THE PERIPHERAL VISION OF NORMAL CHILDREN AND OF CHILDREN TREATED FOR CATARACTS

By

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A Thesis

Submitted to the School of Graduate Studies

in Partial Fulfillment of the Requirements

for the Degree

Doctor of Philosophy

McMaster University

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THE PERIPHERAL VISION OF NORMAL AND DEPRIVED CHILDREN

DOCTOR OF PHILOSOPHY (1992)
(Psychology)

McMASTER UNIVERSITY
Hamilton, Ontario

TITLE: The peripheral vision of normal children and of children treated for cataracts

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NUMBER OF PAGES: xiii, 394

I assessed the development of peripheral vision and the effects thereon of deprivation. First, I examined the visual field in 28 5-year-olds, 24 6-year-olds, 20 7-year-olds, and 16 adults using kinetic perimetry with the Goldmann perimeter and a 6.4' light of either 31.8 cd/m^2 or 318 cd/m^2. Five-year-olds saw as far superiorly as did the group of older subjects. But even at 7 years of age, children did not see as far temporally or inferiorly as did adults. When the 5-year-olds were retested at 7 years, their fields had grown and were as large as those of the original 7-year-olds. I also examined sensitivity across the field in 20 7-year-olds, 20 8-year-olds, 12 9-year-olds, and 20 adults using static perimetry with the Octopus perimeter and a 25.8' light which varied in intensity from 0 to 1000 asb. Sensitivity in the near, mid, and far periphery was adultlike by 7 years of age, with later development at 0° centrally.

Children treated for dense and central cataracts were assessed identically except that the deprived eye was focused for the testing distance by a contact lens. Each deprived eye exhibited a restricted field, especially temporally, and a less sensitive field, especially at 0° centrally. More restricted fields were found for children with longer than with shorter deprivation, except superiorly, and for children with monocular than with binocular deprivation, at least for tests with the dim light. Less sensitive fields at 0° centrally were found for children with developmental than with congenital deprivation. Finally, only children with unilateral congenital deprivation exhibited larger losses at 20° nasally than at 30° temporally. Control

experiments indicated that the losses of peripheral vision could be explained only partly by optical factors.

The results indicate that the development of peripheral vision continues throughout childhood, at least for tests using a small stimulus. Deprivation affects peripheral vision, especially those parts of the field slowest to develop (e.g., the temporal field, 0° centrally, and the near nasal field). It is likely that the underlying pathways are not mature until after 5 years of age.

Acknowledgements

I wish to acknowledge the contribution of several people to the development of and subsequent completion of my doctoral dissertation. First, I appreciate the many hours spent by Dr. Daphne Maurer, my supervisor, in working with me. Her belief in the importance of research and her scholarly approach to it both challenged and encouraged me. I also thank Dr. Terri Lewis for her enthusiastic interest in all aspects of my education. Several members of faculty also provided much valuable assistance. I thank Dr. Lorraine Allan and Dr. Ron Racine, my committee members. I also acknowledge Dr. Craig Hawryshyn and Dr. John Platt.

I am grateful for the efforts of my many subjects and of their parents, and especially for the many hours so willingly provided by J.W., one of my visually deprived subjects. I thank Dr. Henry Brent for his helpful insights concerning the medical aspects of my work. I also appreciate the efforts of Peggy Brent who taught me how to carry out perimetry and Bob Barclay who provided some of the equipment for my work. For the use of their facilities, I thank the Departments of Ophthalmology at The Hospital for Sick Children, St. Joseph's Hospital, and McMaster University Medical Centre. I also acknowledge the financial support of NSERC and OGS.

I am most grateful for the encouragement of many individuals. I appreciate the support of the students in my first year class, especially that of Maeve Walsh and Robb Smith. I also appreciate the encouragement of other friends, such as Rochelle Cole, Tony Comeau, John Keith, Sylvie Malenfant, Marianna Stark, Paul Wheaton, Micheline Wylde, Peter Wyngaarden and, most especially, Rodney Boyd. I also appreciate the kindness shown to me by Audrey and Walter Pilon.

Finally, I express my gratitude to my family. I acknowledge the wisdom and support of my parents Winnie and William Bowering and my brother Kevin Bowering. I appreciate the encouragement from my grandmother Beatrice Wheaton and my uncle Harry Wheaton. And I would be remiss if I did not acknowledge Bismarck. Finally, I express my gratitude to David Pilon for his kindness and encouragement.

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"The Eye altering alters all."

William Blake, The Mental Traveller

Peripheral vision is the individual's ability to detect objects other than those at the point of foveal fixation. Once detected with peripheral vision, the eyes can saccade towards the object, thus allowing detailed inspection. For several reasons, peripheral vision is an important ability to assess in humans.

This dissertation investigated the normal development of peripheral vision in children and the influence thereon of visual deprivation from a dense and central cataract. Using standard perimetric techniques, I first assessed the normal development of both the extent of the visual field and of sensitivity across the field. Then, I examined how visual deprivation from cataracts affected these behaviors. In particular, I evaluated how the timing and the duration of deprivation and whether it was monocular or binocular altered peripheral vision. These variables are likely to affect the peripheral vision of deprived humans given their effects in deprived cats and monkeys.

This research examines two theoretical issues of interest in psychology. The first issue is a basic question in developmental psychology: What is the role of experience in development? The second issue is physiological: What is the relationship between brain

and behavior?

With regard to the first issue, I will examine the relationship between the rate of development of peripheral vision and the effects thereon of deprivation. To do so, it is necessary to understand how normal development proceeds. That is, is peripheral vision an ability which is well developed early in infancy or does it mature slowly over an extended time period? Although there exists a wealth of information regarding the development of peripheral vision during infancy (reviewed in Maurer & Lewis, 1991), the data from children are scant and contradictory, with reports ranging from tunnel vision at 6 years of age to adult-like fields at 5 years (Lakowski & Aspinall, 1969; Tomonaga, 1974).

Once the normal development of peripheral vision is understood, we then are ready to assess how abnormal early experience might alter this development. Much of what is known about the effects of abnormal visual experience comes from studies of animals deprived of normal visual experience through lid suture. The lid sutured eye then is reopened and the visual capacity of the deprived eye assessed. From the animal literature, it appears that visual deprivation affects seriously those abilities that are relatively immature shortly after birth but affects little those abilities which are relatively mature early in life. For example, visual acuity is slow to develop and affected seriously by early deprivation in cats and monkeys (Boothe, 1981; Boothe, Dobson, & Teller, 1985; Giffin & Mitchell, 1978). In contrast, the discrimination of form and of the

orientation of stimuli are relatively mature soon after birth and affected little by early deprivation (Dodwell, Wilkinson, & von Granau, 1983; Loop & Sherman, 1977; Riesen, 1982; van Hof-van Duin, 1976).

Similarly, in humans the consequences of visual deprivation from dense and central cataracts can be related to the normal pattern of development. A cataract is an opacity of the crystalline lens of the eye. Treatment requires removal of the crystalline lens and fitting of the eye with an optical correction, which restores nearly normal visual input. A cataract in humans is analogous to lid suture in animals because both types of deprivation prevent the input of patterned stimulation to the retina. In humans, just as in cats and monkeys, visual acuity is slow to develop and is affected adversely by visual deprivation while the perception of form is early to mature and unaffected by early deprivation (Maurer, Lewis, & Brent, 1989). Peripheral vision, like visual acuity, develops slowly and may be affected by early deprivation. This dissertation, then, provides another test of the relationship between the rate of development and the effects of deprivation.

A study of the development of peripheral vision in humans also may provide hints about the maturity of underlying neural pathways. In the cat, the development of peripheral vision (Sireteanu & Maurer, 1982) and its physiological underpinnings (Sherman, 1974c, 1977b) are studied. Although we cannot examine directly the physiological maturity of these neural pathways in

humans, it is possible to make inferences about their development. For example, by studying the normal development of peripheral vision in children, we hopefully could infer the maturity of underlying pathways. By assessing the peripheral vision of children treated for a cataract, we can make guesses about which pathways are plastic and thus susceptible to visual deprivation. In summary, this research may provide further information about how behavior is mediated by the brain.

In Experiments 1, 2, and 3, I examine the normal development of both the extent of the visual field and of sensitivity across the visual field. In Experiments 4 and 5, I describe the effects of visual deprivation on peripheral vision. In particular, these experiments were designed to evaluate how development might be affected by the timing and the duration of deprivation and whether the deprivation was monocular or binocular. Experiment 6 evaluates the possible influence of optical factors upon peripheral vision in individuals treated for cataracts. The first chapter provides an introduction to the relevant literature while the final chapter evaluates the findings and discusses the physiological basis for the behaviors shown.

CHAPTER 1

PREVIOUS FINDINGS ON THE DEVELOPMENT OF PERIPHERAL VISION

This chapter begins with a discussion of the techniques used in studying peripheral vision in babies and in older subjects and of the strengths and weaknesses of each method when used to test children. The chapter then reviews what is known about the development of peripheral vision in humans and in cats. The following section is about the effects of abnormal early experience on development in humans, cats, and monkeys. The chapter closes with a discussion of the implications of these previous findings for research on the development of peripheral vision in normal children and in children treated for dense and central cataracts.

Techniques of study of peripheral vision in children

There are two techniques for assessing peripheral vision: kinetic perimetry, which estimates the extent of the visual field, and static perimetry, which measures sensitivity across the visual field and the extent of the visual field. Both techniques require the subject to look at a central fixation target during the presentation of the peripheral stimulus. In studies of babies, the dependent measure

typically is the infant's first eye movement from center (e.g., Lewis, Maurer, & Kay, 1978; Harris & MacFarlane, 1974; Schwartz, Dobson, Sandstrom, & van Hof-van Duin, 1987). However, sometimes the observer makes a forced choice judgment of the location of the stimulus on the basis of any behavioral cues from the baby (Finlay, Quinn, & Ivinskis, 1982; Schneck, Hamer, Packer, & Teller, 1984). In studies of children, the subject typically is asked to indicate detection by tapping on the surface of the table or by pushing a buzzer (Allan, 1971; Aspinall, 1976; Liao, 1973), although sometimes he[1] is asked to look toward the stimulus when he detects it (Cummings, van Hof-van Duin, Mayer, Hansen, & Fulton, 1988; Whiteside, 1976).

The difference between the techniques of kinetic and static perimetry lies in the mode of presentation of the peripheral target. As the name implies, in kinetic perimetry a stimulus is <u>moved</u> slowly from outside the subject's field of view toward the center of the visual field. At some location in the visual field, the subject indicates that he is aware of the stimulus. The stimulus then is introduced at a new meridian outside the subject's field of view and the extent of the field for that meridian is measured. This technique has been employed to measure the extent of the visual field under a variety of stimulus conditions in infants (Heersema, van Hof-van Duin, & Hop, 1989; Luna, Dobson, Carpenter, & Biglan, 1989; Mohn & van Hof-van

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[1] Throughout this dissertation, I will use the generic form (he or his) to refer to male and female subjects.

Duin, 1986; Schwartz et al., 1987; van Hof-van Duin & Mohn, 1987) and in children (Allan, 1971; Cummings et al., 1988; Liao, 1973; Matsuo, Endo, Yokoi, & Tomonaga, 1974; Suzumura, Furuno, & Matsuo, 1985; Tomonaga, 1974).

In contrast, static perimetry is defined as the presentation of a nonmoving stimulus at different eccentricities within the visual field. The intensity of the stimulus, or its location, are varied over trials until the subject's threshold, or extent of the field, is obtained. Static perimetry has been used with infants (Aslin & Salapatek, 1975; de Schonen, McKenzie, Maury, & Bresson, 1978; Finlay et al., 1982; Guez, 1978; Harris & MacFarlane, 1974; Lewis, Maurer, & Blackburn, 1985; MacFarlane, Harris, & Barnes, 1976; Maurer & Lewis, 1991; Schneck et al., 1984; Tronick, 1972) and with children (Allan, 1971; Aspinall, 1967, 1976; Cummings et al., 1988; Lakowski & Aspinall, 1969; Liao, 1973; Matsuo et al., 1974; Tomonaga, 1974; Whiteside, 1976). In summary, kinetic perimetry measures the extent of the visual field with a moving target while static perimetry, with a static stimulus, assesses thresholds across the field or the extent of the field.

The advantage of kinetic perimetry is that it is a relatively quick procedure, requiring as little as one trial to measure the extent of the visual field along a meridian. Kinetic perimetry has several potential disadvantages when used to test infants (reviewed in Maurer & Lewis, 1991) which will not be discussed given that this dissertation is about tests with children. In contrast to infants,

because of children's better developed cognitive abilities, concentration, and cooperation, kinetic perimetry is a suitable technique to assess the extent of the visual field.

The main advantage of static perimetry is that it can assess sensitivity across the field and also indicate the extent of the field. Thus, static perimetry can provide more information about the peripheral vision of the subject than does kinetic perimetry. The disadvantage of static perimetry is that it can be a lengthy procedure if thresholds are measured at a number of locations across the visual field.

In summary, either static or kinetic perimetry are sensible choices for use with children, with the technique chosen dependent upon what information is needed.

Development of peripheral vision in infants

Although my dissertation is about the development of peripheral vision in children, in this section I will summarize what is known from infants. The study of infants is important for two reasons. First, it allows us to examine how normal development proceeds and to make guesses about the peripheral vision of older children. Second, it allows relation of the effects of deprivation to the normal pattern of development of peripheral vision.

There exist many studies of the peripheral vision of young infants. Studies agree that initially babies detect stimuli only near the center of the field and, with increasing age, they are able to detect the same stimulus presented further in the periphery along the horizontal, superior, and inferior meridia (Aslin & Salapatek, 1975; Finlay et al., 1982; Harris & MacFarlane, 1974; MacFarlane et al., 1976; Maurer & Lewis, 1991; Mohn & van Hof-van Duin, 1986; Tronick, 1972; van Hof-van Duin & Mohn, 1987; but see Schwartz et al., 1987). For example, 1- and 2-month-olds, when tested with stimuli 4° or larger, have a smaller field for all meridia (vertical, diagonal, and horizontal) than do adults (Aslin & Salapatek, 1975; Schwartz et al., 1987). The measured visual field increases until, at 12 months, the upper field is adult-like when subjects are tested with a 6° stimulus (Mohn & van Hof-van Duin, 1986). The remaining meridia are not fully developed until after this age (Heersema et al., 1989).

Although studies agree that development proceeds from the center of the field toward the periphery, studies disagree about the absolute size of the visual field at each age. For example, Harris and MacFarlane (1974) used static perimetry with a 3° light of 137 cd/m^2 and found that the field expanded horizontally from 25° at birth to 35° at 7 weeks. In contrast, Tronick (1972) used static perimetry with a brightly colored, rotating, 6° X 5° X 2° object and reported that babies from 2 until 6 weeks of age did not indicate

detection of the stimulus when it was more than 15° to the side. Why do these studies disagree?

It is likely that studies disagree upon the absolute size of the field at each age because of differences in methodology. Studies differed in the type of perimetry used (e.g., kinetic or static perimetry), characteristics of the peripheral stimulus (e.g., intensity, size, contrast with the background, distance of the stimulus from the baby, whether or not the target flashed) and whether there was a simultaneous competing activity (e.g., non-nutritive sucking by the infant).

First, as discussed earlier, the technique used may influence the measured extent of the field. Kinetic perimetry may lead to underestimation in the extent of the field if young infants are slow to indicate detection of the peripheral stimulus (e.g., Mohn & van Hof-van Duin, 1986; Schwartz et al., 1987). However, both kinetic and static perimetry may overestimate the field if appropriate controls for spontaneous eye movements are not used. Typically, babies with a high rate of spontaneous looking have been eliminated (Mohn & van Hof-van Duin, 1986; Schwartz et al., 1987) or blank control trials, during which no peripheral stimulus is presented, have been added to allow measurement of such eye movements (Aslin & Salapatek, 1975; Lewis et al., 1985; Maurer & Lewis, 1991). Note, however, that not all studies have used appropriate controls, which may lead to errors in the measured size of the field (Tronick, 1972).

Second, the characteristics of the stimulus, such as its intensity or size, have been shown to affect the measured extent of the visual field. For example, Schneck et al. (1984) reported that 4-week-olds detect a 3.1° light as easily as a 17° light out to an eccentricity of 36° if the smaller light is proportionally more intense than the larger stimulus. As well, Guez (1978) found that infants 2 to 5 months of age are able to detect a 1° light at 40° in the periphery if the light is sufficiently intense. The size of the stimulus is important also (Maurer & Lewis, 1991). When 1-month-olds are tested monocularly with a flashing 3° or 6° light, they give no evidence of detecting the 3° light even just 15° from the center of the visual field, but when tested with the 6° light, they orient toward it significantly at all locations tested between 45° in the temporal field (e.g., the right visual field for the right eye) to 30° in the nasal field (e.g., the left visual field for the right eye).

Other factors such as target distance, contrast, and the movement of the stimulus may affect the measured extent of the visual field. de Schonen et al. (1978) have shown that the measured extent of the visual field of 2- and 3-month-olds, but not 4- and 5-month-olds, is reduced when the central target is close to the baby (30 cm) while the peripheral target is far away (90 cm). As well, contrast affects the extent of the field: Infants generally detect a 6° white ball further to the side when it is against a dark background, providing high contrast, than the same stimulus against a lighter

background, which affords less contrast (Mohn & van Hof-van Duin, 1986; van Hof-van Duin & Mohn, 1987). In addition, the extent of the field may be affected by whether the stimulus flashes. In the only study to examine this issue, Tronick (1972) reported that the measured visual field is smallest in infants 2 to 10 weeks of age when the central light not only remains on but also flashes while the peripheral light is stationary. However, if the central light is stationary while the peripheral light flashes, then the competitive effects of the central stimulus are removed - in this condition, fields are largest. Note, however, that there were methodological problems with this study which weaken the conclusions. For example, Tronick used a small sample which weakens the generality of his conclusions. Also, the dependent measure may not have been the infant's first eye movement to the periphery. Rather, the baby may have scanned the visual field and thereby detected the stimulus by chance. As well, there were no blank control trials to evaluate the effects of spontaneous eye movements.

Third, the measured extent of the visual field may vary with the presence of a competing activity, such as non-nutritive sucking by the baby. For example, MacFarlane et al. (1976) found that 5-week-old infants tested with a 3° flashing light orient further in the periphery if they are not sucking on a pacifier. Sucking on a pacifier may have influenced measurement in three ways. First, sucking may have been distracting to the infant and interfered with detection.

Second, the baby's sucking may have affected his motivational state such that even if the baby detected the target, he may not have been motivated to orient to it. Third, sucking may have increased the latency of the infant's eye movements to the peripheral stimulus. In this experiment, once the peripheral stimulus was presented, infants were allowed only 5 seconds in which to indicate detection. If sucking increased the latency of an eye movement, then 5 seconds may not have been sufficient response time for the baby.

To date, most studies have tested infants under binocular viewing conditions (Aslin & Salapatek, 1975; de Schonen et al., 1978; Guez, 1978; Harris & MacFarlane, 1974; MacFarlane et al., 1976; Schneck et al., 1984; Schwartz et al., 1987; Tronick, 1972). Studies using monocular viewing conditions provide information not only about the gradual expansion of the visual field but also about the development of detection in the nasal visual field (e.g., the left visual field for the right eye) versus the temporal visual field (e.g., the right visual field for the right eye).

The few monocular studies (Finlay et al., 1982; Lewis et al., 1985; Maurer & Lewis, 1991; Mohn & van Hof-van Duin, 1986; van Hof-van Duin & Mohn, 1987) suggest that detection in the temporal visual field precedes detection in the nasal visual field early in infancy. For example, the data from van Hof-van Duin and Mohn (1987) suggest that the extent of the nasal field of infants 1 to 5 weeks of age tested with a 6° stimulus expands more slowly than

does the extent of the temporal field. From 2 months onwards, there also is a slower rate of development of the nasal versus the temporal fields (Heersema et al., 1989; Mohn & van Hof-van Duin, 1986). As well, Finlay et al. (1982) reported that 3-month-olds can detect a bar out to at least 45° in the temporal visual field, the farthest position tested, but only out to 20° in the nasal visual field. However, methodological problems make this study difficult to interpret (see Maurer & Lewis, 1991). Also, two studies by Lewis and Maurer indicate that during early infancy the rate of development of detection in the temporal field is faster than the rate of development of detection in the nasal field (Lewis et al., 1985; Maurer & Lewis, 1991).

In the first study, Lewis et al. (1985) measured the ability of 1- and 2-month-old infants to detect lines 25.6° long but of varying widths located at 20° nasally and 30° temporally. One-month-olds detected the narrowest line shown them, a line only 1.5° wide, at 30° in the temporal field but showed no evidence of detecting a line even 12.8° wide at 20° in the nasal field. This indicates, then, at least an 8-fold difference in sensitivity at 30° temporally versus 20° nasally for 1-month-olds. In contrast, 2-month-olds detected a line 0.75° wide at 20° nasally but gave no evidence of detecting the same line at 30° temporally. Better sensitivity at 20° in the nasal field than at 30° in the temporal field is the pattern shown by normal adults (Allan,

1971; Aspinall, 1967; Fahle & Schmid, 1988; Frisen & Glansholm, 1975; Lakowski & Aspinall, 1969; Liao, 1973).

In the second study, Maurer and Lewis (1991) traced the development of peripheral vision in 1- to 6-month-olds tested with a 3° flashing light presented along the horizontal meridian. At one month of age, babies gave no evidence of detecting the stimulus anywhere in the field, even at 15°, the closest location from center. At older ages, the babies oriented further and further towards the periphery until at 6 months of age the fields were approximately adult-like. Moreover, babies were more likely to orient to stimuli in the temporal field than toward the same object in the nasal field. For example, 2-month-olds oriented out to 30° temporally but did not indicate detection of the same stimulus even at 15° nasally. Three-month-olds oriented out to 60° temporally and no farther than 15° nasally. Together, the studies of Lewis and Maurer suggest that during the first few months, babies are much more sensitive to stimuli in the temporal visual field than in the nasal field.

Overall, then, the work with infants indicates that there are two patterns of development. First, the visual field expands with age. However, the absolute limit of the measured visual field at any age is affected by the manner in which the baby is tested e.g., stimulus size, intensity, or the presence of a competing activity. If the light is large and flashing, then even 6-month-olds have approximately adult-like visual fields. However, from the work with babies, it is unknown

whether adult-like visual fields would be found with a small nonflashing light. Second, the rate of development of the temporal visual field is faster than the rate for the nasal visual field in early infancy.

Development of peripheral vision in children

Four studies (Cummings et al., 1988; Matsuo et al., 1974; Tomonaga, 1974; Whiteside, 1976) have assessed the peripheral vision of children using a fairly large stimulus (from 10' to 1.9°). Two of the studies required the subject to indicate detection by looking toward the target (Cummings et al., 1988; Whiteside, 1976) while the other two studies do not report the behavioral response required of the subject (Matsuo et al., 1974; Tomonaga, 1974).

Tomonaga (1974) presented lights varying in size from 10' to 1.9° both statically and kinetically and found full visual fields at 5 years of age. These data also were reported in the article by Matsuo et al. (1974). Whiteside (1976) presented a 1° light statically and found adult-like fields at 6 years, the youngest age tested. Cummings et al. (1988) presented smaller lights (26' or 42') statically or kinetically and found that the size of the visual field of a small group of children from just under 3 years to 5 years of age was adult-like only for the larger target. Overall, these results indicate that the measured extent of the visual field is affected by the size of the

stimulus used in testing and that the visual field continues to develop until early childhood, under some testing conditions.

Most studies of older children have used much smaller stimuli so as to avoid the possibility that large stimuli would be so far above threshold that they would obscure any subtle differences in ability between children and adults. The studies which have tested children with a small stimulus (i.e., 6') have discrepant results with reports ranging from full fields at 5 years of age (Tomonaga, 1974) to tunnel vision at 6 years (Lakowski & Aspinall, 1969). Lakowski and Aspinall (1969; Aspinall, 1967) tested 33 Scottish children, 6 to 11 years of age, by presenting a 6.4' light statically at various locations along the horizontal meridian of the Goldmann perimeter and asking the child to respond whenever he detected the light. (The Goldmann perimeter is a standard ophthalmological tool used to measure visual fields. For a further description, see Experiment 1.) The field for the only 6-year-old tested extended out to only 15°, and not until 11 years of age was the field nearly adult-like. In an unpublished study, Allan (1971) moved a 6.4' light in from the side of the Goldmann perimeter and asked the child to indicate detection of the target. Allan found that the extent of the field is already nearly adult-like at 5 to 6 years of age, except for a 9° restriction in the temporal visual field (relative to an adult field of approximately 75°). All other studies of young children which used a light of the same size (e.g., 6') were conducted in Japan (Liao, 1973; Matsuo et al., 1974; Suzumura et al., 1985;

Tomonaga, 1974). Like Allan (1971), these investigators, using either kinetic or static perimetry, found fairly broad fields in 5- to 6-year-olds. (A possible exception to this statement is the study conducted by Suzumura et al. (1985). In this study, it is difficult to determine when the fields are adult-like as the results from children in the first decade of life were combined.)

Differences in results between Lakowski and Aspinall (1969) and other studies are not explainable on the basis of the behavioral response required of the subject. For example, some studies required that once the subject detected the peripheral stimulus, he look towards it (Cummings et al., 1988; Whiteside, 1976) while other studies required the subject to maintain central fixation and to indicate detection via tapping (Allan, 1971; Lakowski & Aspinall, 1969; Liao, 1973). Note that studies requiring children to tap reported both small (Lakowski & Aspinall, 1969) and nearly adult-like fields (Allan, 1971). Nor do racial differences (Japanese versus Caucasian children) account for the differences in peripheral vision, as they do for the oblique effect (Annis & Frost, 1973). Studies of Caucasian children have reported both large (Allan, 1971) and small (Lakowski & Aspinall, 1969) fields, as have studies of Japanese children (Liao, 1973; Tomonaga, 1974). Possibly, Lakowski and Aspinall (1969) found smaller visual fields than did other investigators because Lakowski and Aspinall used a small sample of children, which limits the generality of the findings. In summary,

none of the differences across the described studies, with the exception of the small sample used by Lakowski and Aspinall (1969), are likely to contribute to the difference in results between Lakowski and Aspinall (1969) versus other reports.

From the literature, then, it appears that the visual field becomes adult-like before early childhood when subjects are tested with a sufficiently large stimulus. However, it is uncertain when the visual field becomes adult-like when subjects are tested with a small stimulus. One goal of this research was to assess the visual fields of children with visual pathology using small stimuli so as to detect any differences in function between abnormal and normal eyes. Given that the patients were in the controversial age range, it therefore was important to establish normative standards for large groups of children tested identically to the children with visual pathology.

Moreover, there has been no report of interocular differences. It is necessary to know the size of interocular differences in normal children in order to evaluate whether any difference in the peripheral vision between a deprived eye and the fellow nondeprived eye of a patient constitutes a real difference. Also, assessment of interocular differences in the visual field may be used to trace the relative rate of development of each hemisphere (for further discussion of this point, see Experiment 1).

