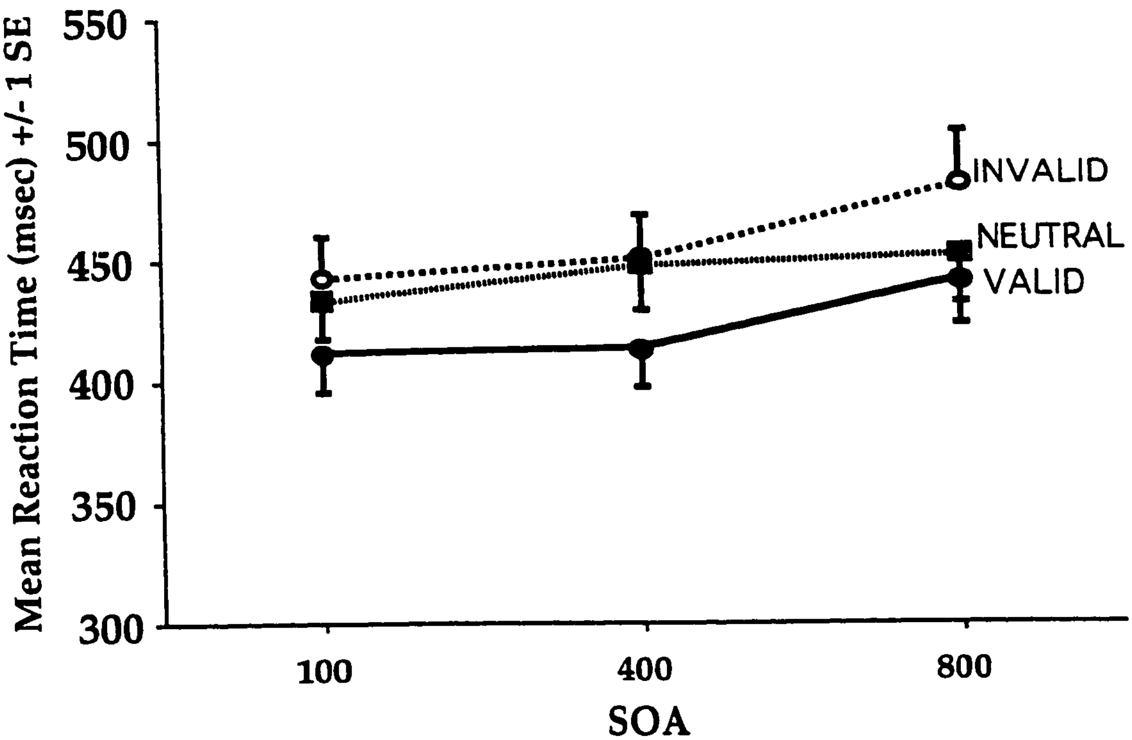

Figure 6. Mean Reaction Time on Valid trials (filled circles), Invalid trials (open circles), and Neutral trials (filled squares) as a Function of SOA, collapsed across age. Means are connected to facilitate comparison of the effects at different SOAs.

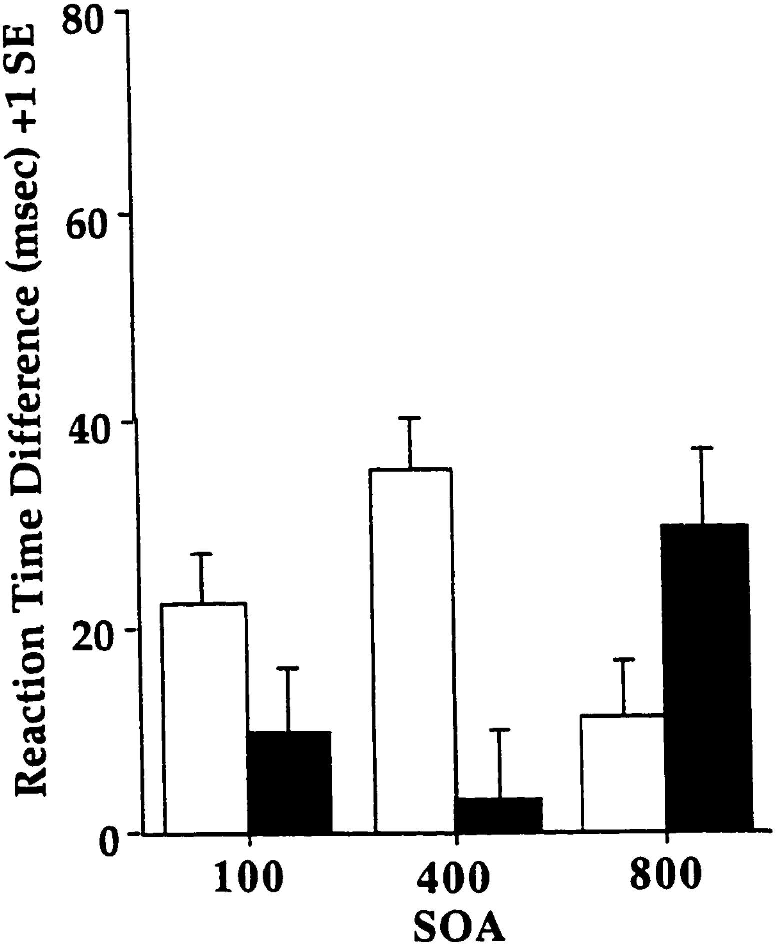

Figure 7. Benefit (as indexed by Neutral - Valid RT, white bars) and Cost (as indexed by Invalid - Neutral RT, black bars) as a function of SOA.

Supplementary Analyses

Results for Inter-Ocular Differences. Results from the ANOVA on inter-ocular differences are shown in Table 7. Figure 8 illustrates that there was no significant difference in reaction time between right eyes and left eyes (non-significant main effect of Eye) and no significant difference in covert orienting between right and left eyes at any SOA (non-significant interactions between Eye and Cue and among Eye, Cue and SOA). None of the other interactions with eye were significant.

Results for the Effects of Practice. Table 8 shows the results from the ANOVA on the effects of practice. Ten-year-olds were significantly faster during the second test session than during the first session (significant main effect of Session). Figure 9 illustrates that there were no significant differences during the two sessions in the effects of cues on covert orienting at any SOA (non-significant interactions between Session and Cue and among Session, Cue and SOA). There was no interaction between Session and SOA nor Session and Visual field and none of the higher order interactions with Session were significant.

Discussion

The results indicate that 8-year-olds, 10-year-olds, and adults were able to use these endogenous cues to covertly orient attention. Although children were generally slower to respond than adults, under these conditions, they

Table 7.
Inter-Ocular Differences in Covert Orienting in 10-year-olds.

Results of the 4-way ANOVA: Eye, SOA, Cue type, Visual Field

Effect	*F*	*df*	*p*
Eye	.35	1,23	.56
SOA	**7.19**	**2,46**	**<.01**
Cue type	**13.08**	**2,46**	**<.001**
Visual Field	.04	1,23	.83
Eye X SOA	1.64	2,46	.20
Eye X Cue type	.55	2,46	.58
SOA X Cue type	.64	4,92	.64
Eye X Visual Field	.28	1,23	.60
SOA X Visual Field	.37	2,46	.69
Cue type X Visual Field	1.23	2,46	.30
Eye X SOA X Cue type	.28	4,92	.89
Eye X SOA X Visual Field	1.40	2,46	.26
Eye X Cue type X Visual Field	2.49	2,46	.09
SOA X Cue type X Visual Field	1.32	4,92	.27
Eye X SOA X Cue type X Field	.56	4,92	.69

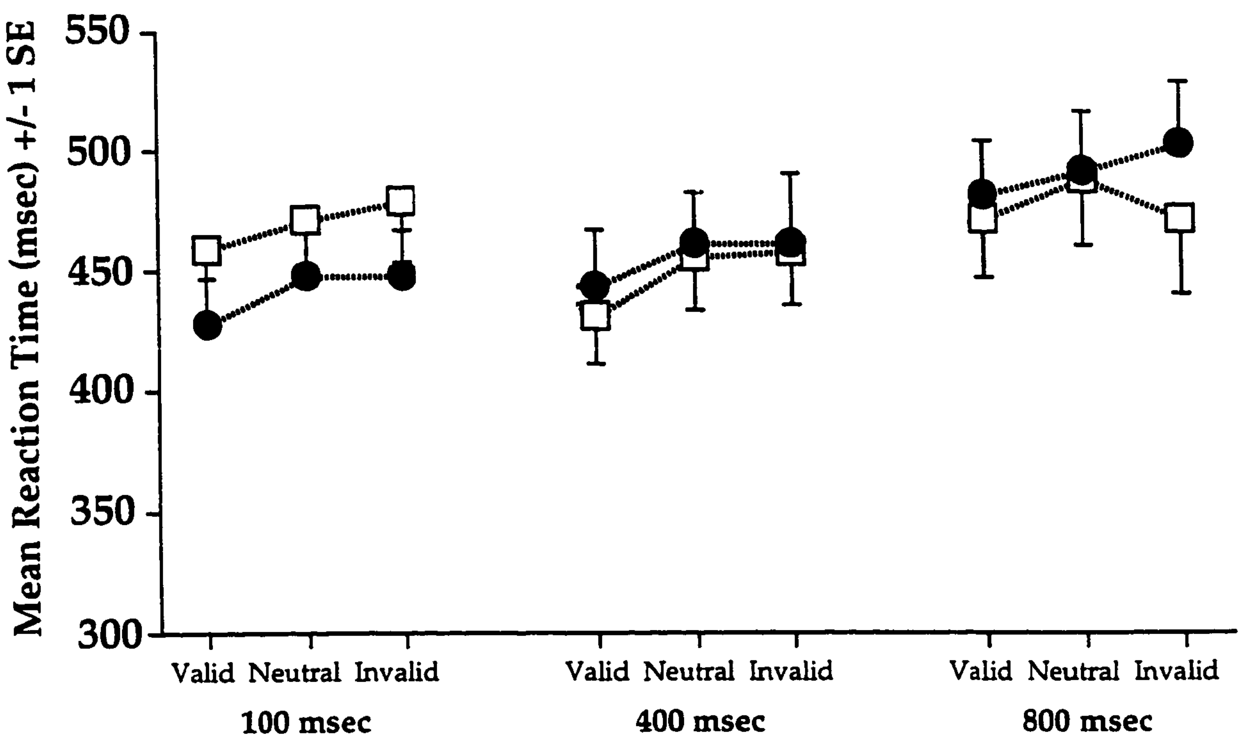

Figure 8. Comparison of reaction times for right eyes (filled circles) and left eyes (open squares) of 10-year-olds as a function of cue type and SOA. Means are connected to facilitate comparisons of the effect of cue in the two eyes.

Table 8.
Effects of Re-Testing Covert Orienting in 10-year-olds.

Results of the 4-way ANOVA: Session, SOA, Cue type, Visual Field

Effect	*F*	*df*	*p*
Session	**7.91**	**1,23**	**<.01**
SOA	**4.67**	**2,46**	**<.01**
Cue type	**11.32**	**2,46**	**<.0001**
Visual Field	.25	1,23	.61
Session X SOA	1.35	2,46	.27
Session X Cue type	1.54	2,46	.22
SOA X Cue type	.82	4,92	.52
Session X Visual Field	.13	1,23	.72
SOA X Visual Field	1.69	2,46	.19
Cue type X Visual Field	1.37	2,46	.33
Session X SOA X Cue type	.76	4,92	.55
Session X SOA X Visual Field	1.14	2,46	.33
Session X Cue type X Visual Field	.08	2,46	.92
SOA X Cue type X Visual Field	.77	4,92	.55
Session X SOA X Cue type X Field	1.96	4,92	.11

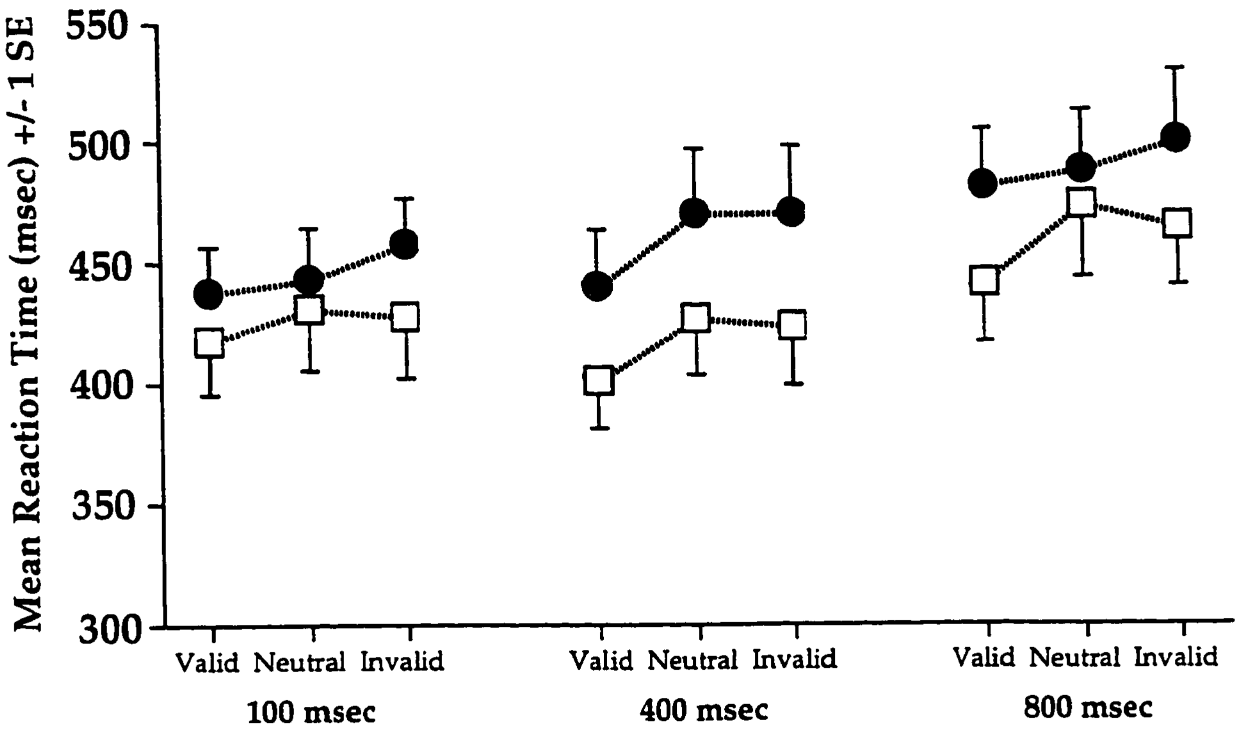

Figure 9. Comparison of reaction times on the first session (filled circles) and on the repeated test (open squares) of 10-year-olds as a function of cue type and SOA. Means are connected to facilitate comparisons of the effect of cue during the two sessions.

showed comparable *benefits* of valid cueing and *costs* of invalid cueing. Although SOA interacted with the response to the cue, it did so similarly at all ages, a result that suggests that the time course of covert orienting initiated by these cues does not change after 8 years of age.

Table 5 illustrates that children (especially 10-year-olds) had a higher false alarm rate (i.e., percentage of responses on catch trials) compared to adults. Because children were making so many responses on catch trials, it was important to first rule out the possibility that the findings were affected by the children's responding after the cue and without detecting the target. To investigate this issue, I analyzed the data only from participants (n = 18 out of 24) with a false alarm rate of less than 3.0 % in an ANOVA with one between-subject factor, Age (8-year-olds, 10-year-olds, adults) and three within subject factors: SOA (100, 400, 800 msec), Cue type (invalid, neutral, valid), and Visual field (upper, lower). The results did not yield effects of cue or cue by SOA interactions different from the type that I report in this chapter. The results showed that there were no age differences in covert orienting nor age differences in the effects of SOA on covert orienting. Thus, age changes in false alarm rate did not alter the pattern of results.

In this procedure, children appear adult-like in their ability to covertly orient attention by 8 years of age. The results differ from many of the previous studies, all of which had smaller sample sizes, that showed developmental change in validity effects, costs, and/or benefits (Akhtar &

Enns, 1989; Brodeur, 1993; Enns & Brodeur, 1989; Pearson & Lane, 1990; Wainwright-Sharp, 1995). Specifically, many of the previous studies showed that children similar in age to those in the present study were slowed more than adults by invalid cueing (i.e., Akhtar & Enns, 1989; Enns & Brodeur, 1989; Pearson & Lane, 1990).

These results also differ from previous studies that tested with a range of SOAs. Previous studies found age differences in the effects of SOA on validity effects, costs, and/or benefits (Brodeur, 1993; Pearson & Lane, 1990). For example, Pearson and Lane (1990) reported an increasing cost of invalid cueing in 8-year-olds, as SOA increased from 83 msec to 300 msec, but a flat function in older participants. In contrast, Brodeur (1993) reported the opposite result: an effect for adults but not for children. Unlike either of these studies, I found that there were no age differences in the influence of SOA on the *benefit* of valid cueing nor on the *cost* of invalid cueing between age 8 and adulthood.

It is unlikely that differences in sample size, variance, and power could explain why I found no age difference in validity effect while other studies found larger effects in children than adults. I tested more participants in each age group than any previous study: There were 24 per group compared to 13 to 21 in previous studies (Akhtar & Enns, 1989; Brodeur, 1993; Enns & Brodeur, 1989; Pearson & Lane, 1990; Wainwright-Sharp, 1995). Only one previous study reported variance but there is no reason to expect greater variance in my studies of children in which the target was easier to detect than in previous studies. In the one study reporting variance (Pearson and Lane, 1990) the standard errors were larger than in my sample. Thus, while previous studies were able to detect significant age differences in the size of the validity effect that were as small as 22 msec (Enns & Brodeur, 1989), my study had equal or greater power to detect a significant difference in results when one existed.

There are three major differences between this study and previous studies that could possibly explain the discrepant results. First, the previous studies required discrimination rather than simple detection of a large target. This could be an important difference that may explain the findings on age differences. For example, in my simple detection procedure children may be able to shift attention quickly enough on invalid trials to detect the large target but not to discriminate between two targets. Second, unlike previous studies, these procedures involved tests along the vertical meridian, and it is possible that covert orienting develops differently along different meridia. Third, in none of the previous studies were the data corrected for a reaction time bias (see Chapter 2). Like previous studies, I also found age differences in covert orienting when I used medians and did not correct these data for a reaction time bias (Goldberg, Maurer, & Lewis, 1996). The results based on medians showed that 8-year-olds had larger *costs* of invalid cueing, the difference in reaction time between invalid and neutral trials, than older participants. There were no age differences in the *benefit* of valid cueing nor in the influence of SOA on covert orienting. This finding suggests that in 8-year-olds, the invalid condition was affected more than other conditions, and other ages, by variance and positive skew. Without Van Selst and Jolicoeur 's (1994) outlier elimination procedure to eliminate a median bias, I would have concluded mistakenly that there were age differences in the effects of the cue on invalid trials. Thus, the failure of previous studies to correct their data for a reaction time bias is a possible explanation for the discrepant results.

All of these possibilities suggest that the neural mechanisms for covert orienting are functional by age 8, but are perhaps limited to certain parts of the visual field (i.e., vertical meridian) or easily detected stimuli. In the General Discussion (Chapter 6) I will consider what it is that develops after age 8 by relating these findings and those of Chapter 5 to the literature.

This study also suggests that there are two different mechanisms involved in attention, with different time courses. At all ages tested, the time courses for the *benefit* of valid cueing and *cost* of invalid cueing were different, with the benefit waning by the 800 msec SOA and the cost increasing at that point. Although there was no significant cost of invalid cueing in Chapter 2, the results may not be contradictory because the cost was of similar size to that in Chapter 3 and marginally significant (p = .07). In Chapter 4, I discuss possible mechanisms that might be involved in these different time courses of benefit versus cost.

The results on inter-ocular differences in covert orienting indicate that covert shifts of attention are the same when tested in the right and left eye. This information suggests that if there are different outcomes in each eye after binocular deprivation, that it may not be due to normal differences between the two eyes, but rather to differences in the visual history of the two eyes.

The results on the effects of practice indicate that re-testing influences only the rate of responding but not the overall pattern of covert orienting. Thus, testing in the first eye of a patient treated for bilateral cataract should not influence measures of covert shifts of attention from testing of the second eye of the same patient.

Conclusions from Covert Orienting Experiments in Chapter 3

This study of the normal development of covert shifts of attention eliminated problems in previous studies by matching the area of the cues on neutral trials to the cues on valid and invalid trials, and correcting for a reaction time bias. It also involved larger groups of participants than previous studies. Under these conditions, there were no age differences in the effects of valid and invalid cues from 8 years to adulthood, nor any age differences in the effects of SOA. These findings suggest that the neural mechanisms important for covert orienting, including the parietal cortex, are mature enough to allow adult-like performance under some conditions, except that responses are slower. These data from normal participants provide a good estimate of "normal performance" to compare to patients treated for cataract (in Chapter 4) who range in age from 8.2 years to 25.5 years.

CHAPTER 4

COVERT ORIENTING IN PATIENTS TREATED FOR BILATERAL CONGENITAL CATARACT

In this chapter, I present my studies of visual attention in children treated for bilateral congenital cataract.[5] In this introduction, I first summarize what is known about the effects of visual pattern deprivation on the development of vision from a functional and neuro-anatomical point of view. I then link information on the effects of deprivation to hypotheses about neural mechanisms involved in human visual attention.

A cataract is an opacity in the lens of the eye that scatters light and blurs images on the retina. When it is dense and central, it prevents any focussed patterned input from reaching the retina. Treatment for the cataract occurs by surgical removal of the cataract and the lens of the eye, rendering the eye aphakic (without a natural lens). The aphakic eye is then fitted with contact lenses or glasses to focus visual input on the retina.[6] In humans treated for unilateral cataract during early childhood, patching the normal eye is

[5] Pilot results from children treated for unilateral cataract are presented in Appendix C.
[6] Recently, intraocular lenses have been used in some centers but that was the case for none of the participants included in this thesis.

recommended to reduce competition and to force usage of the previously deprived eye. I will consider the effects of such deprivation on visual development and of comparable deprivation on vision and brain development in animals.

Visual Deprivation Studies with Humans

To assess the influence of patterned visual input on the development of vision and of the brain, researchers have studied the effects of pattern deprivation in both eyes (binocular deprivation) and in one eye (monocular deprivation). Studies of humans show that such deprivation affects some visual functions more than others (see Maurer et al., 1989; Maurer & Lewis, 1993). Maurer and colleagues have hypothesized that there is a general principle about the effects of visual deprivation: The visual functions that are affected most severely by deprivation in humans are those that are immature at birth (e.g., symmetrical OKN, stereopsis, visual acuity, contrast sensitivity and peripheral vision). The functions that tend to be affected least by deprivation are those that are relatively mature at birth (e.g., gross discrimination of form and color vision).

The effects of deprivation on the development of visual acuity have been studied most extensively (e.g., Birch & Stager, 1988; Drummond, Scott, & Keech, 1989; Maurer et al., 1989; Maurer & Lewis, 1993; Robb, Mayer, & Moore, 1987; Scott, Drummond, Keech, & Karr, 1989). Most of these studies show that Snellen acuity is worse following relatively longer periods of deprivation (e.g., 5 months or more) than following shorter periods of deprivation. Studies of the effects of the timing of deprivation indicate that children deprived from birth have worse Snellen acuity compared to children deprived at a later age. The studies show that there is an interaction between the duration and timing of deprivation, such that the outcome is particularly bad when the deprivation begins at birth and is long lasting. In addition, a comparison of Snellen acuity in children treated for monocular versus binocular deprivation from birth and with comparable durations of deprivation, indicates that in humans, as in kittens and monkeys (see below), vision is relatively better after binocular deprivation than after monocular deprivation. Furthermore, the research shows that the potentially poorer outcomes following monocular deprivation can be tempered by extensive patching of the normal eye.

Visual Deprivation Studies with Animals

The effects of the timing and the duration of deprivation have been studied in animals by varying the time during the animal's life when the deprivation began and how long it lasted. I focus my review on studies with cats, since they are the most frequently studied, and with monkeys, since they are the species with the visual system most similar to humans (Boothe, 1981; Mitchell, 1989; Hubel, 1979). In these studies, animals were visually deprived by occlusion, by suturing the eye(s) shut, or by being reared in the dark. Here, I concentrate on deprivation caused by suturing shut the animal's eye(s) because this type of deprivation is closest to the type of pattern deprivation in humans with dense and central cataract(s).

It has been shown that early visual deprivation has deleterious effects on the visual system in both cats (e.g., Wiesel & Hubel, 1963 a, b, 1965; Daw, Fox, Sato, Czepita, 1992) and monkeys (e.g., Crawford, Blake, Cool, von Noorden, 1975; Blakemore & Vital-Durand, 1986). Recordings of single cells in layer IVc of the visual cortex of the normal cat and monkey, and in layers adjacent to layer IVc, indicate that all cells encountered respond to stimulation of one or both eyes (Hubel & Wiesel, 1977). Following bilateral deprivation in kittens that began 6 to 18 days after birth and lasted 2.5 - 4.5 months, approximately one-third of the cells tested in the visual cortex responded normally to visual stimuli; about one-third of the cells were responsive but abnormal (without precise receptive field properties); and about one-third were unresponsive (Wiesel & Hubel, 1965).

The effects of bilateral lid suture were different in adult monkeys that had been deprived for the first 2 to 16 weeks of life. There was a shift in ocular dominance, so fewer neurons responded when stimulated by either eye and most responded only to stimulation of one eye (Crawford, Pesch, von Noorden, Harwerth, & Smith, 1991). These findings were independent of how long the monkeys had been deprived. Thus, bilateral deprivation in monkeys impairs neurons' responsiveness to stimuli presented simultaneously to both eyes while leaving neurons' responsive to stimuli presented to one eye or the other. In addition, the abnormalities in visual cortex after binocular deprivation in cats and monkeys are relatively less severe when the deprivation occurs later in life compared to when it is present from birth (reviewed in Mitchell, 1981; Movshon & Van Sluyters, 1981).

The effects of monocular deprivation seem to be more severe than the effects of binocular deprivation. Studies of kittens monocularly deprived early in life show there is a shift in the normal cortical balance between the two eyes in favor of the nondeprived eye (e.g., Wiesel & Hubel, 1963a, b, 1965, Daw et al., 1992). Considerably fewer cells can be activated by visual stimuli presented to the deprived eye than to the nondeprived eye. However, following monocular deprivation for the same duration during adulthood, many cells can be driven by the deprived eye (Wiesel & Hubel, 1963b). Thus, the effects of deprivation are less severe when it occurs later in life.

Early monocular deprivation also affects the ocular dominance distribution in the monkey (e.g., Crawford et al., 1975). Following monocular deprivation during infancy, there is a marked decrease in the number of cells in the visual cortex that can be activated by visual stimuli presented to the deprived eye, and the disruption is greater following longer periods of deprivation. Deprivation does not have effects on the retina and, at least in monkeys, does not have any physiological effects on the lateral geniculate nucleus (Blakemore & Vital-Durand, 1986; Boothe et al., 1985). Hence, any behavioral deficits are presumably due to damage to the visual cortex or higher cortical areas.

Behavioral data on the effects of the timing and duration of deprivation agree with the physiological findings already described. Behavioral data show that the visual system can be disrupted when there is abnormal visual input during a *sensitive* period, and the damage is worse the longer the deprivation lasts during that period (reviewed in Boothe, 1981; Mitchell, 1989; Hubel, 1979). For example, data on the effects of the timing and duration of deprivation on visual acuity indicate the worst outcomes are for deprivation beginning soon after birth, and lasting for the longest duration (e.g., Boothe et al., 1985; Giffin & Mitchell, 1978; Mitchell, 1981).

Behavioral data also indicate that monocular deprivation has a more drastic effect on visual development of the deprived eye compared to binocular deprivation. A number of studies have shown that the poorer behavioral outcomes following monocular deprivation can be reduced with the method of "reverse occlusion," that is by occluding or removing the nondeprived eye (reviewed in Boothe, 1981; Mitchell, 1981; Mitchell & Murphy, 1984). These findings suggest that after monocular deprivation, the deprived eye has reduced ability to compete with the good eye for connections in the brain.

Behavioral studies of cats and monkeys show that some visual functions are affected more severely by deprivation than are other visual functions. These impaired visual functions include visual acuity (e.g., Giffin & Mitchell, 1978), spatial contrast sensitivity (e.g., Crawford et al., 1993; Harwerth et al., 1981; Harwerth, Smith, Paul, Crawford, & von Noorden, 1991; Mitchell & Murphy, 1984), peripheral vision (e.g., Sherman, 1973), and optokinetic nystagmus (e.g., Van Hof-van Duin, 1976). However, there is relatively little impairment in the gross discrimination of form (e.g., van Hof-van Duin, 1976) and temporal contrast sensitivity (Harwerth et al., 1991). A general principle that explains these results is that the visual functions that are affected most severely by deprivation are immature at birth and are slow to develop, whereas the functions that are more mature at birth are relatively unaffected by deprivation (Maurer et al., 1989).

Effects of Deprivation on Neural Mechanisms Involved in Attention

Early visual deprivation also has an adverse effect on areas of the brain involved in attention. Studies of monkeys deprived from birth by bilateral lid suture indicate that binocular deprivation dramatically reduces the sensitivity of parietal cells to visual stimuli, much more than it affects cells in the primary visual cortex (Carlson et al., 1987; Hyvarinen & Hyvarinen, 1979; 1982; Hyvarinen et al., 1981). Although there are other areas of the brain involved in attention (e.g., pulvinar nucleus of the thalamus, frontal lobes), there is evidence only on how deprivation affects the frontal cortex, and this evidence is only from rats (Schleibs, Bigl, & Biesold, 1982). More details on the possible role of these other areas are provided in Chapters 5 and 6.

The effects of binocular deprivation on cells in the parietal cortex were described briefly in Chapter 1; more detail is provided here. The research from monkeys binocularly deprived from shortly after birth shows that binocular deprivation dramatically reduces the sensitivity of parietal cells 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). Both eyelids of monkeys were sutured shut during the first week of life and were then opened at 7, 10, or 11 months of age. Results showed that only 2% of the cells tested in the parietal cortex were activated by visual stimuli and 1% by both visual and somatic stimuli–these were reductions of 92% and 97%, respectively, compared to those observed in control monkeys at 15 months of age. However, the number of cells activated by (a) minor motor movements, or by (b) feeling or touching objects, increased by 117% and 53%, respectively. This result suggests that when visual input to the parietal cortex is absent early in life, its neural space is taken over by input from another modality (e.g., Carlson et al., 1987; also see Rauschecker, Egert, & Hahn, 1987).

Although Hyvarinen and colleagues did not measure visual attention directly, the monkeys' responses to visual stimuli were clearly impaired. When tested immediately after the deprivation ended, monkeys were able to detect a moving light spot in a dark room but not able to track the movement of large objects or to grasp large objects. Deprived monkeys never developed a response to a quickly approaching hand and never chattered their teeth in response to a threatening face of the examiner (Carlson, 1990). Deprived monkeys had abnormal visually-guided behavior during the 12 months following the end of deprivation: They had trouble reaching accurately for objects, often bumped into objects, and moved carefully around their environment using tactile cues to explore their surrounds (Carlson, 1990; Hyvarinen et al., 1981; Hyvarinen & Hyvarinen, 1979). In summary, the monkeys' responses to visual stimuli were clearly impaired after deprivation had ended.