In addition to interocular differences, assessment of the reliability of findings from young children is important to establish

when these data will be used for making diagnostic decisions about patients. To date, there have been only two reports of the reliability of measurements from young children (Cummings et al., 1988; Liao, 1973). Cummings et al. (1988) claimed good test-retest reliability but note that their sample was composed of only a few children with not all subjects successfully completing both test sessions. Liao (1973) also reported good test-retest reliability but, like Cummings et al. (1988), the measurement of reliability was based on only a few of his original sample of children. Another goal of my dissertation, then, was to assess reliability in a much larger sample of children at a variety of ages.

In order to develop normative standards for tests with small stimuli and to assess reliability and interocular differences, I have examined the development of the extent of the field using kinetic perimetry on the Goldmann perimeter (see Chapter 2). To examine the development of sensitivity across the visual field, I have used static perimetry on the Octopus perimeter (see Chapter 2). Both approaches are necessary to describe thoroughly the development of peripheral vision: kinetic perimetry to describe the extent of the visual field and static perimetry to describe thresholds across the visual field. Moreover, because kinetic perimetry is a quicker procedure and requires less cognitive sophistication of the subject than does static perimetry, kinetic perimetry can be used to test a younger population than can static perimetry. This is important in

tracing the development of peripheral vision and for identifying abnormalities in even young children with visual pathology.

Effects of visual deprivation on peripheral vision in humans

The assessed patients were children treated for a dense and central cataract, which is an opacity of the crystalline lens of the eye. A cataract, which is analogous to lid suturing in animals, allows examination of the effects of visual deprivation upon humans. This section will provide a brief background about cataracts, then discuss the scant literature on the effects of a dense and central cataract on the development of peripheral vision, and, finally, indicate how my experiments complemented previous work.

The crystalline lens is onion-like, composed of concentric layers of lens fibers enclosed by a clear membranous capsule. The innermost fibers of the lens form a compact area referred to as the nucleus while the outer fibers are much softer and form the cortex of the lens. Any agent which interferes with the transparency of the cells will result in some form of cataract (Scheie & Albert, 1969). Although there is variability in the morphology of cataracts, any cataract diagnosed as dense and central will limit the input of patterned stimulation onto the retina. Note, however, that some light potentially may travel through the periphery of the crystalline lens and reach the retina. Treatment involves removal of the crystalline

lens, rendering the eye aphakic, and fitting of the eye with optical correction, which provides nearly normal visual input. Such input is said to be only nearly normal because the eye is focussed by the optical correction at only one distance and can no longer accommodate to objects at other distances. Because of the lack of accommodation, visual deprivation is said to continue even after treatment but to be less severe than if the eye had been left untreated. To explain potential abnormalities of the visual field, then, we must consider not only the influence of the initial deprivation from the dense and central cataract but also the contribution of the subsequent aphakia. This issue will be addressed further in Chapter 3.

It is common, but erroneous, lore that cataracts only occur in individuals past middle age for cataracts also may be present at birth or develop during infancy or early childhood. In fact, the incidence of congenital cataracts is reported as one in 250 newborns (Chace, Merritt, & Bellows, 1950), although this figure may vary according to epidemics of maternal rubella. Although there are multiple causes for a cataract during infancy, in a significant proportion of cases the etiology is unknown (Merin & Crawford, 1971). Between 8.3 and 16.0% of all congenital cataracts are hereditary (Falls, 1943; Merin & Crawford, 1971), being transmitted typically via autosomal dominant or recessive inheritance but sometimes via sex-linked inheritance. The cataract may appear as an isolated defect, with the individual

otherwise normal, as occurs in galactokinase deficiency, or it may be part of a group of anomalies, as occurs in galactosemia, Marfan's Syndrome, and Down's Syndrome. Cataracts also may be nonhereditary. Prenatal causes of cataract include maternal exposure to rubella, toxoplasmosis, or other infectious diseases while perinatal and postnatal causes include, but are not limited to, retrolental fibroplasia, hypoglycemia, hypocalcemia, irradiation, chronic drug usage (e.g., steroids), or a traumatic eye injury. Of nonhereditary congenital cataracts, the most frequent cause is rubella, accounting for 20% of cases (Merin & Crawford, 1971). With sufficiently early diagnosis and treatment, some types of cataracts are preventable, e.g., the cataracts which occur in galactosemia or galactokinase deficiency may be avoided if galactose is eliminated from the young infant's diet (Gardner & Shoch, 1987; Merin, 1986).

There exists very little information regarding the effect of a dense and central cataract on the development of peripheral vision. Kinetic perimetry has been used to study the extent of the visual field in only two patients (Mohn & van Hof-van Duin, 1983; Moran & Gordon, 1982). The first patient was treated for a unilateral congenital cataract at 19 years of age and tested with a 1° light of 17 cd/m^2. The results showed that the temporal, superior, and inferior fields were approximately normal in extent but the nasal field in the deprived eye was not, extending only out to 20° instead of to the normal 60° (Moran & Gordon, 1982). The second patient was a 4-

year-old child binocularly deprived from birth who had been operated upon but never optically corrected, not even for the test of the visual field (Mohn & van Hof-van Duin, 1983). The child was tested with large white balls mounted on black sticks (STYCAR balls) and exhibited losses in the nasal field but not elsewhere. The amount of the loss of the nasal field was not provided in the article. It was surprising that the patient's visual fields were as normal as reported, especially given the lack of optical correction. Possibly, the large test stimuli may have masked some of the possible losses in the patient's visual fields.

Static perimetry has been used to test sensitivity along the horizontal meridian of several patients treated for dense and central cataracts (Lewis, Maurer, & Brent, 1986; Maurer, Lewis, & Brent, 1983; Mioche & Perenin, 1986; Moran & Gordon, 1982). Two patients monocularly deprived from birth to either 5 months or 19 years, and tested with either a 6.25' or a 1° light, respectively, exhibited decreased sensitivity in the deprived eye compared to the nondeprived eye, especially in the nasal visual field (Maurer et al., 1983; Moran & Gordon, 1982). In contrast, the results are different from four children who had normal visual experience for the first year and then were monocularly or binocularly deprived for less than 6.5 months. Although their deprived eyes exhibited lower sensitivity than did normal eyes, the deprived eyes did not have greater losses in the nasal visual field compared to the temporal

visual field (Lewis et al., 1986; Maurer et al., 1983). Similar findings have been reported for two binocularly deprived patients, one subject deprived from birth to 11 months and the other from 3 years of age to shortly thereafter. These patients, when tested with a 27' light, exhibited uniformly decreased sensitivity along the horizontal meridian within the central 60°, with the amount of loss similar across subjects (Mioche & Perenin, 1986). In summary, the results from the few patients tested indicate that, relative to a normal eye, the deprived eye has decreased sensitivity along the horizontal visual field and, in subjects monocularly deprived from birth, there is especially poor sensitivity in the nasal visual field (Lewis et al., 1986; Maurer et al., 1983; Mioche & Perenin, 1986; Moran & Gordon, 1982).

Although early deprivation from cataract affected peripheral vision in every one of the 11 deprived eyes tested, because of the few eyes assessed, it is difficult to speculate on the effects of the timing and the duration of deprivation and whether it was monocular or binocular. These variables are likely to be important. Studies of monocularly deprived monkeys indicate that earlier onset or longer deprivation cause larger losses in the visual field than do later onset or shorter deprivation (Hendrickson, Boles, & McLean, 1977; Joseph & Casagrande, 1980; Sparks, Mays, Gurski, & Hickey, 1986; Wilson, Lavallee, Joosse, Hendrickson, Boothe, & Harwerth, 1989). Similarly, studies of dark reared cats and of monocularly

deprived cats indicate that longer deprivation or earlier onset of deprivation lead to more restricted fields, respectively (Bisti & Carmignoto, 1986; Kalil, 1978; Maire-Lepoivre, Przybyslawski, & Gary-Bobo, 1988; Sherman, 1973, 1974a, 1974b). As well, we might expect the effects of monocular deprivation upon peripheral vision to be more deleterious than binocular deprivation. When deprivation begins from birth, monocularly deprived cats have more restricted fields than binocularly deprived cats (e.g., Tumosa, Tieman, & Hirsch, 1982; Sherman, 1973, 1974a, 1974b, 1977a; Sherman & Sprague, 1979; Smith, Holdefer, & Reeves, 1982; but see Heitlander & Hoffmann, 1978; Rizzolatti, cited in van Hof-van Duin, 1977; and van Hof-van Duin, 1977). Moreover, evaluation of the effects of monocular versus binocular deprivation allows differentiation of the influence upon peripheral vision of deprivation per se (which arises from either monocular or binocular cataracts) from the effects of interocular competition (which occurs only in cases of monocular deprivation).

Examination of children treated for a unilateral cataract also provides an opportunity to evaluate the effects of occlusion of the nondeprived eye upon the peripheral vision of each eye. In the sample of patients that I studied, when a child was treated for a unilateral cataract, parents were instructed by the ophthalmologist to patch the fellow nondeprived eye 50% of the waking time during the first 5 years of life in order to reduce interocular competition and

force usage of the previously deprived eye. Whether compliance with this instruction affects peripheral vision in humans is unknown although studies of cats suggest that the relative amount of viewing time allowed to each eye affects the extent of the visual field (Tieman & Hirsch, 1983; Tumosa, Nunberg, Hirsch, & Tieman, 1983; Tumosa, Tieman, & Hirsch, 1980; Tumosa et al., 1982). An additional aim of this dissertation, then, was to investigate the influence of patching history of the nondeprived eye upon the peripheral vision of both the deprived and the nondeprived eye in children treated for a unilateral cataract.

In addition, except for the report on one patient treated for a unilateral congenital cataract (Moran & Gordon, 1982), there is no information on the effects of visual deprivation on other than the horizontal meridian. Relevant work from monocularly deprived cats and monkeys (van Hof-van Duin, 1977; Sparks et al., 1986) indicate that losses are not limited to the horizontal meridian. It is likely, then, that visual deprivation in humans also will affect areas other than the horizontal meridian.

To summarize, then, it is likely that earlier deprivation, longer deprivation, or monocular deprivation may have more deleterious effects on peripheral vision than later deprivation, shorter deprivation, or binocular deprivation. Moreover, there may be some effect of occlusion of the nondeprived eye on the peripheral vision of children treated for a unilateral cataract. To evaluate each of these

hypotheses, I examined the extent of the field with kinetic perimetry on the Goldmann perimeter (Experiment 4) and sensitivity across the field with static perimetry on the Octopus perimeter (Experiment 5). Both approaches are necessary to describe thoroughly the peripheral vision of children treated for cataracts. Moreover, because kinetic perimetry is a quicker and simpler task for the subject, kinetic perimetry can be used to test a younger population than can static perimetry. This is important for identifying abnormalities in even young children with visual pathology and, not inconsequently, for increasing the sample of testable children. Finally, because of the brevity of its trials, static perimetry may be used to test children with even marked spontaneous nystagmus (jiggly eye movements). (Such children are untestable with kinetic perimetry on the Goldmann perimeter because of their inability to maintain a central fixation throughout a sometimes lengthy trial.) These data are presented in Chapter 3.

Development of peripheral vision in cats

If normal development proceeds similarly in cats and in monkeys as it does in humans, then cats and monkeys may be used as a model for understanding the neural basis of normal development in human infants. As well, animal models then may be

used to predict the behavioral and physiological effects of visual deprivation in humans.

To date, there has been no study of the normal development of the visual field in monkeys and only one study of development in kittens (Sireteanu & Maurer, 1982). In this study, kittens had one eye occluded and their attention was attracted to the center of the visual field by presenting a central visual or auditory stimulus. Once the kitten was looking centrally, the central stimulus was removed or silenced and a large stimulus, such as a shape made from black construction paper, a red ball of yarn, or a black ring, was introduced in the periphery. The stimulus was said to be detected if the kitten moved its eyes by the appropriate amount directly to the target more often than it did on the blank control trials when no stimulus was presented. Note that, as in studies of infants, the experimenter will underestimate the size of the field if the animal <u>detects</u> the stimulus but does not <u>orient</u> toward it. Like human infants, kittens exhibited two patterns of development. First, with increasing age, kittens oriented toward stimuli further from the center of the field until, at approximately 8 weeks of age, the field was adultlike in extent. Second, during this developmental period, detection in the temporal visual field preceded detection in the nasal visual field.

In summary, because the development of peripheral vision is similar in infants and in kittens, study of deprived cats may provide

clues about the effects of visual deprivation on peripheral vision in humans and its likely neural mediation.

Effects of visual deprivation on peripheral vision in cats

Typical experiments on visual deprivation in animals have involved suturing shut one or both eyes early in life for a period of time, then opening the eye(s) and assessing the capacity of the deprived eye(s). In cats, peripheral vision is affected adversely by monocular or binocular visual deprivation beginning at the time of normal eye opening or by monocular deprivation beginning after eye opening, although studies disagree about the amount of the restriction (Bisti & Carmignoto, 1986; Heitlander & Hoffmann, 1978; Kalil, 1978; Kossut, Michalski, & Zernicki, 1978; Maire-Lepoivre et al., 1988; Rizzolatti, cited in van Hof-van Duin, 1977; Sherman, 1973, 1974a, 1974b, 1977a; Sherman & Sprague, 1979; Smith et al., 1982; Tumosa et al., 1982; van Hof-van Duin, 1977; van Hof-van Duin, cited in Zablocka, 1983; Zablocka, 1983). To date, there have been no studies of the effects of binocular deprivation beginning after the time of normal eye-opening.

Following monocular lid suture in cats beginning either at the time of normal eye opening or at 5 weeks of age, all studies report that the nondeprived eye exhibited a normal field, extending from approximately 90° temporally to 30° or 45° nasally (Bisti &

Carmignoto, 1986; Sherman, 1973, 1974a, 1974b; Sherman & Sprague, 1979; Smith et al., 1982; Tumosa et .al., 1982). However, the deprived eye had a permanently restricted visual field. The deprived eye never gave evidence of detecting a large piece of food anywhere in the nasal visual field but did detect objects in parts of the temporal visual field: at worst, orienting to stimuli located only from 45° to 90° temporally (Sherman, 1973, 1974a, 1974b; Sherman & Sprague, 1979; Smith et al., 1982; Tumosa et al., 1982) and, at best, orienting to stimuli from 90° temporally throughout the temporal field (Bisti & Carmignoto, 1986; Heitlander & Hoffmann, 1978; Rizzolatti, cited in van Hof-van Duin, 1977; van Hof-van Duin, 1977). Those cats who achieved the best outcome included cats sutured at the time of normal eye opening (Heitlander & Hoffmann, 1978; Rizzolatti, cited in van Hof-van Duin, 1977; van Hof-van Duin, 1977) as well as cats deprived for 3 months beginning 5 weeks after birth (Bisti & Carmignoto, 1986).

Some of the worst outcomes following monocular deprivation beginning at the time of normal eye-opening were obtained by Sherman. Sherman (1973, 1974a, 1974b) and Sprague (Sherman & Sprague, 1979) reported that the responses of the deprived eye initially were less brisk and less accurate than those of the nondeprived eye. Even after prolonged usage, the deprived eye was able to detect objects only from approximately 45° to 90° temporally and exhibited decreased responsiveness to stimuli located at 90°

temporally. Similar results have been reported by Tumosa et al. (1982) and Smith et al. (1982). Moreover, in these studies, there was little intersubject variability in the extent of the visual field, even though the duration of deprivation ranged from 3.5 months (Tumosa et al., 1982) to, possibly, 44 months. (The duration of deprivation is not explicit in the article by Sherman & Sprague, 1979.) This indicates that when cats are tested with large stimuli, the duration of monocular lid suturing may not differentially affect the extent of the visual field along the horizontal meridian when deprivation begins from the time of normal eye opening and lasts for an extended period, perhaps even as long as 44 months.

Improvement of the peripheral vision of the monocularly deprived eye occurs after bilateral lesions of the occipitotemporal cortex combined with a split of the commissure of the superior colliculus or smaller unilateral lesions of cortical areas 17, 18, and 19 contralateral to the deprived eye. Following these lesions, detection in the visual field for the monocularly deprived eye improved from the monocular segment (45° to 90° temporally) to include the entire temporal hemifield (Sherman, 1974a; Sherman & Sprague, 1979). However, the expansion of the visual field for the deprived eye was accompanied by loss of the field for the nondeprived eye. The fellow nondeprived eye now behaved like the monocularly deprived eye, detecting objects only in the temporal hemifield and ignoring stimuli in the nasal field (Sherman, 1974a; Sherman & Sprague, 1979).

The phenomenon of restoration of the visual field also has been observed in normally reared cats following lesions. Following an unilateral lesion of the visual cortex, normally reared cats exhibit a total contralateral hemianopia. Following either a split of the collicular commissure or lesioning of the contralateral substantia nigra pars reticulata (Sherman, 1974c, 1977b; Wallace, Rosenquist, & Sprague, 1989; Wallace, Rosenquist, & Sprague, 1990), there is restoration of detection in the hemianopic field. The phenomenon of recovery of the visual field, referred to as the Sprague effect (Sprague, 1966), provides clues about the underlying physiology of peripheral vision in the cat.

Wallace et al. (1990) have argued that the cat's visual cortex and substantia nigra pars reticulata interact to contribute to peripheral vision. Each superior colliculus receives excitatory input from the ipsilateral visual cortex and inhibitory input from the contralateral substantia nigra pars reticulata through the collicular commissure. Removal of the unilateral visual cortex leads to the loss of excitatory input to the ipsilateral superior colliculus, creating an imbalance in stimulation to this superior colliculus, and a disruption of normal behavior. Removal of the suppressive input from the contralateral substantia nigra pars reticulata, either through ablation of this structure or of its pathways through the superior colliculus and the collicular commissure, restores the balance of input to the superior colliculus ipsilateral to the ablated visual cortex. With the

removal of unbalanced stimulation, more normal functioning becomes possible. However, one point unaddressed by Wallace and coauthors is which visual structure(s) now must be mediating the detection of stimuli in the previously hemianopic nasal visual field, given the removal of one visual cortex. Possibly, the projection from the temporal retina ipsilateral to the lesioned visual cortex to the contralateral lateral geniculate nucleus and visual cortex is sufficient for such detection.

The Sprague effect has implications for understanding the pathways affected by monocular lid suture in cats. Sherman (1974a) and Sprague (Sherman & Sprague, 1979) claim that once the pathway from the retina through the lateral geniculate nucleus to the visual cortex (corticotectal pathway) is destroyed, monocularly deprived cats then use the pathway from the retina directly to the superior colliculus (retinotectal pathway) to mediate visually guided behavior in the temporal visual field. Supposedly, the retinotectal pathways must be suppressed in monocularly deprived cats by the visual cortex and, following lesions of the cortex, there is release of suppression. However, even though the retinotectal pathway usually is suppressed in monocularly deprived cats, this pathway must have developed normally. This must be true because the fields of normal cats with occipitotemporal lesions, who are relying on the retinotectal pathways, are identical to those of monocularly deprived cats following occipitotemporal lesions (Sherman, 1974a, 1974c, 1977b;

Sherman & Sprague, 1979). In summary, the preoperative behavior of monocularly deprived cats reflects the functioning of abnormal corticotectal pathways and suppression of normal retinotectal pathways. Following cortical lesions, the behavior of monocularly deprived cats is mediated by their normal retinotectal pathway. This finding has implications for understanding the physiological mediation of peripheral vision in monocularly deprived children and will be discussed in the final chapter.

Studies disagree about the size of the temporal field in monocularly deprived cats. As discussed, Sherman and colleagues report that the temporal field of cats monocularly deprived from the time of normal eye opening extends from only 45° temporally to 90° temporally (Sherman, 1973, 1974a, 1974b; Sherman & Sprague, 1979; Smith et al., 1982; Tumosa et al., 1982). In contrast, Heitlander & Hoffmann (1978), Rizzolatti (cited in van Hof-van Duin, 1977) and van Hof-van Duin (1977) reported that following monocular lid suture from the time of normal eye opening until 6 to 10 months of age, the deprived eye responds normally throughout the temporal hemifield with no suggestion of decreased responsiveness in the far temporal field. Moreover, the superior and inferior visual fields in these monocularly deprived cats are restricted (van Hof-van Duin, 1977).

Differences across studies in the size of the temporal field in the deprived eyes are not explainable by whether the stimulus

moved or was stationary. Though Sherman (1973, 1974a, 1974b), Sherman and Sprague (1979), Smith et al. (1982), and Tumosa et al. (1982) used a moving stimulus while van Hof-van Duin (1977) chose a stationary stimulus to assess the extent of the field, Heitlander and Hoffmann (1978) and van Hof-van Duin (1977) found no difference in field size when cats were tested with either a moving or a stationary stimulus. Nor are discrepancies explained by differences in the size of the stimulus used because all experimenters used relatively large stimuli. Finally, the experimental discrepancies are not explainable by differences in the duration of deprivation because van Hof-van Duin (1977) and Heitlander and Hoffmann (1978) used periods ranging from 6 to 10 months, which were similar to those used by the other investigators. Overall, these studies indicate that the monocularly deprived eye of the cat does not orient toward stimuli in the nasal visual field but does detect and orient toward stimuli in at least parts of the temporal visual field. Discrepancies findings across studies about the size of the temporal field are unexplainable.

Not only does continuous occlusion from lid suture restrict the visual field but, under some circumstances, so can occlusion interleaved with periods of normal experience. Tumosa et al. (1980, 1982) reared cats in darkness from birth for periods of up to one month. Upon removal from darkness, one eye was occluded while the fellow eye was left open for 8 hours. On alternate days, the occluded

eye then was opened for periods of 1 to 7 hours and the fellow eye occluded. This procedure, in which one eye has more visual experience than the fellow eye, is referred to as alternating and unequal monocular occlusion. Beginning at 2 months of age, the peripheral vision of the kittens was tested. These cats had a normal field for the more experienced eye but a restricted field for the less experienced eye. Moreover, the amount of the loss of the visual field for the less experienced eye depended upon the amount of the imbalance in stimulation between the eyes. When there was a minimal discrepancy in the amount of looking time of each eye, the less experienced eye lost only part of the nasal visual field but with progressively larger imbalances in experience, there was a loss of the entire nasal field. Finally, when experience for the less experienced eye was eight times shorter than that for the more experienced eye, then the cats lost their nasal visual field and exhibited decreased responsiveness in the most peripheral portion of the temporal field. However, normal visual fields usually were achieved in each eye after one type of alternating monocular experience - when each eye received equal durations of stimulation. Under this condition, even when each eye received only eight hours of experience per day, then the visual field of each eye was normal in extent (Tumosa et al., 1982). However, when the duration of experience was decreased further to only one hour per day to each eye, the eyes sometimes

(Tumosa et al., 1982) but not always (Tumosa et al., 1980) showed a decrease in responsiveness in the far temporal field.

These data indicate two conclusions. First, the duration of visual stimulation may be important. When each eye experienced little experience, although the amounts were equal, there was decreased responsiveness in the temporal periphery. Second, the relative amount of stimulation to each eye will influence the extent of the field. The more experienced eye, which had a competitive advantage, had a normal field while the less experienced eye had a restricted nasal field. Moreover, the greater the imbalance in stimulation, then the larger the loss of the nasal field. Clearly, then, not only is the nasal visual field susceptible to a continuous period of lid suture, as discussed earlier, but it also is vulnerable to alternating and unequal monocular occlusion.

It also is appropriate to ask whether allowing a previously deprived eye prolonged and uninterrupted visual experience without competition from the fellow eye will alter peripheral vision. This may be accomplished in a procedure known as reverse suturing, which involves opening the lid sutured eye and lid suturing the nondeprived eye. Because the previously deprived eye is not competing with the now closed eye for visual stimulation, it is at a competitive advantage. Studies disagree about the effects of reverse suturing on the development of the field (Heitlander & Hoffmann, 1978; Sherman, 1973, 1974b; Smith et al., 1982; van Hof-van Duin,

1977). Sherman (1973, 1974b) claimed that reverse suturing of the formerly nondeprived eye for several months beginning as early as 5 months did not alter the extent of the visual field (also see Smith et al., 1982). In contrast, following several months of reverse suturing beginning at 6 months of age, van Hof-van Duin (1977) reported increased responsiveness in the binocular field (that part of the field normally viewed by both eyes) on the same side as the opened eye (also see Heitlander & Hoffmann, 1978). The difference in outcome across studies is not explainable by differences in either the duration of the initial lid suture or the reverse suturing or by differences in the age at which the reverse suturing began. Given the discrepant results of reverse suturing in deprived cats, it is difficult to predict whether occlusion of the fellow nondeprived eye in children treated for a unilateral cataract would lead to improved peripheral vision.

To summarize, unequal alternating monocular occlusion does affect the visual field yet reverse lid suture following a continuous period of lid suture may or may not. Two possible explanations exist. First, experiments which have investigated the effect of reverse suture employed this procedure following at least 5 months of monocular deprivation (Heitlander & Hoffmann, 1978; Sherman, 1973, 1974b; Smith et al., 1982; van Hof-van Duin, 1977). In contrast, alternating occlusion began much earlier, at one month of age. The difference in the age at which the procedures began may account for the different outcomes. An alternative explanation exists,

however. The alternating occlusion followed a period of dark rearing from birth and there is some suggestion that dark rearing may alter the effects of later monocular deprivation (Mower, Berry, Burchfiel, & Duffy, 1981; Mower, Caplan, & Letsou, 1982). If this is true, then the competitive effects from occlusion interleaved with periods of normal experience may have been sufficient to alter development because it followed dark rearing. Either or both of these possibilities (e.g., timing or dark rearing) may explain why development was altered by the apparent interocular competition from unequal occlusion but there was no recovery in some cases following the elimination of competition by reverse suturing. To distinguish between the two explanations, implementation of the reverse suturing should begin earlier in life and/or alternating occlusion should be begun at the time of normal eye opening.

In summary, the results from monocularly deprived cats indicate that the nasal, superior, and inferior visual fields are susceptible to early abnormal experience and, under some conditions, detection in the temporal visual field also is impaired. Studies disagree about the effects of reverse suturing on the development of the visual field.

To date, all binocular deprivation in the cat has been from the time of normal eye opening. Like monocular lid suturing, binocular deprivation from the time of normal eye opening also restricts the extent of the cat's field but, again, studies are inconsistent as to the

extent of the loss (Sherman, 1973, 1974b, 1977a; Kalil, 1978; Kossut et al., 1978; Maire-Lepoivre et al., 1988; van Hof-van Duin, cited in Zablocka, 1983; Zablocka, 1983). The largest losses of peripheral vision have been reported by Sherman and Kalil and will be described first.

Sherman (1973, 1974b, 1977a) reported that following binocular lid suturing from the time of normal eye opening, cats were blind initially, even when tested with large stimuli. Subsequently, cats indicated detection of stimuli only in the temporal hemifield with, relative to normal cats, decreased responsiveness to stimuli in the far temporal field. Moreover, as occurred for cases of unilateral lid suture, the duration of bilateral lid suture, which ranged from 5 to 16 months, did not affect the extent of the field (Sherman, 1973, 1974b, 1977a).

Moreover, the deficits in the visual field following binocular deprivation were permanent and unaffected by interocular competition. Even when one eye had a competitive advantage by being open, while the fellow eye continued to be lid sutured for several additional months, the field for the opened eye did not recover and there was no ultimate difference in the size of the field for the two eyes (Sherman, 1974b). How does this result reconcile with the finding that competition from unequal periods of alternating occlusion led to losses in the extent of the visual field? Exactly as before, two arguments may be advanced. First, the competitive

advantage enjoyed by the opened eye of the binocularly deprived cat began relatively late, at 5 months (Sherman, 1974b). In contrast, the unequal periods of alternating occlusion began much earlier in life, at 1 month. Second, the alternating occlusion was preceded by dark rearing, which was not the case for the binocularly deprived cats. Thus, either or both of these variables may explain why competition from unequal periods of alternating occlusion, but not elimination of competition from opening only one eye of a binocularly lid sutured cat, led to interocular differences in the visual field.

In addition to exploring the behavior of deprived cats, Sherman also has investigated its likely physiological basis. Following large bilateral or unilateral occipitotemporal lesions in binocularly deprived cats, there is no effect on peripheral vision - these cats continued to orient to stimuli throughout the temporal hemifield. This indicates that binocularly deprived cats, unlike normally reared cats, do not depend upon pathways through the visual cortex for peripheral vision. However, subsequent ablations of the contralateral superior colliculus in these binocularly deprived cats led to permanent blindness in the hemifield contralateral to the lesioned colliculus, indicating that binocularly deprived cats depend upon pathways through the superior colliculus for peripheral vision (Sherman, 1977a).

Binocular deprivation may be achieved not only by lid suturing but by rearing an animal in darkness. Using a different paradigm

than Sherman (1973, 1974b, 1977a), Kalil (1978) found that cats binocularly deprived by dark rearing[2] for 2 to 4 months from birth were blind initially and then exhibited some recovery. Those cats with the longest periods of dark rearing continued to exhibit losses throughout the entire nasal field and also some loss of detection in the far periphery of the temporal field. Similarly, Maire-Lepoivre et al. (1988) reported that longer periods of dark rearing led to more restricted visual fields. Although the pattern of loss of the visual field is similar in both paradigms, studies of dark reared cats, in contrast to studies of lid sutured cats, indicate that longer deprivation leads to more restricted visual fields (Kalil, 1978; Maire-Lepoivre et al., 1988). With regard to children treated for a cataract, studies of lid sutured cats are more likely to be predictive of outcome than are studies of dark reared cats.

In contrast to the findings of Sherman (1973, 1974b, 1977a) and the most deprived cats raised by Kalil (1978), who reported detection only in the temporal hemifield, other experimenters report less restricted fields in binocularly deprived cats (Kossut et al., 1978; Maire-Lepoivre et al., 1988; van Hof-van Duin, cited in Zablocka, 1983; Zablocka, 1983). Zablocka (1983) reported that hood rearing in cats, a procedure which is similar to bilateral lid suture, for the first

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[2] Dark rearing appears to be analogous to lid suturing because both types of visual deprivation prevent the input of patterned stimulation onto the retina. However, binocular lid suturing and dark rearing have different effects on visual development in the cat (Mower et al., 1981).

6 months of life led to decreased responsiveness only to stimuli located at the periphery of the nasal and temporal visual fields. Similar findings have been reported for cats dark reared for 4 to 7 months (van Hof-van Duin, 1992; van Hof-van Duin, cited in Zablocka, 1983) or for 6.5 months to 12 months (Maire-Lepoivre et al., 1988). Finally, Kossut et al. (1978) tested cats hood reared for the first 6 to 8 months and then transected at the pretrigeminal level (to allow restraint in a stereotaxic apparatus). Kossut et al. (1978) reported that these cats were able to detect a peripheral stimulus out to 40° in the nasal and in the temporal field (the farthest location tested) and then to track it. This result indicates that hood reared cats have fields extending at least to 40° temporally and, more surprisingly, to 40° nasally.

The differences in results are not explicable in terms of methodology. Though Kalil (1978), Kossut et al. (1978), Maire-Lepoivre et al. (1988), and Sherman (1973, 1974b, 1977a) used a moving stimulus while van Hof-van Duin (cited in Zablocka, 1983) probably used a stationary stimulus to assess the extent of the field, Zablocka (1978) found no difference in field size when cats were tested with either a moving or a stationary stimulus. Nor are discrepancies explained by differences in the size of the stimulus used because all experimenters used large stimuli. Finally, the experimental discrepancies are not explainable by differences in the duration of deprivation used by Sherman and colleagues versus by

van Hof-van Duin and colleagues because both groups of investigators used short and long durations of deprivation ranging from 4 to 16 months.

In conclusion, visual deprivation in the cat (whether monocular or binocular or unequal periods of alternating experience) will permanently affect the extent of the visual field. Generally, losses will be in the nasal visual field and, under some conditions of deprivation, in the temporal field. At least in monocularly deprived cats, losses also occur in the superior and inferior visual fields. Moreover, also in monocularly deprived cats, short deprivation beginning after the time of normal eye opening is less deleterious than similar deprivation beginning at the time of normal eye opening. Similar studies have not been attempted in binocularly deprived cats. As well, longer duration of dark rearing causes more abnormal visual fields. In comparison, when lid suture is used, there is a suggestion that deprivation from 3.5 perhaps up to 44 months in monocularly deprived cats and from 5 to 16 months in binocularly deprived cats may not differentially affect the extent of the field. However, to date, there has not been a systematic evaluation of the effects of the duration of visual deprivation on peripheral vision which uses extremely short periods of lid suture and contrasts these results to longer periods of deprivation. Finally, we can form a tentative conclusion about the effects of monocular versus binocular deprivation on development. After monocular deprivation, the

deprived eye never gives evidence of detecting stimuli anywhere in the nasal visual field but does detect objects in parts of the temporal visual field - at worst, detecting stimuli only from 45° to 90° temporally and, at best, detecting stimuli from 90° temporally to the center of the field. Following binocular deprivation, cats either respond only throughout the temporal hemifield or there are losses in both the nasal and temporal periphery. In general, it appears that the consequences of monocular deprivation on the extent of the visual field will be as, or more severe, than those of binocular deprivation.

Effects of visual deprivation upon peripheral vision in monkeys

Although the cat has been used extensively as a model to predict the effects of abnormal visual experience in humans, the monkey remains the preferred species because of the greater genetic similarity between the human and monkey. To date, there are four reports of the effect of monocular lid suture upon the development of the visual field of monkeys but no studies of normal development or of the effects of binocular deprivation.

A comparison of the four publications on monocular lid suture beginning from birth in monkeys suggests that the development of the visual field depends on the duration of deprivation. Sparks et al. (1986) deprived monkeys for short periods of 1 to 2 weeks or for

long periods of 18 to 26 months beginning in the second week of life. Short-term deprivation had no effect in either the nondeprived or deprived eye, with the monocular field extending from 60° nasally to 100° temporally and from 60° superiorly to 60° inferiorly. Long-term deprivation had no effect on the extent of the field in the nondeprived eye but in the deprived eye there was no response anywhere in the field even to large objects. Wilson et al. (1989) also used long-term occlusion of 19 to 27 months beginning within the first two weeks. In addition, they removed the crystalline lens of the fellow eye and then immediately optically corrected the then aphakic eye with a contact lens. Like Sparks et al. (1986), Wilson et al. (1989) also tested with large stimuli but found more variable results for the previously sutured eye: The occluded eye of one monkey was blind while for two other monkeys, there was no response in the nasal field and reduced levels of responsiveness to stimuli in the temporal field. Furthermore, the temporal field for the aphakic eyes of two of these monkeys was slightly restricted. Possibly, the better vision found by Wilson et al. (1989) was because the fellow eye was not normal but aphakic. As a result, the deprived eye was at less of a competitive disadvantage than if the fellow eye had been completely normal, as was true for the monkeys tested by Sparks et al. (1986).

Intermediate periods of deprivation from birth have been shown to have different effects on the visual field than do either short-term or long-term deprivation. Monkeys in these studies were

tested only along the horizontal meridian. Monkeys were deprived for 9 months beginning in the third week (Hendrickson et al., 1977), for 10 to 13 months beginning at 1 month (Wilson et al., 1989), or for 8 to 13 months beginning in the first week (Joseph & Casagrande, 1980), with all of the nondeprived eyes subsequently being reverse sutured. Hendrickson et al. (1977) found that the deprived eye responded, at most, to stimuli only out to 20° in the nasal field and out to 45° in the temporal field. Like Hendrickson and colleagues, Wilson et al. (1989) also reported that the nasal and temporal fields were restricted in extent but that response levels were normal. However, the results from Joseph and Casagrande (1980) are discrepant from these two studies. Joseph and Casagrande (1980) reported that their monkeys were initially blind and then saw only from 45° to 90° temporally (and nowhere in the nasal field), with the majority of the monkeys also exhibiting decreased responsiveness in the far temporal periphery. There was no improvement after reverse suturing but following enucleation of the nondeprived eye the field expanded to include the entire temporal hemifield. The results may be discrepant across studies because Joseph and Casagrande used prosimians (galagoes) and not the Old World macaque monkeys used by other experimenters. Moreover, because prosimian primates are less similar to humans than are Old World monkeys (Oxnard, 1984), these results may be less applicable to research with visually deprived humans.

Not only is the duration of deprivation important but the timing of the deprivation also affects the development of the visual field in monkeys. Wilson et al. (1989) reported that 18 months of monocular deprivation beginning as late as 3 to 5 months had a different effect than did long deprivation of either 19 to 27 months beginning within the first two weeks after birth (Wilson et al., 1989) or 18 to 26 months of deprivation beginning in the second week (Sparks et al., 1986). Monkeys who experienced long deprivation beginning between 3 and 5 months of age saw as far to the side as did the normal monkey but responded less consistently. However, monkeys who experienced long deprivation early in life had, at best, a restricted visual field and decreased responsiveness (Wilson et al., 1989) or, at worse, were blind (Sparks et al., 1986). Joseph and Casagrande (1980) reported more subtle differences in behavior when the timing of the deprivation was varied. They reported that either 9 months of deprivation beginning at 5 weeks or 8 to 13 months beginning at 1 week left vision only from 45° to 90° temporally. Moreover, there was no beneficial effect of reverse suturing following deprivation in either condition. However, unlike the galagoes with early onset deprivation, those with later onset deprivation did not show decreased responsiveness in the far temporal periphery of the visual field. Note that, as stated earlier, the results of Joseph and Casagrande (1980) may be less applicable

to visually deprived humans than the results of Wilson et al. (1988) and Sparks et al. (1986) because of the species of monkey studied.

In summary, both the duration and the timing of the deprivation are important variables influencing peripheral vision, with longer deprivation and earlier deprivation having more severe consequences than shorter or later deprivation.

How do the results from monocularly deprived monkeys compare to the data from monocularly deprived cats and humans and what predictions may be made about the effects of deprivation in humans?

First, all studies, whether of monocularly deprived monkeys, cats, or humans, agree that the peripheral vision of the fellow nondeprived eye is normal.

Second, all studies of monkeys, cats, and humans indicate that if the duration of deprivation is long, lasting for more than several weeks, then the peripheral vision of the visually deprived eye will be abnormal. However, if the monocular deprivation is short, lasting for 1 to 2 weeks beginning in the second week of life, then monkeys have a normal visual field (Sparks et al., 1986). To date, no study has systematically varied the duration of deprivation in monocularly or binocularly lid sutured cats although studies of dark reared cats indicate that longer deprivation has more deleterious effects upon peripheral vision. Likewise, at present, we cannot draw any conclusions about the importance of the duration of deprivation on

development in deprived humans because of the limited sample tested. It is likely, though, that deprived humans will behave like deprived monkeys and that the duration of deprivation will influence peripheral vision.

Third, earlier onset of visual deprivation leads to poorer peripheral vision in monocularly deprived monkeys and in monocularly deprived cats but relevant studies have not been conducted in binocularly deprived animals. For monocularly deprived humans, some information exists which hints that timing may be an important influence (Lewis et al., 1986; Maurer et al., 1983). The role of the timing of the deprivation upon peripheral vision requires evaluation in a large sample of monocularly and binocularly deprived humans.

Fourth, to date, the influence of patching upon peripheral vision in deprived humans has not been evaluated and is difficult to predict from the literature on animals.

Finally, in cats the effects of monocular deprivation from birth are at least as serious as those of binocular deprivation. Whether this also is true in monkeys is unknown as relevant studies of binocular deprivation are lacking. In humans, there are clues that unilateral deprivation from birth, but not bilateral deprivation, may affect especially the nasal visual field. This outcome needs to be evaluated in a larger sample.

General summary

Previous studies of infants and kittens indicate that peripheral vision is immature early in life and develops from the center out with better detection in the temporal visual field than in the nasal visual field early in life. The rate of this development proceeds much faster in kittens than in infants. Moreover, the absolute limits of the measured field at any age will be influenced by the test stimulus used. For example, when tested with a large, flashing light, even 6-month-old infants have approximately adult-sized visual fields. In contrast, when schoolaged children are tested with small stimuli, then there is disagreement about when there is complete development of the visual field. Finally, visual deprivation from a dense and central cataract during this developmental period affects the extent of nasal, temporal, superior, and inferior visual fields and also sensitivity along the horizontal meridian of the field. Yet much remains unknown about the effects of the timing and of the duration of visual deprivation and the relative severity of monocular versus binocular deprivation upon peripheral vision. Studies of deprived monkeys and cats suggest that these variables are likely to be important.

To further investigate the normal development of peripheral vision and the effects thereon of visual deprivation, a series of experiments have been carried out and are reported in subsequent

chapters. Chapter 2 reports on the development of peripheral vision in normal children while Chapter 3 discusses the influence thereon of visual deprivation. The final chapter summarizes and evaluates the research findings and explores the physiological basis of the observed behaviors.

CHAPTER 2
DEVELOPMENT OF PERIPHERAL VISION IN NORMAL CHILDREN

This chapter presents the results from three experiments which examined the normal development of peripheral vision. Experiment 1 compares the extent of the visual field in 5-, 6-, and 7-year-olds, and in adults. Because the results of the first experiment indicate that the visual field continues to develop after 5 years of age, Experiment 2 reports on the longitudinal development of the visual field in 5-year-olds who were retested at 7 years of age. Experiment 3 examines the normal development of sensitivity across the visual field in a different sample of children and adults.

Experiment 1
The development of the extent of the visual field during childhood

Four studies have assessed the peripheral vision of children using a large stimulus (Cummings et al., 1988; Matsuo et al., 1974; Tomonaga, 1974; Whiteside, 1976). Cummings et al. (1988) reported that when tested with a 26' light presented kinetically, the extent of the field for a group of children aged 2 to [illegible] years was not adult-like.

In contrast, when the same children were tested with a 42' light presented statically, then their fields were as large as those of adults. In agreement with the latter result, Whiteside (1976) reported that 6-year-olds have adult-like fields when tested with a 1° light presented statically. Similarly, Tomonaga (1974; data also reported in Matsuo et al., 1974) reported that full visual fields are observed by 5 years of age when children are tested with 10' to 1.9° lights presented statically and kinetically.

Several investigators have measured the extent of the visual field in children using a small stimulus of 6' (e.g., Allan, 1971; Aspinall, 1967; Lakowski & Aspinall, 1969; Liao, 1973; Matsuo et al., 1974; Suzumura et al., 1985; Tomonaga, 1974). The results from these studies are inconsistent. In some studies, schoolaged children had relatively full fields (Allan, 1971; Liao, 1973; Matsuo et al., 1974; Suzumura et al., 1985; Tomonaga, 1974) while in other studies the fields were restricted (Aspinall, 1967; Lakowski and Aspinall, 1969). For example, using similar testing conditions, Tomonaga (1974) reported no further development of the field after 5 years of age while Lakowski and Aspinall (1969) found that the field for the only 6-year-old tested extended out to only 15°. Clearly, when the visual field is adult-like in extent for small stimuli is unresolved.

Not only is there controversy about the development of the extent of the field but there has been no report of interocular differences and only two reports of the reliability of measurements, and those in only a few children (Cummings et al., 1988; Liao, 1973).

Data on interocular differences are important for two reasons. First, information about interocular differences in normal and related eyes is necessary for making diagnostic decisions about differences in performance between a visually deprived eye and a fellow nondeprived eye of a patient. Second, data on interocular differences will allow investigation of whether the developmental rates of the hemispheres of normal children differ. Data on reliability in young children is important because it allows assessment of the stability of behavior over time.

I used kinetic perimetry with the Goldmann perimeter to examine the monocular development of the visual field along eight meridia in large groups of Caucasian 5-year-olds, 6-year-olds, 7-year-olds, and adults. Each subject was tested with a 6.4' light of two intensities: 31.8 cd/m^2 and 318 cd/m^2. To measure interocular differences, I tested both eyes of each subject on the first visit. To assess reliability, I retested one eye during a second visit. My investigation is important for two reasons. First, examination of the visual field of young children may add to what is known about the development of peripheral vision and its neural underpinnings and, second, these results will produce a normative standard for the assessment of the visual fields of children treated for dense and central cataracts.

Method

Subjects. The final sample of subjects consisted of 28 5-year-olds (mean age = 5.1 yrs, range = 5.0 - 5.3 yrs), 24 6-year-olds (mean age = 6.1 yrs, range = 5.8 - 6.2 yrs), 20 7-year-olds (mean age = 7.0 yrs, range = 6.8 - 7.2 yrs), and 16 adults (mean age = 24.9 yrs, range = 20.6 - 31.6 yrs).

To be included in the final sample, each subject had to pass a screening exam and complete three valid tests of the visual field (see Procedure). Because this was a study of normal development, any individual with a history of refractive error or of strabismus (misalignment of the eyes) should be eliminated because either condition potentially may affect the development of peripheral vision (Benedetto & Cyrlin, 1985; Fankhauser & Enoch, 1962; Ferree, Rand, & Monroe, 1929; Fusco, D'Aietti, & Verriest, 1983; Jacobson, Sandberg, & Berson, 1983; Johnson & Leibowitz, 1974; Mahendrastari & Verriest, 1985; Mehdorn, 1986; Serra, 1983; Sireteanu & Fronius, 1989).

A history of refractive error (either myopia, which is nearsightedness, or hyperopia, which is farsightedness) may result in later poor acuity (Atkinson & Braddick, 1988). To detect myopia or amblyopia (reduced vision which cannot be improved with optical correction), each individual was asked to read the Snellen eye chart monocularly from 6 meters. Although the median visual acuity (the ability to resolve fine detail) of a 5-year-old is slightly better than

20/25 as measured by the Snellen eye chart, there exists much variability in the acuity of 5-year-old children (Weymouth, 1963). Therefore, a 5-year-old passed the screening exam if he had a Snellen acuity of at least 20/30 in each eye while for a subject 6 years and older this criterion was tightened to at least 20/20 in each eye. To detect hyperopia greater than 3 diopters (D), each subject was asked to reread the Snellen eye chart monocularly through a +3 D lens. Because the +3 D lens will focus light at 33 cm in front of the retina in an emmetropic eye (an eye with no refractive error), acuity for letters 6 meters away should be worse with the lens than without it. However, if the eye is hyperopic by 3 D or more, then the light will be focussed closer to the retina with the lens than without it. The acuity of the hyperopic eye looking through the lens should then either remain the same or improve. For this reason, at each age, poorer acuity for each eye looking through a +3 D lens than without the lens was required.

To detect subjects with strabismus or a history of strabismus, which interferes with the normal development of binocular vision (Banks, Aslin, & Letson, 1975), stereoacuity was assessed with the Titmus Fly Test and binocular fusion with the Worth Four Dot Test. Stereoacuity is the smallest horizontal disparity of retinal image that permits a perception of relative depth (Keeney, 1970) while fusion is the perception of one object even though there is stimulation of different retinal points in each eye (Burian, 1972).

Most, but not all, 5-year-olds can achieve a stereoacuity of 40 seconds on the Titmus Test, which is the smallest disparity available at a testing distance of 40 cm (Heron, Dholakia, Collins, & McLaughlan, 1985; Romano, Romano, & Puklin, 1976). Therefore, for the purpose of the screening exam, a 5-year-old was required to have a stereoacuity of at least 80 seconds, the second smallest disparity. Older subjects needed a stereoacuity of 40 seconds to pass the screening examination.

Finally, to detect either suppression of vision in one eye under binocular viewing conditions, or double vision when only one object is present (diplopia), each subject was tested with the Worth Four Dot Test at far. No normative data exist for the development of fusion as assessed by the Worth Four Dot Test. However, because the visual cortex of even the 3-month-old infant appears to summate signals from corresponding points of the retinae (Braddick, Atkinson, Julesz, Kropfl, Bodis-Wollner, & Raab, 1980), and because even 6-month-old infants appear to perceive only one object on a test of fusion, as assessed by a preferential looking paradigm (Birch, Shimajo, & Held, 1985), each subject was required to exhibit evidence of fusion of stimuli on the Worth Four Dot Test at 6 meters.

After passing the screening exam, each subject had to complete three valid tests of the visual field. A field test was considered invalid if the blind spot could not be plotted (i.e., the subject did not maintain central fixation as the light moved from the nonseeing to the seeing area of the visual field), if the subject gave inconsistent

responses when a meridian was replotted, or if the subject said that he did not always respond immediately upon perception of the target.

An additional 36 subjects were excluded from the final sample because they refused to participate in the screening exam (n = 1 5-year-old), because they did not pass the screening examination (n = 5 5-year-olds, 12 6-year-olds, 9 7-year-olds, and 1 adult), because their field tests were invalid (n = 8 5-year-olds, 1 6-year-olds), or because of procedural error (n = 1 5-year-old).

Apparatus and stimuli. The Goldmann perimeter was used (see Harrington, 1981) to assess the extent of the visual field (see Figure 1, all figures are presented in Appendix A). The target intensities were 31.8 cd/m^2 (target I2e) and 318 cd/m^2 (target I4e); background illumination was 10.0 cd/m^2 and target diameter was 0.25 mm^2 (6.4' when viewed from 30 cm).

Procedure. I explained the procedure to the subject and obtained parental or individual consent. First, I administered the screening exam. If the subject failed the screening exam, the session was terminated. If the subject passed the screening exam, I used standard clinical procedures (Harrington, 1981) for the test of the visual field: the subject had one eye patched, was seated in front of the Goldmann bowl with his chin resting on the chin rest and the

restraining head strap in place. After the subject had adapted to the lighting conditions of the Goldmann bowl for 5 minutes, he was instructed to maintain fixation on the fixation circle at the center of the bowl and to tap immediately on the surface of the table when first aware of the test target in the periphery. I showed the target to the subject prior to each test and then moved the target from the periphery of the bowl toward the center at a rate of approximately 3° per second. Locations tested always included at least the following meridia: 5°, 45°, 90°, 135°, 180°, 225°, 270°, 315°, and 355°. (The temporal field is represented by 5° and the representation proceeds counterclockwise so that 45°, 90°, and 135° are in the superior field, 180° is in the nasal field and 225°, 270°, 315°, and 355° are in the inferior field. Because the perimeter has an excised section at the 0° meridian, the field extent for 0° could not be measured directly and was calculated as the average of the results at the 5° and 355° meridia.) The subject was unaware of the meridian at which the light would be introduced on any trial. The order of testing of the meridia and inter-trial duration were random. The extent of the field at each meridian was assessed three times with the average value representing the extent of the visual field. In addition, the subject was asked periodically to indicate the approximate location of the light on the trial just completed in order to verify that the target had been perceived. Trials in which the subject could not indicate accurately the location were discarded and later repeated.

After the extent of the field was plotted with both the intense (I4e) and dim (I2e) targets, 10 to 15 static spot checks of the central 30° for each eye were completed with the dim target as a check for central scotomas (areas of the visual field where the light is undetected). The light was flashed on and off and, as before, subjects were told to tap immediately after detecting the flash. Finally, I plotted the nasal, temporal, superior, and inferior extent of the blind spot using the dim target at approximately 15° in the temporal field and 5° below the horizontal meridian. The subject's task was to respond when he was first aware of the light moving from the nonseeing to seeing area of the field.

So as to monitor the subject's fixation of the central fixation target, I observed the subject's eye through a telescope which extended from the central fixation point to the back of the Goldmann perimeter. Trials on which the subject did not maintain fixation were discarded and that meridian was retested later in the examination. A loss of fixation was defined as movement of the pupil of the subject's eye by more than 2 mm (less than 0.4°) as measured by a grid centered in the telescope. In addition, because the size of the pupil may affect measurements of peripheral vision (e.g., Bedwell & Davies, 1977; Lindstrom, Tredici, & Martin, 1968; McCluskey, Douglas, O'Connor, Story, Ivy, & Harvey, 1986), pupil size was measured with the telescope grid at the start and completion of a test.

Each subject was tested on two different days. On the first day, each eye was tested with the intense and dim targets to allow

measurement of the extent of the field for each eye and to determine if there were interocular differences. Half of the subjects in each age group had their right eye tested first, and half the left eye. All subjects completed four tests: the first eye was tested with the intense target, the second eye with the intense target the second eye with the dim target, and finally the first eye with the dim target. On the second day, one eye was retested to determine reliability, first with the intense target, then with the dim target. For half of the subjects, the eye which had been tested first on Day 1 was retested and for the other half of the subjects, the eye which had been tested second on Day 1 was retested.

Results

Each subject was able to detect each presentation of the light in the central field (other than in the area of the blind spot). Very few trials were repeated, and these only in the youngest subjects, because the subject was unable to indicate the location of the peripheral stimulus after tapping on the table. As well, the horizontal diameter of the subject's pupils was within normal limits, measuring between 4 and 7 mm (Mikelberg, Drance, Schulzer, & Wijsman, 1987). [The effect of pupil size on peripheral vision will be evaluated in Experiment 6.]

The results were analyzed using a 4-way ANOVA with age (four ages) as the between subject factor and test (three tests),

intensity (two intensities) and meridian (eight meridia) as within subject factors (see Table 1, all tables are presented in Appendix B).

1(a) Effect of age:

There was a main effect of age: Older subjects detected the target significantly further to the side (p = .00015). Because I wished to carry out a limited number of comparisons tracing the development of the extent of the field with age, and subsequently to use these data as normative standards for children with visual pathology, planned comparisons were chosen. Planned orthogonal comparisons showed that 5-year-olds did not detect the target as far to the side as did the group of older subjects [$F(1,84) = 24.52$, $p <$ .000001]. In contrast, 6-year-olds detected it as far to the side as the group of 7-year-olds and adults [$F(1,84) = 2.20$, $p = .14$], and there was no difference between 7-year-olds and adults [$F(1,84) = 0.28$, $p = .60$]. Finally, a planned comparison (trend analysis) of the data from 5- and 6-year-olds revealed that although the extent of the field increased from 5 to 6 years, the shape of the field remained the same [$F(7,88) = 0.81$, $p = .58$].

(b) Effect of meridian:

There was a main effect of meridian ($p <$ .000001). Because I wished to compare the extent of the visual field along the eight measured meridia, I chose to use Tukey's HSD tests. Tukey's HSD

tests revealed that the field was largest temporally (p's < .01) and smallest superiorly (p's < .05).