The Role of the Parietal Cortex in Visual Attention

There are three main cortical connections of the posterior parietal cortex that are related most directly to visual-attentional processing, all of which are reciprocal: (1) visual cortex; (2) temporal cortex; and (3) prefrontal cortex. Cells in the posterior parietal cortex project to two subcortical areas: the superior colliculus and thalamus (e.g., LaBerge, 1995a, b). The monkey posterior parietal lobe is divided into two major areas: (1) a superior lobe that contains somatosensory area 5; and (2) an inferior lobe (positioned around the intraparietal sulcus) that includes visual area 7a, somatosensory area 7b, visual lateral intraparietal area (LIP), and dorsal prelunate area (e.g., LaBerge, 1995a, b). Neurons in monkey posterior parietal cortex are known to respond to different types of sensory input: visual, oculomotor, somesthetic, somatomotor, vestibular, and auditory (Hyvarinen, 1982). Hyvarinen and Poranen (1974) found an increase in activation when visual or somatic stimuli were presented within the receptive field of neurons in area 7 compared to when no stimuli were presented at all: Approximately one-third of the cells were activated best by visual stimuli presented alone (visual cells); about one-third were activated by either manual reaching (somatomotor), manipulating objects (somatosensory), or tracking (visual cells); and the remaining cells were activated by looking and reaching (11%), by touching and looking (6%), or by other stimuli (11%).

Many lines of evidence indicate that the parietal cortex also plays a functional role in visual-spatial attention (reviewed in Colby, 1991; Hyvarinen, 1982; LaBerge, 1995a, b; Mesulam, 1981). Single unit recordings from neurons in monkey posterior parietal cortex indicate that there is an increase in firing when the monkey maintains visual fixation on a central target while attending covertly to a peripheral target within a cell's receptive field (Steinmetz, & Constantinidis, 1995; Steinmetz et al., 1994). Studies of human adults using Positron Emission Tomography (PET) show greater increases in blood flow in the superior parietal region (areas 5 plus 7 in humans) compared to other brain regions (e.g., frontal, visual, motor cortices) when attention is shifted covertly to a peripheral location by a peripheral cue or by a central cue (Corbetta et al., 1993; Petersen et al., 1994). The activation is greater in the hemisphere contralateral to the location toward which attention shifted than in the ipsilateral hemisphere. Unilateral damage to the posterior parietal cortex, in both the monkey and human, results in a disruption of attention in the contralateral half of extrapersonal space (e.g., Hyvarinen, 1982; Mesulam, 1981). For example, a patient may fail to shave one side of his face, or to eat food from one side of his plate. Patients with bilateral posterior parietal damage have difficulty in visual-spatial attention. For example, a patient may have difficulty observing more than one visual object at a time (simultagnosia) (e.g., Hyvarinen, 1982).

Posner and colleagues (e.g., Posner et al., 1978; Posner & Petersen, 1990) have used a spatial cueing task like that described in Chapters 2 and 3 to examine the effects on attentional abilities of damage to the parietal cortex. Tests of monkeys (Petersen & Robinson, 1986) and human adults with unilateral parietal damage (Baynes et al., 1986; Petersen et al., 1989; Posner et al., 1984; Posner et al., 1987) indicate that they have longer than normal reaction times on invalid trials, when attention must be *disengaged* from the cued location and then shifted to the target at the uncued location (Posner et al., 1982; Posner, et al., 1984; Posner, et al., 1987; Posner, 1988, 1990). Specifically, there are extremely long reaction times when attention is cued to the *good* visual field (ipsilateral to the parietal lesion) and the patient is required to shift attention from the good visual field to detect a target that is presented in the *bad* visual field (contralateral to the parietal lesion). This

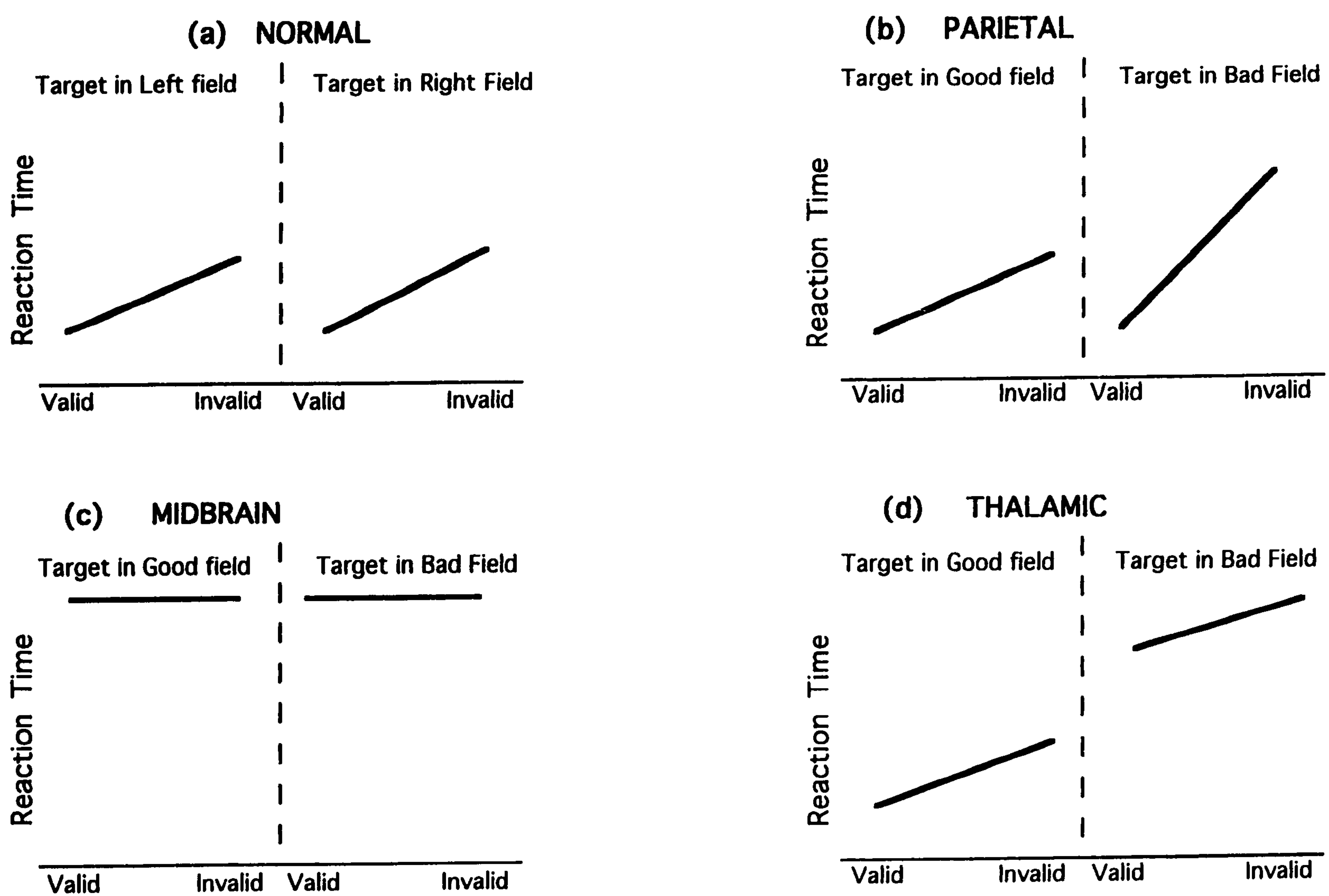

Figure 10. Observed patterns of performance in normals (a) and in patients after brain damage (b, c, d) on a Posner Task (Based on Posner 1988, Figure 3).

pattern of deficit for patients with parietal damage is shown in Figure 10b. (Note that this is the pattern of deficit following damage in adulthood; in the General Discussion, I consider the effects of infantile lesions on visual-spatial attention in a Posner task.)

Groups of patients with damage to parts of the brain other than the parietal cortex show different patterns of attentional deficits on a Posner task. Patients with unilateral damage to the midbrain are slower than normal following both valid and invalid cues, in both visual fields (Posner et al., 1982). This pattern of deficit for patients with midbrain damage is shown in Figure 10c. Posner argues that these reaction time data indicate impairments in *moving* attention to a target or cued location both contralateral *and* ipsilateral to the lesion.

Another pattern of deficit is shown in Figure 10d for patients with unilateral lesions of the thalamus involving the pulvinar. When the target is in the good visual field, these patients perform normally. When the target appears in the bad visual field, patients are slowed more than normal both with correct cueing on valid trials and incorrect cueing on invalid trials (Posner, 1988). According to Posner, these data suggest that thalamic patients have deficits in *engaging* attention in the visual field contralateral to the lesion. This pattern is different from that shown by parietal patients since thalamic patients show increased reaction time on *both* valid trials and invalid trials in the contralateral visual field.

Taken together, the literature suggests that early visual experience may be important for the normal development of attention because attention is slow to develop (see Chapter 3); damage to the parietal cortex during adulthood disrupts it (Posner et al., 1982; 1984; 1987; Posner, 1988; 1990); and neurons in the parietal cortex develop abnormally in the absence of normal binocular visual input (Carlson et al., 1987; Hyvarinen & Hyvarinen, 1979; Hyvarinen et al., 1981). In this chapter, I used the spatial cueing task described in Chapter 3 to provide the first examination of whether early binocular deprivation interferes with the normal development of human visual-spatial attention. I also tested a few patients treated for unilateral congenital cataract, but because there was little basis on which to make predictions from the literature and, the initial testing indicated no deficits, I did not pursue that testing. Results from the tested patients treated for unilateral congenital cataract are in Appendix C.

Method

Participants

The final sample consisted of 32 eyes of 16 patients treated for bilateral congenital cataract. Table 9 provides clinical details for the 32 eyes included in the sample. Patients were included if they were at least 8 years of age during the 24 months during which testing was conducted. Patients were included in this sample with bilateral congenital cataract only if they had dense and central cataracts in both eyes identified on the first eye exam prior to 6 months of age. We assume that a child diagnosed with cataracts before 6 months of age was deprived since birth because it would be unusual for a dense and central cataract to develop rapidly between birth and 6 months of age.

Specific criteria were used in order to select patients likely to have complete pattern deprivation. Patients were included in the sample treated for cataract(s) if, on the first eye exam, the ophthalmologist determined the cataract interfered seriously with vision because: (1) The eye did not fix or follow a light, (2) the cataract completely blocked the view of the fundus through an undilated pupil, (3) no red light reflex was visible through an undilated pupil and/or (4) the cataract looked dense and central. Patients were excluded from the sample if their cataract was not dense (dull red reflex visible through the cataract), located only in the periphery of the lens, or smaller than 5 mm.

About 1 week after surgery to remove the cataract, the aphakic eyes (without a natural lens) were measured for contact lenses. About 1 week later, the patients had the contact lenses fitted and the power checked. In all but two patients, contact lenses were fitted on both eyes on the same day. Most children wore their contact lenses successfully and those who did not switched to glasses. Treatment was followed by years of nearly normal visual experience. Refractive error at the time of testing is shown in Table 9.

Table 9.
Clinical Details at the Time of Covert Orienting Test for the 16 Children Treated for Bilateral Congenital Cataract.

Name (Age in years)	Refraction (diopters)	Age at Diagnosis (months)	Months (days) of Deprivation	Snellen Acuity[7]
L. M.	OD +12.50	0.0	3.1 (94)	6/24
(8.2)	OS +16.50	0.0	3.1 (94)	6/15
B. B.	OD +13.75	3.4	5.4 (165)	6/60
(9.4)	OS +14.00	3.4	5.4 (165)	6/21
A.L.	OD +16.50	2.0	5.4 (165)	6/15
(9.8)	OS +15.50	2.0	5.4 (165)	6/30
A. H.	OD +16.25	1.2	2.1 (64)	6/60
(10.9)	OS +15.75	1.2	2.1 (64)	6/15
J. G.	OD +17.00	0.0	2.7 (83)	6/18
(10.9)	OS +23.00	0.0	2.7 (83)	6/12
S. S.	OD +12.75	2.0	7.5 (228)	6/18
(11.5)	OS +12.00	2.0	7.5 (228)	6/60
K. M.	OD +11.25	1.0	2.3 (71)	6/6
(11.9)	OS +13.25	1.0	2.3 (71)	6/9.5
C. B.	OD +17.00	1.1	3.0 (91)	6/7.5
(12.1)	OS +23.00	1.1	3.0 (91)	6/60
J. J.	OD +14.50	0.6	1.8 (53)	6/12
(12.9)	OS +18.50	0.6	1.8 (53)	6/9
A. D.	OD +16.50	0.0	3.2 (97)	6/24
(13.1)	OS +15.25	0.0	3.2 (97)	6/30
C. P.	OD +10.25	1.9	6.1 (187)	6/9
(14.5)	OS +11.75	1.9	6.1 (187)	6/15

[7] Snellen Acuity is based on the measurement closest to my testing date.

Table 9 (continued).
Clinical Details at the Time of Covert Orienting Test for the 16 Children Treated for Bilateral Congenital Cataract.

Name (Age in years)	Refraction (diopters)	Age at Diagnosis (months)	Months (days) of Deprivation	Snellen Acuity
I. W.	OD +11.50	0.0	5.0 (151)	6/60
(15.1)	OS +13.50	0.0	8.6 (264)	6/7.5
A. C.	OD +11.75	2.9	6.4 (196)	6/12
(16.2)	OS +12.25	2.9	5.3 (161)	6/15
A. R.	OD +17.0	0.2	3.5 (103)	6/30
(16.8)	OS +12.5	0.2	3.5 (103)	6/30
M. D.	OD +11.25	0.0	4.1 (129)	6/7.5
(17.2)	OS +14.2	0.0	4.1 (129)	6/18
S. G.	OD +8.50	3.3	19.5 (586)	6/18
(20.4)	OS +9.75	3.3	19.5 (586)	6/18

OD = Right Eye
OS = Left Eye

The duration of deprivation was taken from birth to the age at which the eyes were first fitted with contact lenses after surgery. Table 9 shows that the eyes in this study were deprived for as long as 19.5 months and as little as 1.8 months (M = 5.1 months).

Patients with some of the abnormalities commonly associated with deprivation such as strabismus (misaligned eyes), horizontal nystagmus (repetitive jerky eye movements in a horizontal direction), and/or microcornea (eye smaller than normal) were included (Maurer & Lewis, 1993). Children with some additional serious problems that could interfere with vision (e.g., glaucoma, detached retina) were excluded. Patients were excluded from the sample if they had continuing deprivation following surgery because they did not wear their optical correction regularly. Specifically, children who wore their optical correction less than 75% of the time during the first 7 years of life and/or stopped wearing their optical correction prior to 7 years of age were excluded.

Children were also excluded if there was significant evidence of developmental delay (scores more than two standard deviations below the normal mean when tested with the Bayley Scale of Infant Development at age 2 and/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.

Stimuli

The stimuli were the same as those used with normal children in Chapter 3. They had been made large enough to be visible to patients treated for cataract, who are known to have reduced vision. The rationale for their design was as follows:

Cues

I determined the size of the cues based on previous measurements of linear letter acuity in this cohort (Maurer & Lewis, 1993). Linear letter acuity resembles my task because it requires the discrimination and recognition of symbols (letters) with central vision. For 95% of the cohort, it is better than 6/240. To receive that score participants must resolve letters formed from lines 40 minutes wide (where 60 minutes = 1 degree of visual angle). Therefore, I made the cues from lines that were at least one degree high and one degree wide. The border around all cues was at least 1 degree thick. Thus the cues were designed to be visible to patients with at least 6/360 linear letter acuity.

Targets

The only data available on peripheral acuity for this cohort are contrast sensitivity data for gratings presented along the horizontal meridian, with their nearest edge 10 degrees in the periphery (the closest distance to center that was tested in this cohort; Tytla, Lewis, Maurer, & Brent, 1992). There are no similar data in the vertical periphery on such patients. At least 95% of the sample of children tested by Tytla et al. (1992), consisting primarily of those children with good fixation, had a grating acuity (the smallest stripe that could be resolved at 100% contrast) of at least 1.5 cycles per degree at 10 degrees in the periphery. Thus, they were able to resolve stripes 20 minutes wide. The near edge of the target was 5 degrees from the center of the central stimulus. To ensure that the target was well above threshold, I made the black and white checks 2 degrees high and wide.

Targets were displaced vertically from center, even though most other studies have used horizontal displacement. Vertical displacement was used in order to minimize the variability in the distance of fixation from the target for the children treated for bilateral congenital cataracts, many of whom have a manifest nystagmus that is predominantly horizontal.

Procedure

The procedure was the same as for normal participants in Chapter 3 except:

(1) The patients' eyes were corrected optically for the testing distance. Patients wore contact lenses or glasses but, since their eyes are without a natural lens, they are unable to accommodate to focus visual input at varying distances. In order to optically correct the aphakic eye(s) for the testing distance of 36 cm, patients were fitted with lenses inserted into trial frames over their contact lenses or glasses. The ophthalmologist determined whether the current contact lenses corrected each eye for infinity and, if not,

how much dioptric power would have to be added or subtracted to achieve that result. He did so by using retinoscopy or by seeing whether the patient's visual acuity at far could be improved by adding trial lenses with more or less power. If the regular contact lens focused an eye at infinity, then I placed a +2.75 diopter (D) lens in front of it to focus the eye at 36 cm.[8] Additional or less power, as necessary, was given if the current contact lens did not focus an eye at infinity. This was calculated by adding +2.75 diopters to the power needed over the contact lens to focus the eye at infinity. For example, if the ophthalmologist determined that the eye needed -1.0 D over the contact lens to focus at infinity, then a +1.75 power lens was used in the trial frame (-1.0 D + 2.75 D = + 1.75 D). If the child was wearing bifocals, then the current distance correction needed for focusing the eye at infinity plus the power needed for focusing at the testing distance was inserted into the trial frames, and the child did not wear the bifocals.

(2) Half of the patients in the group treated for bilateral cataract were tested first with the eye with the better acuity based on the Sheridan Gardiner Test and the other half were tested first with the eye with the worse acuity. If both eyes had the same acuity, I alternated whether the first eye tested was the right eye or the left eye. This procedure was used to balance across eyes with

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[8] If an eye is focussed at infinity, the additional diptric power needed to focus it at 36 cm is equivalent to the inverse of the distance in meters. Thus, the dioptric power needed to focus the eye at 36/100 meters = 100/36 diopters = +2.75D.

better versus worse acuity any benefits from learning or impairments because of fatigue.

(3) All but one patient completed the testing in one day. For that patient, testing was discontinued on the first day because of a problem with the computer. We repeated the entire protocol on the second day.

(4) Patients were not given the screening test designed to exclude normal participants with eye problems.

Data Analyses

As in previous chapters, I eliminated trials with eye movements or anticipations. I used the outlier elimination procedure to calculate mean reaction times for each participant and for each condition. I decided to treat the two eyes of the patients as independent in order to have more data and because each eye can have a different history of visual deprivation (see Ian W., Adam C. in Table 9). To verify that it was reasonable to treat the two eyes as independent, I first established that there was no significant correlation between the validity effect in the two eyes.

Eyeballing the data showed that patients were slower than normal participants overall. Because the measure of covert orienting is in the effects of cueing, not absolute reaction time, I analyzed the differences in reaction time on valid and invalid trials (validity effect), rather than the actual reaction times. If the absolute times to respond on valid and invalid trials were put into an ANOVA, the analyses would indicate just that the groups differed. This result is not of interest because it might reflect differences between normals and patients in the speed of seeing the cue, or of making a motor response to the target, rather than a difference arising from attention. Because it is reasonable to assume that any such differences will have equal effects on valid and invalid trials, it is appropriate to eliminate them by subtracting the reaction time on valid trials from invalid trials. The residual will reflect what is different between the two types of trials, which can only have to do with covert orienting of attention. In the case of valid cues, attention is directed to the correct location of the upcoming target; in the case of invalid cues, attention must be disengaged away from an expected location of an upcoming target, moved and then engaged at a new location. Therefore, the calculation of a *validity effect* (Invalid - Valid RT) is a reasonable approach to evaluate covert orienting in groups of participants that differ in the time needed to respond. I subsequently tried to determine whether any differences in validity effect came from differences in benefit (valid - neutral RT) and/or cost (invalid - neutral RT). I used these as supplementary rather than primary analyses because I could not be certain that the neutral trials provided an equivalent baseline for patients and normal participants (e.g., Jonides & Mack, 1984).

I compared the patients to the normal participants reported in the previous chapter (Chapter 3). Because there were no age differences in covert orienting on this task in normal participants from 8 years to adulthood and because the age distribution of patients ranged from 8.2 years to 25.5 years, I decided to combine all data from normal participants to gain the best estimate of "normal" performance.

To examine the effects of duration of deprivation on covert shifts of attention, I split the data from the patients treated for bilateral cataract into two groups based on the number of months of deprivation. I decided to split the data at 4 months because the literature on the normal development of attention (see Chapter 3) shows that a number of attentional abilities, including the ability to disengage attention easily, improve dramatically around 4 months of age (Johnson et al., 1991). One group (n = 16 eyes, of 8 patients; mean age = 12.1 years; range 8.2 - 16.8 years) had a history of shorter visual deprivation lasting for less than 4 months (mean duration = 2.7 months; range 1.8 - 3.4 months). The second group (n = 16 eyes, of 8 patients; mean age = 14.3 years; range 9.4 - 20.4 years) had a history of longer visual deprivation lasting for more than 4 months (mean duration = 7.6 months; range 4.2 - 19.5 months).

Results

Preliminary Analyses

Sheridan-Gardiner near acuity for all eyes ranged from 6/6 to 6/120. Patients treated for bilateral congenital cataract performed well on the post-tests: They demonstrated that they understood the meaning of the cues (e.g., "helper," "fooler," or "no clue") with an average score of 9.7 correct out of 10 trials (range 8 - 10), during which eye movements occurred on an average of only 0.2 out of 10 trials (range 0 - 2). They also demonstrated an ability to discriminate between 1 or 2 boxes in the periphery. The average score was 9.8 correct out of 10 trials (range 8 - 10), during which eye movements occurred on an average of 0.2 out of 10 trials (range 0 - 2).

Data on the frequency of eye movements, responses on catch trials, and anticipations of the target during the main task are shown in Table 10. Patients treated for bilateral cataract did not differ from normal participants on any of these measures. The percentage of data eliminated by the outlier elimination procedure (Van Selst & Jolicoeur, 1994) was 3.30% for eyes treated for bilateral cataract(s) compared to 3.11% for normal participants (in Chapter 3).

There was no significant correlation of the size of the validity effect between the two eyes of patients treated for bilateral cataract ($p > .10$). Therefore, the data from the two eyes were treated as independent. Table 11 shows the results from an ANOVA on validity effects (Invalid - Valid RT), with 1 between-subject factor, Group (Bi-long, Bi-short, normals), and 2 within-subject factors: SOA (100, 400, 800 msec), and Visual field (down, up).

Effect of Group. There was a significant interaction between Group and SOA ($p < .001$). Figure 11 illustrates that patients performed normally when the cue appeared 100 or 400 msec before the target. At the 800 msec SOA,

Table 10.
Mean Percentage (Standard Error) of Eye Movements, Responses on Catch Trials, and Anticipations of the Target in Patients Treated for Bilateral Congenital Cataract, Unilateral Congenital Cataract, and Normal 8-year-old, 10-year-old and Adult Participants.

	Eye Movements	
Bilateral Congenital	3.1%	(0.3%)
Unilateral Congenital	4.7%	(0.7%)
Normal 8-year-olds	4.6%	(0.5%)
Normal 10-year-olds	4.4%	(0.5%)
Normal Adults	1.0%	(0.3%)

	Responses on Catch Trials	
Bilateral Congenital	10.7%	(1.7%)
Unilateral Congenital	10.0%	(3.1%)
Normal 8-year-olds	15.1%	(2.4%)
Normal 10-year-olds	21.1%	(2.5%)
Normal Adults	5.4%	(1.4%)

	Anticipations of the Target	
Bilateral Congenital	0.4%	(0.1%)
Unilateral Congenital	0.3%	(0.1%)
Normal 8-year-olds	0.6%	(0.2%)
Normal 10-year-olds	0.7%	(0.2%)
Normal Adults	0.5%	(0.2%)

Table 11.
Covert Orienting after Binocular Deprivation Lasting Less or More Than 4 Months.

Results of the 3-way ANOVA on Validity effects: Group, SOA, Visual Field

Effect	*F*	*df*	*p*
Group	.46	2,101	.63
SOA	.86	2,202	.42
Visual Field	.02	1,101	.96
Group X SOA	**5.22**	**4,202**	**< .001**
Group X Visual Field	1.90	2,101	.15
SOA X Visual Field	.17	2,202	.83
Group X SOA X Visual Field	.38	4,202	.82

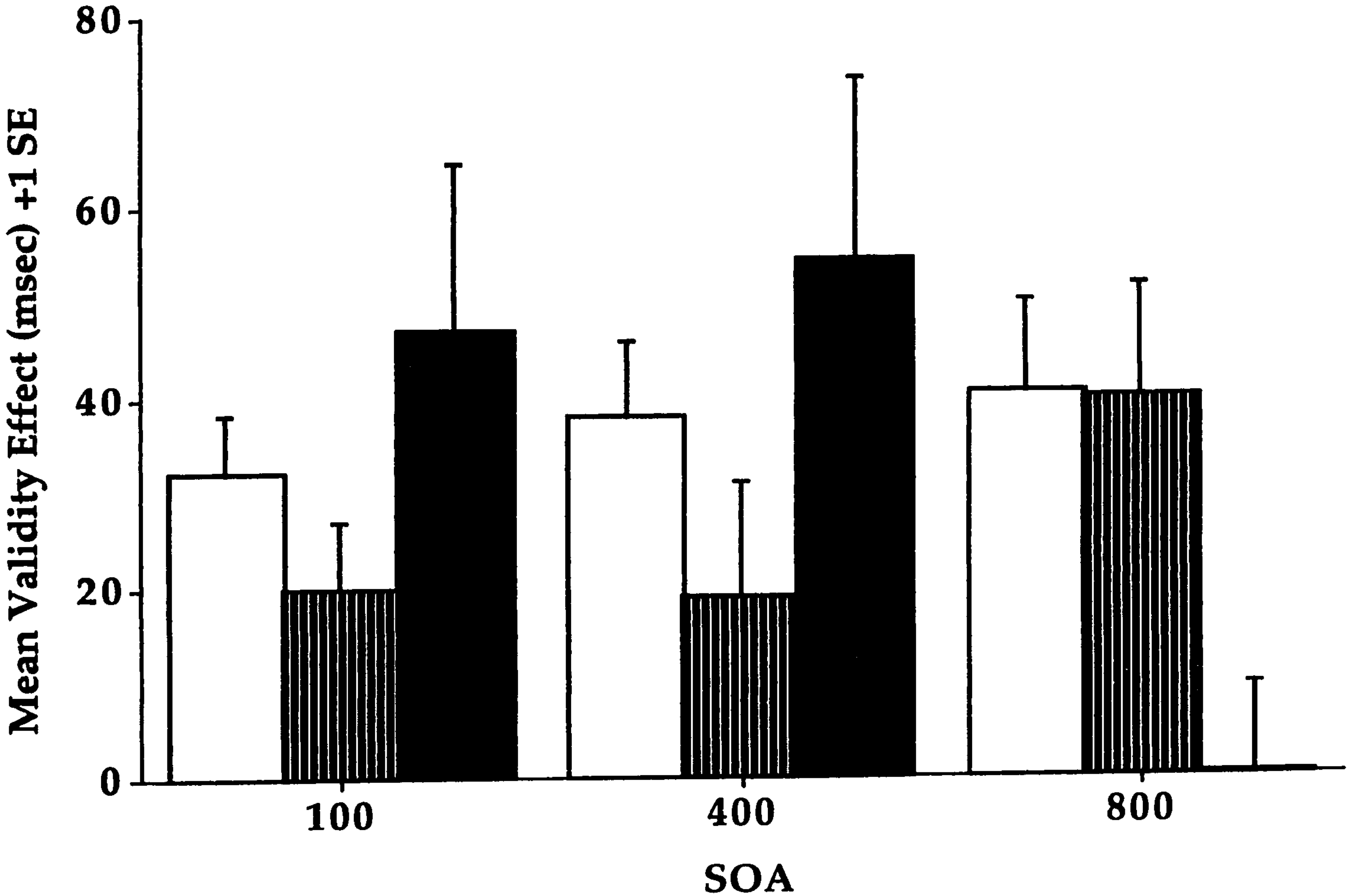

Figure 11. Validity effects as a function of SOA for Normals (white bars), Bilaterally Deprived Patients with Short Deprivation (striped bars) and Bilaterally Deprived Patients with Long Deprivation (black bars).

patients with longer deprivation (> 4 months), unlike normals and patients with shorter deprivation, responded no more quickly on trials with valid cues than on those with invalid cues (Analyses of simple effects, and Tukey post-tests, $ps < .01$).

A graph of the mean reaction time on valid, invalid, and neutral trials for each group of participants at each SOA is shown in Figure 12. The data suggest no cost of invalid cueing at the 800 msec SOA in patients with longer periods of bilateral deprivation, but this was difficult to analyze further because the neutral trials did not seem to provide an appropriate baseline for this group: at two SOAs the mean reaction time on neutral trials was actually longer than the mean reaction time on invalid trials. Similar problems with neutral trials have been reported in the literature (Jonides & Mack, 1984) and have been observed in some conditions previously in this thesis.

Figure 13 shows scatterplots of the validity effect as a function of the duration of deprivation for trials at the 100 msec, 400 msec, and 800 msec SOAs. The figure illustrates that at the 800 msec SOA, patients with longer periods of deprivation have smaller validity effects.