There also was an interaction of meridian and age (p < .000001). At most meridia, the extent of the field was larger at the older ages. For example, at 0° temporally, the group of adults detected the light 11.5° further to the side than did the 5-year-olds. The literature has concentrated on examining the extent of the horizontal and vertical meridia in children and in animals. Because I wished to increase the power of the tests and to compare my findings to the literature, I examined the development of the extent of the field with age along the horizontal and vertical meridia using planned comparisons. Planned orthogonal comparisons revealed that 5-year-olds did not see the target as far to the side as did the group of older subjects at 0° temporally [$F(1,84) = 28.79$, $p = .000001$], 180° nasally [$F(1,84) = 26.71$, $p = .000001$], or 270° inferiorly [$F(1,84) = 28.37$, $p = .000001$] but did see as far at 90° superiorly [$F(1,84) = 1.66$, $p = .20$]. Six-year-olds did not see the target as far to the side as the group of 7-year-olds and adults at 0° temporally [$F(1,84) = 7.27$, $p = .008$] or at 270° inferiorly [$F(1,84) = 8.26$, $p = .005$] but did see as far at 90° superiorly [$F(1,84) = 2.04$, $p = .15$] and 180° nasally [$F(1,84) = 1.96$, $p = .16$]. Even 7-year-olds did not see as far to the side as did adults at 0° temporally [$F(1,84) = 10.02$, $p = .0025$] or at 270° inferiorly [$F(1,84) = 5.55$, $p = .0197$], with the size of the difference being 6.1° and 3.5°, respectively. However, 7-year-olds had larger fields than did adults by 4.9° at 90° superiorly [$F(1,84) =$

10.59, p = .0014] and there was no significant difference in the size of the field at 180° nasally [$F(1,84)$ = 1.16, p = .29].

(c) Effect of intensity:

The extent of the field was influenced by target intensity, with a larger field obtained with the intense light (p < .000001). Also, intensity interacted significantly with meridian (p < .000001). Because there has been no previous investigation of how stimulus intensity affects the extent of the field along different meridia, I chose to use post hoc tests. Tukey's HSD tests revealed that the extent of the field was smaller superiorly, particularly with the dim light (all p's < .01). However, the pattern of responding to each intensity of light did not differ significantly with age (p = .14), nor did the overall pattern of responding by age to the different intensities at each meridian (p = .20).

(d) Effect of test:

There was no main effect of test (p = .30). Because these data will be used to evaluate the peripheral vision of children with eye disorders and because most children in the clinical setting will have only one test of their visual field, the means for just the first test of the visual field are presented in Tables 2 and 3. Also, there were no significant interactions of test by age (p = .49), test by age by intensity (p = .63), test by meridian (p = .34), test by age by meridian (p = .36), or test by meridian by intensity (p = .53). However, there

was a significant interaction of test by intensity (p = .00049). Tukey's HSD tests indicated that there were no differences in the extent of the field for tests using the same intensity (all p's > .05) and the extent of the field was always larger for tests using the intense light than tests using the dim light (all p's < .01) (see Figures 2 - 7).

Finally, there was a significant interaction of test by age by intensity by meridian (p = .00071). Given the complexity of the interaction, its probable clinical insignificance, and possible unreliability, it was not analyzed further. Nonetheless, some description of the four way interaction may be worthwhile (see Figures 2 - 7).

Regardless of intensity and test, each age exhibits the following pattern: The temporal field is largest, the superior field is smallest, and the extent of the nasal and inferior fields fall between these extremes. For the first and second tests of the dim light and for the second test with the intense light, the adults have the largest fields except superiorly where their field is as small as that of the 5-year-olds (Figures 2, 4, & 6). The same pattern exists for the third test with the dim light and the first and third tests with the intense light except that the nasal field of the adults is not larger than that of the 7-year-olds (Figures 3, 5, & 7). Note that for all figures the biggest difference in the fields of the adults versus the 7-year-olds lies in the temporal field (Figures 2 - 7). Other commonalties exist across figures. In general, 5-year-olds have the smallest fields at all but the superior meridia. Note, though, in Figure 6, that the nasal field of the

5-year-olds is similar to that of the 6- and 7-year-olds. Finally, the extent of the field of the 6-year-olds is fairly similar to that of the 7-year-olds (Figures 2, 6, & 7) although it is sometimes smaller at some locations, such as nasally and inferiorly (Figures 3, 4, & 5).

Although statistically significant, treatment differences of only a few degrees are unlikely to be clinically significant, given that they may be within the realm of measurement error. Examination of the reliability data for each meridian and intensity of light at each age indicates that the extent of the field on the test and retest typically differed by 1° to 4° (see Tables 13 and 14). This suggests that although the differences observed in the interaction of age with test with intensity with meridian are statistically significant, they are within the realm of measurement error and not clinically significant. The independent replication of the interaction of age by test by intensity by meridian would constitute a convincing demonstration of its robustness.

(2) Interocular difference scores:

Calculation of interocular difference scores in normal subjects has two purposes. First, for the patient treated for a unilateral cataract, the diagnosis of abnormality is made by comparing the size of the field for the abnormal eye to that of the fellow nondeprived eye. This approach is more sensitive than comparing the field of the abnormal eye to that of group norms, given the variability in the size of the visual field across normal individuals (see this experiment). In

order to determine whether any difference in the size of the field in the deprived eye versus the fellow nondeprived eye is a significant difference, it is necessary to know what constitutes normal interocular variation. Second, the difference scores indicate whether each hemisphere in normal subjects develops at approximately the same rate.

Interocular difference scores can be calculated as signed difference scores and as absolute difference scores. The signed difference scores will indicate whether the right eye generally has a larger field than the left eye, or vice versa. It is important to be aware of whether one eye is consistently larger than the other eye in the normal sample when evaluating the effects of deprivation upon a right eye versus upon a left eye in patients. In contrast, the absolute difference scores will indicate whether there is a tendency for one eye to have a larger field than the fellow eye and is independent of whether the eye with the larger field is consistently the right (or the left) eye. Should there be much variation in the size of the field between normal eyes, then this may serve to distort appreciation of whether true differences exist between a patient's deprived and nondeprived eye.

Note that there exists a problem in interpreting non-zero interocular difference scores. If there is no difference in the extent of the field of two related eyes then the difference score should be zero. If the difference score is not zero then it need not be because of a real difference between the two eyes but could arise because of

measurement error. Nonetheless, these difference scores set a baseline for the difference in the extent of the visual field in two related normal eyes, which is important for diagnostic reasons in patients.

The signed interocular difference scores were calculated by averaging across subjects the difference of the extent of the field for the subject's left eye (either test 1 or 2) from the extent of the field for the same subject's right eye (the remaining test, either test 1 or 2) for each meridian and light intensity (Tables 4 & 5). A negative score means that the extent of the field for the left eye was greater than the extent for the right eye at that meridian while a positive score indicates the opposite finding.

For tests with the dim light, the average interocular difference scores were small, ranging from -3.5° to 3.9° (see Table 4). Moreover, the scores were similar across age. For example, the difference scores ranged from -2.4° to 0.86° for 5-year-olds and from -2.6° to 2.9° for adults. On average, then, the size of the field is similar in the right eye and in the fellow left eye and there does not appear to be a systematic effect of age. Similar conclusions may be drawn from the test with the intense light: For example, the difference scores ranged from -1.1° to 1.1° for 5-year-olds and from -2.4° to 2.9° for adults with the scores for the adults representing the most extreme scores across all ages (see Table 5). These data were not statistically analyzed because the difference scores were very small and similar across age.

When making diagnostic decisions in patients, it is necessary to know not only the average difference but also the largest interocular difference in the extent of the field between a normal right eye and a normal fellow left eye. The largest interocular difference score provides an index of the maximum amount of difference in the size of the field between two eyes that would be considered normal. Assuming that the normative sample is sufficiently large and representative of the population, an interocular difference in a patient treated for unilateral cataract which is greater than the largest interocular difference will indicate abnormality, then.

The most extreme interocular score at each age, which represents the most aberrant 5% of values, was eliminated from the data in the previous calculation of the signed interocular difference scores so as to minimize the effects of extremely atypical scores. Tables 6 and 7 present the second largest interocular difference scores, regardless of sign. The interocular differences were large: For tests with the dim light, scores ranged from 5° at 270° inferiorly for adul. to 20° at 90° superiorly for 5-year-olds (Table 6). For tests with the intense light, scores ranged from 13°, found at two locations for 5-year-olds, to 14°, found at three locations for 5- and 6-year-olds (Table 7). This indicates, then, that substantial differences in the extent of the field between two related normal eyes may occur. As such, interocular differences in the extent of the field of children treated for a unilateral cataract which are smaller than these values may be considered to be within normal limits.

The absolute interocular difference scores were calculated by averaging across subjects the absolute value of the difference of the extent of the field of the right eye (test 1 or 2) minus that of the fellow left eye (the remaining test, either test 1 or 2) for each meridian and light intensity (see Tables 8 & 9). In other words, this calculation involved finding the absolute value of the signed interocular difference scores calculated previously. The range was from 2.6° to 6.0° for the dim light (Table 8) and from 2.4° to 5.8° for the intense light (Table 9). Moreover, unlike for the signed scores, some variation in the size of the absolute scores exists across age. For the dim light, the scores for the adults range from 2.4° to 4.6° while for the 5-year-olds the scores tend to be larger, ranging from 3.7° to 6.0° (Table 8). Similarly, for the intense light, the score for the adults range from 2.4° to 4.8° while for the 5-year-olds the scores tend to be larger, ranging from 4.1° to 5.8° (Table 9).

Given that there was some suggestion of variability in the size of the absolute difference scores across age, with the 5-year-olds appearing to have larger scores than did the adults, a 3-way ANOVA was used. The between group factor was age (4 ages) and the within group factors were intensity (2 intensities) and meridian (8 meridia) (see Table 10). There was a main effect of age (p = .0038). For two reasons I chose to use post hoc tests to investigate the variable of age, unlike my earlier planned comparisons of age. First, I have no expectations from the literature concerning the influence of age upon interocular difference scores. Second, the interocular difference

scores are not being used for normative purposes. Tukey's HSD test showed that the absolute interocular difference scores of 5-year-olds were larger than those of adults by nearly 1.4° ($p < .01$) but that there were no other significant differences among the ages (all p's > .05). There was a significant main effect of meridian ($p = .0127$). Tukey's HSD tests showed that the difference score at 45° superiorly was significantly larger than at 90° superiorly or at 225° inferiorly (p's < .05). However, there were no significant effects of intensity ($p = .44$), age by intensity ($p = .75$), age by meridian ($p = .93$), intensity by meridian ($p = .73$), or age by intensity by meridian ($p = .15$). In summary, the results indicate that 5-year-olds, but not other ages, have larger absolute interocular difference scores than do adults.

(3) Range of the extent of the field:

Assessment of the range of the size of the visual field across subjects within an age provides information about variability within the group. This information is helpful when examining the data from patients. For example, if there is much variability in the extent of the visual field for normal subjects of the same age, and this variability is larger than the within subject variability, then this would suggest that the more conservative approach when evaluating data from a patient treated for a unilateral cataract would be to use the fellow nondeprived eye, rather than the data from normal volunteers, as the normal standard. Obviously, this approach assumes, and requires

verification, that the peripheral vision of the fellow nondeprived eye of patients is indeed normal.

The smallest value, representing the most aberrant 5% of values, was discarded for each intensity and meridian for test 1. The data from test 1 were chosen because there likely will be only one assessment of the visual field for each patient in a clinical setting. The range of the field was calculated as the largest extent of the field minus the smallest extent of the field for each meridian and light intensity in test 1 (Tables 11 and 12). The range at each age was large: For tests with the dim light, scores ranged from 13° at 270° inferiorly for adults to 29° at 45° superiorly for 6- and 7-year-olds (Table 11). For tests with the intense light, scores ranged from 9° at 225° inferiorly for adults to 32° at 45° superiorly for 5-year-olds (Table 12).

(4) Test-retest reliability:

Test-retest differences for individual subjects were calculated by averaging across subjects the differences of the extent of the field for the repeat test (test 3) from the extent for first test of the same eye (either test 1 or test 2) for each meridian and light intensity (Tables 13 & 14). Test-retest reliability was high at each age and for each light intensity. For the dim light, the highest difference was 3.7° located at 225° inferiorly for adults (Table 13). For the intense light, the largest difference was also 3.7° but at 315° temporally for 6-year-olds (Table 14).

(5) Analysis of the blind spot:

To assess whether subjects used similar criteria to indicate detection in the center of the field, the size and the location of the blind spot were analyzed with a 2-way ANOVA. The between group factor was age (4 levels) and the within group factor was the description of the area and location of the blind spot (3 levels: area of the blind spot, temporal and the inferior location of the central most point of the blind spot). Specifically, the area for the blind spot was calculated using the formula for an ellipse: Area = pi * length of the major radius * length of the minor radius. The parameters for the central most point of the blind spot were its location in the temporal field and its location in the inferior field.

There was no significant main effect of age [$F(3,84) = 1.86$, $p = .14$] and no significant interaction of age and the measurements of the area and location of the blind spot [$F(6,168) = 1.76$, $p = .11$]. Not surprisingly, there was a significant main effect of the measurements of the blind spot [$F(2,168) = 515.81$, $p < .00001$] : The area of the blind spot was much larger than the value for the temporal or for the inferior location of the central most point (p's $< .01$). The average area of the blind spot was 366.7 mm^2 while the center of the blind spot was located at 15.6° temporally and 2.0° inferiorly.

Discussion

There is development of the extent of the visual field early in childhood. Five-year-olds do not see a small target as far to the side as do older subjects in the temporal, nasal, or inferior fields but do see as far in the superior field. The field grows after 5 years of age but even at 7 years of age children do not see as far to the side as do adults in the temporal or inferior fields but do see as far in the nasal field. Moreover, 7-year-olds have larger superior fields than do adults.

These results differ from those of other investigations using a light of the same size. For example, Matsuo et al. (1974) and Tomonaga (1974) found no further development of any part of the visual field after 5 years of age which, in this experiment, was true of only the superior visual field. Note that these studies were conducted in Japan. Possibly, my results differ from these studies because of racial differences in the subjects assessed. For example, Annis and Frost (1973) reported that the visual acuity of Euro-Canadians, unlike that of Cree Indians, was higher for gratings oriented vertically and horizontally as opposed to obliquely. Possibly, as occurs for visual acuity, racial differences may influence the development of peripheral vision or behaviors important to its assessment.

Other studies report that development continues even after 6 years of age (Aspinall, 1967; Lakowski & Aspinall, 1969; Liao, 1973)

while my study found that after this age development occurred only in the temporal and in the inferior field. (My findings cannot be compared to those of Allan (1971) because Allan combined the results from 5- and 6-year-olds.) My results may differ from studies which found smaller fields, possibly because care was taken to ensure that subjects were not fatigued or uncooperative.

The smaller temporal fields of the 5-year-olds relative to the older subjects are not likely to have been caused by greater fatigue, poorer attention, different response criteria, or poorer visual acuity. First, for all subjects included in the sample, fixation was steady enough at the end of each test to plot the blind spot. Second, subjects of all ages appeared to use similar criteria to indicate detection, at least in the center of the field: The size and location of the plotted blind spot did not differ significantly with age. Moreover, the criteria of the 5-year-olds also likely was similar to that of older subjects in at least one portion of the peripheral field: the superior field. Third, reliability was excellent and interocular differences were relatively small at all ages (see Tables 13 & 14; 4, 5, 8, & 9). Fourth, the standard errors were similar across all ages (see Tables 2 & 3).

Differences in visual acuity or stereoacuity between 5-year-olds versus older subjects also probably cannot explain the difference in the extent of their fields. Although 5-year-olds had generally poorer visual acuity and stereoacuity than did older subjects, their superior fields were as large. Further evidence that visual acuity may not influence field size comes from studies of

visually deprived eyes: In children treated for binocular cataracts, there is no correlation between the extent of the loss in peripheral vision and visual acuity (Bowering, Maurer, Lewis, Brent, & Brent, 1989).

Differences in the visual field between 5 years and older ages are not likely to be related to improvements in understanding of the task, attention, visual acuity, or stereoacuity. Rather, changes in the visual field with age may be related to development in any of the neural structures which have been shown to influence peripheral vision. In the primate, these structures include the retina, lateral geniculate nucleus, visual cortex, superior colliculus, frontal eye fields, and posterior parietal cortex (see Chapter 4). Little is known about the development of many of these structures in humans or even in primates, such as the superior colliculus or frontal eye fields, and, as such, it is difficult to determine whether they are contributing to the slow development of the visual field. In contrast, some structures, such as the retina and the lateral geniculate nucleus, are well developed by the schoolaged years (Abramov, Gordon, Hendrickson, Hainline, Dobson, & LaBossiere, 1982; Hickey & Peduzzi, 1987; Yuodelis & Hendrickson, 1986) and, therefore, unlikely to be contributing to the slow development of peripheral vision. The physiological basis of the observed behavior will be examined further in Chapter 4.

The results also indicated that adults had a significantly smaller superior field than did 7-year-olds. This is not the first

report that the rate of development of the superior visual field may be different than elsewhere in the visual field. For example, Heersema et al. (1989) reported that the superior visual field of infants and toddlers tested kinetically with a 6° light is fully developed many weeks before the other meridia. Similarly, constriction of the visual field, which begins during middle age (Suzumura et al., 1985), especially affects the superior visual field (Haas, Flammer, & Schneider, 1986). At this time, there is no physiological explanation of why the superior field develops early or is especially likely to be lost. Moreover, the adults assessed in this study were young. As such, it is reasonable to assume that they would have experienced minimal changes to their visual structures as a result of aging or environmental damage (such as from ultraviolet light) and that their all parts of their visual fields would be full. Rather, differences in the extent of the superior field with age may depend upon the physical structure of the brow, which, being more prominent in adults than in children, may block the adult's detection of light in the upper portion of the superior field. For this reason, the superior fields of the older children may be fuller than those of the adults. That physical dimensions influence the extent of the superior field is documented. For example, adults with adult-onset blepharoptosis (droopy eyelids) have restricted superior fields (Cahill, Burns, & Weber, 1987).

This research also provides some information about the relative development of each hemisphere in the early schoolaged

years. The small interocular differences detected suggest that the right hemisphere is developing at the same rate as the left hemisphere, as are the right and left eyes of individual subjects (Tables 4, 5, 8, & 9). Furthermore, the difference scores were fairly similar across age, except for the absolute interocular difference scores, indicating that there was not an age-related pattern of development. In addition, note that there was a large range of normal values at all ages for each meridian and light intensity, which was greater than the range of interocular differences (Tables 6, 7, 11, & 12). For patients treated for unilateral eye disorders, then, maximum sensitivity in diagnosis likely is obtained by comparing the field extent of the deprived eye to the performance of the fellow nondeprived eye.

Finally, in a clinical setting, children may be retested during each of several medical appointments in order to determine whether performance remains stable or changes, as may occur after diagnosis of an additional medical complication. There exists the concern that performance may improve because of repeated testing and the practice effect may mask actual losses of function. For this reason, evaluation of test-retest differences in normal subjects is important. There was excellent test-retest reliability at all ages (Tables 13 & 14), indicating that improvement with practice, at least for a small number of tests, is unlikely to affect the measured extent of the visual field. My research does not address the question of the effects on performance of practice spaced over a large number of tests.

In summary, the visual field is adult-like in extent by approximately 7 years of age when subjects are tested with a 6.4' light of either 31.8 or 318 cd/m^2, except for small differences in the extent of the temporal and inferior fields. The relatively slow development of peripheral vision may be attributed to maturation of any of the neural structures which mediate detection in the temporal periphery. Both the normative standards garnered in this study and the implication that neural development is continuing during early childhood are important for evaluating the impact of visual deprivation on the development of the visual field.

Experiment 2

Longitudinal development of the extent of the visual field in children

Experiment 1 indicated that 5-year-olds have immature fields but at 6 years of age, the visual field is virtually adult-like at most locations, except in the temporal field and in the inferior field where even 7-year-olds do not have full fields. However, in the superior field, 7-year-olds see further than do adults. To confirm the findings of the first experiment, the original sample of 5-year-olds from Experiment 1 was retested at 7 years of age.

Method

Subjects. Twenty-seven 7-year-olds were tested (mean age = 7.0 yrs, range 6.8 - 7.3 yrs) with one child from the original sample of 28 subjects lost to follow-up. All children successfully completed the field tests. However, five children exhibited poor visual acuity on the screening exam. Because subsequent ophthalmological examinations revealed no ocular pathology, the data from these children were included in the analyses.

Procedure. Each child first was evaluated with the screening procedure established in Experiment 1. Regardless of the child's performance on the screening examination, his visual fields were tested. However, if the child failed the screening exam (as defined by the criteria established for 7-year-olds tested in Experiment 1), he was referred to an ophthalmologist. If the ophthalmologist diagnosed an eye problem, then the child's data were eliminated from the analyses. Otherwise, all data were to be included in the analyses.

As in Experiment 1, the first eye was tested with the intense target (intensity 318 cd/m^2), the second eye with the intense target, the second eye with the dim target (intensity 31.8 cd/m^2), and, finally, the first eye with the dim target. The eye tested first at 7 years was the eye that was tested first at 5 years of age. All aspects of the procedure remained identical to Experiment 1 except that

subjects were not required to return for a second visit to assess test-retest reliability, which was high at all ages in Experiment 1.

Results

For each intensity, the mean extent of the field along each meridian for each test was calculated (for the means of test 1 for the 5- and 7-year-olds, see Table 15).

(1) Extent of the field at 5 and 7 years:

The first question was whether the measured fields were larger at 7 years of age than at 5 years of age in the same subjects. To answer the question, the data were analyzed with a 4-way ANOVA with age (two ages), test (two tests), intensity (two intensities), and meridian (eight meridia) as within subject factors (see Table 16).

(a) Effect of age:

The ANOVA showed a main effect of age: The subjects detected the target significantly further to the side at 7 years of age than at 5 years of age ($p < .00001$).

(b) Effect of meridian:

There was a main effect of meridian ($p < .00001$). Tukey's HSD revealed that the field was largest at 0° temporally (p's $< .01$). At 90°

superiorly, the field was smaller than at most other meridia, except for 225° inferiorly (p's < .01).

Also, there was an interaction of age and meridian (p < .00001). Planned comparisons indicated that the 7-year-olds saw further to the side than did the 5-year-olds at 0° temporally [$F(1,182)$ = 185.72, p < .00001], 90° superiorly [$F(1,182)$ = 18.21, p < .00001], 180° nasally [$F(1,182)$ = 61.35, p < .00001], and at 270° inferiorly [$F(1,182)$ = 63.53, p < .00001].

(c) Effect of intensity:

The extent of the field was influenced by target intensity, with a larger field obtained with the intense light (p < .00001). Also, intensity interacted significantly with meridian (p < .00001). Tukey's HSD Tests revealed that for the dim light, the field was smaller in the superior visual field than in other meridia (all p's < .01). For the intense light, the field was equally small in the superior and nasal fields, and smaller in the superior and in the nasal field than in other meridia (all p's < .01). However, the pattern of responding to each intensity of light did not differ significantly with age (p = .68).

In contrast, the overall pattern of responding to the different intensities at each meridian did vary significantly with age (p = .0124). Unplanned comparisons (Scheffe tests) indicated that at both 5 and 7 years of age, there was a significant interaction of intensity by meridian [$F(31,182)$ = 8.35, p < .00001; $F(31,182)$ = 10.79, p < .00001]. Tukey's tests indicated that at 5 years, the field was smaller

superiorly for tests with the dim light than elsewhere, except for the test of the dim light at 225° inferiorly (all p's < .05). At 7 years, the field was smaller superiorly for tests with the dim light than elsewhere (all p's < .05)

(d) Effect of test:

There was a main effect of test (p = .0035), with the field larger by less than a degree on the second test. There was no significant interaction of test by age (p = .50), test by meridian (p = .69), test by intensity (p = .80), test by age by meridian (p = .43), test by meridian by intensity (p = .31), test by intensity by age (p = .07), or test by age by intensity by meridian (p = .13).

(2) Extent of the field for the experienced 7-year-olds versus the inexperienced 7-year-olds and adults:

To determine how the field of these experienced 7-year-olds compared to the fields of the inexperienced 7-year-olds and the adults tested in Experiment 1, the data were analyzed with a 4-way ANOVA with group (three levels: experienced 7-year-olds, inexperienced 7-year-olds, adults) as a between subject factor and test (two tests), intensity (two intensities), and meridian (eight meridia) as within subject factors (see Table 17). Note that because the data are being reused, thus increasing the number of F tests for these data, there exists an increased likelihood that some results of this analysis may be significant by chance alone.

(a) Effect of age:

The ANOVA showed no main effect of age: Adults did not detect the target significantly further to the side than did either group of 7-year-olds (p = .63).

(b) Effect of meridian:

There was a main effect of meridian (p < .00001). Tukey's HSD test revealed that the largest extent was at the temporal meridian of 0° (p's < .01) and the smallest at the superior meridian of 90° (p's < .01).

Also, there was an interaction of age and meridian (p < .00001). Planned comparisons at the four principle meridia revealed that adults had a larger field than did the group of naive and experienced 7-year-olds at 0° temporally [$F(1,60)$ = 12.34, p = .0012] and 225° inferiorly [$F(1,60)$ = 6.47, p = .013] but had smaller fields at 90° superiorly [$F(1,60)$ = 16.23, p = .0004]. However, there was no difference in the extent of the field at 180° nasally [$F(1,60)$ = 1.01, p = .32]. At all locations except for 0° temporally, the difference in the extent of the field for the experienced 7-year-olds versus the inexperienced 7-year-olds was less than two degrees, a difference that may be within the realm of measurement error. Given that the temporal portion of the visual field is last to develop (see Experiment 1), a comparison of performance between the experienced and inexperienced 7-year-olds was planned for 0° temporally. At this location, a planned comparison revealed that the field for the

experienced 7-year-olds was significantly larger than the field for the inexperienced 7-year-olds by 3.4° [$F(1,60) = 5.22$, $p = .024$].

(c) Effect of intensity:

The measured extent of the field was influenced by target intensity, with a larger field obtained with the intense light ($p < .00001$). In addition, intensity interacted significantly with meridian ($p < .00001$). Despite this interaction, Tukey's HSD tests indicated that for either light, the field was smaller in the superior visual field (all p's $< .01$).

Although the pattern of responding to each intensity of light did not differ significantly with age ($p = .08$), there was a significant interaction of age by intensity by meridian ($p = .0242$). This significant three way interaction then was analyzed for each age group. First, for the adults, there was a significant interaction of intensity by meridian [$F(7,105) = 35.86$, $p < .00001$]. Tukey's HSD tests indicated that, for each light, the field was smallest superiorly (p's $< .01$). Second, for the experienced 7-year-olds, there was a significant interaction of intensity by meridian [$F(7,182) = 59.74$, $p < .00001$]. Tukey's HSD tests indicated that for tests with the intense light the field was smallest, and equally so, at 225° inferiorly and at 90° superiorly (p's $< .01$). For tests with the dim light, the field was smallest superiorly (p's $< .01$). Third, for the inexperienced 7-year-olds, there was a significant interaction of intensity by meridian [$F(7,133) = 32.65$, $p < .00001$]. Tukey's HSD tests indicated that for

tests with both lights the field was smallest, and equally so, at 225° inferiorly and at 90° superiorly (p's < .01).