Discussion

Under these conditions, patients treated for bilateral cataract before 4 months of age, the age at which the ability to easily disengage attention

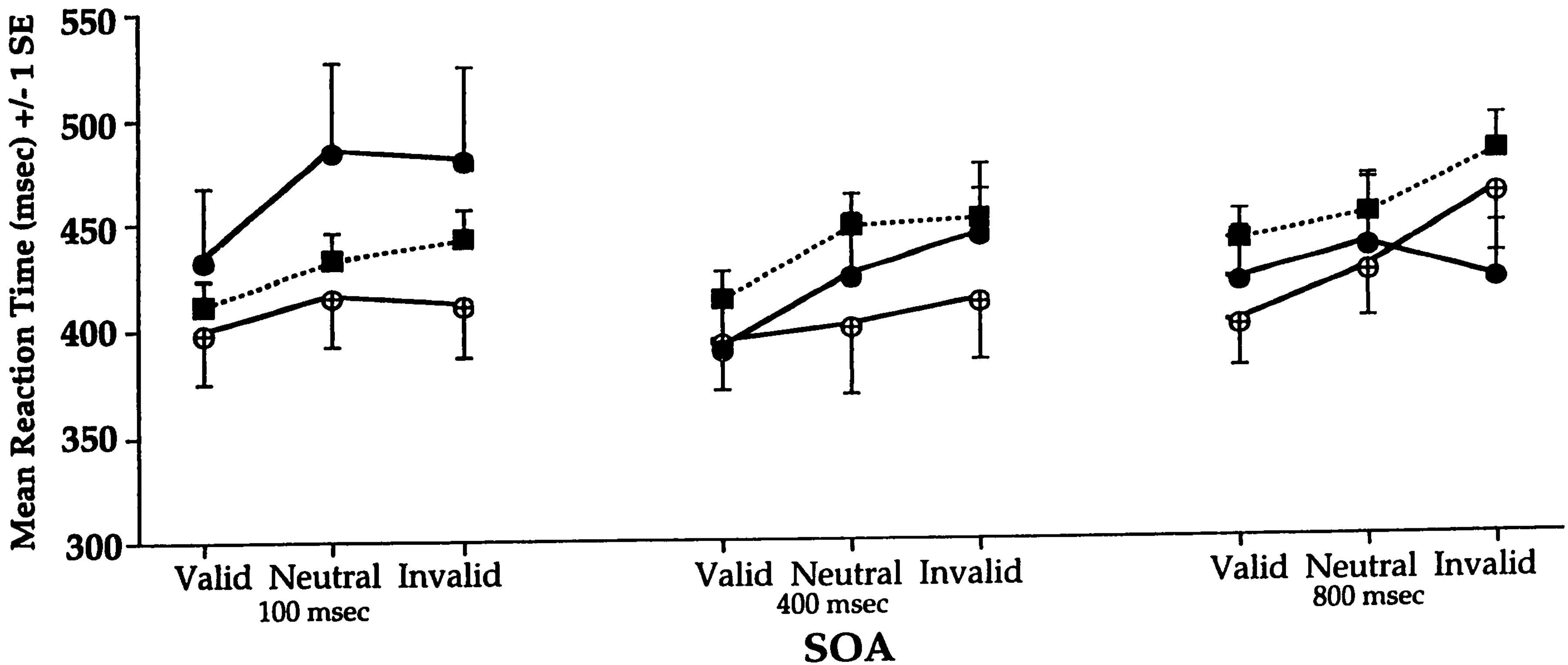

Figure 12. Reaction time as a function of cue type and SOA for Normals (filled squares), Bilaterally Deprived Patients with Short Deprivation (open circles), and Bilaterally Deprived Patients with Long Deprivation (filled circles). Means are connected to facilitate comparisons of the conditions at each SOA.

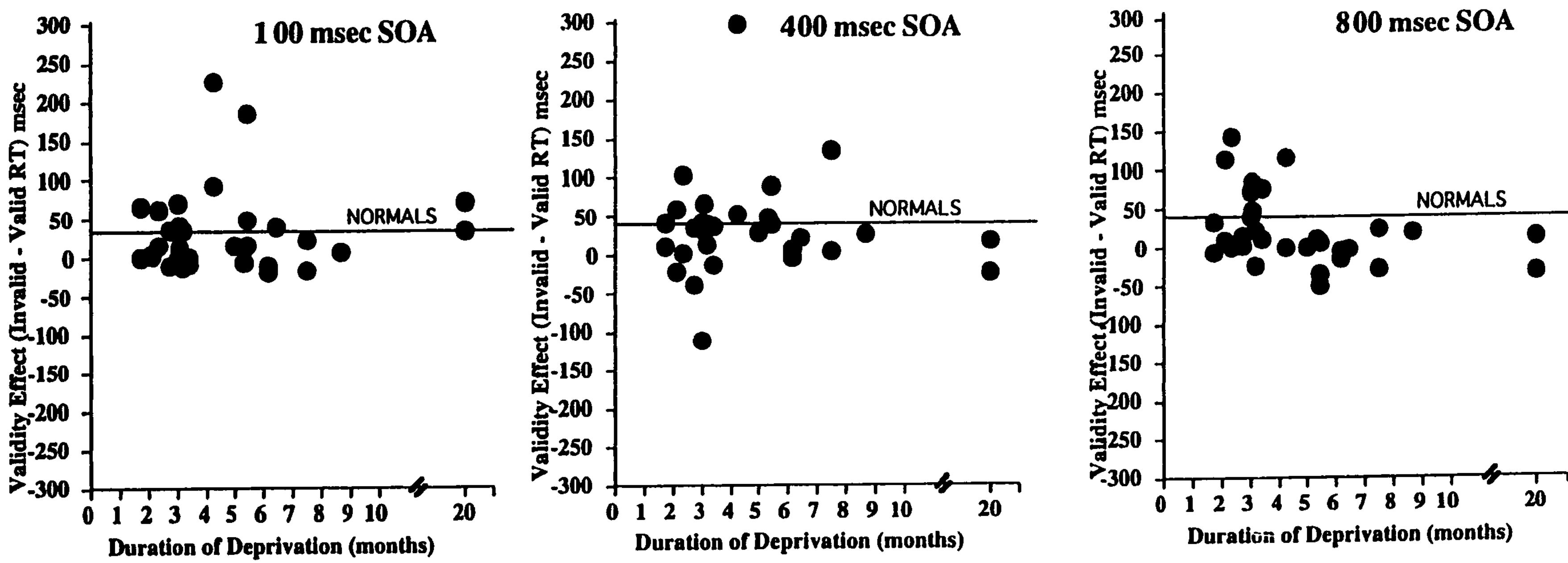

Figure 13. Scatterplot of Validity Effects (Invalid - Valid RT) at the 100 msec SOA (left graph), 400 msec SOA (middle graph), and 800 msec SOA (bottom graph) for Bilaterally Deprived Patients as a function of the Duration of Deprivation (months). On each graph, the line indicates the normal value. Each dot represents the result for one deprived eye.

appears to emerge (e.g., Johnson et al., 1991), performed normally. Patients with deprivation lasting longer than 4 months, performed normally at 100 and 400 msec SOAs but abnormally at an 800 msec SOA. In particular, they showed no effect of the cue presented 800 msec before the target. This result suggests that 4 months of binocular deprivation from birth interferes with the ability to maintain attention on a peripheral location after a long delay.

Before ascribing the deficit to attention, it is important to consider possible non-attentional reasons for the deficit at the 800 msec SOA. First, it is possible that the deficit at the 800 msec SOA could be caused by patients with longer deprivation making more responses not based on detection of the target than patients with shorter deprivation. However, it is unlikely that the deficit was caused by false alarms, because there were no between-group differences in the frequency of responses on catch trials at any SOA. Second, it is possible that the deficit at the 800 msec SOA could be caused by problems in understanding the task, or in seeing the cues and/or targets. This is an unlikely explanation because these patients performed normally at the 100 and 400 msec SOAs. The normal performance at the early SOAs indicates that these patients did not have a problem in shifting attention. The abnormal performance at the 800 msec SOA indicates that they had difficulty maintaining attention at a cued location.

The results can be understood in the framework developed by Nakayama and MacKeben (1989) which distinguishes between a sustained and a transient component of attention. This distinction was justified on the basis of a series of visual search tasks. Participants searched for the presence of an odd target (a black or white bar that was horizontal or vertical in orientation). In simple search cases, the odd target differed from distractors in orientation. In conjunctive search cases, the odd target differed from distractors in orientation and color. There were two types of cueing conditions: In the *sustained* cueing condition, the cue (a red square surrounding the location of the upcoming target) was visible all of the time. In the *transient* cueing condition, the cue appeared at a short variable delay (0 - 500 msec) before the presentation of the array containing the target. Sustained cueing did not enhance performance in the simple search condition. According to the authors, this is because the "target by itself can be singled out and marked by the pre-attentive system; the addition of a cue can add little to target visibility" (page 1636). Sustained visual cueing enhanced perception of the target in the conjunctive search condition. Transient visual cueing enhanced perception of the target in the conjunctive condition even further. Transient cues were most effective when they appeared 50 - 150 msec before the target. There was a striking fall-off in performance when the cue lead time was longer than 100 - 200 msec. This result shows that attention can be directed to a peripheral location, but not held there. The authors argued that the results together indicate two components of visual-spatial attention: a sustained and a transient component, and that different mechanisms may be involved in each. Additional experiments showed that the transient component of attention was relatively uninfluenced by the expectancy of a target, retinal eccentricity of a target, flickering of the cue, and changes in the paradigm to a vernier task. The authors argued further that different neural pathways possibly mediate the two types of attention: The sustained component is determined by visual attention at higher cortical levels (e.g., V4, inferotemporal, parietal, and/or frontal) as compared to the transient component of attention that is determined by lower levels (e.g., superior colliculus, V1).

This perspective can be applied to my paradigm by assuming that even though the cues remained on throughout the trial, attention was moved covertly as soon as the cue appeared and had to be sustained at the cued location until the target appeared. The results of Chapter 4 suggest that, in the group with longer deprivation, there was a deficit in the sustained component of attention, as performance was abnormal at the longest SOA. Further, there appears to be no deficit in the transient component of attention, as performance was normal at the two shorter SOAs. We would predict, based on this account, that the effects of longer periods of bilateral deprivation are determined by abnormalities at higher cortical levels (e.g., V4, inferotemporal, parietal, and/or frontal) rather than at lower levels.

Although the results indicate a deficit after binocular deprivation, they are not exactly what I had predicted. I originally predicted that deficits following binocular deprivation would be similar to those in adult patients following damage to the parietal cortex, because binocular deprivation in monkeys damages cells in the parietal cortex (Hyvarinen & Hyvarinen, 1979). Most of the data on the effect of lesions in humans are gathered from patients with unilateral parietal damage. Patients with unilateral parietal lesions show larger-than-normal validity effects when the target appears in the visual field contralateral to the lesioned hemisphere. The pattern of results at the 800 msec SOA in this chapter after longer binocular deprivation were the opposite: no validity effect at all. This indicates that the effects of more than 4 months of early visual deprivation on the brain and attention differ from the effects of unilateral damage to the parietal cortex in adults. It is important to note that adults with unilateral parietal damage were tested under different conditions (e.g., smaller cues, smaller targets, horizontal placement). To confirm that the deficits are different in children treated for cataract than in adults following unilateral parietal damage, patients with the two types of damage would have to be tested with the same procedures.

In some respects, the effects after longer binocular deprivation are like those reported in one case report of a patient with bilateral parietal lesions (Verfaellie, Rapscak, & Heilman, 1990). The results from this one case are important to discuss as damage from bilateral parietal lesions may be most similar to that of binocular deprivation. In a task with exogenous cues, and 1200, 1400, and 1600 msec SOAs, the patient with bilateral parietal lesions showed no validity effect in the lower visual field but a significant validity effect in the upper visual field at all SOAs tested. In one respect, this finding is comparable to that observed here for the group with longer periods of deprivation. These patients failed to show any validity effect at the longest (800 msec) SOA, but unlike the patient with bilateral parietal damage, this was true for both the upper and the lower visual field.

A better understanding of the results may come from considering the possibility that there are different consequences of early versus late brain injury. This hypothesis is well documented for studies of language development (e.g., Bates, Thal, & Janowsky, 1993; Thal, Marchman, Stiles, Aram, Trauner, Nass, & Bates, 1991). For example, children with early brain injury to Broca or Wernike's areas tend not to show the same patterns of language impairments (e.g., dissociation between comprehension and production deficits) as seen in adults with similar types of brain damage.

An examination of the literature on visual-spatial attention in children with infantile lesions may explain why children treated for binocular deprivation do not perform like adults with unilateral damage to the parietal cortex (Craft, Schatz, Glauser, Lee, & DeBaun, 1994a; Craft, White, Park, and Figel, 1994b; Johnson, Tucker, Stiles, & Trauner, in press). Craft et al. (1994b) used a spatial cueing task with children (mean age = 7 years) to examine whether the deficits from early infantile brain lesions are similar to those described by Posner (1988) in adults. They investigated visual-spatial attention in 36 normal children and in 33 children with perinatal injury (prior to 6 weeks of age) resulting in spastic diplegic cerebral palsy. Based on MRI, patients were judged to have bilateral lesions anterior to the central sulcus (n =6), posterior to the central sulcus (n = 10), both anterior and posterior (n = 8), or no observable lesion (n = 9).[9]

Craft et al. (1994b) found abnormalities in visual-spatial attention in the group with anterior lesions, but not in the group with posterior lesions. Thus, although children with infantile bilateral posterior lesions were generally slower than normal children, they had validity effects of normal size at both 100 msec and 800 msec SOAs, unlike adults with unilateral or bilateral posterior lesions. In contrast, children with bilateral infantile anterior lesions failed to show a validity effect in the right visual field at the 100 msec SOA and showed larger-than-normal validity effects in the left visual field at the 100 msec SOA and in both visual fields at the 800 msec SOA (similar to the pattern illustrated in Figure 10b). These findings suggest a role of anterior areas in disengaging attention. There are no data on adults with bilateral frontal lesions but adults with unilateral frontal lesions, restricted to the dorsolateral prefrontal cortex, perform normally in a spatial cueing task (Rafal, 1996). Overall, the results suggest that the neural mechanisms for visual-spatial attention may be different in infants and adults, with infants

[9] Patients with diffuse lesions had longer-than-normal reaction times in the left visual field. Patients without any observable lesions did not differ from normal children.

relying more on anterior areas of the brain and adults relying more on posterior areas of the brain.

Johnson et al. (in press) provide additional evidence that the anterior/frontal area may be a critical neural substrate for visual-spatial attention during infancy. Johnson et al. examined spatial cueing in infants (around 9 - 10 months of age) following unilateral posterior or anterior lesions. The methods were identical to Johnson and Tucker (1996) and the data from normal 7-month-old infants were used as a comparison group. Babies with unilateral posterior lesions performed normally. However, unlike normal babies and babies with unilateral posterior lesions, babies with left anterior lesions did not benefit from valid cueing at a shorter SOA (133 msec); however, they did show the normal pattern of inhibition of return (IOR) at the longer (700 msec) SOA.

Additional evidence for the role of the anterior/frontal brain region in visual attention early in life comes from recent ERP studies in infants (Csibra, Johnson, & Tucker, in preparation) and adults (Csibra, Johnson, & Tucker, 1997). Targets were shown in the left or right visual field and were presented either concurrently with the fixation stimulus (overlap trials), or presented after a temporal gap following the offset of the fixation stimulus (gap trials). Both 6-month-olds and adults were slower to make an eye movement to the target on overlap trials than on gap trials. Unlike adults, infants did not show pre-saccadic parietal positivity. Much like adults, infants did show more positive voltage over the posterior left frontal region in the gap condition than in the overlap condition. The results suggest that by 6 months of age, frontal structures are functional during some aspects of visual attention such as disengaging visual fixation while other structures, such as parietal cortex, do not affect behavior.

In summary, unlike patients with shorter deprivation, patients treated for bilateral congenital cataract, past 4 months of age, are impaired on a test of visual attention, specifically on the ability to sustain attention. The findings suggest that visual experience during early infancy is critical for the sustained component of attention to develop normally. Based on the literature, I originally predicted that the deficits would be similar to those found in adults with unilateral parietal lesions. However, studies of children with infantile brain lesions (e.g., Craft et al., 1994a, b; Johnson et al., in press) provide a possible explanation for the different results. Taken together, the results suggest different neural systems controlling visual-spatial attention in infants than in adults. Normal babies may rely more on the frontal cortex (especially left structures, Csibra et al., in preparation) for attentional abilities during infancy, and only later come to depend on the parietal cortex (like adults). Obviously, after infantile parietal lesions, the frontal cortex can continue to play this role. Early deprivation might affect the development of attention not, or not only, through effects on the visual responsiveness of the parietal cortex, but primarily through effects on anterior or frontal systems. Not only has the frontal cortex been related to the development of many aspects of attention during infancy (e.g., anticipatory looking, IOR, see Chapter 3), but it has a more protracted developmental period than parietal, occipital, and temporal cortical areas of the brain (Huttenlocher, 1994; Chugani, 1994) during which it could be affected by lack of input. Finally, although there are no studies in humans or in primates on the effects of visual deprivation on the frontal cortex and frontal functions, there is one study in rats showing that visual deprivation reduces neuro-chemical binding levels in the frontal cortex (Schleibs et al., 1982).

The results show that early visual deprivation interferes with the normal development of attention on an easy task, which did not reveal age differences in normal participants (see Chapter 3). In the next chapter, I examine whether there may be deficits even after shorter binocular deprivation that are revealed if the task is more difficult.

CHAPTER 5

COVERT ORIENTING AND THE ABILITY TO IGNORE DISTRACTORS

The results of Chapter 4 indicated that patients with more than four months of binocular deprivation have an abnormally small validity effect at a 800 msec SOA but not at a 100 or 400 msec SOA. One explanation for these impairments is that patients with more than four months of deprivation have trouble sustaining attention. Patients with shorter periods of binocular deprivation performed normally on the covert orienting task. These findings motivated me to implement a more difficult task that might reveal deficits following shorter (and monocular) deprivation. The second purpose of using a more difficult task was to clarify the nature of the deficit in the group with longer deprivation. Thus, in Chapter 5, I made the task more difficult by adding a distractor-filtering component to the covert orienting task by flanking the target with distractors. To better measure any deficits, I measured accuracy, as well as reaction time, by requiring target discrimination rather than detection.

In this new 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 in order to ignore the distractors. 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.

A variety of tasks encountered by both children and adults require the ability to ignore distracting information so that other information can be processed more efficiently. For example, when driving a car, an adult needs to attend to relevant information, such as the road ahead, despite the presence of other distracting stimuli, such as children playing in the car. Whether it is learning to read, to play the piano, or to drive a car, it is important for both children and adults to inhibit the processing of distracting information and focus attention on important information.

Evidence from a variety of paradigms including Stroop color-word naming, same/difference judgments, and speeded classification, suggests that the ability to ignore distracting information improves during childhood (e.g., Enns, 1990; Enns & Akhtar, 1989; Enns & Cameron, 1987; Lane & Pearson, 1982; Plude, Enns, & Brodeur, 1994; Ridderinkhof & van der Molen, 1995; Strutt, Anderson, and Well, 1975; Tipper, Bourque, Anderson, & Brehaut, 1989; Well, Lorch, & Anderson, 1980). Generally, children's responses are influenced more than those of adults by distracting information. For example, Strutt et al. (1975) asked children aged 6, 9, and 12 years, and adults to sort cards with pictures of target stimuli on them. The presence of distractors slowed the sorting of six-year-olds significantly more than that of older children. This result suggests that older children are better able to ignore distracting information than younger children.

The typical paradigm used to examine the ability to ignore distractors involves speeded classification. In a speeded classification task, the participant is instructed to make one response to one target stimulus and a different response to a second target. The participant's reaction times to a target are compared when the target is surrounded by distractors that are mapped to the same response as the target, and when it is surrounded by distractors that are mapped to a different response than the target. In a speeded classification task, Eriksen and Eriksen (1974) found that adults were slower to respond to targets surrounded by distractors of the opposite category than the same category. Moreover, response-incompatible distractors had the largest effect on performance at the closest spacing between targets and distractors. These data show that adults' reaction times can be influenced by distracting information.

Two studies have used tasks similar to that used by Eriksen and Eriksen (1974) to investigate age differences in the effects of distracting stimuli on reaction times of adults and children (4, 5, and 7 years old, Enns & Akhtar, 1989; 5 - 12 years old, Ridderinkhof & van der Molen, 1995). Both studies showed that, like adults, children are slowed by incompatible distractors. However, the magnitude of the slowing was far greater for children 4-to-7 years old (Enns & Akhtar, 1989) and 5-to-9 years old (Ridderinkhof & van der Molen, 1995) than for adults. Older children, between 10 and 12 years of age, were slowed no more than adults by incompatible distractors (Ridderinkhof & van der Molen, 1995). These findings indicate that younger children are not as good as older children and adults at ignoring distracting information.

Ridderinkhof and van der Molen (1995) also used the speeded classification task to examine whether the processes involved in ignoring incompatible information change during development. EMG and the lateralized readiness potential (LRP) of the EEG were used as indices of response activation, and P300 latency was used as an index of the duration of perceptual analysis. The physiological results showed that there was a delay in EMG and LRP onset on incompatible trials compared to compatible trials, and that the size of the delay decreased with age between 5 years and adulthood. The latency of the P300 was longer on incompatible trials than on compatible trials (see also Coles, Gratton, Bashore, Eriksen, & Donchin, 1985). This effect on P300 was the same for children and adults. Together, the data suggest that in adults and children distractors affect both perceptual processing and response execution but in children the effect on response execution is larger than in adults. Thus, the source of the developmental decrease in the effect of incompatible distractors is from a decrease in the effect of distractors on response execution and not a change in their effect on perceptual processing.

To investigate age differences in the ability to ignore distracting information under more complex circumstances, distractors have been added to the spatial cueing task. Age differences in the effects of the combination of cueing and of distracting information have been examined in studies with exogenous cues (Akhtar & Enns, 1989) and endogenous cues (Brodeur, 1995). Akhtar and Enns (1989) used a two-alternative choice reaction time task with endogenous cues. Five-, 7-, and 9-year-old children and adults were asked to make one of two responses depending upon whether a square or a plus sign appeared on the computer screen, without moving their eyes. The cue could be valid, invalid, or neutral. On a given trial, a target shape (+) was presented either alone or surrounded by distractors that were compatible with the target (+++) or incompatible with the target. On valid trials, distractors did not have a deleterious effect on reaction time at any age. However, on invalid trials, 5-, 7-, and 9-year-old children were slowed considerably more than adults by incompatible distractors. One possible explanation is there was no effect of distractors on valid trials at any age because the task was probably easy, with attention already focused on the location of the target. However, there may have been large age differences in the effects of distractors on invalid trials because the task is harder when participants have to ignore distractors surrounding stimuli presented in an unattended location.

The only study of age differences in covert shifts of attention with endogenous cues and distracting information (Brodeur, 1995) is hard to interpret because the results were inconsistent across the age groups studied (ages 6, 9, and adult) and many details are missing from the brief unpublished report. Clearly, there is more to learn about the combined effects of cueing and distractors on response time. In this chapter, I look at the joint effects of endogenous cues and distractors on normal development and possible disruptions following early visual deprivation.

The developmental changes that I have just summarized may depend not just on the parietal cortex but also on the frontal cortex. Posner and Petersen (1990) distinguish between two attention systems: a posterior attention system involving the parietal cortex and an anterior attention system involving the frontal cortex.

At least in adults, the frontal cortex is important for executive aspects of attention such as planning, working memory, inhibition, and maintenance of anticipatory set/preparedness to act and these tasks often make use of visual information (e.g., Denckla, 1996; Duncan, 1986). For example, adults with damage to the frontal cortex are impaired on tasks such as the Wisconsin Card Sorting Test, Word Fluency, Go-No-Go task, Tower of London, that require these abilities (reviewed in Denckla, 1996; Duncan, 1986).

These findings suggest that the anterior-frontal system plays a key role in maintaining attention, inhibiting a motor sequence and/or in inhibiting attention to distractors in order to focus on important stimuli. In contrast, the posterior-parietal system is important for shifting attention, at least in adults (see Chapter 2).

The evidence reviewed earlier in this chapter suggests that there is slow development on tasks that depend upon frontal structures. In particular, the ability to focus attention on important information while ignoring distractions becomes adult-like after age 9 (Akhtar & Enns, 1989; Enns, 1990; Enns & Akhtar, 1989; Enns & Cameron, 1987; Lane & Pearson, 1982; Plude et al., 1994; Ridderinkhof & van der Molen, 1995; Strutt et al., 1975; Well et al., 1980). There is additional evidence from other tasks for the late development of abilities that are related to the frontal cortex. Levin and his colleagues (Levin, Culhane, Hartmann, Evankovich, Mattson, Harward, Ringholz, Ewing-Cobbs, & Fletcher, 1991) used a variety of tasks related in adults to the frontal cortex (for example, Tower of Hanoi/London, Wisconsin Card Sort) and found that performance did not reach adult levels until adolescence, that is, much later than for tasks tapping the parietal attentional system (see Chapter 3). Together, the literature suggests that performance reaches adult-like levels at a slower rate on frontally-mediated tasks than on tasks related in adults to the parietal system.

The behavioral data fit with what is known about relative rates of neural development. Several lines of evidence show that the anterior attention system has a longer period of development than the posterior system. Anatomical data indicate delayed myelination and synaptogenesis in the frontal cortex relative to other brain regions (e.g., Conel, 1939-67; Huttenlocher, 1994; Lecours, 1975). PET findings of glucose metabolism indicate that the frontal cortex has a longer developmental period than parietal, occipital, and temporal areas of the brain (Chugani, 1994).

One interpretation of the data in Chapter 4 is that long binocular deprivation damages the frontal cortex, which plays a role in regulating attention during infancy. If this is true, then we would expect abnormalities on other functions depending upon the frontal cortex. Among those deficits may be impairments in maintaining attention, inhibiting a motor sequence and/or in inhibiting attention to distractors in order to focus on important stimuli. Because of the later development of abilities which in adults depend upon the anterior system than those that depend on the parietal system, it is possible that functions mediated by the two systems are affected differently by binocular deprivation.

In the studies below, 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. On half of the trials, the target was flanked by distractors that were compatible with the response to the target while on the other half of the trials the target was flanked by distractors that were incompatible with the response to the target. I decided to use only one SOA in order to have more data from each condition. Targets were always presented 400 msec after the onset of a central arrow. I selected a 400 msec SOA for two reasons. First, my own data presented in Chapter 2 showed that the size of the validity effect is largest at a 400 msec SOA. Second, Müller and Findlay (1988) showed that the size of the validity effect is largest at a 400 msec SOA in a discrimination task with endogenous cues. There is a study with children using a discrimination task but exogenous cues that found very large validity effects at a 200 msec SOA, the only SOA tested (Akhtar & Enns, 1989). However, a 200 msec SOA may be too short of a time interval for children treated for cataract to utilize information from endogenous cues in a complex task.

In this chapter, I report studies with normal 10-year-olds and adults, and with patients. The normal development of covert orienting in a choice-discrimination task with targets surrounded by distractors may differ from the findings in Chapter 3. Performance may be different on the present task simply because it is a more difficult task, or because it depends on different brain systems. The data from patients will help to determine whether there are deficits following deprivation from birth lasting both less than and more than four months. The data may also clarify the nature of the deficits.

Method

Participants

The final sample of normal participants consisted of 24 10-year-old children (mean age = 10.0 years, range = 9.8 - 10.2 years) and 24 adults (mean age = 20.4 years, range = 18.6 - 26.2 years). The inclusion criteria for normal participants were described in Chapter 2. The information on the normal participants is reported in the bottom half of Table 3. An additional five participants were excluded from the final sample because they failed the vision screening examination (two 10-year-olds; one adult), made errors on more than 50% of the trials (one 10-year-old), or fell asleep during the task (one adult).

The sample of patients consisted of 30 eyes of 15 children treated for bilateral congenital cataract from the same sample reported in Chapter 4. Detailed characteristics of the patients are provided in Table 12.[9] The patients had a mean duration of deprivation of 4.5 months (range 1.8 - 7.6 months). For comparison, 30 eyes of fifteen age-matched children (+/- 3 months) were tested; details about the comparison group are provided in Table 3. One additional child was excluded because she failed the vision screening exam.

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[10] As in Chapter 4, since there were not enough data to be able to examine potentially important variables such as the amount of patching of the good eye and the duration of deprivation, the data from patients treated for monocular deprivation are included in Appendix C.

Table 12.
Clinical Details at the Time of the Distractor Test for the 15 Children Treated for Bilateral Congenital Cataract

Name (Age in years)	Refraction (diopters)	Age of Diagnosis (months)	Months (days) of Deprivation	Snellen Acuity[1]
L. M.	OD +12.50	0.0	3.1 (94)	6/18
(8.9)	OS +16.50	0.0	3.1 (94)	6/15
A. L.	OD +16.50	2.0	5.4 (165)	6/12
(10.4)	OS +15.50	2.0	5.4 (165)	6/18
A. H.	OD +16.25	1.2	2.1 (64)	6/60
(10.9)	OS +15.75	1.2	2.1 (64)	6/15
J. G.	OD +17.00	0.0	2.7 (83)	6/18
(11.1)	OS +23.00	0.0	2.7 (83)	6/12
B. B.	OD +13.75	3.4	5.4 (165)	6/21
(11.1)	OS +14.00	3.4	5.4 (165)	6/21
S. S.	OD +12.75	2.0	7.5 (228)	6/18
(13.1)	OS +12.00	2.0	7.5 (228)	6/60
A. D.	OD +16.50	0.0	3.2 (97)	6/24
(13.1)	OS +15.25	0.0	3.2 (97)	6/30
J. J.	OD +14.50	0.6	1.8 (53)	6/12
(13.4)	OS +18.50	0.6	1.8 (53)	6/9
C. B.	OD +14.5	1.1	3.0 (91)	6/9
(13.8)	OS +18.5	1.1	3.0 (91)	6/60
G. S.	OD +28.50	2.6	6.1 (187)	6/60
(15.6)	OS +24.50	2.6	6.1 (187)	6/60
I. W.	OD +11.50	0.0	5.0 (151)	6/30
(15.8)	OS +13.50	0.0	8.6 (264)	6/7.5
A. R.	OD +16.25	0.2	3.5 (103)	6/30
(15.8)	OS +12.50	0.2	3.5 (103)	6/30

[1] Snellen Acuity is based on the measurement closest to my testing date.

Table 12 (continued).
Clinical Details at the Time of the Distractor Test for the 15 Children Treated for Bilateral Congenital Cataract

Name (Age in years)	Refraction (diopters)	Age of Diagnosis (months)	Months (days) of Deprivation	Snellen Acuity
C. P.	OD +10.25	1.9	6.1 (187)	6/7.5
(16.0)	OS +11.75	1.9	6.1 (187)	6/15
A. C.	OD +11.75	2.9	6.4 (196)	6/12
(16.9)	OS +12.25	2.9	5.3 (161)	6/15
M. D.	OD +11.25	0.0	4.1 (129)	6/9
(17.2)	OS +14.2	0.0	4.1 (129)	6/18

Apparatus & Stimuli

The apparatus was the same as described in Chapter 2.

Cues

The cues were the same as described in Chapter 2.

Targets

A plus and a circle were chosen because children treated for cataract have little difficulty discriminating these forms (Maurer et al., 1989). These shapes were presented with their centers 9.0 degrees (57.0 mm) directly above or below the center of the central stimulus. The nearest edge of the shape was 5 degrees (31.5 mm) from the center of the central stimulus. At this distance, the shapes were well within the visual field for children treated for cataract. Bowering (1992) reported that the superior and inferior visual fields of children in a similar sample treated for cataract extend well beyond 20 degrees peripherally for stimuli much smaller than 1 degree.

The choice of the shapes' size was based on linear letter acuity in the center of the visual field combined with information on peripheral contrast sensitivity. Linear letter acuity in the center of the field for a similar sample was 6/192 or better for 95.3% of the eyes treated for bilateral congenital cataract and 76.5% of the eyes treated for unilateral congenital cataract (Maurer et al., 1989). The letters on this line have a stroke width of 32 minutes of arc or .53 degrees.

Unfortunately, there are no data on discrimination acuity in the *periphery* in children treated for cataract. Data in normal adults indicate that letters need to be three times larger in order to be discriminated 5 degrees from center than in the center of the visual field (Fahle & Schmid, 1988). Peripheral contrast sensitivity for gratings for a similar sample of children treated for bilateral cataract indicated that losses were a constant proportion of normal sensitivity at all locations within the central 60 degrees (Tytla et al., 1992).[10] Based on this evidence, I determined that shapes need to be made of lines 1.6 degrees (.53 degrees multiplied by three) to be discriminated at 5 degrees in the periphery and have an overall size of 8 degrees high and 8 degrees wide. To determine the overall size of the target shape, I took the minimum stroke width, 1.6 degrees, and multiplied by 5, because the ratio of the overall size of a letter on the Snellen eye chart to the stroke width of a letter is 5-to-1.

The distractor shapes were identical to the targets and were aligned horizontally beginning 1.6 degrees (10.0 mm) from the nearest edge of the target. I used the same logic as described above in making these choices. The nearest edges of the distractor stimuli were 7.4 degrees (47.0 mm) from the center of the central stimulus.

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[11] Losses in children treated for unilateral cataract were greatest in the central field with smaller losses at all locations tested in the periphery.

Procedure

The procedure was developed from pilot studies with normal participants that are described in Appendix D. The method was the same as described in Chapter 2 with the following exceptions: (1) 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. (2) On half of the trials, the target was flanked by distractors that were compatible with the response to the target. On the other half of the trials, the target was flanked by distractors that were incompatible with the response to the target. (3) Participants were tested only at a 400 msec SOA to maximize the amount of data collected in each condition of interest. (4) Participants were provided with auditory feedback ("great" or "whoops") regarding the accuracy of their responses. The feedback was auditory in order not to interfere with the processing of visual information.

Participants were tested on a total of 200 trials (5 blocks of 40 trials) with one of two targets (plus sign or cross) presented randomly on each trial under a combination of one of three cue conditions (valid, invalid, neutral) and one of two distractor conditions (compatible distractors, 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. On the remaining half of the trials (n= 100 trials total), the target was surrounded by distractors incompatible with the response to the target. Figure 14 shows an example of an invalid trial with incompatible distractors. On half of the trials the target was a circle, while 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.

Participants were asked to use the index finger of each hand to press one of two buttons on a keyboard to respond to the target. One-half of the participants were instructed to use the "v" button to respond to the circle and the "n" button to respond to the plus. The reverse was true for the other half of the participants. Reaction time was measured from the onset of the target to the participant's keypress. The participant's response was coded as correct or incorrect. At the end of each trial participants were shown a blank screen during which they received auditory feedback ("great" or "whoops") regarding the accuracy of their response and the experimenter coded whether the participant had maintained fixation or made an eye movement during the trial.

Instructions & Practice

Participants were first taught which button to press for a circle and which for a plus sign. Participants were then introduced to the task with a demonstration of the types of trials that would occur. The instructions

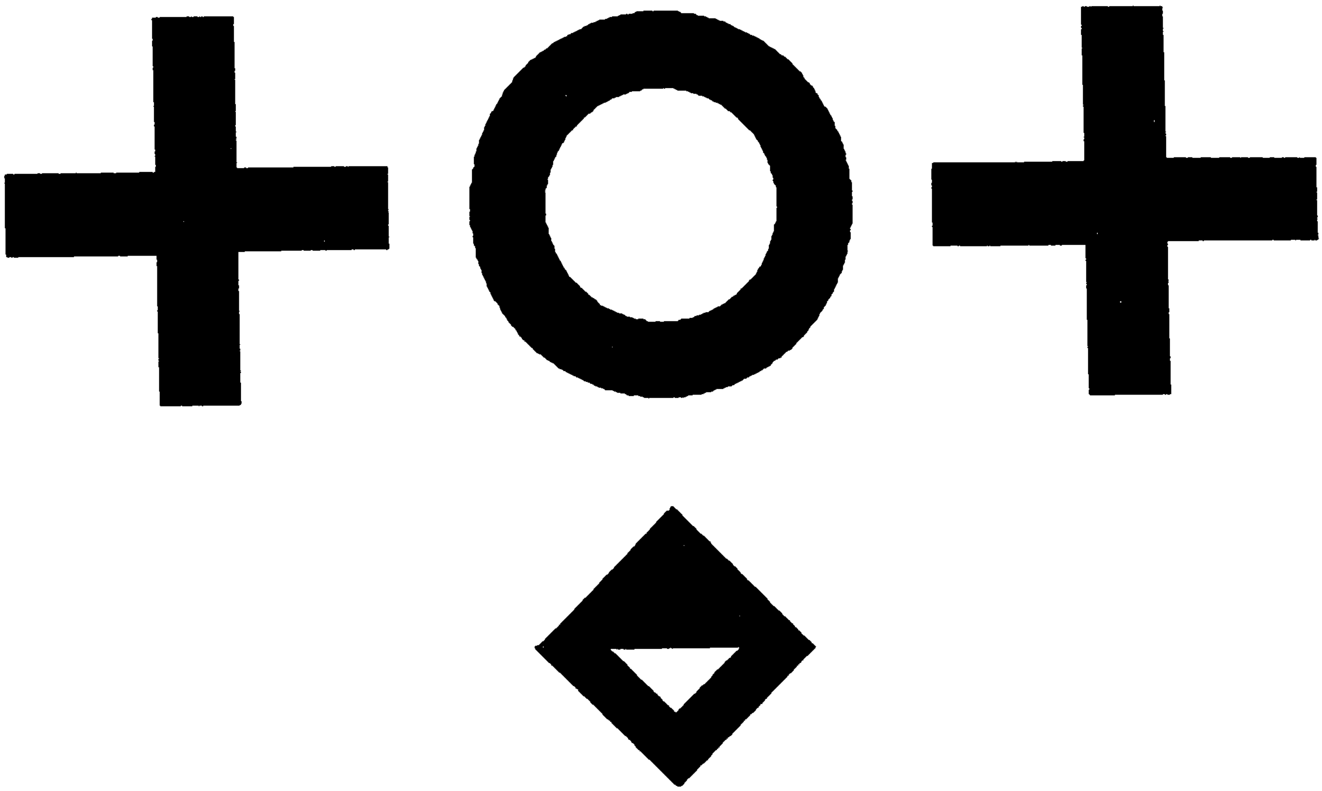

Figure 14. Filtering task: Example of an Invalid trial with Incompatible distractors.

provided to the participants are reproduced in Appendix B. Participants were given a practice session with all types of trials that would be used in the experiment. This procedure familiarized participants with the task, allowed them to practice shifting attention without eye movements, and to practice responding as quickly and accurately as possible. This procedure also allowed the experimenter the opportunity to give participants verbal feedback regarding their fixation and accuracy of performance. Participants were given practice until they completed 10 consecutive trials correctly without eye movements.

Post-tests

After the test, participants were given two post-tests. The first post-test was to determine whether participants could discriminate the target when distracting shapes were present. While fixating centrally, participants were asked to say the name of a target shape (circle or plus) on 10 neutral trials (5 with compatible distractors and 5 with incompatible distractors). The number of correct responses and the number of trials on which subjects made an eye movement were recorded by the experimenter. In addition, to check that participants understood the meaning of the cues, as in Chapter 3, they were presented with 10 trials on which they were to say whether the cue was a "helper" (the arrow helped them find the target), "fooler" (the arrow pointed the wrong way from the target), or "no clue" (the arrow pointed in both directions). Again, the number of correct responses (out of 10) and eye movements were recorded by the experimenter.

Data Analyses

I conducted the main analyses on mean reaction times. For analyses involving interactions with cue, I examined the difference in reaction time on valid versus invalid trials. The reasoning for looking at the validity effect was the same as presented in Chapter 4: patients were slower than normal participants overall. Because the measure of covert orienting is in the effects of cueing, not absolute reaction time, I analyzed the differences in reaction time on valid and invalid trials (validity effect), rather than the actual reaction times. The residual will reflect what is different between the two types of trials, which can only have to do with covert orienting of attention. I subsequently tried to determine whether any differences in validity effect came from differences in benefit (valid - neutral RT) and/or cost (invalid - neutral RT). I used these as supplementary rather than primary analyses because I could not be certain that the neutral trials provided an equivalent baseline for patients and normal participants (e.g., Jonides & Mack, 1984).

For analyses involving interactions with type of distractor, I examined the difference in reaction time on trials with compatible and incompatible distractors. I did so because patients' slowness on both types of trial could arise from a number of factors that have nothing to do with attention, such as the speed of seeing the stimuli or of making a motor response. It is reasonable to assume that those factors would have an equal effect on compatible and incompatible trials. The result of the subtraction provides a measure of the effects on reaction time from an inability to ignore the information in the distractors.

Results

Normal Participants

The percentage of trials on which adults and 10-year-olds made eye movements or errors is shown in Table 13. Adults made eye movements on fewer trials than 10-year-old children (two-tailed t-test, $p < .05$).

Accuracy

Trials on which participants made eye movements were eliminated before analyzing accuracy. The mean proportion of correct responses (+ 1 standard error) for each group, and for each condition, is shown in the top part of Table 14. Clearly, there was a high level of accuracy at both ages in all conditions.

The proportion of correct responses for 10-year-olds and adults was subjected to an ANOVA with one between-subject factor, Age (adults, 10-year-olds), and three within-subject factors: Cue type (valid, invalid, neutral), Distractor type (compatible, incompatible) and Visual field (lower, upper). Table 15 shows the results for the ANOVA.

Table 13.
Mean Percentage of Eye Movements and Correct Responses (1 SE) in Normal Participants and Patients Treated for Congenital Cataract on a Covert Orienting Task with Distractors.

	Eye Movements	
Normal 10-year-olds	8.0%	(1.4%)
Normal Adults	2.6%	(0.9%)
Bilateral Congenital	6.3%	(0.9%)
Unilateral Congenital	4.9%	(1.6%)
Age-Matched Controls	5.2%	(1.5%)

	Correct Responses	
Normal 10-year-olds	96.8%	(0.2%)
Normal Adults	97.5%	(0.3%)
Bilateral Congenital	97.3%	(0.6%)
Unilateral Congenital	96.2%	(0.4%)
Age-Matched Controls	97.1%	(0.3%)

Table 14.
Mean Proportion Correct (1 SE) for Each Group of Participants as a Function of Distractor and Cue Type.

	COMPATIBLE			INCOMPATIBLE		
	INVALID	NEUTRAL	VALID	INVALID	NEUTRAL	VALID
10-year-olds	.97 (.01)	.96 (.01)	.97 (.01)	.96 (.01)	.97 (.01)	.97 (.01)
Adults	.97 (.01)	.97 (.01)	.97 (.01)	.98 (.01)	.98 (.01)	.98 (.01)
Unilaterals	.96 (.02)	.94 (.02)	.96 (.01)	.96 (.01)	.97 (.02)	.98 (.01)
Bilaterals	.97 (.01)	.97 (.01)	.97 (.01)	.97 (.02)	.96 (.01)	.96 (.01)
Age-Matched Controls	.98 (.01)	.97 (.01)	.96 (.01)	.97 (.01)	.97 (.01)	.95 (.01)

Table 15.
Differences between 10-year-old and Adult Participants in their Ability to Respond Accurately to the Target.

Results of the 4-way ANOVA: Age, Distractor type, Cue type, Visual field

Effect	*F*	*df*	*p*
Age	2.76	1,46	.10
Distractor type	3.13	1,46	.09
Cue type	.42	2,92	.65
Visual Field	.68	1,46	.41
Age X Distractor type	.21	1,46	.65
Age X Cue type	.27	2,92	.76
Distractor type X Cue type	1.27	2,92	.28
Age X Visual Field	**6.67**	**1,46**	**<.05**
Distractor type X Visual Field	1.32	1,46	.26
Cue type X Visual Field	1.19	2,92	.31
Age X Distractor type X Cue type	2.36	2,92	.10
Age X Distractor type X Visual Field	.03	1,46	.86
Age X Cue type X Visual Field	.68	2,92	.51
Distractor type X Cue type X Field	1.71	2,92	.18
Age X Distractor type X Cue type X Field	.13	2,92	.88

Effect of Age and Its Interactions. The ANOVA showed that 10-year-olds were as accurate as adults (Main effect of age, $p > .05$). Adults were more accurate when the target was in the lower visual field than when it was in the upper visual field (Age by Field interaction and Analysis of simple effects, $ps < .05$). Ten-year-olds' accuracy was the same for targets in the upper and lower visual fields (Analysis of simple effects, $p > .05$). None of the other interactions with age was significant (all $ps > .05$). In particular, children were not less accurate than adults on invalid trials with incompatible distractors (Age X Cue X Distractor, $p > .05$).

Reaction Time

Reaction times on trials during which participants made eye movements or responded incorrectly were excluded. Because there were so few errors, I did not adjust the reaction times on correct trials for the number of errors (see Akhtar & Enns, 1989).

As in Chapters 3 and 4, mean reaction times were calculated after outliers were eliminated with a moving criterion adjusted relative to the number of trials per condition (Van Selst & Jolicoeur, 1994). The percentage of scores eliminated with the outlier elimination procedure was 2.99% for 10-year-olds and 2.43% for adult participants. The ANOVA had one between-subject factor, Age (adults, 10-year-olds), and three within-subject factors: Cue type (valid, invalid, neutral), Distractor type (compatible, incompatible) and Visual field (lower, upper). Table 16 shows the results of the ANOVA.

Table 16.
Differences between 10-year-old and Adult Participants in the Ability to Shift Attention Covertly and Ignore Distractors as Measured by Reaction Time.

Results of the 4-way ANOVA: Age, Distractor type, Cue type, Visual field

Effect	*F*	*df*	*p*
Age	**121.48**	**1,46**	**< .0001**
Distractor type	**49.65**	**1,46**	**< .0001**
Cue type	**21.17**	**2,92**	**< .0001**
Visual Field	**166.07**	**1,46**	**< .0001**
Age X Distractor type	**5.75**	**1,46**	**.02**
Age X Cue type	2.46	2,92	.09
Distractor type X Cue type	2.09	2,92	.13
Age X Visual Field	1.92	1,46	.17
Distractor type X Visual Field	1.89	1,46	.17
Cue type X Visual Field	1.77	2,92	.18
Age X Distractor type X Cue type	.65	2,92	.52
Age X Distractor type X Visual Field	0.00	1,46	.99
Age X Cue type X Visual Field	1.50	2,92	.23
Distractor type X Cue type X Field	1.14	2,92	.32
Age X Distractor type X Cue type X Field	2.79	2,92	.07

Effect of Age. There was a significant main effect of Age ($p < .0001$). Adults responded faster than 10-year-old children. Age interacted with Distractor type ($p = .02$). Figure 15 illustrates the difference in reaction time between trials with incompatible distractors and trials with compatible distractors. Participants at both ages were slowed significantly by incompatible distractors (Analyses of Simple Effects, *ps* < .05). Because adults were faster than 10-year-olds, I calculated the difference in reaction time between compatible and incompatible distractors for participants in each age group to eliminate the reaction time difference, and conducted an additional ANOVA. The results showed that 10-year-olds were slowed significantly more (50.3 msec) than adults (24.8 msec) by incompatible distractors (Main effect of Age, $p < .01$).

Compatible and incompatible distractors did not differentially influence reaction time on valid, invalid, or neutral trials, at either age (non-significant interaction of Distractor type by Cue type and of Age by Distractor type by Cue type). There were no age differences in how cue informativeness altered reaction time (non-significant interaction of Age by Cue type). There was a significant main effect of Cue. Participants at both ages were significantly faster on valid trials than on neutral trials (Main effect of cue type, Tukey post-tests, *ps* < .01), but not significantly slower on invalid trials than on neutral trials ($p > .05$).

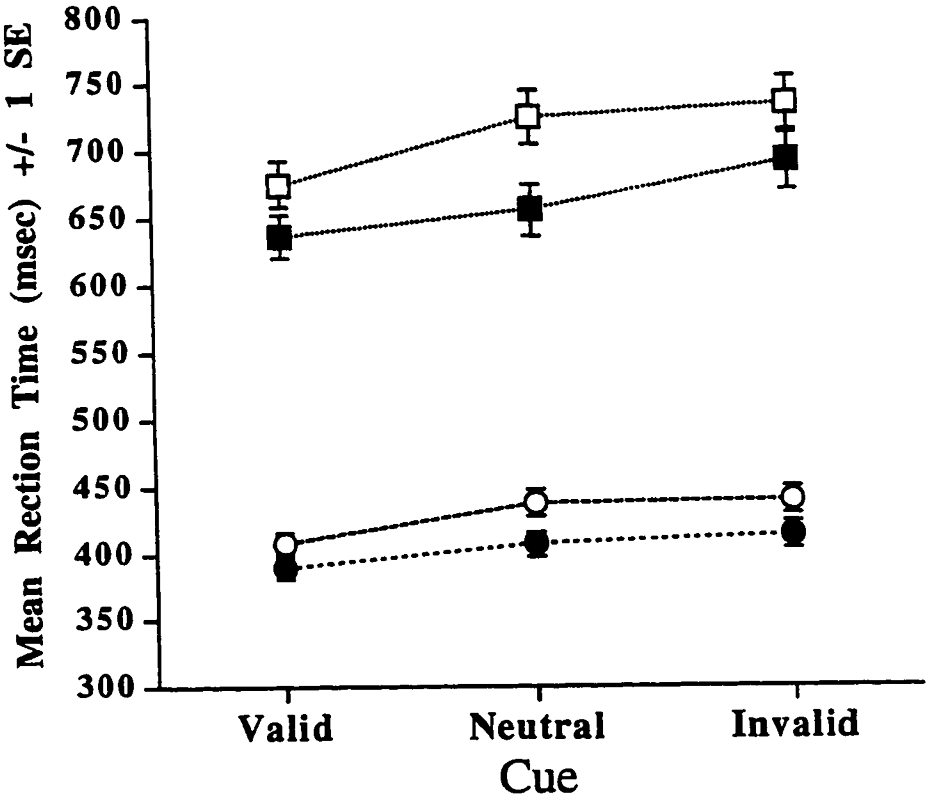

Figure 15. Mean reaction time for 10-year-olds (squares) and adults (circles) on trials with Incompatible distractors (open symbols) and Compatible distractors (filled symbols) as a function of cue type. Results for different cue types are connected to facilitate comparison.

Effect of Distractor. Figure 15 illustrates that participants in both groups were faster on trials with compatible distractors than on trials with incompatible distractors (Main effect of Distractor type).

Effect of Visual field. Participants were faster to respond to targets in the upper visual field (519.4 msec) than the lower visual field (582.2 msec) (Main effect of visual field). Visual field did not interact with any other factor.

Patients Treated for Bilateral Congenital Cataract and Age-Matched Controls

Because of the differences between 10-year-olds and adults, patients were compared to an age-matched control group. Patients treated for bilateral congenital cataract performed well on the post-tests: They demonstrated that they understood the meaning of the cues (e.g., "helper," "fooler," or "no clue") with an average score of 9.9 correct out of 10 trials (range 9-10), during which eye movements occurred on an average of only 0.5 out of 10 trials (range = 0 - 2). Patients also demonstrated an ability to discriminate the target when it was flanked by compatible and incompatible distractors. The average score was 9.9 correct out of 10 trials (range = 9 - 10), during which eye movements occurred on an average of 0.4 out of 10 trials (range = 0 - 3). Data on the frequency of eye movements and correct responses are shown in Table 13. Patients treated for bilateral congenital cataract were able to perform this relatively complex task as well as normal 10-year-olds and age-matched controls.

Mean reaction times were calculated after outliers were eliminated with a moving criterion adjusted relative to the number of trials per condition (Van Selst & Jolicoeur, 1994). The percentage of 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. I included data from both eyes in the analyses to increase sample size, after determining that the difference in reaction time between trials with incompatible distractors and compatible distractors from the two eyes of each patient were not correlated significantly for any cueing condition (i.e., valid, neutral, invalid) (all *p*s > .10).

As in Chapter 4, the patients were divided into those with less than, or more than, four months of deprivation. There were 14 eyes (mean age = 12.4 years; range 8.9 - 15.8 years) in the group with less than four months of deprivation (mean duration = 2.7 months; range 1.8 - 3.4 months) and 16 eyes (mean age = 14.5 years; range 10.4 - 17.3 years) in the group with more than four months of deprivation (mean duration = 5.9 months; range 4.2 - 7.6 months).

Accuracy

The proportion of correct responses for patients and age-matched controls was subjected to an ANOVA with one between-subject factor, Duration of deprivation (longer, shorter), and four within-subject factors: Group (patient, age-matched control), Cue type (valid, invalid, neutral), Distractor type (compatible, incompatible), and Visual Field (lower, upper). Table 17 shows the results of the ANOVA. 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). None of the other main effects nor any of the interactions were significant. The results indicate that patients with longer and with shorter deprivation did not differ from age-matched controls in their accuracy for any condition. The mean proportion of correct responses (+ 1 standard error) for each group, and for each condition, is shown in Table 14. Patients' accuracy exceeded .95 in all conditions.

Reaction Time

Table 18 shows the results from an ANOVA on mean reaction time with one between-subject factor, Duration of deprivation (longer, shorter), and four within- subject factors: Group (patient, age-matched control), Distractor type (compatible, incompatible), Cue type (invalid, neutral, valid), and Visual field (lower, upper).

Effects of Group. Patients were significantly slower than age-matched controls (Main effect of Group).

There was a significant interaction between Group, Distractor, and Cue. To examine this 3-way interaction more closely, I conducted an ANOVA on the difference in reaction time between incompatible and compatible trials (Incompatible - Compatible RT) with two within-subject factors: Group

Table 17.
Differences between Patients and Age-matched Control Subjects in their Ability to Respond Accurately to the Target.

Results of the 5-way ANOVA: Duration of deprivation, Group, Distractor type, Cue type, Visual Field

Effect	*F*	*df*	*p*
Duration of deprivation	2.07	1,28	.16
Group	0.02	1,28	.89
Distractor type	**4.31**	**1,28**	**.047**
Cue type	0.69	2,56	.50
Visual Field	0.23	1,28	.63
Duration X Group	0.00	1,28	.95
Duration X Distractor type	0.03	1,28	.87
Duration X Cue type	0.32	2,56	.73
Duration X Visual Field	0.98	1,28	.33
Group X Distractor type	0.00	1,28	.96
Group X Cue type	0.69	2,56	.51
Group X Visual Field	2.46	1,28	.13
Cue X Visual Field	0.24	2,56	.78
Distractor type X Cue type	0.10	2,56	.91
Distractor type X Visual Field	1.03	1,28	.32
Duration X Group X Distractor	0.02	1,28	.90
Duration X Group X Cue	0.31	2,56	.73
Duration X Group X Visual Field	0.27	1,28	.61
Duration X Distractor X Cue	0.57	2,56	.57
Duration X Distractor X Visual Field	0.04	1,28	.84
Duration X Cue type X Visual Field	0.56	2,56	.57
Distractor X Cue type X Visual Field	1.48	2,56	.24
Group X Distractor X Cue	1.49	2,56	.23
Group X Cue X Visual Field	1.38	2,56	.26
Group X Distractor X Visual Field	0.73	1,28	.40
Duration X Group X Distractor X Cue	0.21	2,56	.81
Duration X Group X Cue X Field	0.65	2,56	.52
Duration X Distractor X Cue X Field	0.62	2,56	.54
Group X Distractor X Cue X Field	1.21	2,56	.30
Duration X Group X Distractor X Field	0.61	1,28	.44
Duration X Group X Dist X Cue X Field	0.03	2,56	.97

Table 18.
Differences between Patients and Age-matched Control Subjects in Responses to the Target as Measured by Reaction Time.
Results of the 5-way ANOVA: Duration of Deprivation, Group, Distractor type, Cue type, Visual Field

Effect	F	df	p
Duration of deprivation	**6.25**	**1,28**	**.02**
Group	**10.58**	**1,28**	**< .01**
Distractor type	**35.38**	**1,28**	**< .0001**
Cue type	**24.69**	**2,56**	**< .0001**
Visual Field	**248.37**	**1,28**	**< .0001**
Duration X Group	**6.25**	**1,28**	**.02**
Duration X Distractor type	1.83	1,28	.18
Group X Distractor type	2.28	1,28	.14
Duration X Cue type	1.61	2,56	.21
Group X Cue type	.63	2,56	.53
Distractor type X Cue type	.46	2,56	.63
Duration X Visual Field	1.93	1,28	.17
Group X Visual Field	.10	1,28	.75
Distractor type X Visual Field	.13	1,28	.72
Cue type X Visual Field	.44	2,56	.64
Duration X Group X Distractor	3.24	1,28	.08
Duration X Group X Cue type	1.13	2,56	.33
Duration X Group X Field	.01	1,28	.91
Duration X Distractor X Cue type	.28	2,56	.76
Group X Distractor X Cue type	**3.86**	**2,56**	**.03**
Duration X Distractor type X Field	3.18	1,28	.09
Group X Distractor type X Field	.58	1,28	.45
Duration X Cue X Visual Field	1.52	2,56	.22
Group X Cue X Visual Field	**3.29**	**2,56**	**< .05**
Distractor type X Cue type X Field	.22	2,56	.80
Duration X Group X Distractor X Cue	.08	2,56	.92
Duration X Distractor X Cue X Field	.07	2,56	.93
Duration X Group X Distractor X Field	.07	1,28	.79
Duration X Group X Cue type X Field	1.51	2,56	.23
Group X Distractor X Cue X Field	.25	2,56	.78
Duration X Group X Dist X Cue X Field	.49	2,56	.61

(patient, age-matched normals), and Cue (valid, neutral, invalid). The ANOVA results are shown in Table 19. Figure 16 illustrates that on invalid trials, patients were slowed more than age-matched controls by incompatible distractors (Group by Cue interaction, Analyses of Simple Effects, *ps* < .05). The effects of distractors did not differ significantly for patients and age-matched controls on valid or neutral trials (*ps* > .10).

There was also a significant Group by Cue by Field interaction. To examine the source of the interaction, I conducted an ANOVA on the difference in reaction times between invalid and valid trials. The results from this ANOVA are shown in Table 20. Figure 17 illustrates that patients showed a normal validity effect in the lower visual field. In the upper visual field they showed a larger-than-normal validity effect (Group by Field interaction, Analyses of Simple Effects, *ps* < .05). The overall pattern illustrated in Figure 18 suggests a deficit in shifting attention to the upper visual field. In patients, correct cueing to the upper visual field does not decrease reaction time relative to neutral cueing (A vs. B). Incorrect cueing to the lower visual field greatly increases reaction time to recognize a target in the upper visual field (B vs. C). Incorrect cueing to the upper visual field does not increase reaction time (D vs. E). All three of these comparisons suggest that there is a deficit in using the central arrow to guide covert orienting toward the upper visual field.

Table 19.
Effects of Distractor and Cue type in Patients vs. Age-Matched Control Subjects.

Results of the 2-way ANOVA on Incompatible - Compatible RT: Cue type, Group

Effect	*F*	*df*	*p*
Group	2.48	1,29	.12
Cue	.52	2,58	.59
Group X Cue	**3.98**	**2,58**	**.02**

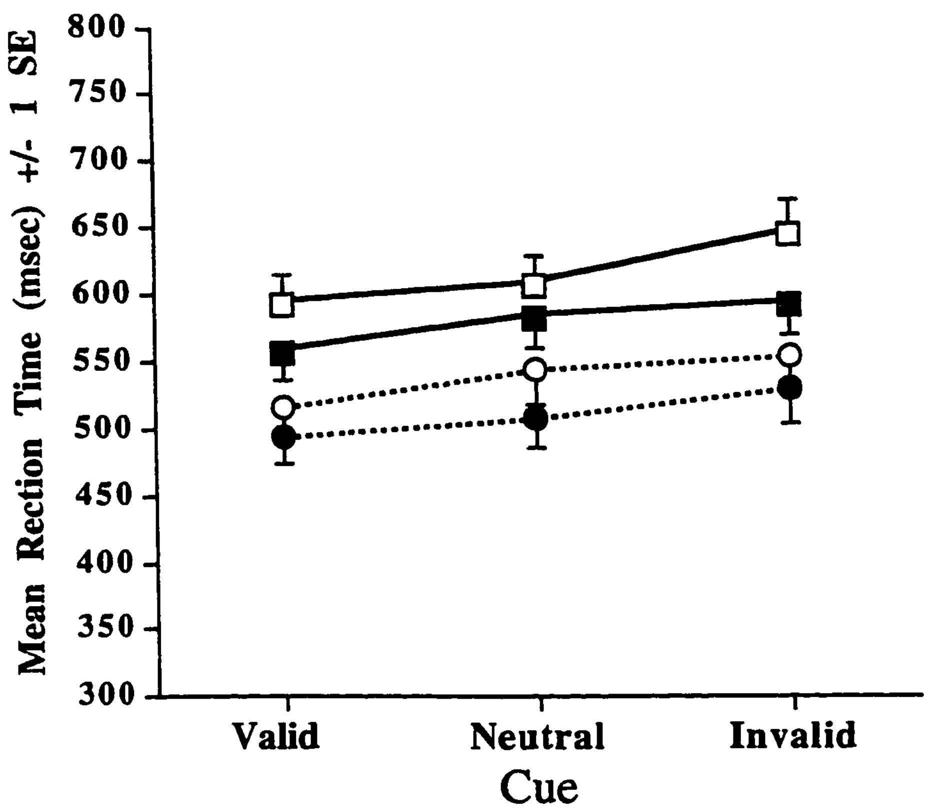

Figure 16. Mean reaction time as a function of cue type on trials with incompatible distractors (open symbols) and compatible distractors (filled symbols) for patients (squares) and age-matched controls (circles). Means are connected to facilitate comparisons of the effect of cue for the different groups.

Table 20.
Effect of Cue type and Visual Field in Patients vs. Age-Matched Control Subjects.