(d) Effect of test:

There was a main effect of test (p = .0224), with the field larger on the second test by, on average, less than 0.5°. There also was a significant interaction of test by age by intensity by meridian (p = .0417). The three way interaction of test by intensity by meridian then was analyzed for each of the three age groups. For adults and for the experienced 7-year-old children, there was no significant interaction of test by intensity by meridian [$F(7,105) = 0.64$, $p > .05$; $F(7,182) = 0.99$, $p > .05$], thereby requiring no further exploration. In contrast, there was a significant three way interaction for the inexperienced 7-year-olds [$F(7,133) = 3.08$, $p = .0051$], which prompted analysis of the interaction of intensity by meridian at each of the two levels of test. For both tests 1 and 2 for the inexperienced 7-year-olds, there was a significant interaction of intensity by meridian [$F(7,133) = 7.99$, $p < .00001$; $F(7,133) = 26.69$, $p < .00001$]. Tukey's HSD test for test 1 revealed that for both lights the field was smallest, and equally so, at 225° inferiorly and at 90° superiorly (p's < .01). For test 2, the field for the dim light was smallest at 90° superiorly (p's < .01). For the intense light, the field was smallest, and equally so, at 225° inferiorly and at 90° superiorly (p's < .01). To summarize the results of the analyses of the significant interaction of age by test by intensity by meridian, there were significant

interactions of intensity by meridian for both tests 1 and 2 for the inexperienced 7-year-old children

There was no significant interaction of test by age (p = .57), test by meridian (p = .35), test by intensity (p = .10), test by age by intensity (p = .31), test by age by meridian (p = .11), or test by meridian by intensity (p = .46).

(3) Interocular difference scores:

The rationale and method for calculating interocular difference scores were presented in Experiment 1. I first will present the results from the calculation of the signed difference scores and then the absolute difference scores.

Regardless of intensity, the signed interocular difference scores of the experienced 7-year-old subjects were small, always less than or equal to 2°, which indicates that, on average, the size of the field is similar in the right eye and the fellow left eye (Table 18). Moreover, sometimes the scores were negative, indicating that the left eye had the larger field, while sometimes the scores were positive, indicating that the right eye had the larger field. Finally, the size of the interocular difference scores were similar for these experienced 7-year-olds and the inexperienced 7-year-olds tested in Experiment 1 (see Table 18).

It is important to determine the maximum size of the interocular differences between a normal right eye and the fellow normal eye because these values aid in evaluating whether

interocular differences in patients treated for a unilateral cataract are abnormal. As in Experiment 1, the most extreme score, representing the most aberrant 5% of scores, was eliminated. The second largest interocular difference (the difference between the extent of the field for the right eye minus the extent of the field for the fellow left eye) for each meridian and light intensity is presented in Table 19. The interocular differences of the experienced 7-year-olds were large: For tests with the dim light, scores ranged from 8° at 270° inferiorly to 12° at 0° temporally. For tests with the intense light, scores ranged from 7° at 0° temporally to 13° at three locations. This indicates, then, that substantial differences in the extent of the field between two related normal eyes may occur. Note that these values were similar to the values obtained by the inexperienced 7-year-olds tested in Experiment 1 (see Table 19).

The absolute interocular difference scores for the experienced 7-year-olds ranged from 2.9° to 5.6° for the dim light and from 2.5° to 5.7° for the intense light (Table 20). The absolute scores indicate that there was a tendency for one eye to have a larger field than the fellow eye regardless of whether the eye with the larger field was consistently the right (or the left) eye. Note, though, that the absolute scores for the experienced 7-year-olds were similar to those of the inexperienced 7-year-olds tested in Experiment 1 (see Table 20).

(4) Range of the extent of the field:

As indicated in Experiment 1, patients tested in a clinical setting typically have only one test of their visual field. In the normal subjects, the range of the extent of the visual field for only test 1 will be assessed. These normative values then may be used to determine if differences in the extent of the field in a patient treated for unilateral cataracts reflect abnormality. To eliminate the most extreme 5% of scores, the smallest extent of the field for each meridian and intensity for test 1 was discarded. The range of the field was calculated as the largest extent of the field minus the remaining smallest extent of the field for each meridian and light intensity in test 1 (Table 21). The potential range of the field in normal subjects was large: For the dim light, there was a maximum difference of 24° at 0° and 315° temporally for the experienced subjects. For the intense light, the maximum value for the experienced subjects was 20° at 270° inferiorly. The range of values obtained by the experienced subjects typically was smaller than those found for the inexperienced 7-year-olds (Table 21).

(5) Analysis of the blind spot:

As in Experiment 1, the area and location of the blind spot was calculated for each subject. The area of the blind spot was calculated by the formula for the area of an ellipse: area = pi * major radius * minor radius. The location was the temporal and inferior coordinates of the center of the blind spot. The area and the location of the blind

spot for the experienced 7-year-olds was compared to that of the inexperienced 7-year-olds and the adults using a 2-way ANOVA with a between factor of group (inexperienced 7-year-olds versus adults versus experienced 7-year-olds) and a within factor consisting of the area of the blind spot and the temporal and inferior coordinates. There was no significant effect of group [$F(2,60) = 0.19$, $p = .82$] or of the interaction of group by the parameters of the blind spot [$F(4,120) = 0.22$, $p = .93$]. Not surprisingly, there was a significant effect of the parameters of the blind spot [$F(2,120) = 721.61$, $p < .00001$]: on average, the blind spot was 392.0 mm^2 in area and located 15.6° temporally and 2.7° inferiorly in the visual field.

Discussion

The major findings of Experiment 1 were replicated in Experiment 2. First, the visual field increases from 5 to 7 years of age in the same subjects. This parallels the finding from Experiment 1 that the visual field increased from 5 to 7 years in different groups of subjects. Moreover, the field is approximately adult-like by 7 years of age, with three exceptions. That is, compared to adults, 7-year-olds do not see as far temporally or inferiorly but see further superiorly. A new finding was that the fields are of the same size in experienced and in inexperienced 7-year-olds, except for a small difference at 0° temporally.

Also, findings from Experiment 1 about the effects of meridian, intensity, and interocular differences were replicated. As in Experiment 1, it was found that the field was largest at 0° in the temporal periphery and, typically, smallest at 90° in the superior visual field. Moreover, the field was largest when measured with the intense light. Furthermore, for each ANOVA in Experiments 1 and 2, there was a significant interaction of meridian and intensity, with the field smallest in the superior field but sometimes equally small in the nasal field. Finally, given that the magnitude of interocular differences were small, it appears that no differences exist in the rate of development of the hemispheres in the experienced 7-year-olds. Moreover, because these values were similar to those of the inexperienced 7-year-olds tested in Experiment 1 (see Table 18) this indicates that there was a similar pattern in the rate of hemispheric development in both groups of subjects.

Some discrepancies between the results of the two experiments did arise. In Experiment 1, there was not a significant main effect of test, although the interactions of test by intensity and test by intensity by age by meridian were significant. Because the original sample of 7-year-old children in Experiment 1 did not improve with practice, the 7-year-old children in Experiment 2 were not expected to show a practice effect. Yet, there was a significant main effect of test in each ANOVA in Experiment 2, although no significant interactions involving test except for the 4-way interaction of test by age by intensity by meridian in the comparison of the 7-year-olds

and adults (see Tables 16 & 17). Note that though the main effect of test in Experiment 2 was statistically significant, the increase in field size of, on average, less than a degree from test 1 to test 2 is not significant practically. Much greater variability than one degree typically is found both between the two eyes of one subject and across subjects of the same age (see Tables 19 & 21). When evaluating the fields of children treated for a cataract, then, the minimal effect of test may not be of much consequence.

Less important discrepancies between the results of Experiments 1 and 2 also occurred. For the first experiment and for the comparison of the two groups of 7-year-olds and adults in Experiment 2, there was a significant interaction of age by test by intensity by meridian, which was not found in the comparison of 5- and 7-year-olds in Experiment 2 (see Tables 16 & 17). Although the 4-way ANOVA of Experiment 1 was not interpreted statistically, the analysis of the significant 4-way ANOVA of Experiment 2 involving the two groups of 7-year-olds and adults revealed that there were significant interactions of intensity by meridian at both levels of tests for the inexperienced 7-year-olds. As well, there were significant interactions of intensity by meridian at each level of age in Experiment 2 (see Tables 16 & 17) but no significant interaction of intensity by meridian by age in Experiment 1.

In summary, Experiment 2 replicated the main findings of Experiment 1 with both experiments allowing the conclusions that the measured visual field does increase from 5 to 7 years of age and,

compared to adults, 7-year-olds do not see as far temporally or inferiorly but see further superiorly. Moreover, the fields of experienced and inexperienced 7-year-olds are of the same size, except for 0° temporally. The replication of results strengthen the generalizability of the conclusions from Experiment 1 and strongly suggests that there indeed is development of the visual field during early childhood when subjects are tested with a small stimulus. Moreover, the replication of findings provides greater confidence in the use of the normative data for the evaluation of the extent of the visual field in children treated for dense and central cataracts.

Experiment 3
The development of sensitivity across the visual field during childhood

Static perimetry allows assessment of thresholds across the visual field and also can indicate the boundary of the visual field for that particular stimulus. As such, the results from tests using static perimetry complement the results from kinetic perimetry on the Goldmann perimeter presented in Experiments 1 and 2 and provide further information about the normal development of peripheral vision. Because of the brevity of trials, static perimetry is the preferred method for assessment of peripheral vision in children with nystagmus (jiggly eye movements), a condition which

frequently occurs in patients treated for cataracts. As such, the gathering of normative data using the technique of static perimetry is especially important.

Relatively little is known about the normal development of sensitivity as assessed with static perimetry because most investigators have tested only along the horizontal meridian (Allan, 1971; Aspinall, 1967, 1976; Lakowski & Aspinall, 1969; Liao, 1973; Matsuo et al., 1974; Tomonaga, 1974; Whiteside, 1976). The sole study to examine development along several meridia of the visual field assessed only a few subjects and used large stimuli (Cummings et al., 1988). Examination of these studies suggests three conclusions. First, studies of peripheral vision using small stimuli have produced limited and contradictory information regarding the age at which adult-like thresholds are reached (e.g., Lakowski & Aspinall, 1969; Tomonaga, 1974). Given that the extent of the visual field requires many years to develop (see Experiments 1 and 2), then sensitivity across the visual field also may be slow to develop. Second, these studies of children, like those of adults, do agree that visual sensitivity decreases with increasing eccentricity from the center of the visual field (Allan, 1971; Aspinall, 1967; Aulhorn & Harms, 1972; Fahle & Schmid, 1988; Frisen & Glansholm, 1975; Lakowski & Aspinall, 1969; Liao, 1973; Matsuo et al., 1974; Tomonaga, 1974; Whiteside, 1976). Moreover, there are differences in sensitivity in the nasal versus in the temporal hemifield. For example, one-month-old infants detect a narrower line located at 30° temporally than at

20° nasally. In contrast, 2-month-old infants exhibit better detection at 20° nasally (Lewis et al., 1985). Better detection at 20° nasally than at 30° temporally is the pattern typically shown by children (Allan, 1971; Liao, 1973) and adults (Allan, 1971; Aspinall, 1967; Fahle & Schmid, 1988; Frisen & Glansholm, 1975; Lakowski & Aspinall, 1969; Liao, 1973).

To investigate the normal development of sensitivity across the visual field, static perimetry was performed with the Octopus 500 perimeter, a standard clinical tool (see Procedure), on large groups of 7-, 8-, and 9-year-olds, and adults. Based on the previous literature in normal infants and in children treated for cataracts, I also chose to examine in more detail thresholds at 20° nasally and at 30° temporally. As well, interocular differences and reliability were assessed at each age by testing both eyes of each subject and by retesting one eye, respectively. To date, there have been two reports of the reliability of measurements with children, and these in only a few children (Cummings et al., 1988; Liao, 1973), and no report of interocular differences.

Method

Subjects. The final sample of subjects consisted of 20 7-year-olds (mean age = 7.0 yrs, range = 6.9 - 7.2 yrs), 20 8-year-olds (mean age = 8.0 yrs, range = 7.8 - 8.2 yrs), 12 9-year-olds (mean age = 9.0

yrs, range = 8.8 - 9.2 yrs) and 20 adults (mean age = 26.5 yrs, range = 16.9 - 37.7 yrs).

To be included in the final sample, subjects had to pass a screening exam and complete validly all required tests of the visual field (see Procedure). As in Experiment 1, subjects passed the screening exam if they had a Snellen acuity at 6 meters of at least 20/20 in each eye, poorer acuity for each eye with a +3 D lens than without it (to control for hyperopia greater than 3 D), fusion at 6 meters on the Worth Four Dot Test, and a stereoacuity of 40 seconds on the Titmus Fly Test. These criteria were chosen because even normal children younger than 7-year-olds typically achieve a monocular visual acuity of at least 20/20 (Weymouth, 1963), a stereoacuity of 40 seconds (Heron et al., 1985; Romano et al., 1976), and likely are able to fuse (Birch et al., 1985; Braddick et al., 1980). Finally, a field test was considered invalid if the subject failed more than 50% of either the false positive or false negative catch trials (see the section on Programs for definition of these terms).

An additional 13 subjects were excluded from the final sample because they did not pass the screening examination (n = 4 7-year-olds, 2 8-year-olds, 1 9-year-old, and 1 adult), the field tests were invalid (n = 2 7-year-olds) or incomplete (n = 2 7-year-olds), or because of malfunction of the computer (n = 1 9-year-old).

Apparatus. The Octopus 500 perimeter (see Figure 8) was used to assess sensitivity across the visual field (see Bebie, Fankhauser,

Jenni, Rhyner, Weijland, 1983; Silverstone & Hirsch, 1986; Whalen & Spaeth, 1985).

Stimuli and lighting conditions of the bowl. The range of the target intensities was 5.1 log units, from a minimum intensity of 0.008 apostilbs (asb) (0.0025 cd/m^2) to a maximum intensity of 1000 asb (318 cd/m^2). Thresholds for the Octopus are expressed in decibels (db), with 10 db equaling one log unit. The decibel log scale is related inversely to the intensity scale, with the lowest target intensity of 0.008 asb equalling 50 db and the highest intensity of 1000 asb equalling 0 db (see Table 22). A loss of sensitivity of 20 db is equivalent to a loss of 2 log units, which means that the stimulus must be 100 times more intense than normal for the subject to detect it (Silverstone & Hirsch, 1986).

The target diameter was 0.43° (0.25 mm^2 or 25' when viewed from 45 cm), the stimulus duration was 0.1 seconds, and the background illumination of the perimeter was 4 asb (1.3 cd/m^2). The location of the light within the bowl on each trial was determined randomly by the computer.

Programs of the Octopus perimeter.

Each subject was tested with a fast bracketing procedure (program #24) and a normal bracketing procedure (program #62) (Bebie et al., 1983; Bebie, 1985; Fankhauser, Bebie, & Flammer, 1988, Whalen, 1985), with only the latter type of program precisely

measuring each threshold. Because children are unlikely to have the stamina and attention span required for the precise measurement of each location across the entire field, the fast bracketing program was chosen for assessment of the whole visual field while selected areas in the field were precisely measured with the normal bracketing program.

The testing strategy labelled fast bracketing determined whether the subject's sensitivity at a particular location was normal or not relative to normative data in adults collected previously by the manufacturers of the Octopus perimeter. Only if the threshold was abnormal, defined as a loss of sensitivity of more than 4 db relative to the normative threshold in adults at that location, did the Octopus perimeter quantify the amount of loss. This measurement was carried out by bracketing the abnormal threshold, first in steps of 4 db and then in steps of 2 db of stimulus luminance. The threshold was the average of the intensity of the last detected and undetected stimuli. This procedure required from two to five stimulus presentations at each test location. The fast bracketing program (program 24) assessed sensitivity at intervals of 15° across the field, which produced thresholds for 13 locations in the central field (0° - 30°), 36 locations in the paracentral field (30° - 60°) and 27 locations in the peripheral field (60° - 84°).

In contrast, the testing strategy labelled normal bracketing used the double bracketing procedure to measure precisely the subject's threshold at each location, whether the threshold was

abnormal or not. This procedure required about five stimulus presentations at each test location. The normal bracketing program (program 62) measured sensitivity precisely at 21 locations, each location separated by 3°, in any 12° by 12° area within the visual field.

At the start of each trial, the computer beeped to alert the subject, regardless of whether or not the trial was a real trial or a catch trial. A positive catch trial was a trial on which the computer signalled the presentation of a stimulus by beeping but no stimulus was presented. If the subject responded, likely because he either did not understand the instructions or could not inhibit his responding, then this was a "false positive error". In contrast, a negative catch trial was a trial on which there was an alerting beep followed by the presentation of the maximum intensity stimulus (1000 asb) at a location where the subject previously had indicated detection of a stimulus. If the subject failed to respond to the stimulus, likely because of loss of attention, then this was termed a "false negative error". Approximately 10% of all trials were positive or negative catch trials. Because the computer reported each error as it occurred, I could stop the test temporarily and caution the subject. However, if the subject failed more than 50% of either the positive or negative catch trials on a test, then the test was completed but ruled invalid and then repeated.

Procedure. I explained the procedure to the subject and obtained parental and/or individual consent. The subject was tested with standard clinical procedures (Whalen & Spaeth, 1985). The subject had one eye patched, was seated in front of the Octopus hemisphere with his chin resting on the adjustable chin rest and his forehead against the forehead bar, and was asked to fixate the central fixation aid.

I then set the perimeter's electronic probe area to record and monitor the position of the subject's pupil as he fixated the fixation target. A loss of fixation occurred when the probe field was not coincident with the subject's pupil and the computer then discarded and later repeated that trial. Also, the perimeter's videocamera transmitted an enlarged picture of the subject's eye to the monitor. By watching the monitor, I was able to detect whether the subject moved his eye during the trial and, as necessary, I instructed the subject to refixate. Loss of fixation may arise because the subject is fatigued or inattentive or because of nystagmus (uncontrollable, jiggly eye movements). When the subject appeared to be fatigued or inattentive, I halted the test for a few minutes.

After the subject had adapted to the lighting conditions of the perimeter for 5 minutes, he was instructed to maintain fixation on the central fixation aid, to listen for the alerting beep which would signal the onset of a new trial, and to push a button if he was aware of a flash of light in the bowl. The subject was warned that some

trials were catch trials during which there either was no stimulus or an intense light would be presented.

Adults and 7-year-olds were tested on two visits. Because of the amount of testing required, the procedure then was shortened for the 8- and 9-year-olds so as to require only one visit. The following describes the procedure for the 7-year-olds and the adults. On the first visit, each subject was given a screening exam. If the subject passed the screening exam, one eye was tested at 20° in the nasal visual field and at 30° in the temporal field with the normal bracketing procedure (program 62) followed by the same ordering of the two tests for the second eye. Then, the whole visual field of the first eye was tested with the fast bracketing procedure (program 24). The order of presentation of the tests at 20° in the nasal visual field and at 30° in the temporal field was counterbalanced across subjects, as was the eye which was tested first. On the second visit, the first eye was tested at 20° in the nasal visual field and at 30° in the temporal field with the normal bracketing procedure (program 62) with the order of the tests remaining the same as during the first visit, in order to allow assessment of reliability. Then, one eye was tested with the fast bracketing program (program 24). For half the subjects this was the right eye and for the other half this was the left eye, to allow the assessment of reliability or of interocular differences. Finally, the first eye was tested at 0°, and at 30° and 45° in the nasal visual field, and at 15° and 45° in the temporal visual field with the normal bracketing program (program 62). These

locations were chosen to allow assessment of sensitivity at several locations along the horizontal meridian. To avoid the confound of fatigue when interpreting the results, these tests were randomly ordered with the constraint that the most peripheral locations not be tested last and the 0° location not be tested first.

The procedure for 8- and 9-year-old subjects required one visit. The subjects completed all of the above tests in the prescribed order during one session, except for the second test of the fast bracketing program (program 24) and the tests centered at 0°, and at 30° and 45° in the nasal visual field, and at 15° and 45° in the temporal visual field, scheduled for the second visit Although the amount of testing was shortened for the 8- and 9-year-olds, reliability and interocular differences could still be assessed by comparing data from the repeat tests centered at 20° nasally and at 30° temporally.

Data analysis. For several reasons, it is appropriate both to express thresholds logarithmically, rather than linearly, and then to conduct an ANOVA using these logarithmic values.

First, it is appropriate to express thresholds logarithmically because sensitivity to visual stimuli is believed to operate logarithmically and not linearly (Hurvich & Jameson, 1966). The relationship between the probability of detecting a stimulus and the luminance of the stimulus is not linear but curvilinear. At low levels of stimulus intensity, as the intensity of the stimulus increases, the

probability of detecting the stimulus increases rapidly. However, at high levels of stimulus intensity, as the intensity of the stimulus increases, the probability of detecting the stimulus increases slowly. The relationship between the probability of detecting the stimulus and the luminance becomes linear if there is a logarithmic transformation of the luminance of the stimulus. Fechner's Law expresses this relationship as $B = k * \log L$; that is, the just discriminable brightness (B) is equal to a constant (k) multiplied by the log of the stimulus luminance (L).

Second, the aim of an examination using the Octopus perimeter is to compare the sensitivity of an individual with suspected ocular pathology to the sensitivity of a normal individual. The loss in sensitivity of the patient then is expressed logarithmically in decibels. By expressing the loss of threshold logarithmically, it is easy to interpret the meaning of the loss of sensitivity. For example, if the loss of sensitivity in the patient is 20 db or 2 log units, then the patient must be shown a stimulus 100 times more intense than the normal person before the patient gives evidence of detection of a stimulus (see Silverstone & Hirsch, 1986). Expression of sensitivity using a log scale allows ease of interpretation of the results from patients.

Third, the Octopus perimeter measures thresholds using a light which can range over 5 log units in intensity: from .01 asb (a dim light) to 1000 asb (an intense light). Because the range is so large, it is convenient to use a log scale, and not a linear scale, to express the

thresholds (see Silverstone & Hirsch, 1986). For these reasons, then, it is both theoretically appropriate as well as convenient to conceptualize sensitivity on a logarithmic scale.

It also is appropriate to analyze statistically thresholds which have been expressed logarithmically. A log scale does not have a zero point and, therefore, is not an example of a ratio scale but of an interval scale. There exists the concern that it is inappropriate to average data when they are not ratio in scaling. Yet, statistical texts advise using log transforms to deal with non-normal or heterogeneous data (Winer, 1971) and these transformed data then are manipulated statistically. The conclusion, then, is that it is not inappropriate statistically to average logs and analyze them by an ANOVA. In fact, experiments assessing auditory sensitivity typically use the decibel to express intensity and routinely compute averages of the decibel; e.g., Dadson & King (1952).

The thresholds for tests with the normal bracketing program (program 62) centered at 0° centrally, 20° and 30° and 45° in the nasal field, and at 15°, 30° and 45° in the temporal field each were the mean of five thresholds: the primary location and ± 3° displaced horizontally and ± 3° displaced vertically.

With regard to the test of the entire visual field (program 24), location was divided so as to compare performance in the center of the field (0°) relative to more and more peripheral areas of the field (near, mid, and far periphery). The threshold for 0° was the sole measurement located precisely at 0°. By convention, the near

periphery was defined as being that part of the visual field from the center out to 30° (Harrington, 1981). The threshold for the near periphery, then, was the average of the 11 thresholds located at 15° nasally, 30° nasally, 30° temporally, 15° superiorly, 30° superiorly, 15° inferiorly, 30° inferiorly, 15° nasally and superiorly, 15° temporally and superiorly, 15° nasally and inferiorly, 15° temporally and inferiorly. Note that the threshold for 15° temporally was omitted from this analysis because this area contains the blind spot. The threshold for the midperiphery was the average of the nine thresholds located at 45° nasally, 60° temporally, 45° temporally, 45° superiorly, 45° inferiorly, 30° nasally and superiorly, 30° temporally and superiorly, 30° nasally and inferiorly, and 30° temporally and inferiorly. Finally, the threshold for the far periphery was the average of the remaining nine thresholds located at 60° nasally, 75° temporally, 90° temporally, 60° superiorly, 60° inferiorly, 75° inferiorly, 45° temporally and superiorly, 45° temporally and inferiorly, and 60° temporally and inferiorly. Note that there were more data from the temporal field than from the nasal field for the calculation of the average threshold for the far periphery but this reflects the fact that the temporal quadrant is larger than other quadrants of the visual field.

Results

I first will examine the development of sensitivity at 20° nasally and 30° temporally, then the size of interocular differences, the range of values, and the test-retest reliability for these measurements. Then, I will examine the development of sensitivity elsewhere along the horizontal meridian. Finally, I will examine the development of sensitivity across the visual field, test-retest reliability, interocular differences, and the range of values for these measures.

(1) Development of sensitivity at 20° nasally versus 30° temporally:

The development of sensitivity at 20° nasally and at 30° temporally was analyzed with a 3-way ANOVA with age (4 levels: 7-, 8-, 9-year-olds, and adults) as a between group factor and location (2 levels: 20° nasally and 30° temporally) and test (3 levels) as within group factors (see Table 23).

(a) Effect of age:

There was no significant main effect of age: Adults did not detect dimmer lights than did either 7-, 8-, or 9-year-old children ($p = .87$).

(b) Effect of location:

There was a significant main effect of location (p = .0110), with sensitivity higher at 20° nasally than at 30° temporally by nearly 0.4 db. However, there was no significant interaction of age and location (p = .65).

(c) Effect of test:

There was no significant main effect of test (p = .13), or significant interaction of age and test (p = .96), location and test (p = .67), or age and location and test (p = .51).

In summary, adults did not detect dimmer lights at 20° nasally or at 30° temporally than did children 7, 8, or 9 years of age. Moreover, there was no significant effect or interaction of test. The only significant finding was that sensitivity at 20° nasally was slightly higher than at 30° temporally.

(2) Interocular differences at 20° nasally and at 30° temporally:

It also is appropriate to compare interocular differences in sensitivity at 20° nasally and at 30° temporally in each subject. These differences scores are a measure of the normal variation in sensitivity present in two related eyes and will be useful for evaluation of the significance of differences in sensitivity between related eyes in children treated for a unilateral cataract. The rationale for calculating the difference scores was explained in Experiment 1.

The signed interocular difference scores, for tests centered at 20° nasally and at 30° temporally, were calculated as the difference in sensitivity for the subject's left eye (either test 1 or 2) from the sensitivity for the same subject's right eye (the remaining test, either test 1 or 2). The average interocular scores and the standard errors are presented in Table 24. For each location, a negative score indicates that the left eye was more sensitive than the right eye while a positive score indicates that the right eye was more sensitive. The average scores hovered around 0 db and were similar across location and age, indicating that sensitivity was similar in both eyes.

To verify this statement, the data were analyzed by a 2-way ANOVA with a between factor of age (7-, 8-, 9-year-olds, adults) and a within factor of location (20° nasally, 30° temporally). There was no significant main effect of age (p = .68), location (p = .64), or interaction of age by location (p = .78) (see Table 25).