Results of the 2-way ANOVA on Validity Effect: Group, Field

Effect	*F*	*df*	*p*
Group	1.23	1,29	.27
Field	.31	1,29	.58
Group X Field	**35.67**	**1,29**	**.02**

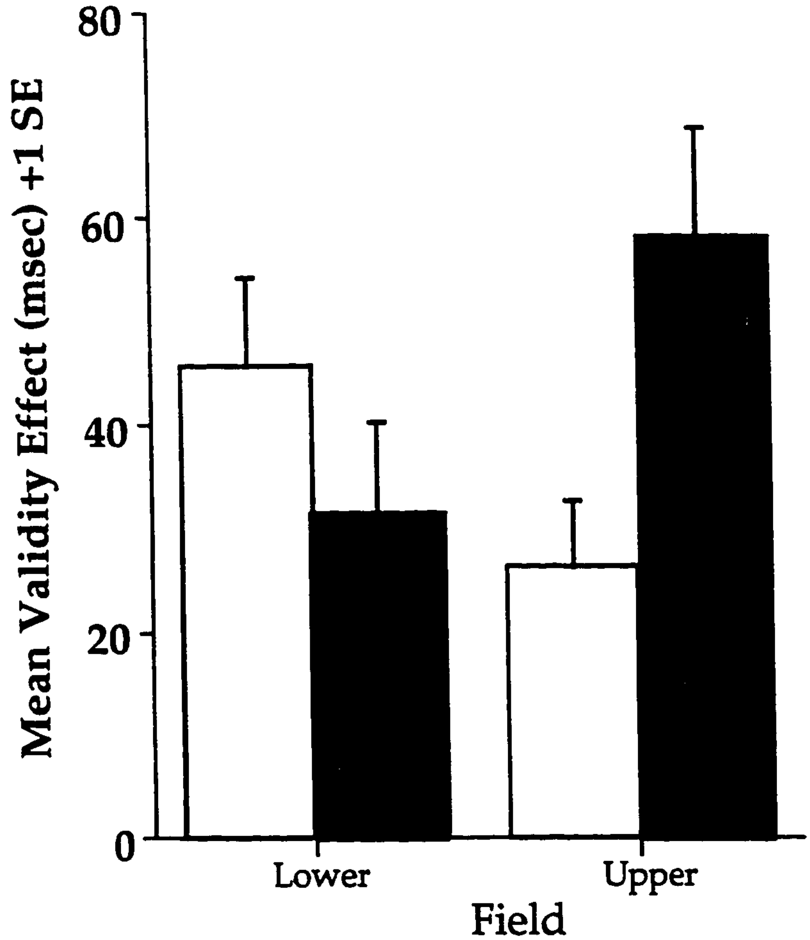

Figure 17. Mean Validity Effect (Invalid - Valid RT) on trials in the lower visual field and upper visual field for patients (black bars) and age-matched controls (open bars).

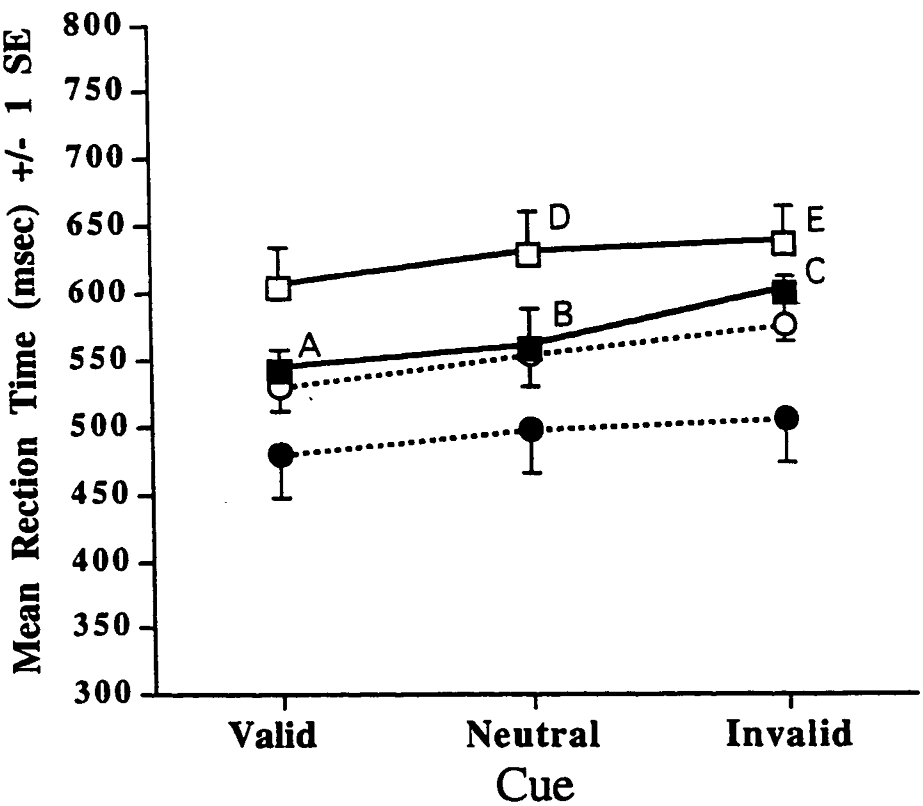

Figure 18. Mean reaction time as a function of cue type on trials in the lower visual field (open symbols) and upper visual field (filled symbols) for patients (squares) and age-matched controls (circles). Means are connected to facilitate comparisons of the effect of cue for the different groups.

Effect of Duration of Deprivation. The interaction between Group and Duration of deprivation was significant. Analyses of simple effects indicated that there was no difference in reaction time between patients with shorter deprivation and patients with longer deprivation ($p > .10$). Patients with longer deprivation were not significantly slower than the age-matched controls for that group ($p > .10$). Similarly, patients with shorter deprivation were not significantly slower than the age-matched controls for that group ($p > .10$). However, the age-matched controls for the group with shorter deprivation were slower than the controls for the group with longer deprivation ($p < .01$), probably because their mean age was lower (11.8 vs. 14.4 years).

Effect of Distractor. There was a main effect of Distractor. Across all conditions, reaction times were significantly slower on trials with incompatible distractors than on trials with compatible distractors.

Effect of Cue. There was a main effect of Cue. Across all conditions, reaction times were significantly slower on invalid trials than on neutral trials and valid trials, and significantly faster on valid trials than on neutral trials (Tukey post-tests, all *ps* $< .01$).

Effect of Visual Field. There was a main effect of visual field. Reaction times were faster to targets in the upper visual field (526.1 msec) than in the lower visual field (583.8 msec).

Figures 19 and 20 show the relationship between the duration of deprivation and the size of the two effects on which patients differed from the normal comparison group: the size of the validity effect in the upper visual field and the effect of incompatible distractors in increasing reaction time on invalid trials. Both graphs indicate larger effects in patients than in the normal comparison group (most ordinate values are positive) but no obvious relationship to the duration of deprivation.

Discussion

Patients Treated for Bilateral Congenital Cataract.

Although patients were slower overall than age-matched control subjects, on valid trials and on neutral trials their reaction times were influenced by distractors no more than those of age-matched control subjects. However, on invalid trials they were slowed more than age-matched control subjects by incompatible distractors. Interestingly, this pattern of effects is similar to one shown during normal development. On invalid trials but not on valid trials, 5-, 7-, and 9-year-old children are slowed more than adults by incompatible distractors (Akhtar & Enns, 1989). The effect of distractors on invalid trials suggests that deprivation interferes with the development of the ability to disengage attention from a miscued location, move attention to a new location, and then narrow the focus of attention on a target while ignoring distractors.

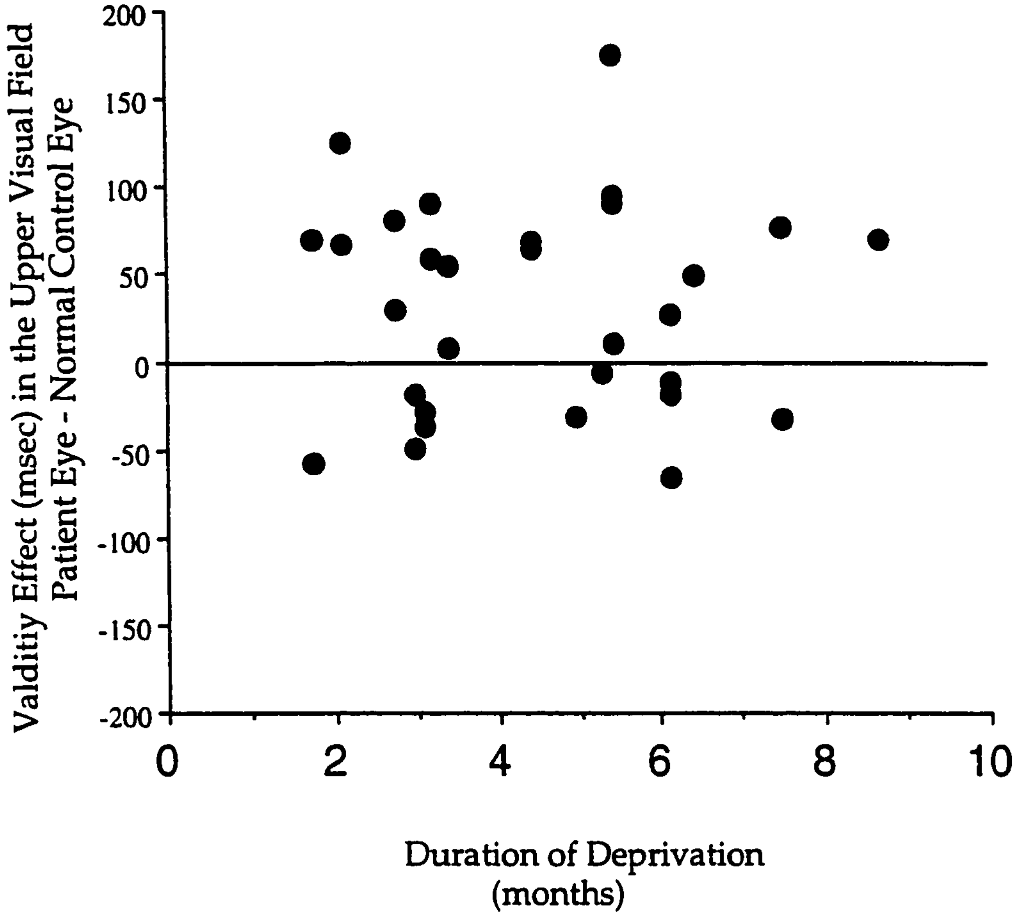

Figure 19. Each dot illustrates the difference between a patient's eye and its normal control eye in the size of the validity effect in the upper visual field plotted as a function of the duration of deprivation. The horizontal line at an ordinate value of zero represents no difference between patient and control. Positive values indicate a larger effect in the patient. Negative values indicate a smaller effect in the patient.

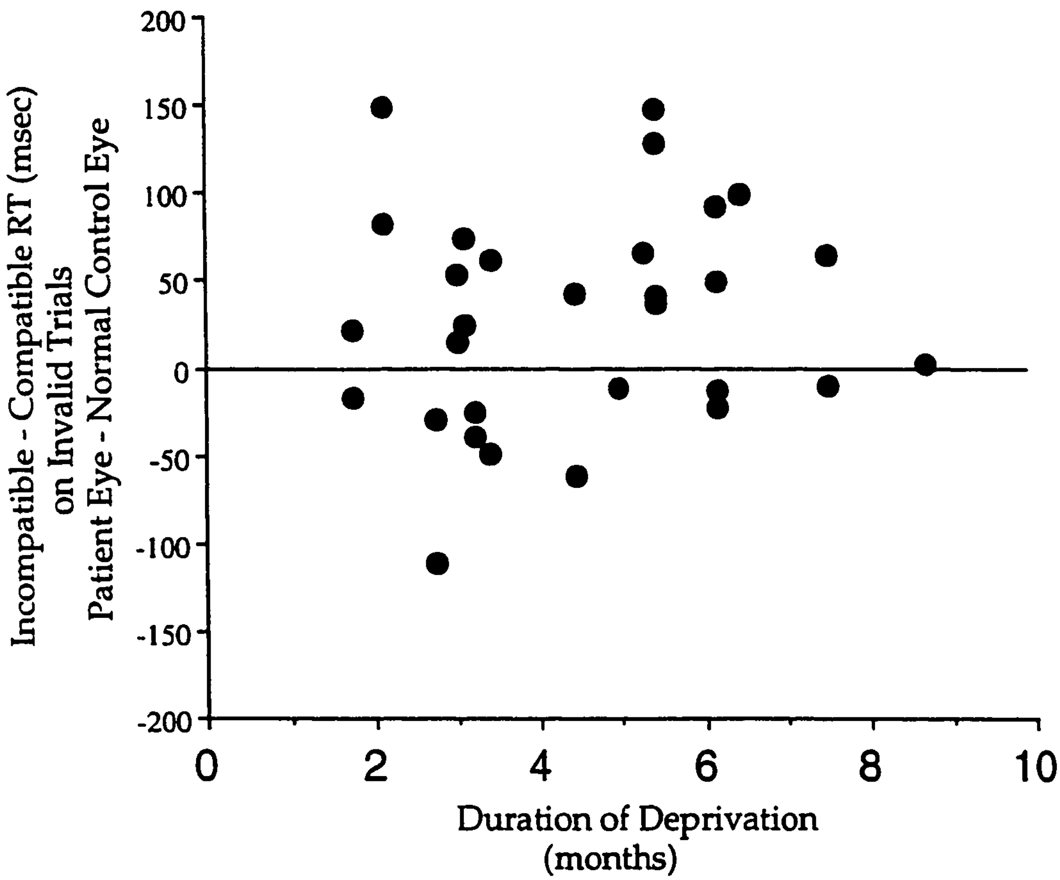

Figure 20. Each dot illustrates the difference between a patient's eye and its normal control eye in the effect of distractors on invalid trials, plotted as a function of the duration of deprivation. The effect of distractors was measured as the difference in RT between trials with incompatible distractors and compatible distractors. The horizontal line at an ordinate value of zero represents no difference between patient and control. Positive values indicate a larger effect in the patient. Negative values indicate a smaller effect in the patient.

The hypothesis presented in the discussion section of Chapter 4 is that early deprivation might affect the development of attention not just through effects on the visual responsiveness of parietal systems but also (or even mainly) through effects on frontal systems. This hypothesis fits with findings of deficits in the pattern of covert orienting toward the upper visual field and not toward the lower visual field. Three aspects of the data indicate such a deficit. First, the validity effect was larger than normal in the upper visual field (compare data points B vs. C in Figure 18). This result suggests that patients were less able than normal controls to switch attention from the incorrectly cued lower visual field to detect that something was in the upper visual field and to recognize which of the two shapes it was. Second, correct cueing to the upper visual field did not decrease patients' reaction time relative to neutral cueing (A vs. B). Third, incorrect cueing to the upper visual field did not increase their reaction time (D vs. E). All three of these comparisons suggest that early visual deprivation interferes with the ability to use the central arrow to guide covert orienting toward the upper field.

Although patients showed deficits in covert shifts of attention toward the upper visual field, both patients and age-matched controls were faster to recognize the shape of the target when it appeared in the upper visual field than when it appeared in the lower visual field. A possible explanation for these seemingly discrepant findings is that arrows require covert shifts of attention controlled by endogenous attentional systems whereas targets summon attention through exogenous systems. Because the two systems depend on different neural mechanisms, deprivation may affect one more than the other.

Previc's (1990) compilation indicates that lesions to monkey parietal cortex lead to deficits in peripersonal space, the lower visual field, where we reach for objects, and that lesions to the prefrontal cortex lead to deficits in the upper visual field, or extrapersonal space. There is converging evidence for the role of the parietal cortex in shifts of attention to the lower visual field from a case study of a patient with bilateral parietal damage: Although she performed normally in the upper visual field, she was unable to benefit from a cue in the lower visual field and hence had no validity effect in the lower visual field (Verfaellie et al., 1990). Together, the deficits found in Chapter 5 in the upper visual field and in ignoring distractors suggest that deprivation affects the development of attention mainly through effects on frontal systems.

In summary, the results with a relatively harder task show that visual experience is important for the development of the ability to shift attention and to ignore distractors. In the next chapter, I discuss the mechanisms that may account for these results.

Normal 10-year-olds versus Adults

Incompatible distractors slowed the reaction time of participants at both ages. However, 10-year-olds were slowed more than the adults by incompatible distractors. My results with 10-year-olds are consistent with those of Akhtar and Enns (1989), who found that children aged 5, 7, and 9 years were slowed more than adults by distracting information. In contrast, a study that required ignoring distractors but without also requiring covert orienting showed the *same* effect of distractors in children 10 - 12 years of age as in adults (Ridderinkhof & van der Molen, 1995). The comparison suggests that 10-year-olds are able to ignore distractors as well as adults under some conditions, but not if they must also covertly orient. In the General Discussion (Chapter 6), I discuss the neural substrates that are likely to be involved in the change between 10 years and adulthood.

The effect of distractors was not altered by the informativeness of the cue (i.e., valid, neutral, invalid) at either age. These findings contrast to those from a study with younger children aged 5, 7, and 9, and with smaller stimuli oriented horizontally (Akhtar & Enns, 1989) in which children were slowed considerably more than adults by incompatible distractors on invalid trials. There are several possible explanations of the discrepant results. First, it is possible that the interaction between validity of the cue and distractors decreases between 9 years of age (the age of the oldest group of children in Akhtar & Enns, 1989) and 10 years of age (the age of the children in this chapter). Second, Akhtar and Enns (1989) did not correct their data for a reaction time bias. A reaction time bias could lead to a different pattern of results, especially if there was more positive skew for younger children in the hardest conditions (e.g., invalid trials with incompatible distractors). Third, unlike Akhtar and Enns (1989), these procedures involved tests along the vertical meridian. In Chapter 6, I discuss the possibility that different brain mechanisms are involved in controlling attention along the horizontal and vertical meridia. Finally, previous studies with adults indicate that the distance between the target and distractors influences the effects of the distractors on reaction time. Eriksen and Eriksen (1974) found that the greatest slowing from incompatible distractors occurred at the smallest spacing (.5 degrees) between the target and the distractors. Therefore, it is possible that Akhtar and Enns' (1989) found significant slowing by incompatible distractors on invalid trials because they used relatively small spacings between the target and the distractors (.2 degrees compared to 1.6 degrees in my study), and at that spacing distractors have a greater effect on children than on adults.

As in Chapter 3, this study showed that, although 10-year-olds were slower than adults, there were no age differences in validity effects (compare Figures 3 and 15). The results suggest that under these conditions covert orienting is functioning at adult levels by 10 years of age.

CHAPTER 6

GENERAL DISCUSSION

In this chapter, I summarize the behavioral findings and discuss the developmental and neurophysiological implications of the results.

What My Studies Add to Our Knowledge about the Effects of Binocular Visual Deprivation

Studies in animals and in humans have shown that early visual deprivation adversely affects visual development (reviewed in Boothe et al., 1985; Movshon & van Sluyters, 1981; Hubel & Wiesel, 1977; Maurer & Lewis, 1993). However, little was known about the influence of early visual experience on the normal development of cognitive ability. In this dissertation, I provided the first examination of how early visual experience influences the development of attention. My studies of attention were guided by predictions based on what is known from studies of children treated for early visual deprivation, monkeys binocularly deprived from birth, and patients with damage to the parietal cortex.

One of the main reasons I suspected that visual experience might be important for the normal development of attention was that functions affected most seriously by deprivation are those that are immature at birth and develop slowly during childhood. In contrast, functions that are spared by deprivation are those that are reasonably mature at birth (Maurer et al., 1989). The abilities to shift attention covertly and to ignore distracting information are not present at birth but develop post-natally. Because these abilities emerge during infancy and continue to develop through the school-aged years, they are likely to be affected by early visual deprivation. In humans with brain lesions, attention is disrupted by damage to the parietal cortex. Moreover, binocular deprivation from birth in monkeys dramatically reduces the sensitivity of parietal cells to visual stimuli much more than it affects cells in the primary visual cortex (Carlson et al., 1987; Hyvarinen & Hyvarinen, 1979, 1982; Hyvarinen et al., 1981). In the absence of such experience in humans–because of bilateral congenital cataracts–attention might not develop normally and, following treatment, behavior on attention tasks might be similar to that shown in patients with parietal damage.

My studies of children treated for bilateral congenital cataracts indicate that visual deprivation affects the development of attention in humans. The manner in which attention is affected in a simple detection task (Chapter 4), is related to how long the deprivation lasted. Patients treated for bilateral cataract before four months of age, the age at which normal infants first show evidence of covert orienting (Johnson et al., 1991, 1994; Johnson & Tucker, 1996), performed normally. Patients with deprivation lasting longer than four months performed normally at 100 and 400 msec SOAs but abnormally at an 800 msec SOA. In particular, they showed no effects of the cues presented 800 msec before the target. In a discrimination task (Chapter 5), patients treated for binocular deprivation (of any duration from 1.8 - 7.6 months) had a larger-than-normal validity effect in the upper visual field but a normal validity effect in the lower visual field. On invalid trials, but not on valid or neutral trials, patients were slowed more than normal controls by incompatible distractors.

It is important to rule out possible explanations for the effects other than binocular deprivation. First, all of the patients had poor acuity. It is unlikely that poor acuity affected performance on my tasks because children performed well on the post-tests and had good performance under some conditions. For example, on the simple detection task patients treated for longer deprivation performed normally at the shorter SOAs and on the discrimination task with distractors, they showed a normal validity effect in the lower visual field and were affected normally by distractors on neutral trials. Additional evidence that reduced acuity did not affect patient's performance on my tasks comes from the fact that (i) children with monocular deprivation and poor acuity performed normally in both attention tasks; (ii) on the simple detection task, deficits at the 800 msec SOA were related to the duration of deprivation and not to acuity (r = -.29, $p > .10$); and (iii) on the discrimination task, there was no significant correlation between acuity and the size of the validity effect in the upper visual field (r = .02, $p > .10$) or the slowing by incompatible distractors on invalid trials (r = .06, $p > .10$). Taken together, this evidence suggests that acuity did not affect performance on these tasks.

Second, it is unlikely that the deficit in attention might have occurred because many of the patients have eye movement abnormalities associated with cataracts such as spontaneous jerky eye movements, otherwise known as nystagmus. Patients with nystagmus cannot maintain perfectly steady fixation. The literature suggests that there is a relation between the systems that control eye movements and covert orienting of attention (Posner et al., 1982; Rizzolatti, Riggio, Dascola, & Umilta, 1987; Sheliga, Riggio, Craighero, & Rizzolatti, 1995). However, it is unlikely that nystagmus impaired performance on these tasks because patients had good performance under some conditions.

I may have discovered that there are deficits in attention following early binocular deprivation that are manifest whenever the task is difficult, no matter how it is made difficult. For example, patients performed abnormally when required to sustain covert orienting, when tested with the combination of invalid cueing and incompatible distractors, and when attention was required to the field opposite where the response occurred (cueing to the upper visual field versus a response down at the keyboard). In that case, my studies revealed overall problems in controlling attention following early binocular deprivation. There is the alternative possibility that the pattern of deficits points to problems in only some aspects of attention following deprivation. One reason for thinking so is that patients (and normal participants) were faster to discriminate targets in the upper visual field than in the lower visual field (see Chapter 5). That finding suggests that the incompatibility between the location of the target and the response did not affect patients' reaction time and that their deficit in the upper but not lower visual field reveals differential effects of binocular deprivation.

What My Studies Add to Our Knowledge about the Normal Development of Attention

Prior to 4 months of age, babies have difficulty in moving their eyes away from a fixation object and toward another object (Atkinson et al., 1992; Hood & Atkinson, 1993; Johnson et al., 1991). However, it is unclear from the developmental studies whether (1) young babies have difficulty disengaging attention and/or (2) young babies have difficulty programming the eye movements used to measure shifts of attention. When children are tested with procedures that eliminate eye movements, they respond more quickly to a target at a correctly cued location than to a target at an incorrectly cued location (Akhtar & Enns, 1989; Brodeur, 1990, 1993; Enns & Brodeur, 1989;

Pearson & Lane, 1990). However, the effect of incorrect cueing on reaction time is greater in children than it is in adults (Akhtar & Enns, 1989; Brodeur, 1993; Enns & Brodeur, 1989; Pearson & Lane, 1990). This pattern suggests that it takes longer for young children than for adults to disengage attention from a miscued location, to move it to the location of a target, to re-engage attention at the target location, and to respond (Posner et al., 1978). While none of these studies has pinpointed the age at which covert attention becomes adult-like, the evidence that young babies have "sticky fixation," combined with the evidence that even young children are harmed more than adults by incorrect cueing, suggests that attention develops slowly during childhood and may be affected by abnormal visual experience during development. To clarify the normal development of attention, I examined the development of covert shifts of attention using two types of tasks, detection (Chapter 3) and discrimination (Chapter 5), under conditions with large cues, large targets, distractors, and tests along the vertical meridian.

The results show that although children were generally slower to respond than adults on both tasks, 8-year-old children (Chapter 3) and 10-year-old children (Chapters 3 & 5) were able to orient attention initiated by these endogenous cues as well as adults. The results under these conditions also suggest that there may be different patterns for the normal development of covert orienting (Chapters 3 & 5) and the normal development of the ability to ignore distractors (Chapter 5). Unlike the development of covert orienting, under these conditions, the ability to ignore distractors was not adult-like by 10 years of age: 10-year-olds were slowed more than the adults by incompatible distractors. Comparisons among studies, however, suggest that children around 10 years of age are able to ignore distractors as well as adults under some conditions (Ridderinkhof & van der Molen, 1995), but not if they must also orient covertly (Chapter 5). The data in Chapter 5 are the first to show with a covert orienting paradigm that the ability to ignore distractors is not yet adult-like by 10 years of age. A study by Akhtar and Enns (1989) with slightly younger children, ranging in age from 8.7 to 9.8 years, also found that children were slowed more than adults on invalid trials by incompatible distractors.

Implications for Our Understanding of the Neural Mechanisms of Attention

Effects of Early Deprivation

The deficits in patients treated for bilateral congenital cataract were not as predicted. I originally predicted that deficits following binocular deprivation would be similar to deficits in adult parietal patients because binocular deprivation in monkeys damages the visual responsiveness of cells in the parietal cortex (Hyvarinen & Hyvarinen, 1979). However, the results after longer binocular deprivation were unlike the larger-than-normal validity effects characteristic of parietal deficits. Instead, patients failed to show a validity effect at a longer SOA. As discussed in Chapter 4, this pattern of deficit suggests that the effects of early visual deprivation on the brain and attention may differ from the effects of damage to the parietal cortex in adults. In the framework developed by Nakayama and MacKeben (1989) the results in Chapter 4 suggest that deprivation causes a deficit in the *sustained* component of attention, and no deficit in the transient component of attention, because performance was normal at the two shorter SOAs. Results from Chapter 5 also suggest that deprivation causes a deficit in the ability to ignore distractors. There was, however, one finding that is like the pattern in parietal patients: a larger-than-normal validity effect in the upper visual field when the task involved discrimination and distractors surrounded the target.

The results from studies of infantile lesions suggest that anterior systems may play a larger role in attention in infants than in adults. For example, children who had had bilateral or unilateral infantile posterior lesions reacted normally to spatial cueing (Craft et al., 1994b; Johnson et al., in press). However, spatial cueing effects were abnormal after anterior lesions, with no benefit of cues (in the right visual field) at short SOAs in children after bilateral infantile lesions (Craft et al., 1994b) and in infants following left frontal lesions (Johnson et al., in press). Also, there was an abnormally large validity effect in children who suffered bilateral infantile anterior lesions when tested at a long SOA or in the left visual field (Craft et al., 1994b). These findings suggest that the neural systems controlling visual-spatial attention are different in infants than in adults. Based on Event-related potentials, Csibra et al (in preparation) suggest that normal babies rely more on the frontal cortex, especially left structures, for attentional abilities during infancy, and only later come to depend on the parietal cortex. Obviously, after infantile parietal lesions, the frontal cortex can continue to play a role in attention. Early deprivation might affect the development of attention not just through effects on the visual responsiveness of the parietal cortex, but also through effects on anterior frontal systems.

The hypothesis that early deprivation might affect the development of attention through effects on anterior frontal systems is supported by the deficits in the upper visual field (Chapter 5). Inspection of the mean reaction times in the patients who had early visual deprivation suggests that their larger-than-normal validity effect in the upper visual field arises from a deficit in using the central arrow to guide covert orienting toward the upper visual field. This result fits with the interpretation of evidence from studies of attention in adults, that parietal lesions are related to deficits in the lower visual field (peripersonal space) while pre-frontal lesions are related to deficits in the upper visual field (extrapersonal space) (Previc, 1990; Verfaellie et al., 1990).

The hypothesis that early deprivation might affect the development of attention through effects on anterior frontal systems is also supported by the deficits in ignoring distractors (Chapter 5). Evidence from adults with frontal lesions indicates that the frontal cortex is involved in focusing and sustaining attention, ignoring interference from distracting information and in inhibiting prepotent responses (Denckla, 1996; Duncan, 1986; Husain & Kennard, 1997; Godefroy, Lhullier, & Rousseaux, 1996; Rueckert & Grafman, 1996; Richer & Lepage, 1996). This system also has a protracted developmental period during which it could be affected by lack of input. For example, glucose utilization findings show that the frontal cortex has a longer developmental period than parietal, occipital, and temporal cortical areas of the brain (Huttenlocher, 1994; Chugani, 1994). In addition, although there are no studies in humans or in primates on the effects of visual deprivation on the frontal cortex and frontal functions, one study in rats shows that visual deprivation does disrupt development of the frontal cortex (Schleibs et al., 1982).

My speculations about neural mechanisms may apply only to tests along the vertical meridian because different neural structures may be involved in shifts of attention along the vertical meridian than along the horizontal meridian. A review of the literature indicates that the frontal cortex is related to attention in the upper visual field while the parietal cortex is related to attention in the lower visual field and in the left and the right visual fields (Previc, 1990; Posner et al., 1982). Because the underlying mechanisms important for attention along the vertical meridian and the horizontal meridian are different, it is possible that I might obtain different results if I conducted my tests along the horizontal meridian.

Normal Development

Combined with the literature, my findings suggest that the neural mechanisms for covert orienting are functioning at adult levels by 8 years of age. The adult-like functioning of the neural mechanisms may be limited to certain parts of the visual field (i.e., vertical meridian) or to easily detected stimuli. According to Posner's theory (1988), my findings indicate that at least under the conditions tested, the component processes of covert shifts of attention (i.e., disengage, move, engage) and the underlying mechanisms, such as the parietal cortices, have matured by 8 years of age.

The findings suggest that under these conditions, the structures of the brain important in ignoring distracting information are not fully developed by 10 years of age. The ability to keep attention away from distractors and focused on important stimuli is related to the frontal attentional system, at least in adults (Denckla, 1996; Duncan, 1986; Husain & Kennard, 1997; Godefroy et al., 1996; Richer & Lepage, 1996). For example, the visual search of one patient with a unilateral frontal lesion was disrupted contralateral to the lesion by distractors in ipsilesional space (Husain & Kennard, 1997). The patient was able to direct his visual search to the contralesional side of arrays only when the density of distractors was low or when targets were highly discriminable. In contrast, the patient responded well to targets ipsilateral to the lesion even when the density of distractors was high. Patients with frontal lesions are also impaired on other paradigms that require the ability to inhibit responses/attention to certain types of information (e.g., Go-No Go, Continuous Performance Test, Wisconsin Cart Sorting Test). For example, on the Go-No Go test, patients with frontal lesions make more frequent responses than normal control subjects to irrelevant stimuli (Godefroy et al., 1996). Finally, the likely contribution of an immature frontal cortex (Huttenlocher, 1994; Chugani, 1994) to 10-year-old's performance in Chapter 5 is also suggested by studies of normal children on tasks related to frontal lobe function such as Wisconsin Card Sorting, Go-No Go, and Tower of London. There is major improvement on these tasks around early adolescence (Levin et al., 1991).