It is important to examine not only the average interocular difference in sensitivity but also to determine the range of such interocular differences. An interocular difference in sensitivity in a patient treated for a unilateral cataract which is greater than this value will indicate abnormality.

Regardless of sign, the largest difference score, which represented the most aberrant 5% of scores, at each location for each age was eliminated. The remaining largest scores are presented in Table 26. For tests at 20° nasally, the largest interocular difference was -4.4 db for the 7-year-olds while the smallest difference was

-1.6 db for the 9-year-olds. For tests at 30° temporally, the largest interocular differences was -8.4 db for the 7-year-olds and the smallest difference was 2.8 db for the 9-year-olds. These results indicate that substantial differences in sensitivity did occur between two related eyes but, generally, the interocular difference scores appeared similar across age.

The absolute interocular difference scores were the averages of the absolute values of the signed difference scores, calculated previously (Table 27). Because the absolute difference scores were small and appeared similar across age, no statistical analysis was conducted. For 20° nasally, the largest absolute differences scores were 1.5 db for 7- and 8-year-olds while the smallest score was 0.77 db for 9-year-olds. For 30° temporally, the largest absolute differences score was 2.6 db for 7-year-olds while the smallest score was 1.4 db for adults.

(3) Range of differences of sensitivity for 20° nasally and for 30° temporally:

It also is important to assess the size of differences in sensitivity across subjects at 20° nasally and at 30° temporally. This information permits evaluation of the amount of variation present in a normal population. The smallest threshold (least sensitive value) at 20° nasally and at 30° temporally at each age was eliminated and the range of sensitivity was calculated as the largest threshold minus the remaining smallest threshold (Table 28). The range was large. For

20° nasally, the largest difference was 10.6 db for the 8-year-olds and the smallest difference was 4.4 db for the 9-year-olds. For 30° temporally, the largest difference was 16.0 db for the 7-year-olds and the smallest difference was 5.4 db for the adults.

(4) Test-retest reliability for 20° nasally and 30° temporally:

Test-retest differences for individual subjects (Table 29) were calculated by averaging across subjects the differences in sensitivity at 20° nasally (and, in a separate analysis, at 30° temporally) for the repeat test (test 3) from the first test of the same eye (test 1). Test-retest reliability was high at 20° nasally and at 30° temporally for each age. For 20° nasally, the largest difference was -0.87 db for the adults. For 30° temporally, the largest difference was -1.1 db for the 7-year-olds.

(5) Development of sensitivity along the horizontal meridian:

The development of sensitivity along the horizontal meridian was analyzed using a 2-way ANOVA with age (2 levels: 7-year-olds and adults) as a between group factor and location (7 levels: 0°; 20°, 30°, and 45° nasally; 15°, 30°, and 45° temporally) as a within group factor. Note that neither the 8- or 9-year-olds were tested along the horizontal meridian at locations other than 20° nasally and 30° temporally. Because the previous analysis revealed no significant main effect or interaction of test, the data used in this analysis were

from the first tests centered at 20° nasally and at 30° temporally (see Table 30).

(a) Effect of age:

There was no significant main effect of age: Adults did not detect dimmer lights than did 7-year-olds ($p = .57$).

(b) Effect of location:

There was a significant main effect of location ($p < .00001$). Because I was interested in comparing sensitivity across the horizontal meridian, I chose to use post hoc testing to compare all areas. Tukey's HSD revealed that the lowest sensitivity was near the blind spot, at 15° temporally (all p's $< .01$) and the greatest sensitivity at 0° in the central visual field (all p's $< .05$). Sensitivity decreased from the center to the periphery, at least in the nasal visual field. For example, sensitivity was higher at both 20° nasally and at 30° nasally than at 45° nasally (p's $< .01$). However, in the temporal field, sensitivity was not significantly higher at 30° than at 45°. (Note that it is not reasonable to compare sensitivity at 15° temporally to elsewhere along the horizontal meridian because 15° temporally is within the area of the blind spot where decreased sensitivity is expected.) Finally, comparison across the hemifields revealed that while sensitivity at 45° temporally was higher than at 45° nasally ($p < .01$), there was no significant difference between 30° temporally and 30° nasally.

In addition to the significant main effect of location, there was a significant interaction of location and age ($p < .00001$). Simple effects indicated that adults and 7-year-olds were equally sensitive at 45° nasally [$F(1,202) = 0$, $p = 1.0$], 30° nasally [$F(1,202) = 0.22$, $p = .64$], 20° nasally [$F(1,202) = 0.61$, $p = .44$], 30° temporally [$F(1,202) = 1.69$, $p = .20$], and 45° temporally [$F(1,202) = 1.69$, $p = .20$]. However, compared to 7-year-olds adults were more sensitive at 0° [$F(1,202) = 8.83$, $p = .003$] but less sensitive at 15° temporally [$F(1,202) = 57.79$, $p < .0001$].

In summary, sensitivity was highest at 0° and gradually decreased with increasing eccentricity, at least for the nasal field. The same pattern was not evident in the temporal field, which may reflect limited sampling of locations. Finally, adults were more sensitive at 0° compared to 7-year-olds but the reverse finding was true at 15° temporally (the area of the blind spot).

(6) Sensitivity across the whole visual field:

Because the strategy used in assessing the whole field was fast bracketing, the test measured precisely only those thresholds which were abnormal relative to the thresholds of adults in the manufacturer's normative sample, defined as a local loss of sensitivity of more than 4 db. In cases where the program did not measure precisely, the subject's threshold was the intensity of the dimmest light actually detected by the subject at that location.

To examine sensitivity across the whole visual field, the data first were summarized as a range of values. Data were tabulated from 0° at 15° intervals along four meridia: the horizontal meridian (from 60° nasally to 90° temporally), the vertical meridian (from 60° superiorly to 75° inferiorly), and the two diagonal meridia (from 30° superiorly and 30° nasally to 60° inferiorly and 60° temporally; from 30° inferiorly and 30° nasally to 45° superiorly and 45° temporally). The least sensitive threshold at each location for each age group was eliminated. The ranges and the percentages of normal thresholds at each location for tests 1 and 2 are presented in Tables 31 to 34. Note that the whole visual field was tested once for the 8- and 9-year-olds but tested twice for the 7-year-olds and adults.

For each of the four meridia assessed on the first test of the visual field, most subjects at each age fell within the normal range for adults, at least in the center of the field. Fewer subjects were within the normal range for adults when tested at the edge of the field relative to more central areas, indicating that there may be more variability in sensitivity at the edges of the field. For example, 21% of 7-year-olds had adultlike sensitivity at 60° nasally while 100% of the same children exhibited adult-like sensitivity at 15° nasally (Table 28). Note, too, that there was a surprisingly low percentage of normal thresholds for tests centered at 0° and at 15° temporally (Tables 31-34). For example, only 68% of adults had normal sensitivity at 0° (Table 31). Finally, the results for the second

test of the whole visual field for the 7-year-olds and the adults typically replicate the results from the first test (Tables 31-34).

The development of sensitivity across the visual field then was analyzed using two different ANOVA's. The first analysis was designed to examine the reliability of the performance of the 7-year-olds and the adults on the two tests of the visual field. Because the whole visual field of the 8- and 9-year-olds was tested only once, their data could not be included in this analysis. The second analysis was designed to compare the performance across the field of the 7-, 8-, and 9-year-olds and adults.

The first analysis was a 3-way ANOVA with a between group factor of age (2 levels: 7-year-olds and adults) and within group factors of test (2 levels) and location (4 levels: 0°, near periphery, midperiphery, and far periphery) (see Table 35).

(a) Effect of age:

There was a significant main effect of age: Adults were significantly more sensitive than 7-year-olds by nearly 1.9 db (p = .0014).

(b) Effect of test:

There was no significant main effect of test (p = .77), age by test (p = .52), or age by test by location (p = .39).

(c) Effect of location:

There was a significant main effect of location ($p < .00001$). Tukey's HSD tests showed that sensitivity was highest at 0° (all p's < .01), and then decreased from the near to the mid to the far periphery (all p's < .01). Finally, there was a significant interaction of age by location ($p < .00001$). Analysis of simple effects showed that adults were more sensitive than 7-year-olds at 0° [$F(1,142) = 43.98$, $p < .0001$] but the two groups were equally sensitive in the near periphery [$F(1,142) = 0.004$, $p = .95$], midperiphery [$F(1,142) = 0.71$, $p = .40$], and far periphery [$F(1,142) = 0.80$, $p = .37$].

In summary, the significant findings were that adults were more sensitive that 7-year-olds but only at 0°. As well, sensitivity decreased from the center to the periphery of the field.

The second analysis examined the development of sensitivity across the visual field for all age groups. The 2-way ANOVA consisted of a between group factor of age (4 levels: 7-, 8-, and 9-year-olds, and adults) and a within group factor of location (4 levels: 0°, near periphery, midperiphery, and far periphery) (see Table 36). Because there was no significant main effect or interaction of test in the previous analysis of the whole visual field of the 7-year-olds and adults, only the data from test 1 for the 7-year-olds and adults were used for this analysis.

(a) Effect of age:

There was a significant main effect of age ($p < .0007$). Planned comparisons were conducted so as to trace the development of sensitivity at successively older ages and then to use these data as norms. Planned orthogonal comparisons indicated that 7-year-olds were as sensitive as the group of 8-year-olds, 9-year-olds, and adults [$F(1,68) = 0.18$, $p = .67$]. In contrast, 8-year-olds were not as sensitive as the group of 9-year-olds and adults [$F(1,68) = 6.57$, $p = .0126$], nor were 9-year-olds as sensitive as adults [$F(1,68) = 10.56$, $p = .0018$].

(b) Effect of location:

There was a significant main effect of location ($p < .00001$). Tukey's HSD tests showed that sensitivity was highest in the near periphery (all p's $< .01$), then at 0° (p's $< .01$), then in the midperiphery ($p < .01$), and lowest in the far periphery.

Finally, there was a significant interaction of age by location ($p < .00001$). Analysis of simple effects showed that the four age groups were equally sensitive in the near periphery [$F(3,269) = 0.04$, $p < .99$], midperiphery [$F(3,269) = 0.04$, $p < .96$], and far periphery [$F(3,269) = 0.74$, $p < .53$], but not equally sensitive at 0° [$F(3,269) = 40.64$, $p < .0001$]. Because there has been no previous examination of the development of sensitivity at 0° centrally, I chose to use Tukey's HSD tests. Tukey's HSD tests revealed that at 0° adults were more sensitive than 7-year-olds ($p < .01$), and that 7-year-olds were more

sensitive than either the 8- or the 9-year-olds (p's < .01). There was no significant difference in performance between the 8- and the 9-year-olds.

(7) Test-retest reliability for the entire visual field:

Test-retest reliability at each location for the 7-year-olds and adults was calculated by subtracting the threshold for the repeat test (test 2) from the threshold for the first test of the same eye (test 1) (see Table 37). Test-retest reliability was high at each location for both the 7-year-olds and the adults, with the poorest reliability found at 0° for the 7-year-olds.

(8) Interocular difference scores for the whole visual field:

Finally, it is appropriate to calculate interocular difference scores for the test of the whole visual field. These scores are important because they provide a baseline of normal variation by which to interpret the data from children treated for unilateral cataract. However, these scores should be interpreted with caution given that only half of the 7-year-olds and the adults, and none of the 8- or 9-year-olds, had a test of the entire visual field of each eye.

Data for the peripheral areas of the visual field (e.g., 0°, near periphery, midperiphery, and far periphery) for these subjects were calculated as discussed above for each eye. The interocular scores for each subject then were calculated by subtracting the sensitivity for the left eye from the sensitivity for the fellow right eye at each of

these four locations. The average of the interocular scores at each location for the 7-year-olds and the adults are presented in Table 38. Although these scores were small and similar across location and age, they should be viewed cautiously given the small sample at each age.

To verify that there were no significant differences, the interocular difference scores were analyzed by a 2-way ANOVA with a between factor of age (7-year-olds and adults) and a within factor of location (0°, near periphery, midperiphery, and far periphery) (see Table 39). There were no significant effects of age (p = .28) or of location (p = .89) and no significant interaction of location by age (p = .26).

The largest interocular difference, regardless of sign, for each location for the 7-year-olds and the adults was eliminated. The remaining largest interocular difference scores are presented in Table 40. The range of the interocular difference scores were large, especially at 0°. For example, at 0°, the range of the interocular difference scores were 13 db and 6.0 db for 7-year-olds and adults, respectively. Given the small sample, the results should be interpreted with caution but do indicate that substantial differences in sensitivity may occur between two related normal eyes tested at the same location.

The <u>absolute interocular difference scores</u> were the averages across subjects of the absolute values of the signed difference scores, calculated previously (Table 41). The absolute interocular scores

were small, indicating that there was not a tendency for one eye, regardless of whether it was the right eye or the left eye, to be more sensitive than the fellow eye.

(9) Range of sensitivity across the whole visual field:

So as to eliminate the most aberrant 5% of scores, the smallest threshold was eliminated. The range of the field across subject then was calculated by subtracting the remaining smallest threshold from the largest threshold for each location and age (Table 42). The range tended to be small, except at 0°. For example, at 0°, the largest range was 28 db for the 7-year-olds while the smallest range was 7.0 db for the adults.

Discussion

These results indicate that, when tested with a small stimulus, children as young as 7 years of age appear to be as sensitive as adults along the horizontal meridian and in the near, mid, and far periphery, with the exceptions of 15° temporally (the area of the blind spot) and 0° centrally. At 15° temporally, adults are less sensitive than 7-year-olds. The development of sensitivity at 15° temporally was not assessed at other ages. As well, at 0°, 7-year-olds are more sensitive than 8- or 9-year-olds but less sensitive than adults. Finally, interocular differences and test-retest reliability were similar across age (Tables 24, 26, 27, 29, 37, 38, 40, 41). This

suggests that there is little difference in the rate of interhemispheric development and little improvement with practice, at least for a limited number of tests.

This is the first report of the normal development of sensitivity in the center of the visual field. There are two possible explanations of why sensitivity at 0° was poorer in children than adults. First, the central area may be the last part of the visual field to develop fully, with complete development not occurring until sometime after 9 years of age. Combined with the results of Experiments 1 and 2, this finding suggests that the center of the field and the edges of the temporal field are slowest to develop and hence may be most vulnerable to deprivation. Second, the poorer sensitivity of children at 0° centrally may reflect differing abilities of children and adults to detect stimuli under low contrast. The central fixation aid was a relatively intense spot of light centered at 0° and the light to be detected by the subject flashed upon the central fixation aid. Because the contrast of the light flashing upon the central fixation aid was less than the contrast produced by the light flashing elsewhere in the bowl, detection of the target at 0° may have been more difficult for all ages, but particularly so for the children. There is evidence that children are less able to detect stimuli of low contrast than are adults (see Boothe et al., 1985). Note, however, that 7-year-olds had better detection at 0° centrally than the 8- or the 9-year-olds. The explanation that children's contrast sensitivity is different than that of adults does not explain why 7-year-olds had better detection than

did the 8- and 9-year-olds at 0°. This finding remains inexplicable and requires independent replication.

In contrast to the results at 0°, 7-year-olds were more sensitive than adults at 15° temporally, the area of the blind spot (Duke-Elder, 1962). Seven-year-olds may be more sensitive than adults near the blind spot for either of two reasons. First, like infants (Maurer, 1975), the blind spot of the 7-year-old children may not be located on the same area of the retina as it is for adults. Compared to adults, the infant's blind spot is shifted further into the temporal field (Maurer, 1975). Perhaps, when the light is centered on 15° temporally, it may fall within the blind spot for adults but on the the nasal edge of the blind spot for children, as would occur for infants. However, this seems unlikely because kinetic perimetry reveals that the size and location of the blind spot is similar in 7-year-olds as in adults (see Experiment 1). Second, possibly children have less steady central fixation than do adults and detected the light by chance whenever they looked toward the temporal field. However, as a group, children should be as likely to look toward the nasal field as toward the temporal field and, therefore, there should not be an increased likelihood of detecting a light in the temporal field. Moreover, there was a similar number of repeated trials for the 7-year-olds as for the adults, which suggests that the children do not have more frequent losses of fixation. To summarize, it is unknown why 7-year-olds are more sensitive than adults at 15° temporally.

It also was noted in this experiment that, relative to the normative data for the adult established by the manufacturers of the Octopus perimeter, a high percentage of children and adults tested in this experiment had abnormal thresholds at 0° and 15° temporally. It should be pointed out that relatively few normal individuals were tested for the collection of the normative data for the Octopus perimeter (Weijland, 1987). Possibly, the normative data from the Octopus perimeter are unrepresentative of the population as a whole.

It also was noted that there was a tendency for thresholds to decrease from the near to mid to far periphery, with greater variability in sensitivity found at the edges of the visual field. Moreover, there was slightly better sensitivity at 20° nasally than at 30° temporally. These results parallel previous findings (Allan, 1971; Aspinall, 1967; Aulhorn & Harms, 1972; Fahle & Schmid, 1988; Frisen & Glansholm, 1975; Lakowski & Aspinall, 1969; Lewis et al., 1985; Liao, 1973; Matsuo et al., 1974; Tomonaga, 1974; Whiteside, 1976).

This is the first study examining the normal development of sensitivity across the entire visual field using small stimuli. Other than at 0° centrally and 15° temporally, I found that children were as sensitive as adults. This finding suggests two conclusions. First, that sensitivity, at least in the periphery of the visual field, is well developed early in childhood. This finding complements previous reports that even children 5 or 6 years of age are as sensitive as adults along the horizontal meridian (Tomonaga, 1974; Whiteside, 1974). My finding is in stark contrast to the report of Lakowski and

Aspinall (1969) that 6-year-old children have tunnel vision. Possibly, the discrepancy may reflect the fact that I took care not to fatigue my subjects. Second, my results indicate that the peripheral areas of the visual field are fully developed before the central area. The effects of deprivation upon a slowly developing area of the field (the central field) versus a quickly developing area (the peripheral field) will be explored in Experiment 5.

Yet, a note of caution regarding the results for the test of the whole visual field is warranted. Although children appear to be as sensitive as adults in the near, mid, and far periphery, the fast bracketing strategy does not measure performance precisely. Should the thresholds be measured precisely, perhaps adults would be more sensitive than children at many locations in the visual field. To evaluate this possibility, subjects would need to be tested with a normal bracketing strategy. As explained previously, detailed examination of the entire field using a normal bracketing strategy is not feasible with young children. Moreover, when subjects were tested with a normal bracketing strategy at 20° nasally and at 30° temporally, no significant differences were found between children and adults. This finding suggests that children indeed are likely to be as sensitive as adults across the peripheral visual field. Finally, one aim of this experiment was to establish normative standards. The fast bracketing strategy used is sufficient to detect differences in sensitivity between normal children and children treated for a

cataract, provided that losses in sensitivity in the patients are larger than 4 decibels.

In conclusion, except for the performances at 0° centrally and at 15° temporally, when tested with a small stimulus, children 7 years of age and older appear to be as sensitive as adults across the visual field, including at 20° nasally and at 30° temporally. Experiments 1 and 2 indicated that the extent of the field was slow to develop. The present results indicate that sensitivity in the center of the visual field also requires a prolonged period of development. My results also suggest that there is little difference in the rate of interhemispheric development and little improvement with practice, at least for a limited number of tests. Finally, these results provide normative standards by which to evaluate the visual fields of children treated for dense and central cataracts.

CHAPTER 3
PERIPHERAL VISION IN CHILDREN TREATED FOR CATARACTS

This chapter presents the results from two experiments which examined the peripheral vision of children treated for a dense and central cataract and the results from a control experiment which investigated the possible contribution of optical parameters to the noted effects on peripheral vision.

Predictions of the peripheral vision of children treated for dense and central cataracts are guided by studies of animals that were visually deprived by lid suture.

First, all studies of monocularly deprived monkeys or cats agree that the peripheral vision of the fellow nondeprived eye is normal (Bisti & Carmignoto, 1986; Joseph & Casagrande, 1980; Sherman, 1973, 1974a, 1974b; Sherman & Sprague, 1979; Smith et al., 1982; Sparks et al., 1986; Tumosa et al., 1982; Wilson et al., 1989).

Second, all studies of monkeys and cats indicate that if the duration of deprivation is long, lasting for more than several weeks, then the peripheral vision of the visually deprived eye is abnormal (Bisti & Carmignoto, 1986; Heitlander & Hoffmann, 1978;

Hendrickson et al., 1977; Kalil, 1978; Kossut et al., 1978; Joseph & Casagrande, 1980; Maire-Lepoivre et al., 1988; Rizzolatti, cited in van Hof-van Duin, 1977; Sherman, 1973, 1974a, 1974b, 1977a; Sherman & Sprague, 1979; Smith et al., 1982; Sparks et al., 1986; Tumosa et al., 1982; van Hof-van Duin, 1977; van Hof-van Duin, cited in Zablocka, 1983; Wilson et al., 1989; Zablocka, 1983). However, if the monocular deprivation is short, lasting for 1 to 2 weeks beginning in the second week of life, then monkeys have a normal visual field (Sparks et al., 1986). To date, no study has systematically varied the duration of deprivation in monocularly or binocularly lid sutured cats although studies of dark reared cats indicate that longer deprivation has more deleterious effects upon peripheral vision (Kalil, 1978; Maire-Lepoivre et al., 1988).

Third, earlier onset of visual deprivation leads to poorer peripheral vision in monocularly deprived monkeys and in monocularly deprived cats (Bisti & Carmignoto, 1986; Sparks et al., 1986; Tumosa et al., 1982; Wilson et al., 1989) but relevant studies have not been conducted in binocularly deprived animals.

Finally, in cats the effects of monocular deprivation from birth are more serious than those of binocular deprivation (Sherman, 1974b; but see van Hof-van Duin, 1977). Studies of binocular deprivation in monkeys are lacking.

To date, there has been relatively little examination of the effects of deprivation from dense and central cataracts upon peripheral vision in children. Kinetic perimetry has been used to

study the extent of the visual field in one patient treated for a unilateral congenital cataract and that patient was treated at 19 years of age (Moran & Gordon, 1982). When tested with a 1° stimulus, compared to his nondeprived eye, the deprived eye of the patient had a temporal, superior, and inferior field which were approximately normal in extent but a nasal field which was restricted to 20°. Similarly, there exist few reports using static perimetry: Including the above noted patient, only nine eyes treated for a cataract have been assessed and these assessments were only along the horizontal meridian. These included tests of two patients monocularly deprived from birth to either 5 months or 19 years, one eye from one patient binocularly deprived from birth to 11 months, one eye from one patient binocularly deprived for a short period of time beginning at 3 years of age, as well as five eyes from four children unilaterally or bilaterally deprived for less than 6.5 months beginning after 7 months of normal experience (Lewis et al., 1986; Maurer et al., 1983; Mioche & Perenin, 1986; Moran & Gordon, 1982). Relative to normal eyes, the deprived eyes had decreased sensitivity along the horizontal meridian. Moreover, in patients treated for unilateral congenital cataract, but not in patients treated for bilateral cataracts, there were especially large losses in the nasal visual field.

These studies indicate that the peripheral vision of deprived eyes is abnormal. Yet, because of the small sample tested to date, there is no knowledge of the effects of deprivation on sensitivity other than along the horizontal meridian. As well, no firm conclusions

can be drawn about the effects of the timing or duration of deprivation.

Moreover, there has been no study of the effects of occlusion of the nondeprived eye in children treated for unilateral cataracts. In such cases, parents are instructed by the ophthalmologist to patch the fellow nondeprived eye 50% of the waking time for the child's first 5 years of life in order to force usage of the previously deprived eye. In their study of children treated for a unilateral congenital cataract, Maurer et al. (1989) defined compliance with the patching regime as being excellent (patching of the nondeprived eye 40% to 50% of the time), fair (patching 20% to 39% of the time), or poor (patching 0% to 19% of the time). They found that less patching of the nondeprived eye was correlated with decreased visual acuity in the deprived eye (Maurer et al., 1989). The patching of the nondeprived eye was not correlated with its visual acuity (Lewis, Maurer, Tytla, Bowering, & Brent, 1991). Like visual acuity (Boothe et al., 1985; Maurer et al., 1989), peripheral vision develops slowly over several years in normal children (see Experiments 1, 2, and 3). As occurs for visual acuity, patching of the fellow nondeprived eye early in life may affect the peripheral vision of the previously deprived eye in children treated for a unilateral cataract.

The first goal of this research was to increase the sample of deprived children assessed so as to test the typicality of previous findings. A secondary goal was to examine, to the extent possible, the effects upon peripheral vision of the variables of the timing and

duration of deprivation, whether the deprivation was monocular or binocular, and the effects of occlusion. The effect of the timing of visual deprivation was assessed by comparing the visual fields of children with similar durations of deprivation but different ages of onset. For example, this analysis compared the data from children whose deprivation lasted for only a few months but whose onset of deprivation varied widely, beginning at birth or during infancy or even after school age. The duration of visual deprivation was assessed by comparing the visual fields of children with similar ages of onset but different durations of visual deprivation. For example, the analysis compared data from children diagnosed at birth but treated at different ages. The effects of monocular versus binocular deprivation was assessed by comparing the fields of children treated for unilateral versus bilateral cataracts, given that the deprivation was similar in age of onset and duration. Using the definition of compliance with patching suggested by Maurer et al. (1989), the influence of patching was evaluated by comparing the fields of the deprived eyes for children who were excellent, fair, and poor patchers.

Moreover, I chose to assess the effects of deprivation on the extent of the field and on sensitivity along the horizontal meridian, as has been done in previous studies, and also to explore its effect on sensitivity across the visual field. In Experiment 4, I used kinetic perimetry with the Goldmann perimeter to assess the extent of the visual field along eight meridia. In Experiment 5, I used static

perimetry with the Octopus perimeter to assess sensitivity across the visual field. The normative data for evaluating the results from the deprived children were reported in Experiments 1 and 3. Experiment 6 consisted of a series of investigations designed to explore the potential influence of optical factors upon the peripheral vision of deprived subjects. The role of these factors is important to evaluate because they potentially may limit the size of the visual field or sensitivity within the field.

Experiments 4, 5, and 6 are important for several reasons. As discussed in Chapter 1, one aim of the dissertation is to understand the effects of visual deprivation in humans. Previous research has shown that visual acuity is immature at birth and develops slowly over several years (Boothe et al., 1985; Maurer et al., 1989). Moreover, during this developmental period, visual acuity is vulnerable to deprivation from cataracts (Boothe et al., 1985; Maurer et al., 1989). In Experiments 4 and 5, I examined the effects of visual deprivation on peripheral vision. This research provides another test of the generality of the developmental principle that slowly developing behaviors are affected by abnormal experience. Second, the results may provide clues about the physiological changes underlying the development of peripheral vision. If particular parts of the visual field of the deprived eye are especially affected by visual deprivation, then we can draw some conclusions about the initial immaturity and vulnerability of the pathways which mediate detection in these parts of the visual field. Finally, these data may

have practical implications for the treatment of children diagnosed with dense and central cataracts. For example, if earlier onset of deprivation and/or longer deprivation are shown to have more deleterious effects on peripheral vision, then this indicates the necessity for earlier and/or swifter treatment of the cataract.