Other studies indicate that in addition to the frontal cortex, circuits involving the pulvinar nucleus of the thalamus are involved in ignoring distracting information. Animals and humans with damage to the pulvinar have difficulty sustaining attention on a target in the presence of distractors (Desimone & Duncan, 1995). The pulvinar has direct connections with the posterior parietal cortex (posterior attention system), occipital cortex, dorsolateral prefrontal cortex, ventrolateral prefrontal cortex (anterior attention system), and temporal areas of the brain (LaBerge, 1995a, b). A quantitative magnetic resonance study of T1-weighted images of neural structures from brains of children at various ages showed that the pulvinar undergoes maturational changes up to 11.4 years of age, at which point it is adult-like (Steen, Ogg, Reddick, & Kingsley, 1997). My findings that 10-year-olds were worse than adults in ignoring distracting information is not surprising in light of the evidence that neither the pulvinar nor the frontal cortex are mature by 10 years of age.

In summary, the results show that in a discrimination task with easily detected targets, the ability to shift attention covertly, but not the ability to ignore distractors, is adult-like by 10 years of age. The different rates of development on these tasks are consistent with PET studies of glucose metabolism that indicate that the frontal cortex has a longer developmental period than parietal, occipital, and temporal areas of the brain (Chugani, 1994).

Future Studies on the Role of Experience in the Development of Cognitive Ability

To test the generality of these deficits in patients, and hence their neural basis, future studies might examine children treated for cataracts on tasks related to frontal lobe function. If children treated for cataract perform abnormally on such tasks, then it would confirm that early deprivation impairs functions mediated by the frontal attentional system. To understand the neural basis of the deficits I found after deprivation, it would be important to use my procedure in children with infantile or late-onset, parietal or frontal lesions to determine whether normal babies rely on the frontal cortex for attentional abilities during infancy, and only later come to depend on the parietal cortex like adults. To examine whether different neural mechanisms are involved in shifts of attention along the horizontal versus vertical meridia, it would be interesting for future studies to examine the performance along the horizontal meridian of patients with bilateral deprivation. In addition, to explore other conditions under which patients treated for binocular deprivation show deficits, future studies might test children treated for bilateral cataract in a discrimination task at other SOAs (such as 800 msec) and with smaller spacings between the distractors and the target.

Future studies might also examine the influence of visual deprivation on attention in other modalities. After binocular deprivation in monkeys (e.g., Hyvarinen & Hyvarinen, 1979), there is an increase in the percentage of cells driven by somatosensory stimuli in the parietal cortex. In humans following binocular deprivation, there might also be an increase in the percentage of cells driven by somatosensory stimuli in the parietal cortex and performance might be better than normal on spatial cueing tasks with non-visual cues. Tests of attention in other sensory modalities might help us find out whether visual experience is important for the normal development of attention in modalities other than vision. Finally, to be able to define a "sensitive period" during which experience is necessary for the normal development of attention, future research should examine patients who had normal visual development and then suffered from deprivation later on during childhood. It is reasonable to predict at least two possible outcomes in children whose deprivation occurs later on in life. If the damage occurs after a normal period of visual development, then no damage to the parietal cortex, and consequently no attention deficits, may be observed. Alternatively, a pattern of deficits similar to those in adults with parietal lesions may be observed.

Future Studies on the Normal Development of Attention

One developmental-cognitive neuropsychological implication of the results reported in this thesis is that the parietal attentional system is mature before the frontal system. On this basis, we would expect 10-year-olds to perform at adult levels on other tasks that depend upon the parietal cortex (e.g., shifts of attention in other modalities, visual search, smooth pursuit) and yet differ from adults on tasks that depend upon the frontal cortex (e.g., planning, working memory, inhibition, maintenance of anticipatory set/preparedness to act). To test the hypotheses that different mechanisms are involved in these two types of attention, and that these mechanisms develop at different rates, future research might compare the performance of normal children on tasks that are thought to involve parietal and/or frontal cortex function.

Conclusions

These are the first studies to examine the role of experience in the normal development of attention. The findings show that deprivation does disrupt aspects of the attentional system that are involved in shifting and maintaining attention. In a simple reaction time task, the ability to sustain attention was abnormal after longer periods of deprivation. In a more complex task, patients with binocular deprivation of any duration were impaired in the ability to shift attention to the upper visual field and in the ability to ignore distracting information. Together these findings indicate that the normal development of attention is sensitive to experience early in life. The results add to the evidence that deprivation especially disrupts abilities that are immature at birth and that develop slowly during childhood (Maurer et al., 1989).

These studies on the normal development of attention showed that while covert orienting is developed by 8 years of age, at least under some conditions, the ability to ignore distracting information is not yet fully developed even by 10 years of age. This result suggests that the parietal system, involved in the ability to shift attention covertly in adults, and the frontal system, involved in the ability to ignore distractors, develop at different rates.

Finally, the tasks that I have designed to study attention in children treated for cataracts can be used for testing other populations. The cues and targets are large enough so that the tasks can be used to examine the effects of other types of eye disorders. Also, the tasks are simple enough that they can be used in testing the attentional abilities of children with developmental disorders, patients with brain injury, and the elderly.

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Appendix A

Preliminary Studies in Normal Participants

I conducted four pilot studies with normal participants before deciding on the final procedure to use with normal children and patients treated for cataract. Table 1 shows how I varied the predictiveness of the cue and the percentage of catch trials across these studies.

In Pilot Study 1, I used catch trials and two SOAs, intermixed in a random order. When SOAs are intermixed in a procedure that includes catch trials, like Pilot Study 1, the participant's anticipation of the target may be stronger at a shorter SOA than at a longer SOA. For example, at the onset of a given trial, the probability of a valid trial was .66 and that of a catch trial was .17. However, during a trial, once the interval of the shorter SOA had elapsed without the onset of a target, the probability of a valid trial shifted to .57. The probability that the current trial was a catch trial increased from .17 to .29. Consequently, a participant may have been less expectant of a target at a longer SOA.

In Pilot Study 2, catch trials were omitted and two SOAs were tested in an intermixed and random order. In studies without catch trials and intermixed SOAs (e.g., Sorensen et al., 1994), the predictive value of the cue may also decrease with increasing SOA. At the shortest SOA in Pilot Study 2, the cue predicted the location where the target would appear 80% of the time. At the longest SOA, because there were no catch trials, the participant may have been well prepared to respond to the target because the time interval predicted the occurrence of the target 100% of the time.

In Pilot Studies 3 and 4, the predictiveness of the cue was held constant at each SOA by presenting the SOAs in separate blocks. In Pilot Study 3, each block of trials at the 100 and 800 msec SOAs had eight catch trials. The cue predicted the location of the target 80% of the time. Pilot Study 4 was identical to Pilot Study 3 but there were 16 catch trials at each SOA (10% of the trials). The cue predicted the location of the target 75% of the time. With this series of four Pilot Studies, I developed the procedure of presenting trials in separate blocks according to SOA. This is the procedure that I used in Chapter 2 to examine the time course of covert orienting in adults and to pick the most informative SOAs to study age differences in covert orienting (Chapter 3) and covert orienting in children treated for cataract (Chapter 4). The results of these pilot studies are based on medians rather than on the mean outlier elimination procedure in the final experiment.

Across all four studies, adults seldom responded on catch trials (M = 5.6%, range 0-12.5%). Eight-year-olds (Pilot Study 1) made more responses on catch trials than adults (M = 15.1%, range 0-37%). Trials on which participants moved their eyes (adults M = 2.1%, range 0-5.6%; 8-year-olds M = 4.6%, range 0.6-12.5%) or anticipated the appearance of the target (adults M = 0.5%, range 0-2.5%; 8-year-olds M = 0.5%, range 0-0.7%) were eliminated from the data analyses.

Participants

Participants were four groups of adult undergraduate students (Pilot Studies 1-4) and one group of 8-year-old children (Pilot Study 1). Separate groups of 24 participants were tested in each study and at each age. Half of the participants were male and half were female. All participants passed the same vision screening examination as described in Chapter 2. The procedure was the same as in Chapter 2 with the exceptions described below for each of the studies.

Pilot Study 1

Purpose

Pilot Study 1 was designed to examine (1) the validity effect in normal children and adults and (2) the consistency and reliability of the validity effect when participants are retested within a two-week period.

Procedure

Normal 8-year-old children and adults were tested with 288 trials at each of two SOAs (100 or 800 msec) in two experimental sessions. The two sessions were separated by at least one day, but no more than 14 days. Targets were presented either 100 or 800 msec after a cue, with the time intervals randomized across trials. There were 160 valid, 40 invalid, 40 neutral, and 48 catch trials, half at each SOA.

Results

I conducted an analysis of variance with Age (8 yrs vs. adult) as a between subject variable and Session (first, second), Cue type (invalid, neutral, valid), SOA (100, 800 msec), and Visual field (upper, lower) as within subject variables. The results are shown in Table 21.

<u>Validity effect in normal children and adult participants.</u>

There was a significant 3-way interaction between Age, Cue, and SOA ($p < .05$). To identify the source of this interaction, I conducted separate ANOVAs for 8-year-olds and adults with Cue type and SOA as within subject factors. Results of the ANOVA for 8-year-olds are shown in Table 22. There was a significant main effect of Cue type ($p < .0001$). Eight-year-olds were 49.8 msec faster on valid trials than on invalid trials (Tukey post-tests, $p < .01$). This significant validity effect (invalid - valid RT) is shown in Figure 21. Additionally, Tukey post-tests indicated that reaction times were significantly faster on valid trials than on neutral trials (Benefit, $p < .05$) and not significantly slower on invalid trials than on neutral trials (Cost, $p > .05$). There was a significant main effect of SOA ($p < .0001$). Eight-year-olds responded faster to trials at the 800 msec SOA than at the 100 msec SOA. The interaction between Cue type and SOA was not significant.

Results from the ANOVA for adults are shown in Table 23. There was a significant Cue type by SOA interaction ($p < .001$). Analyses of simple effects indicated a significant effect of Cue type at the 100 ms SOA ($p < .05$) but not at

Table 21.
Differences Between 8-year-olds and Adults in Covert Orienting Initiated by Endogenous Cues – Sessions 1 & 2.

Results of the 5-way ANOVA: Age group, Session, Cue type, SOA, Visual Field

Effect	*F*	*df*	*p*
Age	**99.44**	**1, 46**	**< .00001**
Session	**84.92**	**1, 46**	**< .00001**
Cue	**35.27**	**2, 92**	**< .00001**
SOA	**58.19**	**1, 46**	**< .00001**
Field	.04	1, 46	.83
Age X Session	**6.73**	**1, 46**	**< .05**
Age X Cue	**4.87**	**1, 92**	**< .01**
Session X Cue	2.18	1, 92	.12
Age X SOA	**23.29**	**1, 46**	**< .00001**
Session X SOA	1.47	1, 46	.23
Cue X SOA	2.07	2, 92	.13
Age X Field	.89	1, 46	.35
Session X Field	**5.09**	**1, 46**	**< .05**
Cue X Field	.01	2, 92	.98
SOA X Field	1.03	1, 46	.32
Age X Session X Cue	1.22	2, 92	.30
Age X Session X SOA	1.51	1, 46	.22
Age X Cue X SOA	**3.32**	**2, 92**	**< .05**
Age X Session X Field	3.15	1, 46	.08
Age X Cue X Field	.58	2, 92	.56
Age X SOA X Field	1.38	1, 46	.24
Cue X SOA X Field	2.20	2, 92	.11
Session X Cue X SOA	1.33	2, 92	.27
Session X Cue X Field	.35	2, 92	.70
Session X SOA X Field	2.48	1, 46	.12
Age X Session X Cue X SOA	2.58	2, 92	.08
Age X Session X Cue X Field	.62	2, 92	.54
Age X Session X SOA X Field	3.04	1, 46	.08
Age X Cue X SOA X Field	.04	2, 92	.96
Session X Cue X SOA X Field	.04	2, 92	.95
Age X Session X Cue X SOA X Field	.30	2, 92	.74

Table 22.
Results from 8-year-old Participants on First Experimental Session.

Results of the 2-way ANOVA: Cue type, SOA

Effect	*F*	*df*	*p*
Cue	**13.34**	**2, 46**	**< .00001**
SOA	**37.14**	**1, 23**	**< .00001**
Cue X SOA	2.69	2, 46	.07

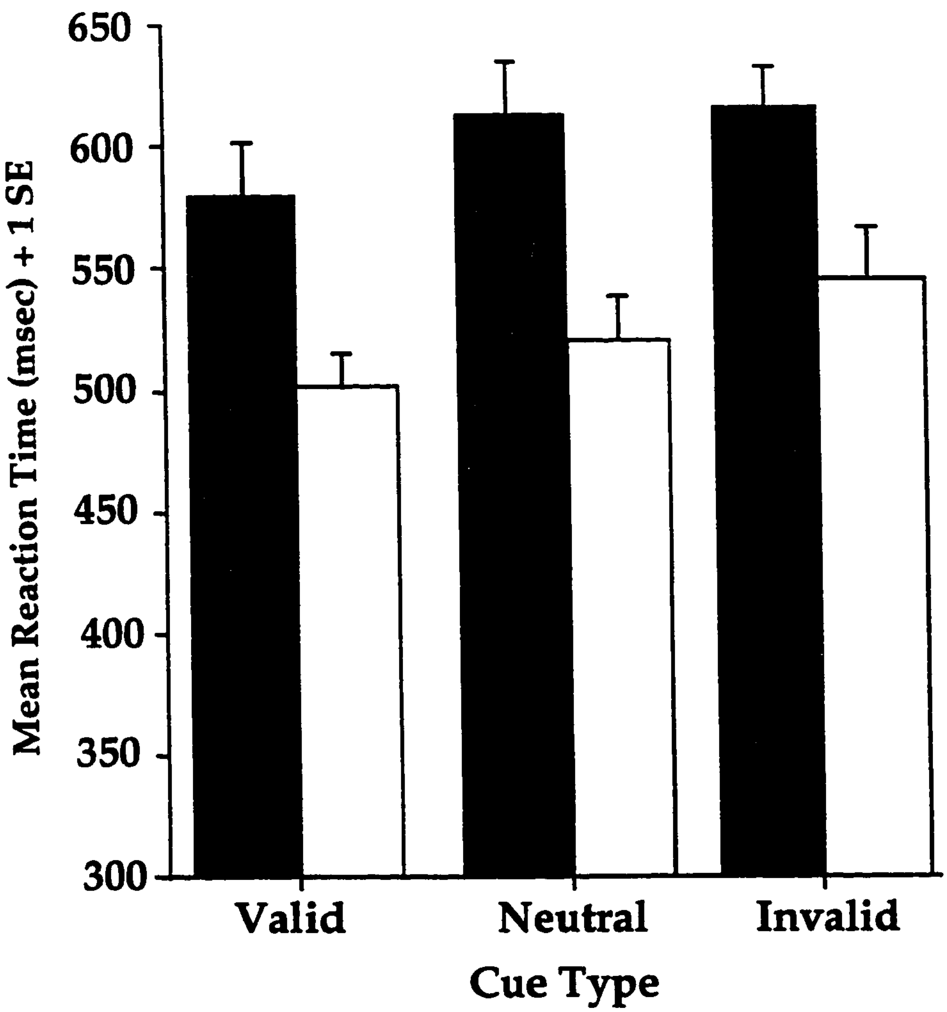

Figure 21. Mean Reaction time as a function of cue type for 8-year-old children at the 100 msec SOA (black bars) and at the 800 msec SOA (white bars).

Table 23.
Results from Adult Participants on Data From First Experimental Session.

Results of the 2-way ANOVA: Cue type, SOA

Effect	*F*	*df*	*p*
Cue	**18.21**	**2, 46**	**< .00001**
SOA	**15.60**	**1, 23**	**< .001**
Cue X SOA	**9.49**	**2, 46**	**< .001**

the 800 msec SOA. Figure 22 illustrates the significant validity effect at the 100 msec SOA. Reaction times were 32.5 msec faster on valid trials than on invalid trials (Tukey post-test, $p < .01$). Additionally, Tukey post-tests indicated that reaction times were significantly faster on valid trials than on neutral trials (Benefit, $p < .01$) and slower on invalid trials than on neutral trials (Cost, $p < .05$).

Consistency and reliability of the validity effect.

Table 21 shows that there were no age differences in the consistency of the validity effect (Age X Cue X Session, $p > .05$). Overall, participants were faster on trials from the second session than on trials from the first session (Main effect of Session, $p < .0001$). Although session interacted with age ($p < .05$), I was unable to identify the source of the interaction using analyses of simple effects. Session also interacted with visual field ($p < .05$). Analyses of simple effects indicated that reaction times during the first session were longer than during the second session, to targets in both visual fields (both *p*s $< .05$). Analyses of simple effects also showed that reaction times did not differ for targets in the upper and lower visual fields ($p > .05$); thus, the source of the interaction is unclear.

Discussion

Practice influenced only the speed of participants' responding. Practice had no influence at any age on the size of the validity effect. Adults showed significant validity effects at the 100 msec SOA but not at the 800 msec SOA

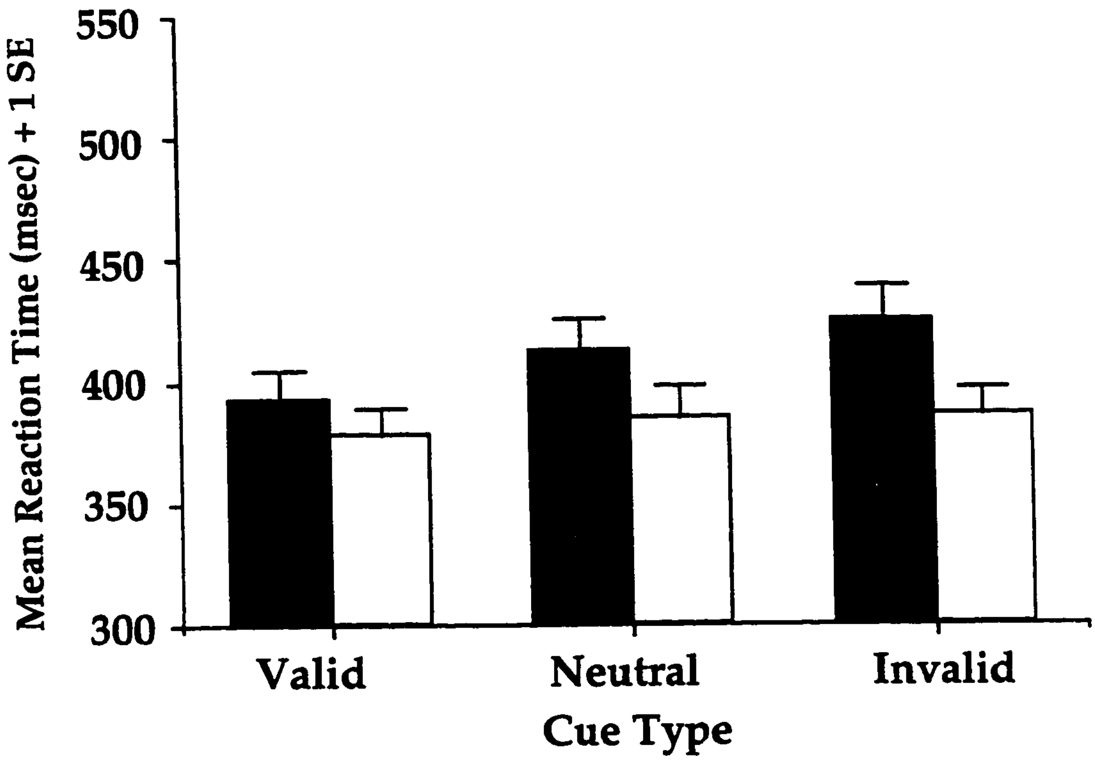

Figure 22. Mean Reaction time as a function of cue type for adult participants at the 100 msec SOA (black bars) and the 800 msec SOA (white bars).

while 8-year-old children showed significant validity effects at both SOAs. One study in the literature that also used endogenous cues and a simple reaction time task obtained similar findings: Moran, Thaker, Smith, Cassady, & Layne-Gedge (1992) found significant validity effects at only a 100 msec SOA and not at a 800 msec SOA. However unlike this study, Carter, Robertson, Chaderjian, Celaya, & Nordahl (1992) found significant validity effects at both 100 and 800 msec SOAs. A possible explanation why the results from Carter et al., (1992) differ from this study and Moran et al., (1992) is that a third, 0 msec SOA was also included (Carter et al., 1992). The addition of a third SOA makes the testing conditions different and can influence the predictive value of the cue among SOAs, and hence the participant's anticipation of the target. Another possible explanation for differences between Pilot Study 1 and previous studies in the literature is that Pilot Study 1 used larger cues and targets, and vertical placement of the stimuli on the computer screen.

Pilot Study 1 differs from the two previous studies that also used simple reaction time procedures, 100 and 800 msec SOAs, and endogenous cues, because it included catch trials. In Pilot Study 1, there were catch trials and the two SOAs were intermixed in a random order. When SOAs are intermixed in a procedure that includes catch trials, like Pilot Study 1, the participant's anticipation of the target may be stronger at a shorter SOA than at a longer SOA. Consequently, a participant might have been less expectant of a target at a longer SOA.

The possibility that the participant's anticipation of the target decreases with increased SOA may explain why adults showed significant validity effects on trials at the 100 msec SOA, but failed to show significant validity effects on trials at the 800 msec SOA. In previous studies with catch trials and intermixed SOAs, at the shortest SOA, the cue predicted the location where the target would appear more than 50% of the time, and the probability of the trial being a catch trial was .15 (Shulman et al., 1979) or .20 (Remington & Pierce, 1984; Shepherd & Müller, 1989). At the longer SOAs, the probability of the trial being a catch trial was higher and hence the predictiveness of the cue lower. At the longest SOAs predictiveness was well less than 50%. The results from these studies showed that the effects of valid, invalid, and neutral cueing varied across SOA. These findings are discussed more thoroughly in Chapter 2.

A possible explanation why 8-year-olds showed significant validity effects at both SOAs, is that they may have been less likely to use time as a cue for the appearance of a target. In Pilot Study 2, I attempted to equate predictiveness of the cue at the two SOAs by eliminating catch trials.

Pilot Study 2

Purpose

Pilot Study 2 was designed to examine the validity effect in normal adults in a procedure that attempted to equate and increase the overall predictiveness of the cue at both SOAs. In Pilot Study 2 catch trials were eliminated and two SOAs were tested in an intermixed and random order. As discussed previously, in studies without catch trials and intermixed SOAs (e.g., Sorensen et al., 1994), the predictive value of the cue may also decrease with increasing SOA. At the longest SOA, because there were no catch trials, the participant may be well prepared to respond to the target because the time interval predicts the occurrence of the target 100% of the time.

Procedure

Adults were tested in one experimental session of 240 trials with no catch trials. There were 160 valid trials, 40 invalid trials, and 40 neutral trials, half at a 100 msec SOA and half at a 800 msec SOA. At the shorter SOA the cue predicted the location where the target would appear 80% of the time.

Results

An ANOVA was used to compare data in adults from Pilot Study 2 to data from the first session of Pilot Study 1 in which the cue was less predictive at the 100 and 800 msec SOAs. The between-subject factor was Study (Study 1, Study 2) and the within-subject factors were Cue type (Invalid, Neutral, Valid), SOA (100 msec, 800 msec), and Visual field (Lower, Upper). The results from the ANOVA are shown in Table 24. There was a significant four-way interaction between Study, Cue type, SOA, and Visual field ($p<.05$) that I analyzed by conducting separate ANOVAs in the upper and lower visual field.

Table 25 shows the results from the ANOVA in the upper field. There was a significant Cue type by SOA interaction ($p < .05$). Analyses of simple effects indicated that there was a significant effect of Cue type at the 100 msec SOA ($p < .001$). Tukey post-tests indicated that participants (combined across Pilot Study 1 and Pilot Study 2) showed a significant validity effect (25.9 msec, $p < .05$). There was no effect of Cue type at the 800 msec SOA ($p > .05$). This is illustrated in the top graphs in Figures 23 and 24. There was also a significant Study by SOA interaction ($p < .0001$). However, analyses of simple effects were unable to uncover the source of the interaction: In both Study 1 and 2, reaction times were significantly faster to targets at the 800 msec SOA than at the 100 msec SOA.

In the lower visual field, the ANOVA indicated a significant three-way interaction between Study, Cue type, and SOA ($p < .01$). This is shown in Table 26. To analyze this interaction more closely, separate ANOVAs were conducted at each SOA with Study and Cue type as within-subject factors. Results of the ANOVA at the 100 msec SOA are shown in Table 27. There

Table 24.
Influence of Cue Predictiveness on Reaction Time.

Results of the 4-way ANOVA: Study, Cue type, SOA, Visual Field

Effect	*F*	*df*	*p*
Study	.26	1, 46	.61
Cue	**24.80**	**2, 92**	**< .00001**
SOA	**104.6**	**1, 46**	**< .00001**
Field	**5.17**	**1, 46**	**< .05**
Study X Cue	.51	2, 92	.60
Study X SOA	**23.26**	**1, 46**	**< .00001**
Study X Field	1.48	1, 46	.23
Cue X Field	1.03	2, 92	.36
Cue X SOA	**7.52**	**2, 92**	**< .001**
SOA X Field	1.28	1, 46	.26
Study X Cue X SOA	**3.28**	**2, 92**	**< .05**
Study X Cue X Field	1.22	2, 92	.29
Study X SOA X Field	1.32	1, 46	.26
Cue X SOA X Field	.14	2, 92	.87
Study X Cue X SOA X Field	**4.22**	**2, 92**	**< .05**

Table 25.
Influence of Cue Predictiveness on Reaction Time to Targets in the Upper Visual Field.

Results of the 3-way ANOVA: Study, Cue type, SOA

Effect	*F*	*df*	*p*
Study	.12	1, 46	.72
Cue	**14.33**	**2, 92**	**< .00001**
SOA	**88.16**	**1, 46**	**< .00001**
Study X Cue	.05	2, 92	.95
Study X SOA	**17.44**	**1, 46**	**< .0001**
Cue X SOA	**3.29**	**2, 92**	**.042**
Study X Cue X SOA	.65	2, 92	.53

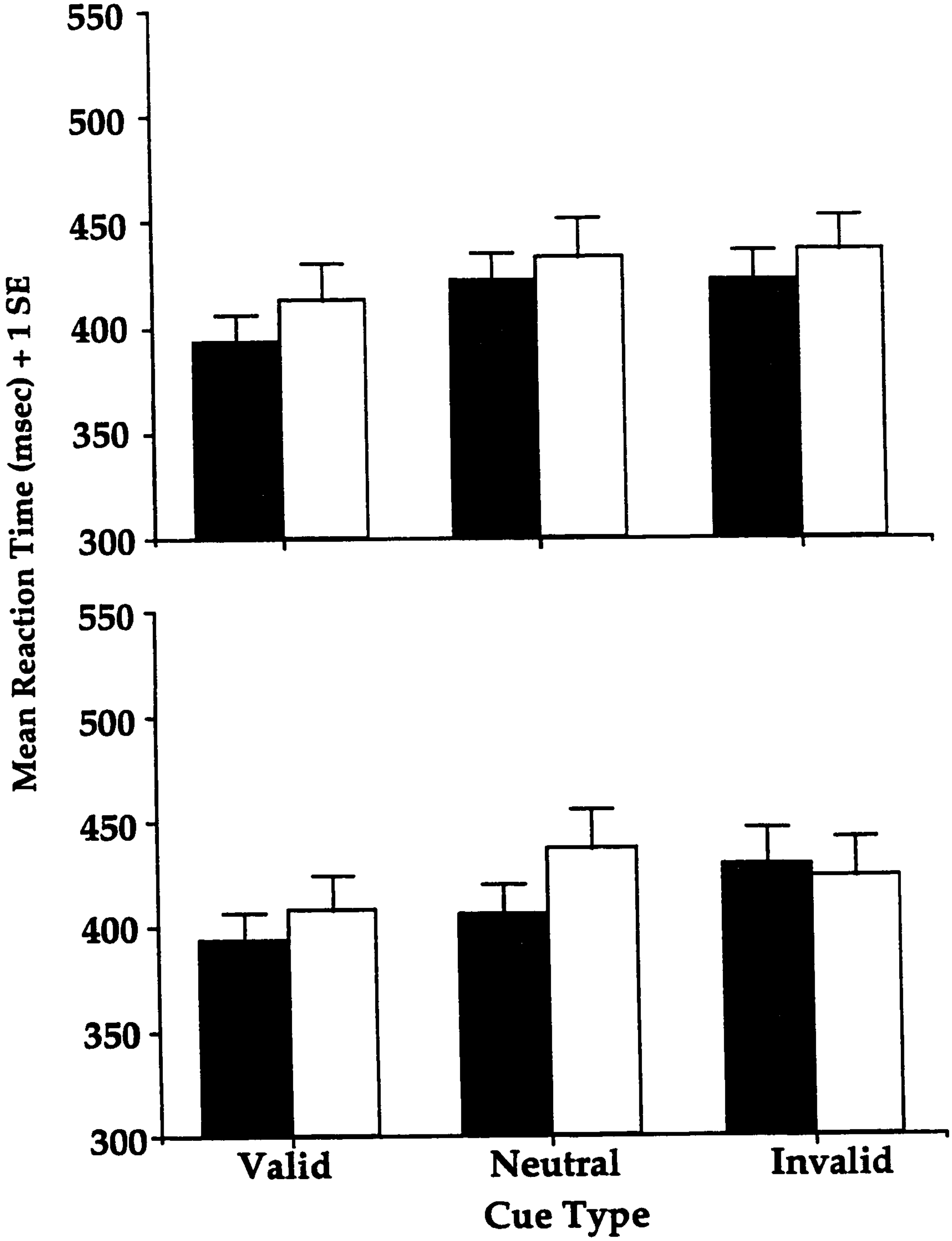

Figure 23. Mean reaction time as a function of cue type for adult participants at the 100 msec SOA. The top graph shows data for the upper visual field and bottom graph for the lower visual field. Black bars show the data for Pilot Study 1 and white bars for Pilot Study 2.

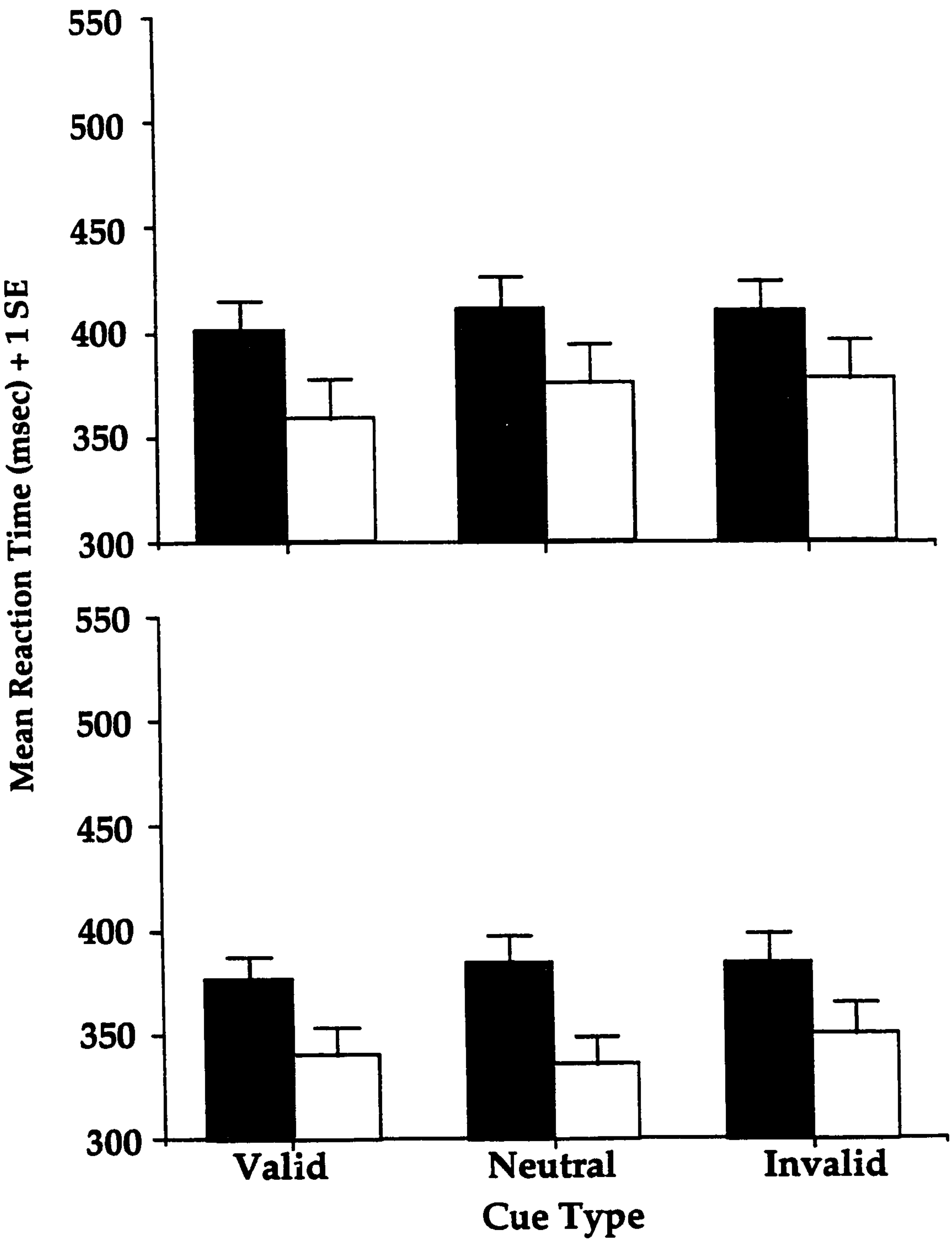

Figure 24. Mean reaction time as a function of cue type for adult participants at the 800 msec SOA. The top graph shows data for the upper visual field and bottom graph for the lower visual field. Black bars show the data for Pilot Study 1 and white bars for Pilot Study2.

Table 26.
Influence of Cue Predictiveness on Reaction Time to Targets in the Lower Visual Field.

Results of the 3-way ANOVA: Study, Cue type, SOA

Effect	*F*	*df*	*p*
Study	.44	1, 46	.51
Cue	**14.46**	**2, 92**	**< .00001**
SOA	**89.41**	**1, 46**	**< .00001**
Study X Cue	.15	2, 92	.16
Study X SOA	**21.99**	**1, 46**	**< .0001**
Cue X SOA	**4.83**	**2, 92**	**< .01**
Study X Cue X SOA	**6.67**	**2, 92**	**< .05**

Table 27.
Influence of Cue Predictiveness on Reaction Time to Targets in the Lower Visual Field at the 100 msec SOA.

Results of the 2-way ANOVA: Study, Cue type

Effect	*F*	*df*	*p*
Study	.36	1, 46	.55
Cue	**15.85**	**2, 92**	**< .00001**
Study X Cue	**7.07**	**2, 92**	**< .001**

was a significant interaction between Study and Cue type ($p < .01$). Analyses of simple effects indicated that there was a significant effect of Cue type in the data from Pilot Study 1 and in Pilot Study 2 ($ps < .001$). The bottom graph of Figure 23 illustrates that validity effects were significant in Pilot Study 1 (36.2 msec, Tukey post-test, $p < .05$) but not in Pilot Study 2 (15.6 msec). The bottom graph of Figure 24 illustrates that there was no effect of Cue type at the 800 msec SOA nor an interaction between Cue type and Study ($ps > .05$). Table 28 shows there was a main effect of Study ($p < .05$) in the ANOVA. At the 800 msec SOA, reaction times were faster for targets in the lower visual field in Pilot Study 2 than in Pilot Study 1.

Discussion

Participants did not show significant validity effects at the 800 msec SOA even when the probability of a valid trial was made to be relatively higher in Pilot Study 2. At the 100 msec SOA, in the upper visual field participants showed significant validity effects in both pilot studies. However, in the lower visual field validity effects were significant only in Pilot Study 1, in which the probability of a valid trial was lower than in Pilot Study 2. Participants in Pilot Study 2 differed from those in Pilot Study 1 on only minor accounts: they were younger (mean age = 20y 7m compared to 25y 3m); they were tested with fewer trials (240 trials compared to 288 trials = 240 trials + 48 catch trials); and they were tested mostly during the day as opposed

Table 28.
Influence of Cue Predictiveness on Reaction Time to Targets in the Lower Visual Field at the 800 msec SOA.

Results of the 2-way ANOVA: Study, Cue type

Effect	*F*	*df*	*p*
Study	**4.54**	**1, 46**	$< .05$
Cue	2.12	2, 92	.13
Study X Cue	1.47	2, 92	.24

to during the evening hours. None of these differences would be expected to influence the results.

There are probably separate reasons why participants in each study did not show significant validity effects at the 800 msec SOA. In Pilot Study 1 that included catch trials, at the 800 msec SOA the probability of a valid trial was lower than at the 100 msec SOA because of the increased likelihood that a catch trial could occur. In Pilot Study 2, I originally thought that I was making the predictive value of the cue equivalent at both SOAs by eliminating catch trials. At the 100 msec SOA, the cue predicted the location where the target would appear 80% of the time. However, at the 800 msec SOA, the time interval predicted the occurrence of a target 100% of the time, and attending to the cue provided no additional benefit because a target always appeared. A smaller and non-significant validity effect at the 800 msec SOA may reflect a waning of covert attention to the cued location with time following the cue, or an increased vigilance or preparedness because a target will always appear. Thus, in both Pilot Study 1 with catch trials and Pilot Study 2 without catch trials, it is possible that the participant's general preparedness for a target changed with increasing SOA.

In the combined analysis of Pilot Studies 1 and 2, participants were faster to respond to targets in the lower visual field (389.5 msec) than the upper visual field (396.5 msec, main effect of visual field, $p < .05$). This effect did not appear in any of the main experiments, and I can provide no explanation for it.

In conducting these pilot studies, my goal was to devise a procedure that kept the predictiveness of the cue constant across SOA, that yielded large validity effects, and that included catch trials in order to measure false positive responding. To accomplish this, in Pilot Studies 3 and 4, I included catch trials and tested with trials at each SOA presented in separate blocks.

Pilot Study 3

Purpose

The purpose of Pilot Study 3 was to evaluate validity effects in a procedure with catch trials and with the predictive value of the cue constant across SOA.

Procedure

The trials at the 100 msec and 800 msec SOAs were presented in separate blocks with the order of SOAs counterbalanced across participants. At each SOA there were 104 valid trials, 20 invalid trials, 20 neutral trials, and 8 catch trials. The cue predicted the location where the target would appear 80% of the time. The probability that the trial was a catch trial was 5%.

Results

Data were analyzed in an ANOVA with Order of blocks (start with 100 msec or 800 msec SOA) as a between-subject variable and Cue type, SOA, and

Visual field as within-subject variables. The results from the ANOVA are shown in Table 29. There was a significant two-way interaction between Cue type and Visual field as well as significant main effects of Cue and Visual field. The interaction is illustrated in Figure 25. Overall, reaction times were faster for targets in the lower visual field than the upper visual field. Analyses of simple effects also indicated a significant effect of Cue type both in the upper visual field and in the lower visual field (both $ps < .05$). In the upper visual field reaction times were faster on valid trials than on invalid trials (validity effect, 34.4 msec); faster on valid trials than on neutral trials (benefit, 19.7 msec) and faster on neutral trials than on invalid trials (cost, 14.7 msec) (Tukey post-tests all $ps < .05$). In the lower visual field reaction times were significantly faster on valid trials than on invalid trials (validity effect, 20.7 msec); faster on valid trials than on neutral trials (benefit, 25.1 msec, Tukey post-tests all $ps < .05$) but not faster on neutral trials than on invalid trials (cost, -4.4 msec).

Discussion

Pilot Study 3, with trials at the 100 msec and 800 msec SOAs presented in separate blocks, showed a significant validity effect that did not interact with SOA. These results indicate that blocking trials by SOA is an effective procedure to obtain significant validity effects at both the 100 and 800 msec SOAs. One disadvantage of the procedure used in Pilot Study 3 is that catch

Table 29.
Influence of Cue Predictiveness on Reaction Time.

Results of the 4-way ANOVA: Order, Cue type, SOA, Visual Field

Effect	*F*	*df*	*p*
Order	.54	1, 22	.47
Cue	**25.82**	**2, 44**	**< .00001**
SOA	.40	1, 22	.53
Field	**17.28**	**1, 22**	**< .001**
Order X Cue	2.22	2, 44	.12
Order X SOA	2.87	1, 22	.10
Cue X SOA	1.66	2, 44	.20
Order X Field	.19	1, 22	.67
Cue X Field	**3.69**	**2, 44**	**< .05**
SOA X Field	.01	1, 22	.97
Order X Cue X SOA	1.02	2, 44	.37
Order X Cue X Field	1.39	2, 44	.26
Order X SOA X Field	.33	1, 22	.57
Cue X SOA X Field	.05	2, 44	.95
Order X Cue X SOA X Field	2.81	2, 44	.07

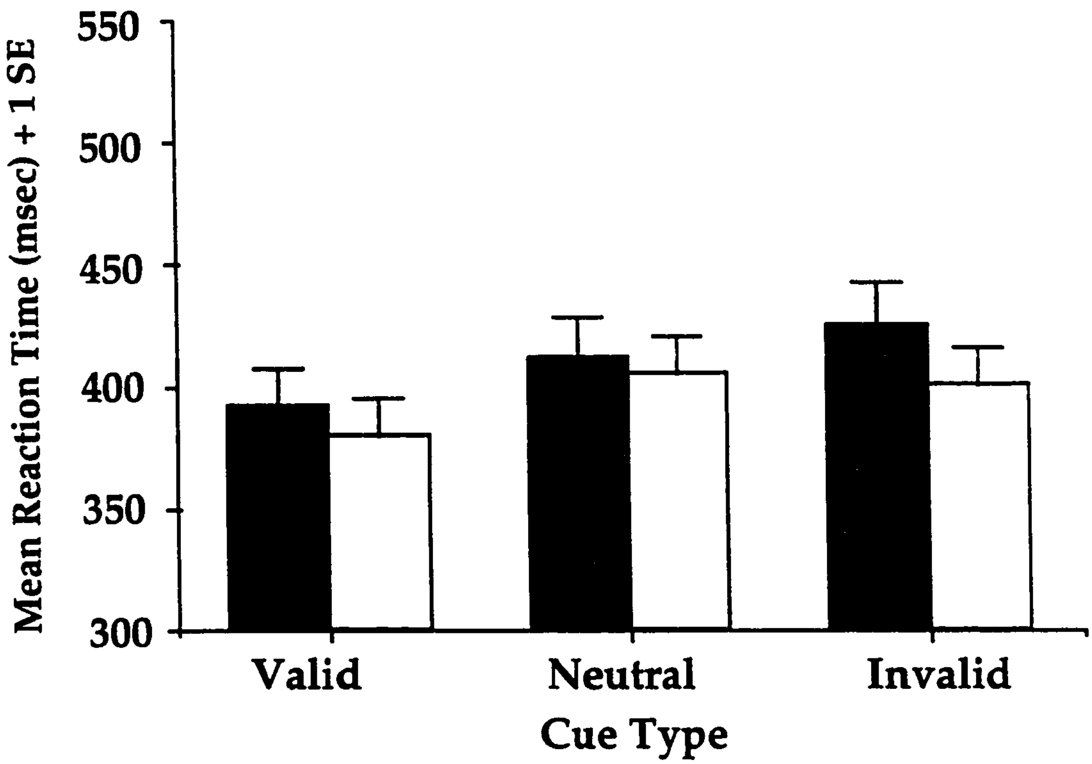

Figure 25. Adult reaction time as a function of cue type with tests of the 100 msec SOA and the 800 msec SOA blocked separately. Black bars show the data for the upper visual field and white bars show the data for the lower visual field.

trials occurred on only 5% of the trials. Since more catch trials would provide a better index of false positive responding, I increased the percentage of catch trials to 10% in Pilot Study 4.

Pilot Study 4

Purpose

The purpose of Pilot Study 4 was to evaluate validity effects in a procedure with trials at the 100 and 800 msec SOAs presented in separate blocks and with catch trials occurring on 10% of the trials.

Procedure

Adults were tested using the same procedures as described in Pilot Study 3 except that the probability that a trial was a catch trial was 10%. There were 16 catch trials. The cue predicted the location where the target appeared 75% of the time.

Results

Data were analyzed in an ANOVA with Order of blocks (start with 100 msec or 800 msec SOA) as a between-subject variable and Cue type, SOA and Visual Field, as within-subject variables. The results are shown in Table 30. There was a significant three-way interaction between Cue type, SOA, and Visual field. There was also a significant interaction between Cue type and SOA and significant main effects of Cue type and SOA (all *ps* $< .05$). The

Table 30.
Influence of Cue Predictiveness on Reaction Time to Targets at Each SOA.

Results of the 4-way ANOVA: Order, Cue type, SOA, Visual Field

Effect	*F*	*df*	*p*
Order	.26	1, 22	.61
Cue	**31.87**	**2, 44**	**< .00001**
SOA	**6.18**	**1, 22**	**< .05**
Field	3.81	1, 22	.064
Order X Cue	.70	2, 44	.50
Order X SOA	2.92	1, 22	.10
Cue X SOA	**4.80**	**2, 44**	**< .05**
Order X Field	1.03	1, 22	.32
Cue X Field	.31	2, 44	.73
SOA X Field	.15	1, 22	.70
Order X Cue X SOA	.08	2, 44	.92
Order X Cue X Field	1.12	2, 44	.33
Order X SOA X Field	.26	1, 22	.61
Cue X SOA X Field	**3.98**	**2, 44**	**< .05**
Order X Cue X SOA X Field	.53	2, 44	.59

three-way interaction between Cue type, Visual field, and SOA was analyzed by conducting separate ANOVAs for each visual field.

In the upper visual field there was a significant main effect of Cue ($p<.00001$). This result is shown in Table 31. Figure 26 (top graph) shows that reaction times on valid trials were significantly faster than on invalid trials (validity effect, 29.5 msec); reaction times on valid trials were significantly faster than on neutral trials (benefit, 16.8 msec) (Tukey post-tests, both $ps < .05$); and reaction times on invalid trials were marginally ($p = .052$) slower than on neutral trials (cost, 12.7 msec). There was also a significant main effect of SOA ($p < .05$); reaction times were slower at the 100 msec SOA (363.6 msec) than at the 800 msec SOA (342.4). The interaction between Cue type and SOA was not significant ($p > .05$).

Table 32 indicates that in the lower visual field the Cue type by SOA interaction was significant ($p<.01$). There was a significant difference in reaction times at the two SOAs on invalid trials (Tukey post-test, $p < .01$) but not on neutral or on valid trials (all $ps > .05$). This is illustrated in the bottom graph of Figure 26.

Discussion

These data indicate that when trials at the SOAs of 100 and 800 msec are blocked separately, and when catch trials account for 10% of the trials, there are significant validity effects at the two SOAs in both visual fields. In the

Table 31.
Influence of Cue Predictiveness on Reaction Time to Targets in the Upper Visual Field.

Results of the 2-way ANOVA: Cue type, SOA

Effect	*F*	*df*	*p*
Cue	**14.10**	**2, 44**	**< .00001**
SOA	**4.58**	**1, 22**	**< .05**
Cue X SOA	2.78	2, 44	.072

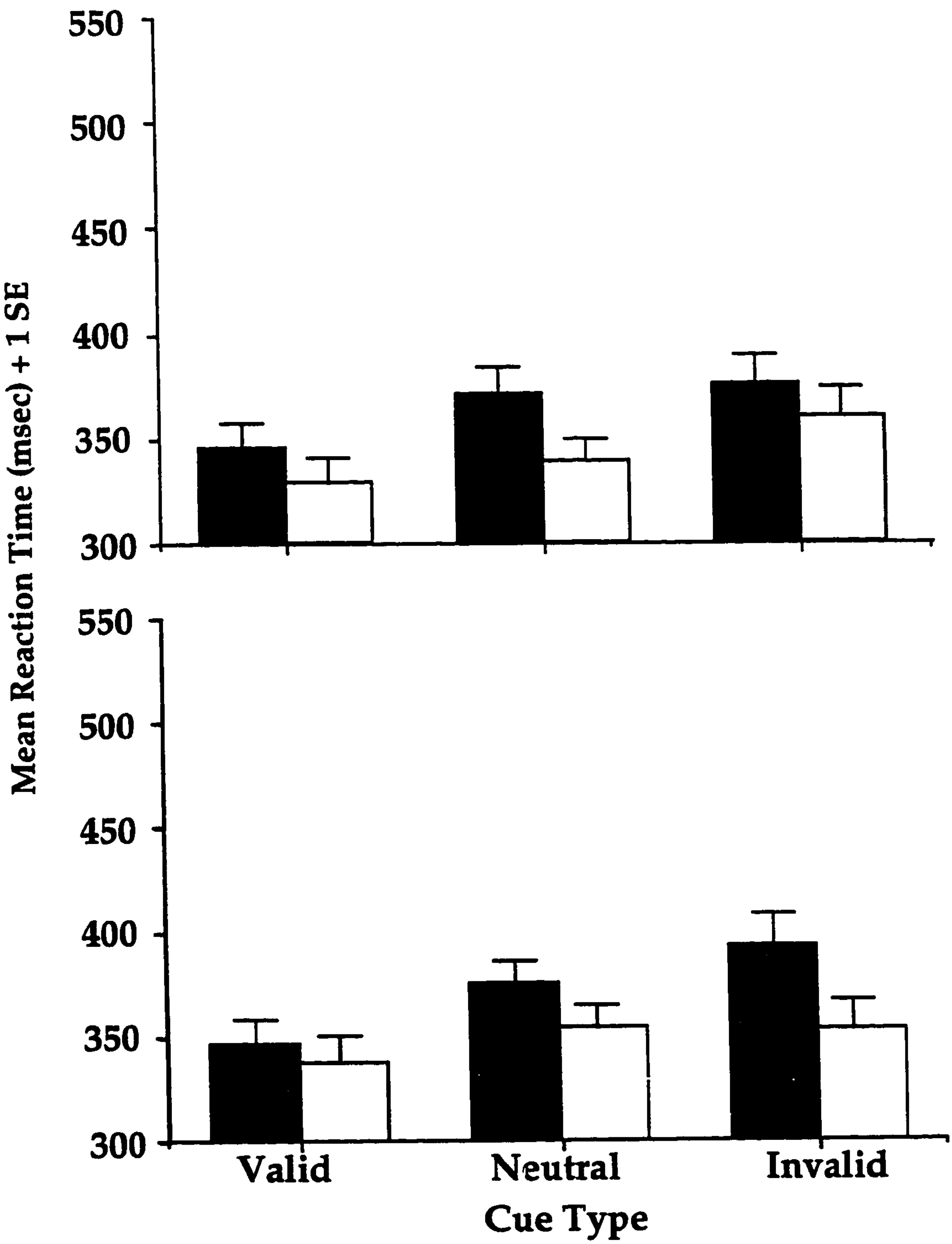

Figure 26. Adult reaction time as a function of cue type with tests of the 100 msec SOA (black bars) and the 800 msec SOAs (white bars) blocked separately. The data for the upper visual field are shown in the top graph and for the lower visual field in the bottom graph.

Table 32.
Influence of Cue Predictiveness on Reaction Time to Targets in the Lower Visual Field.

Results of the 2-way ANOVA: Cue type, SOA

Effect	*F*	*df*	*p*
Cue	**17.35**	**2, 44**	**< .00001**
SOA	**5.79**	**1, 22**	**.025**
Cue X SOA	**5.91**	**2, 44**	**.005**

lower visual field, validity effects are significant at both SOAs but larger at the 100 msec SOA than at the 800 msec SOA.

The data from Pilot Study 3 showed a significant validity effect that did not interact with SOA in both visual fields when the probability of a valid trial was .80 and that of a catch trial was .05. The data from Pilot Study 4 showed significant validity effects at both SOAs when the probability of a valid trial was .75 and that of a catch trial was .10.

Cverall Discussion of Pilot Studies 1 Through 4

This series of studies involved manipulating the methods of the task by varying the presence of catch trials and whether the SOAs were intermixed or presented in separate blocks. In Pilot Studies 1 and 2 there was no validity effect at a 800 msec SOA, perhaps because of a waning of covert attention to the cued location with time following the cue or a change in the participant's general preparedness for a target with increasing SOA. Studies 3 and 4 showed that presenting SOAs in separate blocks is an effective approach to obtain significant validity effects at the 100 and 800 msec SOAs, perhaps because it equates the predictive value of the cue across SOA. Thus, the method for administering the task in Chapters 2 through 4 involved presenting trials at each SOA in separate blocks and measuring false positive responding on 10% of the trials, as in Pilot Study 4.

Appendix B

Instructions for Detection Task

EXAMINER SAYS:

In this experiment you will see several things appear briefly on the computer screen, one after another. On the screen you now see a black diamond in the center and above and below this diamond you see black outlines of two squares.

What we want you to do is to keep looking at the center of this black diamond at all times. When you are looking in the center of the black diamond, I will begin a trial in which a white arrow will appear in the center of the black diamond.

The cue is an arrow that points up, down, or in both directions. Most of the time this cue is correct in letting you know which box will contain a checkerboard.

Your job is to press the button on the keyboard as soon as you see the checkerboard.

Sometimes there is no checkerboard. You want to be sure that you press the button only when you see the checkerboard.

COMPUTER SCREEN SHOWS:

Black diamond & black outlines of squares.

Examiner presses key on keyboard.

Cue comes on inside of the black diamond. It is an arrow that points up to the location where a checkerboard appears.

The demonstration program shows three valid trials with the arrow pointing up and the target appearing in the upper visual field; one catch trial with the arrow pointing up and no target appearing; three valid trials with the arrow pointing down and the target appearing in the lower visual field; and one catch trial with the arrow pointing down and no target appearing in either visual field.

Instructions for Detection Task

EXAMINER SAYS:

Sometimes the cue is an arrow that points in both directions. This means that the checkerboard may appear either in the top or bottom box.

Also, sometimes, there is no checkerboard following the cue. You want to be sure that you press the button only when you see the checkerboard.

Sometimes we are going to trick you. For example, the arrow may point up but the checkerboard may appear in the bottom box. When this happens just be sure to press the button as soon as you see the checkerboard.

We do not want you to move your eye up or down to look at the boxes. You should be able to see something that appears within either box without moving your eyes. Thus, because you do not know which box may contain the checkerboard, your best strategy is to keep looking right in the middle. I will be sitting next to you watching to see that you keep looking in the middle and will remind you to look back in the middle if you happen to make an eye movement.

Remember to try to keep your eye on the middle of the screen and to press the button as fast as you can as soon as you see the checkerboard.

Now lets try some practice trials.

COMPUTER SCREEN SHOWS:

An arrow pointing in both directions inside of the black diamond and a checkerboad then appears in the box in the upper visual field.

A neutral-catch trial is shown with an arrow pointing in both directions with no target appearing in either visual field.

Two invalid trials are shown: (1) the arrow points up and then the target appears in the lower visual field; (2) the arrow points down and then the target appears in the upper visual field.

Instructions for Discrimination Task with Distractors

EXAMINER SAYS:	COMPUTER SCREEN SHOWS:
In this experiment you will see different shapes on the computer screen. What shape do you see?	Black circle centered on screen.
Your job is to push this button ("v" for half of the subjects) when you see this shape.	Screen goes blank
Now what shape do you see? Push this button ("n" for half of the subjects) when you see this shape.	Black plus sign centered on screen.
	Screen goes blank
Now, what we want you to do is to keep looking at the center of this black diamond at all times.	Black diamond shape is shown.
When you are looking in the center of the black diamond, I will begin a trial in which a white arrow will appear in the center of the black diamond.	*Examiner presses key on keyboard.*
The cue is an arrow that points up, down, or in both directions. Most of the time this cue will point to a circle or a plus on the screen.	An arrow is shown inside the black diamond shape.
Your job is to press the button on the keyboard as soon as you see the plus or the circles.	Subject is shown four valid trials: half in each field; half with the target a plus flanked by plusses and half with the target a circle flanked by circles.
But be careful! Sometimes we trick you. Sometimes the arrow points the wrong way. Just keep your eye looking in the middle and try to push the correct button.	Subject is shown two invalid trials: one in each field; one with the target a plus flanked by plusses, the other a circle flanked by circles.
Sometimes the arrow points in both directions. In this case, the shapes will be either on the top or bottom part of the screen.	Subject is shown two neutral trials: one in each field; one with the target a plus flanked by plusses, the other a circle flanked by circles.

Instructions for Discrimination Task with Distractors

EXAMINER SAYS:

Now here I make the task a little harder. What you need to do is press the button that corresponds to the middle shape.

Try to press the correct button as quickly as you can without making any mistakes. Remember, we do not want you to move your eye up or down to look at the shape. Your best strategy is to keep looking right in the middle. I will be sitting next to you watching to see that you keep looking in the middle and will remind you to look back in the middle if you happen to make an eye movement.

Now lets try some practice trials.

COMPUTER SCREEN SHOWS:

Subject is shown valid trials (4) invalid trials (2) and neutral trials (2). The target shape is flanked by incompatible stimuli: plus flanked by circles; circle flanked by plusses.

Appendix C

Covert Orienting after Monocular Deprivation

One study has examined the effects of monocular deprivation on the responsivity of neurons in monkey LGN, striate cortex (area 17), and parietal cortex (area 7) (Hyvarinen, Hyvarinen, Farkkila, Carlson, & Leinonen, 1978). In that study, one monkey was deprived in both eyes for the first month of life and then the right eye opened spontaneously. The deprivation of the left eye continued for 13 more months. The results showed that neurons in parietal area 7 responded only to visual stimuli presented to the right eye. None of the 63 recording sites in parietal area 7 were influenced at all by the left eye. In contrast, recordings from area 17 showed that although most neurons were activated by visual stimulation only of the right eye, two neurons (out of 11) were mildly activated by visual stimuli presented to the left eye. Cells in LGN responded normally to stimulation of either eye. Thus, the results from this one monkey with (hybrid) monocular deprivation show that deprivation from 1 month to 14 months of age, eliminates the binocularity of parietal cells, leaving them responsive only to the non-deprived eye. Based on this evidence, I predicted that after long-term monocular deprivation (unrelieved by patching of the good eye), visual attention will be normal only when tested in the nondeprived eye.

I used the procedure described in Chapter 4 to examine whether patients with a history of monocular deprivation from birth had validity effects that were larger or smaller than normal, and whether the size of any abnormality was affected by SOA. Treatment for unilateral cataract is the same as that discussed for bilateral cataract in Chapter 4 except that parents of children treated for unilateral cataract were instructed to patch their child's good eye throughout early childhood. Depending upon the ophthalmologist involved and the year of treatment, the recommended amount of patching varied across patients from 50% to 75%. Not only was there variation in the amount of recommended patching but compliance also varied across participants. The mean number of hours per day that each patient patched from 1 week after receiving the first contact lens to age 5.0 years is shown in Table 33. Counting began 1 week after the first contact lens because parents are not told to begin patching until then. Counting stopped at age 5 because that is when tapering of patching usually begins and the prescribed amount is varied according to gains and losses in visual acuity of the deprived eye.

Patients

The sample consisted of nine deprived eyes from nine patients treated for unilateral congenital cataract (mean age = 13.9 years; range 8.6 years - 25.5 years). Table 33 shows that these patients had deprivation lasting from 2.4 months to 10.3 months (mean duration = 5.3 months). As in Chapter 4, deprived eyes were tested with their own optical correction and an add appropriate to focus the eye at the testing distance. For tests of the fellow non-deprived eye, patients were allowed to wear their normal optical correction. No additional add was given for the testing distance because the fellow eye

Table 33. Clinical Details at the Time of Covert Orienting Test for the 9 Children Treated for Unilateral Congenital Cataract.

Name (Age)	Refraction	Age of Diagnosis (months)	Months (days) of Deprivation	Snellen Acuity	Sheridan Gardiner Acuity	Mean # hrs/ day Patched
K. A.	OD plano	-	-	6/9	6/6	-
(8.1)	OS +11.00	0.9	2.4 (73)	6/18	6/24	4.0
J. B.	OD plano	-	-	6/6	6/4	-
(9.8)	OS +12.25	0.9	2.5 (76)	6/54	6/60	1.8
A. T.	OD plano	-	-	6/6	6/6	-
(10.1)	OS +17.50	4.9	8.0 (245)	6/60	6/36	3.0
A. M.	OD +19.50	3.0	4.2 (129)	CF @ 6m	6/115	3.8
(10.3)	OS plano	-	-	6/9	6/9	-
B. A.	OD +13.00	4.9	8.5 (259)	6/21	6/9	3.0
(11.4)	OS -3.50	-	-	6/6	6/4	-
N. F.	OD +13.50	1.5	4.1 (124)	6/18	6/36	5.0
(13.7)	OS plano	-	-	6/6	6/6	-
A. M.	OD +0.50	2.9	10.3 (313)	CF @ 6m	6/144	0.1
(16.7)	OS -4.00	-	-	6/6	6/4	-
D. C.	OD -4.50	-	-	6/4.5	6/9	-
(17.5)	OS +11.25	0.0	3.2 (98)	6/15	6/24	5.4
J. W.	OD plano	-	-	6/9	6/6	-
(25.5)	OS +12.50	3.5	4.9 (150)	6/15	6/18	3.0

OD = Right eye OS = Left eye CF= Counting fingers could accommodate. The fellow non-deprived eye was tested if it had an acuity of at least 6/7.5 with appropriate optical correction. Six non-deprived eyes were included in the final sample. None had any visual complications. The remaining three were not included because acuity with appropriate optical correction was worse than 6/7.5 (two patients) or they refused further testing (one patient).