Before presenting my results, it is prudent to note that problems exist when conducting research with this clinical population. For example, several ocular conditions commonly coexist with a cataract, such as microcornea (small cornea), microphthalmia (small eye), strabismus (misalignment of the eye), and nystagmus (jiggly eye movements) (Hiles & Wallar, 1977; Parks, 1982). As such, it was usually not possible to test children who were treated for a cataract but had no other eye condition. The potential effect of associated ocular conditions on peripheral vision will be evaluated in Experiment 6.

When selecting deprived subjects, I applied two criteria concerning these associated ocular conditions. First, subjects included those with secondary strabismus (diagnosis of misalignment of the eye <u>after</u> the onset of the cataract) but not those with primary strabismus (diagnosis <u>before</u> the onset of the cataract). To evaluate the potential effects of secondary strabismus, I will be guided by studies of the effects of primary strabismus upon peripheral vision. Second, when using the technique of kinetic perimetry with the Goldmann perimeter, it was imperative that the deprived eye be able to maintain relatively steady fixation upon the central fixation aid

for several seconds. Otherwise, as the stimulus moves in slowly from the periphery of the perimeter, the subject might detect it by a chance eye movement, thus artifactually increasing the measured extent of his visual field. Steady fixation was defined as less than 2 mm, or 0.4°, of movement as measured by the telescope grid of the Goldmann perimeter. Nystagmus, or uncontrollable jiggly eye movements, often occurs in children whose visual deprivation begins at birth, rendering them untestable with this technique. Because nystagmus is far less common in children whose deprivation is of later onset, then there exists a potential sampling bias when using the technique of kinetic perimetry. The impact of this bias will be discussed later in the chapter. It is important to note that such a sampling bias does not exist when using the technique of static perimetry with the Octopus perimeter. Because of the extreme brevity of each trial, fixation usually is maintained sufficiently long, even in subjects with marked amounts of nystagmus. Moreover, the computer system of the Octopus perimeter monitors the subject's fixation, discarding and later repeating those trials during which fixation was lost.

Subjects excluded from the sample were those children diagnosed with a cataract who were awaiting surgery, and eyes with an additional complication such as a detached retina, corneal scar, pupillary membrane, glaucoma (raised intraocular pressure), persistent hyperplastic primary vitreous (embryonic maldevelopment in which there is a dense white vitreous mass

attached to the optic disc), or otherwise abnormal fundus or ocular media. Although these conditions may occur in the population of children with a cataract, they are not typical of the population, unlike such conditions as microcornea and nystagmus. Therefore, the elimination of children with atypical and potentially serious complications likely will reduce noise in the data. Finally, noteworthy is the fact that because of the strict criteria for inclusion, sometimes only one eye of a child diagnosed with bilateral cataracts was appropriate to test.

Experiment 4
The extent of the visual field in children treated for cataracts

Method

Subjects. The subjects were children treated for dense and central cataracts, which were present either unilaterally or bilaterally, and were congenital or developmental or traumatic in nature (see Table 43). I defined a cataract as dense and central if it met any of the following criteria: It prevented the ophthalmologist from seeing either the fundus of the eye (interior of the globe) or the reflection of light from the retina (i.e, the red reflex), or the eye was unable to fixate upon and follow an object moving through the visual field, or

the cataract was described by the ophthalmologist as dense and central.

The duration of deprivation of these eyes ranged from one to 45 months. I defined the duration of deprivation as lasting from birth in eyes treated for congenital cataracts until the fitting of an optical device following surgery. In eyes treated for developmental cataracts, deprivation was said to last from the age of diagnosis of a dense and central cataract until the fitting of an optical device following surgery. Short deprivation was defined as lasting for 6 months or less while long deprivation was greater than 6 months.

Included in the sample were children treated for a cataract who also had microcornea, microphthalmia, secondary strabismus, and nystagmus. Excluded from the sample were children diagnosed with a cataract who were awaiting surgery, and eyes with an additional complication such as a detached retina, corneal scar, pupillary membrane, glaucoma, persistent hyperplastic primary vitreous, or otherwise abnormal fundus or ocular media. At the time of the test, each child was 5 years of age or older.

To determine the effects of monocular deprivation upon the extent of the visual field (see Table 43), I tested 14 deprived eyes from children deprived from birth for varying periods of time (unilateral congenital group). Because not all of these children returned to the clinic for subsequent testing, only 12 and 9 of their fellow nondeprived eyes were assessed with the intense light and with the dim light, respectively. I also tested 11 deprived and 11

fellow nondeprived eyes of children (unilateral developmental group) who had normal visual experience early in life and then either developed a unilateral cataract (n = 6) or sustained a unilateral eye injury which led to the formation of a cataract (n = 5).

The parents of children treated for a unilateral cataract had been instructed by the ophthalmologist to patch the fellow nondeprived eye 50% of the waking time during the first 5 years of life in order to force usage of the previously deprived eye. Compliance with the patching regime varied from excellent (patching of the nondeprived eye 40% to 50% of the time, n = 8 children), fair (patching approximately 20% to 39% of the time, n = 11 children), to poor (patching approximately 0% to 19% of the time, n = 6 children).

To determine the effects of binocular deprivation on the extent of the visual field (see Table 43), I tested 13 eyes from 10 children bilaterally deprived from birth for varying periods of time (bilateral congenital group). For comparison, I also tested 18 eyes from 11 children who had normal early experience and then developed a cataract in both eyes (bilateral developmental group). Note that both eyes were not tested in some cases because of insufficient time in the clinical setting, or because one eye exhibited marked nystagmus, had an associated condition which rendered it unsuitable for this study (see exclusion criteria), or had been enucleated. Finally, no child had developed bilateral cataracts as the result of trauma.

Apparatus. The Goldmann perimeter and stimuli were identical to those of Experiment 1. Briefly, the stimuli were 0.107° in size and either 318 cd/m^2 (stimulus I4e) or 31.8 cd/m^2 (stimulus I2e) in intensity. The background intensity was 10.0 cd/m^2.

Procedure. With two exceptions, the procedure was identical to that described in Experiment 1. Briefly, each child had one eye occluded and sat in front of the Goldmann perimeter with his chin on the chin rest and forehead encircled by the head strap. The child was instructed to fixate the central fixation aid and to indicate awareness of a peripheral light by tapping upon the table. The light was presented randomly along one of eight meridia (0° temporally, 45° temporally and superiorly, 90° superiorly, 135° nasally and superiorly, 180° nasally, 225° nasally and inferiorly, 270° inferiorly, and 315° temporally and inferiorly). The differences from Experiment 1 were as follows. First, the aphakic eye wore a contact lens of a power designed to focus the eye at the testing distance of 33 cm. Second, because of time constraints in the clinical situation, sometimes both eyes could not be tested during one visit. To ensure that there was a complete test of at least one eye, the chosen eye was tested first with the intense target (I4e) and then with the dim target (I2e). Moreover, the eye selected to be tested first was the deprived eye in unilateral cases and the eye with the better acuity in bilateral cases. If in bilateral cases there was no interocular difference in acuity, then one eye was selected randomly for the test.

On the child's subsequent visit to the clinic, the untested eye was assessed using the same protocol as for the tested eye.

Data analysis. In some of the subsequent analyses, loss scores were calculated for each deprived eye. A loss score was calculated by subtracting the extent of the visual field of the deprived eye from the extent of the field of the fellow nondeprived eye for each of the eight meridia at each intensity of light. The loss score represents the amount of restriction, if any, at that meridian in the visual field of the deprived eye relative to normal. If the nondeprived eye was not tested, as occurred in some unilaterally deprived children, or if the child did not possess a nondeprived eye, as occurred in bilaterally deprived children, then the comparison data were from the group of normal children of the same age (see Experiment 1).

Results

This section will address five questions. First, in children treated for a unilateral cataract, does the fellow nondeprived eye have a visual field of normal extent? This analysis will establish the appropriateness of using the nondeprived eye as a standard by which to evaluate the results from the deprived eye. Second, does visual deprivation alter the extent of the field? Third, what are the effects upon peripheral vision of the variables of the timing and duration of deprivation and whether the deprivation was monocular

or binocular? Fourth, does the history of occlusion of the nondeprived eye affect the extent of the field in the deprived eye in children treated for a unilateral cataract? Fifth, is the typically poorer visual acuity of the deprived eyes of patients, relative to normal individuals (see Maurer et al., 1989), correlated with the measured extent of their fields?

(1) Extent of the visual field of the fellow nondeprived eye of monocularly deprived children:

This question is important to address for three reasons. First, Experiment 1 indicated that there exists much variability in the normal extent of the visual field between subjects within an age but much less interocular variability. As such, evaluation of the loss of the extent of the field for a deprived eye is likely to be more precise when comparing performance to that of its fellow nondeprived eye rather than to population norms. Before doing so, it must be determined that the fellow nondeprived eye indeed has a field of normal extent. Second, upon the recommendation of the ophthalmologist, the nondeprived eye of a monocularly deprived child is occluded, sometimes up to 50% of the waking time, so as to force usage of the deprived eye. Is there a deleterious effect of occlusion upon the field of the nondeprived eye? If there is, then the field of the nondeprived eye should be smaller than that of normal children of the same age. If the field for the nondeprived eye is smaller than normal, using it as the comparison potentially may

minimize my assessment of losses in extent of the field in the deprived eye. Third, evaluation of the extent of the field of the fellow nondeprived eye allows evaluation of the role of competition between a nondeprived and fellow deprived eye. If interocular competition is occurring, then not only might the deprived eye show a restricted field, but the nondeprived eye might exhibit a larger field than that of normal children of the same age. Together, these last two reasons suggest that it is important to consider the possibility that occlusion of the nondeprived eye may cause reduction in the extent of the field for the nondeprived eye while interocular competition may cause expansion in its extent.

In order to determine whether the fellow nondeprived eye of monocularly deprived children was normal, the extent of the visual field of the nondeprived eye was compared to that of normal volunteers (see Experiment 1) with ANOVA's. Because the nondeprived fellow eye of the deprived subjects typically was tested but once, these data were compared to the normative data collected from the first test of the visual field (see Experiment 1).

The data were analyzed by a 3-way ANOVA (Table 44) with a between group factor of group (4 levels) and within group factors of intensity (2 levels) and meridian (8 angles of the field). Group 1 consisted of the adult normal volunteers. Groups 2 to 4 consisted of the nondeprived eyes of patients treated for unilateral cataracts (congenital or developmental), divided respectively into those patients who were excellent patchers, fair patchers, or poor patchers.

Groups 2 to 4 were not matched on the variables of the timing and duration of deprivation because of the small sample. For groups 2, 3, and 4, the mean onset of deprivation was at 15.1, 14.7, and 34.2 months and the mean duration of deprivation was 6.6, 5.1, and 8.6 months, respectively. Moreover, because so few nondeprived eyes of the younger patients were assessed, this analysis contained only data from patients older than 9 years of age.

(a) Effect of group:

There was no significant main effect of group (p = .13).

(b) Effect of intensity:

There was a significant main effect of intensity (p < .00001), with the field for the intense light larger than for the dim light. There was no significant interaction of group by intensity (p = .90).

(c) Effect of meridian:

There was a significant main effect of meridian (p < .00001), with Tukey's HSD tests indicating that the largest extent of the field was found temporally (p's < .01). As well, there was a significant interaction of group by meridian (p = .03). Because there has been no previous investigation of the effect of patching upon peripheral vision, I chose to investigate this finding using simple effects and Tukey's HSD tests. Simple effects revealed that there were significant effects of group only at 45° superiorly [$F(3,98) = 3.45$, p = .019], 90°

superiorly [$F(3,98) = 3.33$, $p = .023$], and 135° superiorly [$F(3,98) = 3.22$, $p = .026$]. At 45° superiorly, 90° superiorly, and at 135° superiorly, Tukey's HSD tests indicated that the group of excellent patchers saw further than did the normal volunteers (p's $< .05$). Moreover, at 90° superiorly, even the fair patchers saw further superiorly than did the normal volunteers ($p < .05$).

There also was a significant interaction of intensity by meridian ($p < .00001$), with simple effects revealing that the extent of the field was larger for the intense light at each meridian [0°: $F(1,25) = 577.55$, $p < .0001$; 45°: $F(1,25) = 181.80$, $p < .0001$; 90°: $F(1,25) = 181.80$, $p < .0001$; 135°: $F(1,25) = 149.52$, $p < .0001$; 180°: $F(1,25) = 134.55$, $p < .0001$; 225°: $F(1,25) = 107.38$, $p < .0001$; 270°: $F(1,25) = 427.62$, $p < .0001$; 315°: $F(1,25) = 315.76$, $p < .0001$). There was no significant interaction of group by intensity by meridian ($p = .95$).

The preceding analysis indicated that the extent of the visual field of the fellow nondeprived eye of monocularly deprived subjects did not differ from that of the normal volunteers except in the superior visual field. Two explanations are possible. First, the early competitive advantage enjoyed by the nondeprived eye may have caused expansion of its field while the subsequent implementation of patching caused shrinkage of its field. Given that the excellent patchers had the largest superior fields, it is unlikely that patching

caused shrinkage of the field. Second, and more likely, patching truly may not affect the size of the field along most meridia.

The extent of the superior field was smaller for the adults than for the nondeprived eye of the patients. One possible explanation is that the superior field is more sensitive to alterations in experience than are other parts of the field. Subsequent analyses in this chapter reveal that this is unlikely to be true. An alternative and more likely explanation is as follows. As reported in Experiment 2, the same adults had a smaller superior field than did the normal 7-year-old children. This difference was attributed to the well developed brow of the adults which may obscure detection of stimuli in the superior field. In the present experiment, the patients were older than 9 years of age who, like the normal children, also may have had a less well developed brow than the adults. The better performance in the superior visual field for the patients compared to the adults may reflect differences in the brow and not differences in performance of nondeprived versus normal eyes. It is appropriate, then, to use the data from the fellow nondeprived eye in monocularly deprived subjects as a control for their deprived eye.

(2) Extent of the visual field of the deprived eyes:

To analyze the data from the deprived eyes of the unilaterally and the bilaterally deprived subjects, loss scores were calculated (see Data analysis). The loss score in degrees represents the amount of

restriction, if any, at that meridian in the visual field of the deprived eye relative to normal.

The most striking result was that, in every case, the extent of the visual field for the deprived eye was restricted, with the average loss across meridia being 23.2°. For the purpose of illustration, Figure 9 presents the extent of the visual field for one eye of a binocularly deprived 24-year-old patient, T. M., and that of a group of normal adult subjects. The represented eye of T. M. was diagnosed with a dense and central cataract at 13.5 months. The duration of deprivation was 3 months and her visual acuity measured with the Snellen eye chart was good, being 20/25. Figure 9 indicates that, for tests with the intense light, the visual field for the deprived eye was restricted along each meridia relative to the normative data.

The data for the group of deprived eyes then were analyzed by a 5-way ANOVA (Table 45) with the between group factors of condition of deprivation (two levels), timing (two levels), and duration (two levels) and the within group factors of intensity (two levels) and meridian (eight levels). The levels of each of these five factors were as follows.

First, condition of deprivation consisted of patients with either monocular or binocular deprivation. Second, timing of deprivation consisted of eyes with a diagnosis of a dense and central cataract at birth (children treated for congenital cataract) or eyes with an early normal history that subsequently were diagnosed with a cataract between 6 and 72 months of age (children treated for developmental

cataract). Third, duration of deprivation was defined as lasting from birth in congenital cases or from the time of diagnosis of a dense and central cataract in developmental cases until fitting of the optical device. Duration of deprivation consisted of eyes with either short duration (defined as less than or equal to 6 months) or with long duration (defined as lasting more than 6 months). Fourth, intensity consisted of tests with the intense light (Goldmann stimulus I4e) and with the dim light (Goldmann stimulus I2e). Finally, meridian consisted of the eight meridia in the visual field at which the subjects were assessed.

(a) Effect of condition:

There was no main effect of condition: Unilaterally deprived and bilaterally deprived subjects exhibited similar restrictions of the visual field (p = .29).

(b) Effect of timing:

There was no main effect of timing: Subjects deprived from birth and subjects deprived beginning after birth showed similarly sized restrictions of the visual field (p = .37). There was no significant interaction of condition by timing (p = .09).

(c) Effect of duration:

There was a main effect of duration (p = .0050): Subjects deprived for six or fewer months exhibited mean losses of 20.1°

while subjects deprived for longer periods exhibited mean losses of 26.3°. There were no significant interactions of duration by condition (p = .16), or duration by condition by timing (p = .73), although the interaction of duration by timing approached significance (p = .059).

(d) Effect of intensity:

The extent of the field loss was influenced by target intensity with a significantly more restricted field obtained for the dimmer light (p < .00001). Moreover, intensity interacted significantly with condition (p < .0077), with an analysis of simple effects revealing that the most restricted fields were found for children treated for unilateral cataracts who were tested with the dim light [$F(1,84)$ = 5.93, p = .017].

There were no significant interactions of intensity with timing (p = .37), intensity by timing by condition (p = .88), intensity by duration (p = .66), intensity by duration by condition (p = .47), intensity by duration by timing (p = .80), or intensity by duration by timing by condition (p = .18).

(e) Effect of meridian:

There was a main effect of meridian (p = .00001), with Tukey's HSD tests revealing that the most restricted fields were located temporally (p's < .01). As well, there was a significant interaction of meridian by duration (p = .0058). To date, there has been no examination of the effects of duration of deprivation on functioning

anywhere in the visual field in visually deprived children. So as to investigate the effects of the duration of deprivation at multiple meridia, I chose to use simple effects. Simple effects revealed that children with longer duration of deprivation had significantly more restricted visual fields at all locations other than 90° and 135° superiorly than did children with short duration of deprivation (0°: $F(1,88) = 17.67$, $p < .0001$; 45°: $F(1,88) = 3.86$, $p = .05$; 90°: $F(1,88) = 2.65$, $p = .107$, 135°: $F(1,88) = 2.47$, $p = .12$; 180°: $F(1,88) = 6.11$, $p = .015$; 225°: $F(1,88) = 6.52$, $p = .012$; 270°: $F(1,88) = 4.58$, $p = .035$; 315°: $F(1,88) = 13.11$, $p < .0001$).

Also, there was a significant interaction of meridian by intensity ($p < .00001$). Simple effects did not reveal the source of this interaction because at each location in the visual field, the field was more restricted for tests with the dim light than with the intense light (0°: $F(1,48) = 11.58$, $p = .001$; 45°: $F(1,48) = 15.84$, $p < .0001$; 90°: $F(1,48) = 20.34$, $p < .0001$, 135°: $F(1,48) = 24.97$, $p < .0001$; 180°: $F(1,48) = 42.31$, $p < .0001$; 225°: $F(1,48) = 76.22$, $p < .0001$; 270°: $F(1,48) = 34.24$, $p < .0001$; 315°: $F(1,48) = 15.36$, $p < .0001$).

There were no significant interactions of meridian by condition ($p = .74$), meridian by timing ($p = .92$), meridian by condition by timing ($p = .41$), meridian by condition by duration ($p = .08$), meridian by timing by duration ($p = .99$), or of meridian by condition by timing by duration ($p = .65$). There also were no significant interactions of meridian by condition by intensity ($p = .80$), meridian by timing by intensity ($p = .87$), meridian by condition by timing by

intensity (p = .26), meridian by duration by intensity (p = .22), meridian by condition by duration by intensity (p = .76), meridian by timing by duration by intensity (p = .08), or of meridian by condition by timing by duration by intensity (p = .69).

To summarize, the extent of the visual field was restricted for every deprived eye, with the largest restriction in the temporal field. Moreover, children with longer deprivation and children with monocular deprivation who were tested with the dim light had more restricted fields.

(3) Effect of patching of the fellow nondeprived eye on the extent of the visual field for the deprived eye in children treated for unilateral cataract:

To combat the effects of visual competition from the nondeprived fellow eye upon the deprived eye, parents were instructed by the ophthalmologist to patch the child's nondeprived eye 50% of the waking time during the first five years of life. However, compliance varied across subjects from poor patching to excellent patching. As such, the influence of patching of the fellow nondeprived eye upon the extent of the visual field of the deprived eye needs to be evaluated.

The influence of patching of the nondeprived eye upon the extent of the field of the deprived eye was analyzed with a 3-way ANOVA (Table 46) with a between factor of patching (3 levels) and

within factors of intensity (2 levels) and meridian (8 levels). The levels of patching included children who were excellent patchers (n = 8), fair patchers (n = 4), and poor patchers (n = 6), with the groups matched for the age of onset (18, 22, and 28 months, respectively) and the duration of deprivation (each group deprived for a mean of 7 months). Given the small sample, the variables of timing and duration of deprivation were not included as factors within the ANOVA.

(a) Effect of patching:

The amount of loss of the visual field was not significantly different across excellent, fair, or poor patchers (p = .68).

(b) Effect of intensity:

There was a larger loss of the field for tests with the dim light (p = .0003) but no significant interaction of patching with intensity (p = .21).

(c) Effect of meridian:

There was a significant effect of meridian (p < .00001), with Tukey's HSD tests revealing that the largest loss were at 0° and 315° in the temporal field (p's < .01). Moreover, there was a significant interaction of meridian with patching history (p = .0105). The effect of patching upon the extent of the field has not been investigated previously. As such, I chose to use simple effects and Tukey's tests to

investigate the interaction. Simple effects revealed that there was a significant effect of patching only at 0° temporally [$F(2,24) = 3.4$, $p = .05$], with Tukey's HSD tests indicating that the least restricted field was found for children who were excellent patchers (p's $< .01$).

There also was a significant interaction of intensity with meridian ($p = .0004$), with Tukey's tests revealing that the most restricted fields were in the temporal meridian for tests with the dim light (p's $< .05$). There was no significant interaction of meridian with intensity with patching ($p = .14$).

In summary, there was an effect of patching only at 0° temporally, with children who were excellent patchers having the largest visual fields.

(4) Effect of visual acuity upon the extent of the visual field in deprived eyes:

One possible explanation for restricted fields in deprived subjects is their poor visual acuity which, in turn, is related to the timing and duration of deprivation, whether the deprivation is monocular or binocular, and, in cases of monocular deprivation, to the patching of the nondeprived eye (Maurer et al., 1989). As such, it may be difficult to determine whether deprivation per se or whether one effect of deprivation, which is poor visual acuity, alters peripheral vision.

To evaluate the influence of visual acuity, the relationship between the $\log_2$ value of the deprived eye's typical visual acuity (n = 46 eyes) and its loss scores at each meridian for tests with the intense and with the dim light were evaluated using Pearson correlations (see Table 47). Because Snellen acuity values are logarithmic, the $\log_2$ value of the acuity was used in the calculation of correlation. Moreover, the deprived eye's median acuity, as opposed to the acuity at the time of the test, was chosen as the typical acuity most representative of the child's day-to-day functioning. Finally, note that for patients who had the extent of the visual field assessed for both eyes, only the acuity of the right eye was used so as to avoid sampling acuities from two related eyes.

With alpha corrected for the number of tests (two-tailed), there were no significant correlations between the $\log_2$ value of the Snellen acuities and the loss scores at any meridian for tests with the intense light (see Table 47). For tests with the dim light, although several correlations approached significance, the only significant correlations between visual acuity and the loss scores were at 225° in the inferior field and at 270° in the inferior temporal field (p's < .001). In summary, these data suggest that the visual acuity of the deprived eye does not affect the extent of the field for tests with the intense light but there appears to be an overall pattern of correlation for the dim light.

Discussion

Several important conclusions are evident from the foregoing study. Most importantly, when tested with a small stimulus, all deprived eyes exhibited a restricted visual field along all meridia, particularly in the temporal field. Previously, kinetic perimetry has been used to study the extent of the visual field in one patient with 19 years of monocular deprivation beginning from birth (Moran & Gordon, 1982). The deprived eye of the patient, when tested with a 1° stimulus, had a temporal, superior, and inferior field which were approximately normal in extent but a restricted nasal field. Possibly the findings of the present research differ from those of Moran and Gordon (1982) because Moran and Gordon used a much larger stimulus to assess the visual field, which may have masked deficits in the patient's temporal, inferior, and superior fields. Given the much larger sample of children, the data from the present research may be more typical of the peripheral vision of children treated for a cataract.

The losses in the extent of the field observed in the patients in the present study are not explainable by the secondary strabismus possessed by most patients as strabismus has no systematic effect upon peripheral vision in humans and monkeys (Aggarwal & Verma, 1980; Fusco et al., 1983; Inoue, Mimuro, Kani, & Ohmi, 1985; Jacobson et al., 1983; Jossee, Wilson, & Boothe, 1990; Mahendrastari & Verriest, 1985; Mehdorn, 1986; Nawratzki & Jampolsky, 1958;

Sireteanu & Fronius, 1989). Moreover, significant differences in the extent of the visual field were found in children treated for unilateral versus bilateral cataracts yet strabismus was equally common in both groups. Rather, there are two possible and not necessarily mutually exclusive explanations of why the visual field was restricted. First, Beasley (1965) found that there was loss in the extent of the field in the aphakic eyes of adults who experienced deprivation from cataracts beginning after the presumed sensitive period was completed. As such, the influence of optical factors in the aphakic eyes of my patients, such as an abnormally shaped pupil or cornea, likely explains some of the loss of their fields. But, given the observed losses of the field were much larger than those reported by Beasley (1965) for aphakic adults, optical factors are unlikely to be solely responsible. Experiment 6 evaluates the basis by which optical factors may affect peripheral vision.

Second, the loss of the visual field may reflect the abnormal functioning of the visual pathways subserving peripheral vision. Previous findings on the normal development of peripheral vision would suggest that the pathways serving the nasal visual field should be most vulnerable to deprivation. Studies of normal babies (Maurer & Lewis, 1991) and kittens (Sireteanu & Maurer, 1982) indicate that there is slow development of all parts of the visual field but especially of the nasal field. Note, however, that the tests of infants and kittens used large stimuli, unlike in my studies, and the differences in temporal and nasal functioning in infants are present

only during early infancy. Given its seemingly prolonged period of development, the nasal field and its underlying pathways may be most vulnerable to deprivation. Indeed, there are especially large losses in the nasal field in visually deprived cats (Sherman, 1974a, 1974b) and in the few previously tested humans treated for unilateral congenital cataracts (Lewis et al., 1986; Moran & Gordon, 1982), although not in the few previously tested patients treated for bilateral cataracts (Mioche & Perenin, 1986). Given these previous results, it was surprising that the present research found that deprivation restricts all parts of the visual field but especially the temporal field. This result is best understood by reviewing the patterns of normal development identified in the previous experiments. First, when normal children are assessed with a small stimulus, the most temporal and inferior portions of the visual field, and not the nasal field, are the last to develop (see Experiment 1). As such, it may be that the pathways underlying detection in the inferior and temporal field are the slowest to develop and hence most vulnerable to deprivation. The neural basis for the observed effects will be discussed in Chapter 4. Second, the finding by Lewis et al. (1986) and Moran and Gordon (1982) that visual deprivation most affected the nasal field may not be typical because each tested only one eye. The difference in results from my patients to deprived cats may represent a species difference and will be examined in the final chapter. In summary, my finding that the largest losses were in the temporal field complements my previous finding that the temporal

field is especially slow to develop. These data provide another test of the general developmental principle that slowly developing behaviors are vulnerable to deprivation.