Data Analyses

I decided to compare the deprived eyes to healthy eyes from normal participants rather than to fellow nondeprived eyes. This makes the analyses consistent with those I did for bilateral cases in Chapter 4, and eliminates the influence of any abnormality in the good eye caused by patching.

Results

Patients demonstrated on the post-tests that they understood the meaning of the cues (e.g., "helper," "fooler," or "no clue") with an average score of 9 correct responses out of 10 (range 7 - 10), during which they made eye movements on fewer than 1 out of the 10 trials (mean = 0.7, range = 0 -2). They also demonstrated an ability to discriminate between 1 or 2 boxes in the periphery. All patients performed perfectly with a score of 10 correct out of 10, and made eye movements on fewer than 1 out of the 10 trials (mean = 0.7, range = 0 - 2). Performance on the experimental trials is shown in Table 9. Patients treated for unilateral cataract made eye movements, responses on catch trials, and anticipations of the target no more frequently than normal participants.

As in Chapter 4, mean reaction times were calculated with outliers removed using a moving criterion based upon the number of trials per condition (Van Selst & Jolicoeur, 1994). The percentage of data eliminated was 3.32% for eyes treated for unilateral cataract compared to 3.11% for normal control participants presented in Chapter 3. I conducted an ANOVA on the validity effect, the difference in reaction time on invalid and valid trials, with one between-subject factor, Group (Unilaterals, Normals) and two within-subject factors: SOA (100, 400, 800 msec) and Visual Field (down, up). The results from the ANOVA are shown in Table 34.

Figure 27 illustrates that there was no difference in the size of the validity effect between patients treated for unilateral cataract and normal participants at any SOA (no significant main effect of group, nor any interaction between group and SOA). In this small sample of patients treated for unilateral congenital cataract, there was no evidence of deficits in covert orienting.

Given the results for patients treated for bilateral congenital cataract in Chapter 4, I wanted to examine the effects of the duration of monocular deprivation on performance. However, there were an insufficient number of participants available to provide enough data to control simultaneously for patching compliance and duration of deprivation. Instead, I used separate

Table 34.
Covert Orienting after Monocular Deprivation.

Results of the 3-way ANOVA on Validity effects: Group by SOA by Visual Field

Effect	*F*	*df*	*p*
Group	.04	1,79	.84
SOA	.47	2,158	.63
Visual Field	2.70	1,79	.10
Group X Visual Field	.32	1,79	.57
Group X SOA	.06	2,158	.94
SOA X Visual Field	1.67	2,158	.19
Group X SOA X Visual Field	1.50	2,158	.21

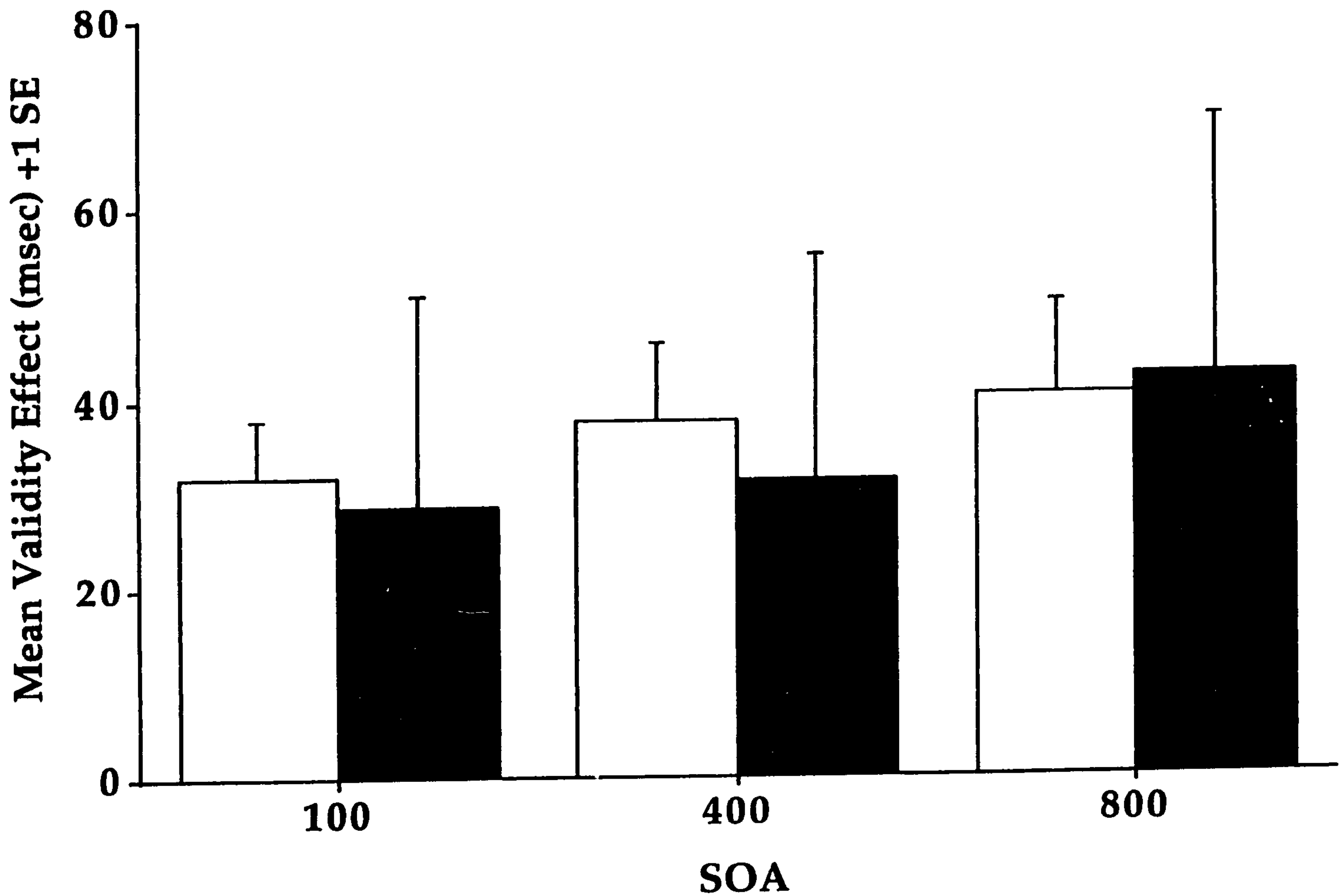

Figure 27. Validity effect as a function of SOA for Normals (white bars) and for Patients Treated for Monocular Deprvation (black bars).

Pearson correlations to examine the relationship between the size of the validity effect at each SOA and (1) the duration of deprivation and (2) hours of patching. There was no significant correlation between the duration of deprivation and the size of the validity effect at any SOA (all *ps*> .10). If I split the patients at four months as I did for patients treated for bilateral cataract (Chapter 4), neither patients treated for longer (n = 6) nor shorter (n = 3) deprivation differ from normal controls in the size of the validity effect (ANOVA, all ps > .10). In addition, there was no significant correlation between hours of patching and the size of the validity effect at any SOA (*ps*> .10).

Discussion

Unlike patients treated for bilateral cataracts after 4 months of age (Chapter 4), patients treated for unilateral cataract showed no deficits compared to normal children in the ability to shift attention covertly. However, the sample was small and there were too few subjects to examine statistically the influence of patching compliance and of duration of deprivation.

My results do not support the hypothesis that visual attention is abnormal when tested in the deprived eye. It is possible that my results can be attributed to differences between my sample and the monocularly deprived monkey described by Hyvarinen et al. (1978). The patients in my sample had shorter periods of deprivation and had periods of forced usage of the deprived eye because of patching of the non-deprived eye.

Close scrutiny of clinical records of the patients treated for unilateral congenital cataract indicates that many of them have a history of strabismus, nystagmus, as well as the poor acuity documented in Table 33. The implications of this are that the deficits in children treated for bilateral deprivation (see Chapter 4) are likely to have been caused by their history of deprivation, rather than by strabismus, nystagmus, or poor acuity.

In summary, these findings suggest that under these conditions patients treated for monocular deprivation have no deficits in the ability to shift attention covertly. In the next section, I report results in patients treated for monocular deprivation on the ability to simultaneously shift attention covertly and ignore distracting information.

The Effects of Monocular Deprivation on the Abilities to Shift Attention Covertly and to Ignore Distracting Information

I used the procedure described in Chapter 5 to examine whether patients with a history of monocular deprivation from birth are impaired in the abilities to shift attention covertly and ignore distractors. I predicted that in a more complex task, there might be deficits when tested in the deprived eye and no deficits when tested in the non-deprived eye.

Participants

The sample consisted of seven deprived eyes of seven patients treated for unilateral congenital cataract (mean age = 14.7 years; range 8.6 - 25.9 years). Table 35 shows that the deprivation in these patients lasted from 2.4 months to 10.3 months (mean duration = 5.9 months). Seven eyes of seven age-matched normal controls (+/- 3 months in age) were tested for comparison. Information about the age-matched normal controls is provided in the bottom half of Table 3.

Patients performed well on the post-tests: They demonstrated an understanding of the meaning of the cues (e.g., "helper," "fooler," or "no clue") with an average score of 9.7 out of 10 trials and few eye movements (mean = 0.3, range = 0 - 2). Patients also demonstrated an ability to discriminate the target when it was flanked by compatible and incompatible distractors. They were able to discriminate the target correctly on 10 out of 10

Table 35. Clinical Details at the Time of the Distractor Test for the 7 Children Treated for Unilateral Congenital Cataract.

Name (Age)	Refraction	Age of Diagnosis (months)	Months (days) of Deprivation	Snellen Acuity	Sheridan Gardiner Acuity	Mean # hrs/ day Patched
K. A.	OD plano	-	-	6/9	6/6	-
(8.6)	OS +11.00	0.9	2.4 (73)	6/18	6/18	4.0
A. M.	OD +19.50	3.0	4.2 (129)	CF @ 6m	6/115	3.3
(10.8)	OS plano	-	-	6/9	6/6	-
B. A.	OD +13.00	4.9	8.5 (259)	6/21	6/9	3.0
(11.4)	OS -3.50	-	-	6/6	6/4	-
A. T.	OD plano	-	-	6/6	6/6	-
(10.75)	OS +17.50	4.9	8.0 (245)	6/60	6/18	3.0
A. M.	OD +0.50	2.9	10.3 (313)	CF @ 6m	6/202	0.1
(17.3)	OS -4.00	-	-	6/6	6/4	-
D.C.	OD -4.50	-	-	6/4.5	6/6	-
(18.1)	OS +11.25	0.0	3.2 (98)	6/15	6/18	5.4
J. W.	OD plano	-	-	6/9	6/6	-
(25.9)	OS +12.50	3.5	4.9 (150)	6/15	6/18	3.0

OD = Right eye OS = Left eye CF= Counting fingers post-test trials during which there were few eye movements (mean = 0.3, range = 0 - 2). Table 12 shows that patients treated for unilateral cataract were able to perform the main task with eye movements and incorrect responses as infrequent as in normal 10-year-old and adult participants and age-matched controls.

Mean reaction times were calculated after outliers were eliminated with a moving criterion adjusted relative to the number of trials per condition (Van Selst & Jolicoeur, 1994). The percentage of data eliminated with the outlier elimination procedure was 2.70% for patients treated for unilateral cataract, and 2.88% for age-matched control participants.

To evaluate whether patients treated for monocular deprivation differed from normal age-mates in the influence of distractors on covert orienting, I conducted an ANOVA on mean reaction times for valid, invalid, and neutral trials under the compatible and incompatible distractor conditions. The ANOVA had 4 within-subject factors: Group (aphakic, age-matched normals), Distractor type (compatible, incompatible), Cue type (invalid, neutral, valid) and Visual field (lower, upper). Because of the small sample, these results are considered preliminary.

Effect of Group. The results in Table 36 show that there was no main effect of Group and that Group did not interact with any factor (*p*s > .10). This finding is illustrated in Figure 28. It indicates that there was no evidence after

Table 36.
Effect of Unilateral Deprivation on the Ability to Orient Attention Covertly and Ignore Distractors Compared to Age-Matched Control Participants.

Results of the 4-way ANOVA: Group, Distractor type, Cue type, Visual field

Effect	*F*	*df*	*p*
Group	.26	1,6	.63
Distractor type	**34.37**	**1,6**	**< .01**
Cue type	**12.93**	**2,12**	**< .001**
Visual Field	**49.67**	**1,6**	**< .001**
Group X Distractor type	.80	1,6	.40
Group X Cue type	2.12	2,12	.16
Distractor type X Cue type	.32	2,12	.73
Group X Visual Field	.24	1,6	.64
Distractor type X Visual Field	1.91	1,6	.22
Cue type X Visual Field	3.01	2,12	.09
Group X Distractor X Cue type	.37	2,12	.69
Group X Distractor type X Field	.04	1,6	.85
Group X Cue X Visual Field	.08	2,12	.92
Distractor type X Cue type X Field	.28	2,12	.76
Group X Distractor X Cue X Field	1.28	2,12	.31

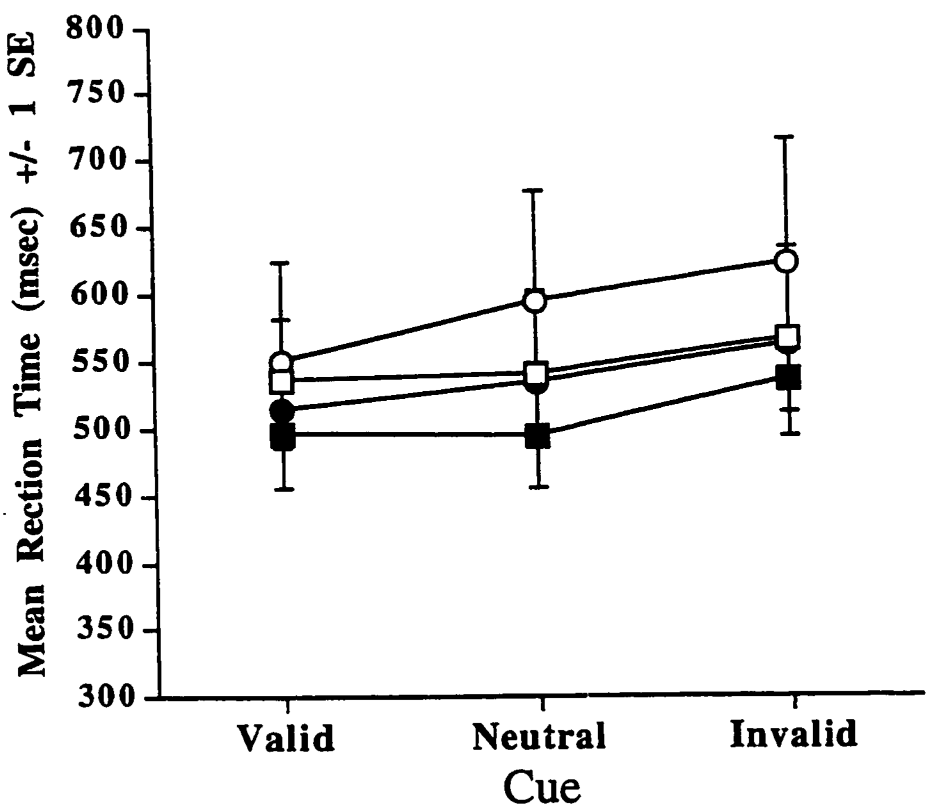

Figure 28. Mean reaction time as a function of cue type on trials with incompatible distractors (open symbols) and compatible distractors (filled symbols) for patients treated for monocular deprivation (squares) and age-matched control participants (circles). Means are connected to facilitate comparisons of the effects of the cue.

monocular deprivation of any differences from normal in the ability to orient attention covertly and to ignore distractors.

Effect of Distractor. There was a main effect of Distractor (p = .001). Across all conditions, reaction times were significantly slower on trials with incompatible distractors than on trials with compatible distractors. Distractor did not interact with any factor (ps > .10).

Effect of Cue. There was a main effect of Cue (p = .001). Across all conditions, reaction times were significantly slower on invalid trials than on neutral trials and valid trials, and significantly faster on valid trials than on neutral trials (Tukey post-tests, all ps < .01). Cue did not interact with any factor (all ps > .10).

Effect of Visual Field. There was a main effect of visual field (p = .001). Reaction times were faster to targets in the upper visual field than in the lower visual field.

There were no significant correlations between the duration of deprivation and the size of the validity effect or filtering effect (Pearson correlation, both ps > .10). In addition, there were no significant correlations between patching compliance and the size of the validity effect or filtering effect (Pearson correlations, both ps > .10).

Discussion

I had predicted that in a more complex task, there might be deficits in visual attention when tested in the deprived eye and no deficits when tested in the non-deprived eye. However, the results showed no evidence in the deprived eye of any differences from normal in the ability to orient attention covertly and to ignore distractors. It is possible that I did not find deficits because my small sample of patients had relatively short periods of deprivation (less than 10.3 months) and had often patched the non-deprived eye, thus encouraging use of the deprived eye.

The results from this study are preliminary because of the small number of patients. They suggest that the abilities to shift attention covertly and to ignore distractors on this task may be normal after treatment for unilateral congenital cataract. Close scrutiny of clinical records of the patients treated for monocular deprivation indicates that many of them have a history of strabismus, nystagmus, as well as the poor acuity documented in Table 35. The implications of this are that the deficits in children treated for bilateral deprivation (see Chapter 5) may be related to their history of binocular deprivation and not to the associated conditions of strabismus, nystagmus, or poor acuity.

Appendix D

Pilot Studies for a Task to Measure the Ability to Ignore Distractors.

In order to finalize a procedure by which to study the ability to ignore distracting stimuli, I conducted several pilot studies with adult participants. These studies involved variations in whether the target (a plus or a circle) was presented alone; flanked by incompatible stimuli (plus flanked by circles, or circle flanked by pluses); flanked by irrelevant stimuli (plus flanked by squares, or circle flanked by squares); or flanked by compatible stimuli (plus flanked by pluses, or circle flanked by circles). The results of these pilot studies are based on medians rather than on the mean outlier elimination procedure used in the final experiment in Chapter 5.

<u>Method 1</u>

Adults (n = 24) were tested on a covert orienting task (see Chapter 2) with distractors flanking the target. On each trial (n = 300), one of two targets (plus sign or cross) was presented: alone (n = 100), surrounded by irrelevant distractors (n = 100), or surrounded by incompatible distractors (n = 100), 400 msec after a valid, invalid, or neutral cue.

<u>Results</u>

I conducted an ANOVA with Cue type, Distractor type, and Visual field as within-subject factors. All of the main effects, the two-way interactions, and the three-way interaction between Cue type, Distractor type, and Visual field were statistically significant ($ps < .001$). To analyze the three-way interaction, separate ANOVAs were conducted for each visual field.

In the upper visual field, participants were faster to discriminate the target when it was flanked by incompatible or irrelevant distractors compared to when it was presented alone (Main effect of Distractor, Tukey post-tests, *ps* $< .05$). The interaction between Distractor type and Cue type was not significant. Participants were slower to respond on invalid trials than on valid or neutral trials (Main effect of Cue type, Tukey post-tests, *ps* $< .05$); reaction times on valid and neutral trials did not differ from each other.

In the lower visual field, the Cue by Distractor type interaction was significant ($p < .001$). Distractors had no effect on reaction times on valid trials. On invalid trials, they increased reaction times only when they were irrelevant (Tukey post-test, $p < .05$). On neutral trials, they created an odd pattern: reaction times were longer when there were incompatible distractors or no distractors than when there were irrelevant distractors (Tukey post-tests, *ps* $< .05$).

Based on the literature I expected incompatible distractors to slow participants' reaction times to the targets more than irrelevant distractors and when the target was presented alone, and to have the greatest effect on reaction time on invalid trials. A possible explanation for the results is that on incompatible trials, participants were provided with added information about the target shape. For example, on incompatible trials, flanking circles mean that the target is a plus, and hence participants were able to discriminate the target more quickly when it was flanked by incompatible distractors than when it was flanked by irrelevant distractors or presented alone.

Method 2

The target alone condition was omitted and was replaced by a compatible condition, such as a plus flanked by plusses. This change was made because previously published results on differences between target alone and distractor conditions are inconsistent (Enns & Akhtar, 1989; Eriksen & Eriksen, 1974). One adult participant was tested with 600 trials. On each trial, one of two targets (plus sign or cross) was presented surrounded by compatible distractors (n = 200), irrelevant distractors (n = 200), or incompatible distractors (n = 200), 400 msec after a valid, invalid, or neutral cue.

Results

The median correct reaction time was calculated for each condition. To evaluate the effects of the cue, the mean reaction time on valid, invalid, and neutral trials was calculated, collapsed across the other conditions. Reaction times were 31 msec slower on invalid trials than on valid trials. To evaluate the effects of the distractors, the mean reaction time on trials with compatible, incompatible, and irrelevant distractors was calculated, collapsed across the other cueing conditions. Reaction times were 4 msec slower on trials with incompatible distractors than on trials with compatible distractors, and 6 msec slower on trials with irrelevant distractors than on trials with compatible distractors. These results show that under these testing conditions, a paradigm including incompatible distractors, compatible distractors, and irrelevant distractors does not produce robust differences between types of distractors.

Method 3

Because I cannot be sure that the irrelevant condition will provide a good baseline against which to compare the incompatible and compatible conditions, I removed the irrelevant condition from the paradigm. Adults (n = 4) were tested with 600 trials. On each trial, one of two targets (plus sign or cross) was presented surrounded by incompatible distractors (n= 300) or compatible distractors (n = 300), 400 msec after a valid, invalid, or neutral cue.

Results

The median correct reaction time was calculated for each condition. Reaction times were 52 msec slower on invalid trials than on valid trials. Participants were 33 msec slower on trials with incompatible distractors than on trials with compatible distractors. These pilot results show that participants were slowed more by invalid cueing than by valid cueing; they were also slowed more on trials with incompatible distractors than on trials with compatible distractors. These findings suggest that this method was an appropriate one to use to examine covert shifts of attention and the ability to ignore distractors in normal participants and patients treated for cataract. This was the method adopted in Chapter 5.

Appendix E

Measurements of Timing Accuracy

Because the experiments in this thesis depend on reaction time, before testing subjects, I determined the accuracy of the reaction times collected by the hardware and software I planned to use. I judged their accuracy in measuring time between two arbitrary events: the presentation of a stimulus on the computer screen and a button press. I measured these events independently with a Hewlett Packard (HP) 5302A 50 MHz universal counter (with an accuracy of 1 msec $\pm$ 0.2 msec) and with SuperLab software running on a Macintosh IIcx (Pilot Studies 1 and 2) or on a Powerbook 160 (Pilot Studies 3 and 4 and Chapters 2 through 5).

The Hewlett Packard (HP) 5302A 50 MHz universal counter was activated when a photo-transistor taped to the center of the computer screen detected the appearance of a target (a small white square) in the center of the computer screen. Timers on the Macintosh IIcx and the Powerbook 160 were activated by a button press on their respective keyboards that also controlled the presentation of the target on the screen.

The timers stopped when I pressed a button on a button box (powered by a 9V battery), that was wired to the contacts of the letter "B" on the keyboard. This is the same key on the keyboard that subjects press during the experiments in this thesis.

I compared reaction times collected on (1) the HP timer versus the Macintosh IIcx (n = 100 comparisons) and (2) the HP timer versus the Powerbook 160 (n = 100 comparisons).

Results of Timing (msec) for a Macintosh IIcx

	MEAN	SD	SE
SuperLab	516.1	134.5	14.1
HP timer	486.1	133.8	14.0
Difference Scores	29.99	8.1	.85

Correlation $r=.998$ $r^2=.996$

Standard error of estimate = $S_y\,(1-r^2)^{1/2} = 133.8\,(1-.996^2)^{1/2} = 8.24$

The means and SDs for the two timing instruments indicate that timing based on the SuperLab software on the Macintosh IIcx is slower on average by 30msec compared to the HP timer. It is likely that SuperLab starts its timer sooner and/or stops its timer later than the HP counter. The variances of the two instruments are nearly identical. The correlation between the two sets of measurements was strong ($r^2 = .996$).

Results of Timing (msec) for a Powerbook 160:

	MEAN (msec)	SD (msec)	SE (msec)
SuperLab	500.3	225.6	22.6
HP timer	504.7	225.5	22.5
Difference Scores	-4.37	6.8	.68

Correlation $r=.999$ $r^2=.999$
Standard error of estimate = $S_y (1-r^2)^{1/2} = 225.5 (1-.9991)^{1/2} = 6.77$

A comparison of the timing measurements from the HP timer and the timing based on SuperLab software on the Powerbook indicates that SuperLab on the Powerbook is faster on average by 4.37 msec. It is possible that the photo-transistor picks up the stimulus on the screen and starts the HP counter 4 msec after SuperLab begins its timer.

The variances of the two instruments are nearly identical. The correlation between the two sets of measurements was strong ($r^2 = .999$). The conclusion is that there is tremendous accuracy with SuperLab when run off the Powerbook.

In summary, the measurement of reaction times are different for the two different computers. Reaction times were approximately 30 msec slower when SuperLab software was run off of the Macintosh IIcx than off of the Powerbook 160. This finding has some implications for the pilot studies reported in Appendix A. Pilot Studies 1 and 2 were run with the Macintosh IIcx and Pilot Studies 3 and 4 were run with the Powerbook 160. Thus, reaction times in the first two pilot studies have a constant error of 30 msec. The experiments reported in Chapters 2 through 5 were run with the Powerbook 160, which agreed almost perfectly with independent measurements taken by the HP timer.

Appendix F

My Ability to Judge Central Fixation and Eye Movements Off-Center

For both of the tasks in this dissertation, I needed to be able to (1) judge that the participant was fixating centrally before the start of each trial and (2) detect whether the participant made an eye movement. Only trials on which there was no eye movement could be used in the reaction time analyses. Therefore, I measured my accuracy for each type of judgment.

Method

On a transparency, I drew 13 horizontal lines spaced so that the separation between them would be one-half of a degree of visual angle when viewed from a distance of 36 cm. I taped the transparency to the computer screen so that the middle line was centered on the central stimulus shown at the beginning of each trial. A research assistant sat 36 cm from the screen, and I sat at her side, similar to the procedure during all of the testing described in this thesis.

Procedure

To test my ability to judge if fixation is central, the research assistant fixated centrally or 1 degree, 2 degrees, or 3 degrees above or below the center of the central stimulus. I was not informed of to the location of her fixation. I reported whether I judged fixation to be central, 1 degree, 2 degrees, or 3 degrees above center or 1 degree, 2 degrees, or 3 degrees below center. Five measures were collected at each of the seven locations, in a random order.

To test the smallest eye movement that I could detect, the research assistant either fixated centrally or made an eye movement 0.5 degree, 1 degree, 1.5 degrees, 2 degrees, 2.5 degrees, or 3 degrees above or below the center of the central stimulus. I was not informed as to whether an eye movement had occurred and if so, to its size and direction. I had to report whether an eye movement occurred, the size of the eye movement, and its direction. Five measurements were collected for each of the 13 conditions presented, in a random order.

Results

I was 100% accurate in judging whether the fixation was center, up, or down, and accurate 88% of the time in judging which of the six locations off center was being fixated. I detected all of the eye movements off center, and judged their direction and size correctly 92% of the time.

Discussion

The results show that I was perfect at judging whether the participant was fixating centrally or whether she was fixating off-center by as little as one degree. I was also able to detect all vertical eye movements that were at least 0.5 degrees. Thus, my measurements of covert shifts of attention are not likely to have been contaminated by trials with overt shifts of 0.5 degrees or more.

Appendix G

Lighting Measurements

I conducted luminance measurements with a Tetronix photometer (model J16) to make sure that the lighting conditions were matched as closely as possible in the testing situations at McMaster University, The Hospital for Sick Children and in patients' homes. I first conducted the measurements at McMaster University because that is where I collected the pilot data and first tested normal participants. I took measures of the luminance across a sheet of gray photographic paper placed in front of the computer monitor in our testing room at McMaster University. I took measures of the same sheet of paper in our testing room at The Hospital for Sick Children. I made every attempt to match the lighting conditions at The Hospital for Sick Children to that at McMaster University (e.g., by setting the position of the computer monitor, drawing the shades, adding overhead light). I made measurements at a number of different positions in the room and determined which position and lighting conditions were closest to those at McMaster University.

Measures taken across gray paper using a Tetronix J16 photometer with a wide angle 8 degree luminance probe (model J6503):

(n= 25 measures – 5 in each of the four corners and in the center of the paper)

	McMaster University	Hospital for Sick Children (HSC)
Mean =	49.2 cd/m^2	49.9 cd/m^2
Range =	45.5 - 53.3 cd/m^2	46.5 - 54.5 cd/m^2
SD =	2.4	1.8

I also took measures at McMaster University and The Hospital for Sick Children of the stimuli against the background of the computer screen and calculated the percent contrast defined as 100 x (light - dark/ light + dark).

Measures with a Tetronix photometer with a narrow angle 1 degree luminance probe (model J6523)

	McMaster	HSC
• Percent contrast of central fixation stimulus	67.7%	60.0%
Luminance of screen with no stimuli present	46.7 cd/m^2	35.4 cd/m^2
Luminance of central fixation stimulus	9.0 cd/m^2	8.9 cd/m^2
• Percent contrast of black box in periphery	58.0%	46.2%
Luminance of screen inside outline of black box	44.5 cd/m^2	32.7 cd/m^2
Luminance of black outline of box	12.2 cd/m^2	12.1 cd/m^2
• Percent contrast black and white checkerboard	59.0%	na
Luminance of white check	46.7 cd/m^2	na
Luminance of black check	12.2 cd/m^2	na

Eight children treated for bilateral congenital cataract were tested in their homes. Four children participated in the Detection Task in Chapter 4 and seven children participated in the Discrimination Task in Chapter 5. A Minolta Spotmeter F was used to measure the contrast of the stimuli on the screen in each home. The testing conditions in the homes were not always optimal. As needed, I added light by using a desk lamp or took away light by hanging a black sheet over the windows. I wanted to be sure that the contrast was similar to that at The Hospital for Sick Children. Although the luminance differed between the hospital and the home environments, the contrast of the stimuli on the screen was within the same range.

<u>Measures on home visits with Minolta Spotmeter F</u>

• Percent contrast of central fixation stimulus	45.3% - 76.4%
Luminance of screen with no stimuli present	20.7 - 33.7 cd/m^2
Luminance of the central fixation stimulus	4.5 - 7.8 cd/m^2

The luminance and contrast measurements indicate that differences between McMaster University and The Hospital for Sick Children are too small to account for any differences between patients and normals. There were no systematic differences between patients tested at home versus The Hospital for Sick Children that might suggest that the reduced luminance and contrast in the home environment would contribute to impaired performance of the patients on some measures.