The present study also indicated some effects of the duration of deprivation and whether the deprivation was monocular or binocular. Other than in the superior visual field, children with longer deprivation exhibited more restricted fields than did children with shorter deprivation. The influence of the duration of deprivation on peripheral vision in humans parallels findings from similar work in monocularly lid sutured monkeys (Sparks et al., 1986) and dark reared cats (Kalil, 1978; Maire-Lepoivre et al., 1988). However, it is unknown why the more severe effects resulting from longer periods of deprivation did not extend to the superior visual field. The extent of the superior field has been measured in monocularly deprived cats (van Hof-van Duin, 1977) and monkeys (Sparks et al., 1986). In the former experiment, monocular deprivation for several months from the time of normal eye-opening led to loss in the superior visual field. In the latter experiment, short-term monocular deprivation from birth had no effect on any part of the visual field while long-term deprivation left the eye blind. These results, especially in deprived monkeys, would suggest that longer deprivation will lead to greater restrictions in all parts of the visual field. Yet, from the work in normal infants (van Hof-van Duin & Mohn, 1987), toddlers (Heersema et al., 1989), and children (see Experiment 1), it is known that the superior visual field reaches

adult size before other portions of the visual field. Possibly, the relatively fast maturation of the superior visual field provides some protection from the effects of prolonged abnormal visual experience.

With regard to the impact of monocular versus binocular deprivation, this study indicated that, at least for tests with the dim light, monocularly deprived eyes exhibited more restricted visual fields than did binocularly deprived eyes. It is possible that the poorer visual acuity typically found in monocularly deprived children than in binocularly deprived children (see Maurer et al., 1989) is able to explain this finding as visual acuity was significantly correlated with loss of the field at some meridia for tests with the dim light. Alternatively, there may be a true difference in the functioning of monocularly deprived versus binocularly deprived children which only is discernible for extremely sensitive tests, such as with a small, dim stimulus. The more severe impact of monocular than of binocular deprivation upon peripheral vision is supported by behavioral work in deprived cats (Sherman, 1973) but the issue has not been studied in deprived monkeys. As well, monocular deprivation has more deleterious effects than does binocular deprivation upon other visual functions in deprived humans, such as color vision, visual acuity, stereoacuity, and contrast sensitivity (Maurer et al., 1989; Maurer & Lewis, in press).

These pieces of evidence suggest that competition between a nondeprived and a deprived eye (as occurs in monocular deprivation) is more harmful to normal functioning than is

deprivation without such competition (as in binocular deprivation). Behavioral work in cats also suggests that the relative amount of looking time that each eye receives is important. For example, when cats are raised with periods of unequal and alternating monocular input between the two eyes, then the more experienced eye has a normal visual field. In contrast, the less experienced eye has a restricted field, with greater restrictions found in those cats with more unequal periods of monocular input (Tumosa et al., 1980, 1982). Similarly, removal of competition through retinal lesions of the nondeprived eye in monocularly deprived monkeys (Hendrickson et al., 1977) or enucleation of the nondeprived eye in monocularly deprived monkeys (Joseph & Casagrande, 1980) or in cats (Smith et al., 1982; but see Heitlander & Hoffmann, 1978) increases the extent of the field for the deprived eye. In contrast, studies of reverse suturing of the nondeprived eye in monocularly deprived cats (Sherman, 1973, 1974b; Smith et al., 1982; but see Heitlander & Hoffmann, 1978; van Hof-van Duin, 1977) and in monocularly deprived monkeys (Joseph & Casagrande, 1980; Wilson et al., 1989) or reverse suturing of one eye in binocularly deprived cats (Sherman, 1974b) indicate that there was no improvement of the peripheral vision of the previously deprived eye. This may be, however, because all reverse suturing was attempted relatively late.

In addition to the evidence from behavioral studies, physiological studies also indicate that monocularly deprived cats and monkeys are more impaired than are binocularly deprived

members of the same species. For example, the binocular segments of the striate cortex are more affected in monocularly deprived cats and monkeys than in binocularly deprived animals (Mitchell & Timney, 1984; Sherman & Spear, 1982).

To summarize, then, the behavioral and physiological evidence suggest that there is some adverse effect of competition from the nondeprived fellow eye in monocularly deprived animals and that this effect sometimes can be minimized if the competitive advantage of the nondeprived eye is removed. My finding of larger losses in monocularly deprived children than in binocularly deprived children is consistent with these findings, as is my finding that excellent patchers have a larger visual field than poorer patchers. However, the effect of patching was significant only at 0° temporally. Three explanations exist as to why there was no significant effect of patching other than at 0° temporally. First, occlusion of the nondeprived eye of children may be similar to reverse suturing in animals, which also usually provides no competitive advantage. Second, perhaps there was such a limited range of patching histories amongst the children, with none of the children patching enough, and relatively few children assessed, that any effect of alteration of the interocular competition upon most locations in the visual fields was not noticeable. Third, 0° temporally is the last part of the field to develop (see Experiment 1). As such, it may have been most sensitive to the effects of the removal of interocular competition through occlusion of the nondeprived eye. Overall, my data suggest

that monocular deprivation either affects different pathways than does binocular deprivation or affects the same pathways more severely. Once the pathways are affected, then there may be some effects of altering competition by adjusting the patching regime. The physiological basis of these behaviors will be addressed in detail in Chapter 4.

Finally, this study indicated that there were no effects of the timing of the visual deprivation: Children diagnosed with congenital cataracts did not exhibit significantly smaller visual fields than did children diagnosed with later onset cataracts. Studies of monocularly deprived cats (Bisti & Carmignoto, 1988), monkeys (Joseph & Casagrande, 1980; Sparks et al., 1986; Wilson et al., 1989), and a few previously studied patients (Maurer et al., 1983) provide some indication that timing may be an influential variable. Given the work in animals and in other deprived humans, it was surprising to find that there were no differential effects upon peripheral vision of the timing of visual deprivation. One possibility is that the timing of deprivation truly does not affect peripheral vision in humans because the sensitive period for peripheral vision lasts for many years. Overall, my finding that the timing of deprivation is unimportant complements the finding that even congenital cataracts do not affect the discrimination of shape or, except in cases of long-term unilateral congenital deprivation, of color (Maurer et al., 1989). However, my finding contrasts with reports of the influence of timing of deprivation upon visual acuity and optokinetic nystagmus (Maurer

& Lewis, in press). Other possible explanations for not finding an effect of the timing of deprivation, discussed below, include weaknesses in the design of the study, bias in the tested sample of children treated for congenital cataracts, and difficulty in pinpointing the parameters of deprivation in some groups of deprived eyes.

First, in my tested sample, the age of onset of deprivation was never later than 72 months, preventing evaluation of the effects of deprivation of extremely late onset. Every eye in my sample had a restricted visual field; perhaps with still later onset, some eyes would have normal peripheral vision.

Second, there may have been no effect of the timing of deprivation upon peripheral vision because my sample of children deprived from birth was unrepresentative of the population. Unlike children treated for cataract of late onset, approximately 50% of the sample of children treated for either unilateral or bilateral congenital cataracts exhibited marked nystagmus upon occlusion of the fellow eye and, therefore, were untestable using the Goldmann perimeter. If marked nystagmus is indicative of an eye with a poor prognosis, it may be correlated with restricted fields. Consequently, I may have eliminated the children treated for congenital cataracts with the most restricted visual fields. To better address the issue of the influence of the timing of deprivation upon peripheral vision, I present in Experiment 5 the data from tests with the Octopus perimeter. Because of the brevity of its trials, the Octopus perimeter can be used to test even children with marked nystagmus. This approach

prevents loss of patients and the subsequent introduction of sampling bias into the results.

Third, unlike studies of lid suture in animals, it may be difficult to pinpoint the precise age of onset of deprivation in children treated for cataracts, especially in some groups of deprived eyes. We can be sure of the age of onset of deprivation in children who receive an eye injury sufficiently severe to cause a cataract. We also can be reasonably sure of the age of onset in children with congenital cataract. In my sample, almost all of the cataracts were diagnosed shortly after birth. Although I cannot be certain that the cataract was present from birth, I can be reasonably confident because cataracts do not develop quickly. But for two reasons, there may be more doubt concerning the age of onset in children who develop cataracts sometime after birth. First, it is important to acknowledge that a developing but not yet dense and central cataract may interfere with vision. As such, by using the age of diagnosis of a dense and central cataract to judge the child's age of onset and duration of deprivation, we may underestimate the true length of deprivation. The second point is related to the first. In cases of a unilateral developmental cataract, the child may have appeared to have seen well because he relied upon his fellow nondeprived eye. Consequently, he may not have been brought to the attention of an ophthalmologist. Lack of medical attention is less likely to occur for children who develop bilateral cataracts because the parent likely will notice that the child appears to see poorly and a subsequent ophthalmological

examination will confirm the suspicion. In cases of children treated for unilateral developmental cataract, then, the age of onset may be earlier and the duration of deprivation longer than can be verified.

These potential biases introduce some uncertainty into the interpretation of data from children treated for developmental cataracts. If some of the children with later onset of deprivation had earlier and, consequently, longer deprivation than what was documented in their medical charts, then this would act to minimize any difference in outcome between children with early onset versus late onset of deprivation and putatively the same duration of deprivation. The resultant effect may be, as was found, that the age of onset of deprivation does not affect the extent of the visual field.

In conclusion, this study found that the visual fields of all children treated for a cataract are restricted, especially temporally, regardless of the timing and duration of deprivation and whether it was monocular or binocular. The effects were largest following longer duration and monocular deprivation. Moreover, the extent of the temporal field was affected by the patching history of the nondeprived eye in children treated for unilateral cataracts. Experiment 5 investigates the influence of deprivation upon thresholds across the field. Experiment 6 examines whether the noted effects upon peripheral vision have an optical basis.

Experiment 5

Light sensitivity across the visual field in children treated for cataracts

As shown by Experiments 1 and 3, both the extent of the visual field and light sensitivity across the field require many years to develop in visually normal children. Given that the extent of the visual field is vulnerable to the effects of deprivation from cataracts (see Experiment 4), I next investigated whether sensitivity across the field also was vulnerable. As reviewed in the introduction, to date, there have been three studies examining the effects of visual deprivation on peripheral vision. Static perimetry has been used to test only along the horizontal meridian two patients monocularly deprived from birth to either 5 months or 19 years, one patient binocularly deprived from birth to 11 months, one patient binocularly deprived from 11 months until 3 years of age, and four patients unilaterally or bilaterally deprived after 7 months of age for less than 6.5 months. The results indicate that the deprived eyes have decreased sensitivity across the horizontal visual field, with only the patients monocularly deprived from birth exhibiting especially large losses in the nasal visual field (Lewis et al., 1986; Maurer et al., 1983; Mioche & Perenin, 1986; Moran & Gordon, 1982).

In the following experiment, I examined the effect of visual deprivation on sensitivity not only along the horizontal meridian but also across the visual field and in a larger sample of children treated

for cataracts than previously tested. To the extent possible, I also examined the influences of the timing and duration of the deprivation and whether it was monocular or binocular. These variables were important to examine because studies of visually deprived cats, monkeys, and humans provide clues that these parameters affect peripheral vision (see Chapter 1). The entire visual field was assessed because studies of monocularly deprived cats (van Hof-van Duin, 1977) and monkeys (Sparks et al., 1986) indicate that the loss of sensitivity is not restricted to the horizontal meridian, the only area previously explored in deprived humans. I also chose to examine sensitivity in more detail at 20° nasally and 30° temporally because visually normal adults (Lakowski & Aspinall, 1969; Liao, 1973) and 2-month-old infants (Lewis et al., 1985), but not 1-month-olds (Lewis et al., 1985), have better sensitivity at 20° nasally than at 30° temporally. Given the pattern of development of sensitivity at 20° nasally versus 30° temporally between 1 and 2 months of age, deprivation during this time versus later in childhood may affect sensitivity differently.

Method

Subjects. As in Experiment 4, the subjects were children treated for dense and central cataracts, which were present either unilaterally or bilaterally, and were congenital or developmental or traumatic in nature (see Tables 48 and 49). Although the criteria for inclusion

were identical to those of Experiment 4, the sample of children was not identical across the two experiments. First, children in Experiment 4 were as young as 5 years of age while, at the time of the test in Experiment 5, each child was 7 years of age or older. Second, because of the brevity of trials with the Octopus perimeter, even children with marked nystagmus could be tested, unlike tests with the Goldmann perimeter (see Experiment 4). Finally, because there were different but overlapping groups of children assessed for the test of the whole visual field (program 24) and for the tests at 20° nasally and 30° temporally (program 62), the subjects composing each assessment will be described separately.

(1) Test of the whole visual field (program 24):

To determine the effects of monocular deprivation (Table 48), I tested nine abnormal eyes and four fellow nondeprived eyes of nine children deprived from birth for varying periods of time (unilateral congenital group). For comparison, I tested five abnormal and four fellow nondeprived eyes of children (unilateral developmental group) who had normal visual experience early in life and then either developed a cataract in one eye (n = 3) or sustained an eye injury (n = 2) which led to the formation of a cataract (Table 48).

The parents of children treated for a unilateral cataract had been instructed by the ophthalmologist to patch the fellow nondeprived eye 50% of the waking time during the first five years of life in order to force usage of the previously deprived eye. Of the

children treated for a unilateral cataract, compliance with the patching regime varied from excellent (patching of the fellow nondeprived eye 40% to 50% of the time, n = 5 children) to fair (patching 20% to 30% of the time, n = 7 children) to poor (patching 0% to 19% of the time, n = 2 children).

To determine the effects of binocular deprivation (see Table 48), I tested 18 eyes from 12 children bilaterally deprived from birth for varying periods of time (bilateral congenital group). For comparison, I also tested 14 eyes from 8 children who had normal early experience and then developed a cataract in both eyes (bilateral developmental group). Note that both eyes were not tested in some cases because of insufficient time in the clinical setting, because an associated condition rendered one eye unsuitable for this study (see exclusion criteria described in Experiment 4), or because one eye had been enucleated. Finally, no child had developed a cataract as the result of trauma.

(2) Tests of 20° nasally and 30° temporally (program 62):

To determine the effects of monocular deprivation (Table 49), I tested twelve abnormal eyes and ten fellow nondeprived eyes of 12 children deprived from birth for varying periods of time (unilateral congenital group). For comparison, I tested nine abnormal and eight fellow nondeprived eyes of children (unilateral developmental group) who had normal visual experience early in life and then either developed a cataract in one eye (n = 3) or sustained an eye

injury (n = 6) which led to the formation of a cataract (Table 49). Of the children treated for a unilateral cataract, compliance with the patching regime varied from excellent (patching of the fellow nondeprived eye 40% to 50% of the time, n = 4 children) to fair (patching 20% to 30% of the time, n = 11 children) to poor (patching 0% to 19% of the time, n = 6 children).

To determine the effects of binocular deprivation (see Table 49), I tested 23 eyes from 14 children bilaterally deprived from birth for varying periods of time (bilateral congenital group). For comparison, I also tested 18 eyes from 12 children who had normal early experience and then developed a cataract in both eyes (bilateral developmental group).

Apparatus. The Octopus perimeter and stimuli were identical to those of Experiment 3. Briefly, the stimulus was 0.43° in size and projected for 0.1 second against a background of 4 apostilbs (1.3 cd/m^2). The intensity of the stimulus ranged from 0.008 apostilbs (0.0025 cd/m^2) to 1000 asb (318 cd/m^2).

Procedure. With three exceptions, the procedure was identical to that of Experiment 3. Each child had one eye occluded and sat in front of the Octopus perimeter with his chin on the chin rest and forehead against the forehead rest. The start of each trial was signalled to the child by a beeping noise. At that time, the child was asked to fixate the central fixation aid and to indicate awareness of a

light presented randomly within the perimeter by pushing a button. Interspersed throughout the assessment were positive catch trials, a trial during which a stimulus was not presented, and negative catch trials, a trial during which the stimulus of maximum intensity which the child had detected at the start of the procedure was presented again.

The three differences from Experiment 3 were as follows. First, the deprived eye wore a contact lens of a power designed to focus the eye at the testing distance of 43 cm. Second, because of time constraints in the clinical situation, sometimes both eyes could not be tested during one visit. The eye selected to be tested first was the deprived eye in unilateral cases and the eye with the better acuity in bilateral cases. If in bilateral cases there was no interocular difference in acuity, then one eye was selected randomly for the test. On a subsequent visit to the clinic, the untested eye was assessed using the same protocol. Third, the tests of the deprived children were not as extensive as those of the normal children. Each deprived subject first was tested with a fast bracketing program (program 24, which assessed the whole visual field) and then with a normal bracketing program (program 62, which assessed a 12° by 12° sector located anywhere within the visual field). The test sectors to which the normal bracketing program was applied were centered on the horizontal meridian at 20° nasally and at 30° temporally with order randomized across subjects. Unlike the tests of the visually normal

children, there were no tests of other portions of the horizontal meridian with the normal bracketing program.

Data analysis. The thresholds for the assessed locations (0° centrally, near periphery, mid periphery, far periphery, 20° nasally, and 30° temporally) were calculated as in Experiment 3. In some of the subsequent analyses, loss scores were calculated for each deprived eye. The loss score represents the amount of loss of sensitivity at that location in the visual field of the deprived eye relative to normal. A loss score was calculated by subtracting the sensitivity of the deprived eye from that of the fellow nondeprived eye for each location assessed. If the nondeprived eye was not tested, as occurred in some unilaterally deprived children, or if the child did not possess a nondeprived eye, as occurred in bilaterally deprived children, then the comparison data were from the group of normal children of the same age (see Experiment 3).

Results

Several questions were posed. First, as indicated previously, it is preferable to compare performance in two related eyes because interocular differences in sensitivity are smaller than the variability across normal individuals at each location across the field. Therefore, I determined whether the fellow nondeprived eye in children treated for unilateral cataracts exhibited normal sensitivity across

the visual field and at 20° nasally and 30° temporally. If so, then it may be used as a standard by which to evaluate the results of the deprived eye in children treated for unilateral cataracts. Second, I examined whether deprivation altered sensitivity across the visual field or at 20° nasally and 30° temporally. These analyses investigated the influence of the timing and duration of deprivation, whether the deprivation was monocular or binocular in nature, and the influence of the patching of the nondeprived eye upon sensitivity. I first will present the results from all tests of the entire visual field and then present the results for tests at 20° nasally and 30° temporally.

(1) Sensitivity across the whole visual field of the nondeprived fellow eye of monocularly deprived children:

In order to determine whether the fellow nondeprived eye of monocularly deprived children was normal, the thresholds for each of the four field sectors (0° centrally, near, mid, and far periphery) of the nondeprived eye was compared to those of normal volunteers (see Experiment 3). The normative comparison group were adults because there often was only one deprived subject tested at each of the younger ages. As well, because the nondeprived eye of the deprived subjects was tested once, their data were compared to the normative data established on the first test of the field (see Experiment 3). Note that this analysis did not evaluate the role of

patching of the nondeprived eye upon its peripheral vision because of the scarcity of fellow eyes assessed.

A 2-way ANOVA with a between group factor of visual history (three levels) and a within group factor of location (four levels) was calculated (see Table 50). Visual history was defined as having had normal visual experience in both eyes (normal volunteers) or having had monocular deprivation beginning at birth (children treated for a unilateral congenital cataract) or beginning some time after birth (children treated for a unilateral developmental cataract).

(a) Effect of visual history:

There was no significant main effect of visual history (p = .65).

(b) Effect of location:

There was a significant effect of location (p < .00001). Tukey's HSD tests revealed that sensitivity was highest at 0° centrally (all p's < .01), and then decreased significantly with increasing eccentricity (all p's < .01). Finally, there was no significant interaction of visual history by location (p = .88).

This analysis indicated that the fellow nondeprived eye of monocularly deprived subjects did not have significantly different sensitivity than that found in normal volunteers. As such, it is appropriate to use the data from the nondeprived eye of the

monocularly deprived subjects as a standard by which to evaluate their deprived eye.

(2) Sensitivity across the whole visual field of the deprived eyes:

The most striking result was that in every case, the deprived eyes were less sensitive than normal. The data were analyzed with a 4-way ANOVA (see Table 51) with between group factors of condition (two levels), timing (two levels), and duration (two levels) and a within group factor of location (four levels). Condition of deprivation consisted of eyes treated for either monocular or binocular deprivation. Timing of deprivation consisted of eyes with a diagnosis of a dense and central cataract at birth (congenital deprivation) or eyes with an early normal history that were diagnosed with a cataract between 6 months and 72 months of age (developmental deprivation). Duration of deprivation consisted of eyes with either short duration (defined as less than or equal to 6 months) or with long duration (defined as lasting more than 6 months). Location consisted of the four areas in the visual field into which the data were divided (0° centrally, near, mid, and far periphery).

(a) Effect of condition:

There was no main effect of condition: Unilaterally deprived and bilaterally deprived subjects exhibited similar losses of sensitivity (p = .93).

(b) Effect of timing:

There was no main effect of timing: Subjects deprived from birth and subjects deprived beginning after birth showed similarly sized losses of sensitivity (p = .12). There was no significant interaction of condition with timing (p = .89).

(c) Effect of duration:

There was no main effect of duration (p = .24), with subjects deprived for 6 or fewer months exhibiting the same sized losses as subjects deprived for longer periods. There were no significant interactions of duration with condition (p = .39), duration by timing (p = .25) or duration with condition with timing (p = .58).

(d) Effect of location:

Sensitivity differed with location (p < .0001). Tukey's HSD tests revealed that the largest loss of sensitivity was obtained at 0° centrally (all p's < .01). The size of the loss was similar across the near, mid, and far periphery.

Moreover, location interacted significantly with timing (p < .00001). An analysis of simple effects revealed that children treated for developmental cataracts had less sensitive fields at 0° centrally than did children treated for congenital cataracts (p < .0001) (see Figure 10). In contrast, there were no significant differences between children treated for developmental cataracts or for congenital

cataracts in the near (p = .86), mid (p = .49), or far periphery (p = .42).

There were no significant interactions of location with condition (p = .19), location by timing by condition (p = .14), location by duration (p = .88), location by duration by condition (p = .99), location by duration by timing (p = .30), or location by duration by timing by condition (p = .56).

To summarize, every deprived eye exhibited a loss of sensitivity. Moreover, children treated for developmental cataracts exhibited larger losses of sensitivity at 0° centrally than did children treated for congenital cataracts.

(3) Normality of the fellow nondeprived eye of monocularly deprived subjects at 20° nasally and at 30° temporally:

In order to determine whether the fellow nondeprived eye of monocularly deprived children was normal, the thresholds at 20° nasally and at 30° temporally of the nondeprived eye were compared to those of normal volunteers (see Experiment 3). The normative comparison group was adults because there were insufficient deprived subjects tested at each of the younger ages to allow analysis. As well, because the nondeprived eye of the deprived subjects was tested once, their data were compared to the normative data established on the first test of the field (see Experiment 3).

A 2-way ANOVA with a between factor of group (four levels) and a within factor of location (20° nasally, 30° temporally) was calculated (see Table 52). Group was defined as having had normal visual experience in both eyes (normal volunteers) or having been a monocularly deprived subject who was an excellent, fair, or poor patcher. Because of an insufficient number of subjects, there was not comparison of performance for children treated for unilateral congenital versus unilateral developmental cataracts.

(a) Effect of group:

There was no significant effect of group (p = .62).

(b) Effect of location:

There was no significant effect of location (p = .70) or interaction of location with group (p = .32).

The analysis indicted that the fellow nondeprived eye of monocularly deprived subjects did not differ significantly in sensitivity from that of normal volunteers. As such, it is appropriate to use the nondeprived eye as a standard by which to evaluate the data of the deprived eye in cases of monocular deprivation.

(4) Sensitivity at 20° nasally and 30° temporally in deprived subjects:

The most striking result was that the sensitivity of the deprived eyes was abnormal in every case. The performance of the deprived eyes was analyzed with a 4-way ANOVA (see Table 53) with between group factors of condition (two levels), timing (two levels), and duration (two levels) and a within group factor of location (two levels). Condition of deprivation consisted of patients treated for monocular or binocular deprivation. Timing of deprivation consisted of eyes with a diagnosis of a dense and central cataract at birth (congenital deprivation) or eyes with an early normal history that were diagnosed with a cataract between 6 and 156 months of age (developmental deprivation). Duration of deprivation consisted of eyes with either short duration (defined as less than or equal to 6 months) or long duration (defined as lasting more than 6 months). Location consisted of tests centered at 20° nasally and 30° temporally.

(a) Effect of condition:

There was no main effect of condition: Unilaterally deprived and bilaterally deprived subjects exhibited similar losses of sensitivity (p = .46).

(b) Effect of timing:

There was no main effect of timing: Subjects deprived from birth and subjects deprived beginning after birth showed similarly sized losses of sensitivity (p = .77). There was no significant interaction of condition with timing (p = .23).

(c) Effect of duration:

There was no main effect of duration (p = .20), with subjects deprived for 6 or fewer months exhibiting the same sized losses as subjects deprived for longer periods. There were no significant interactions of duration with condition (p = .88), duration with timing (p = .32), or duration with condition with timing (p = .15).

(d) Effect of location:

There was no significant main effect of location (p = .08) or significant interactions of location with condition (p = .09) or of location with timing (p = .18). There was, however, a significant interaction of location with condition with timing (p = .003) (see Figure 11). An analysis of simple effects revealed that there was a significant interaction of location by timing for the children treated for unilateral cataracts (p = .0093) but not for the children treated for bilateral cataracts (p = .13). Further analysis of simple effects of the significant interaction revealed that there was not a significant effect of location for children treated for unilateral developmental cataracts (p = .72). In contrast, there was a significant effect of

location for children treated for unilateral congenital cataracts (p = .0001), such that this group had larger losses of sensitivity at 20° nasally than at 30° temporally.

Finally, there were no significant interactions of location with duration (p = .08), location with duration with condition (p = .25), location with duration with timing (p = .08), or location with duration with timing with condition (p = .30).

To summarize, this analysis indicated that only the children treated for unilateral congenital cataracts had larger losses at 20° nasally than at 30° temporally.

(5) Influence of patching of the nondeprived eye on the peripheral vision of the deprived eye:

The amount of patching of the nondeprived eye varied across monocularly deprived children from excellent to fair to poor. Because sensitivity across the visual field and then at 20° nasally and 30° temporally was assessed in different groups of patients, two ANOVA's were necessary to evaluate the influence of patching of the nondeprived eye on the loss scores of the deprived eye.

(i) Effect of patching on loss scores across the field:

The 2-way ANOVA (see Table 54) had a between factor of patching (three levels) and a within factor of location (four levels: 0° centrally, near, mid, and far periphery). The mean duration of

deprivation was similar across groups being 6.1, 7.4, and 7.8 months for the excellent, fair, and poor patchers, respectively. However, the onset of deprivation varied across groups being 12.0, 53.0, and 46.0 months for the excellent, fair, and poor patchers, respectively. Given the small sample, no subjects were eliminated from the analysis so as to allow matching for the age of onset of deprivation.

(a) Effect of patching:

There was no significant effect of patching (p = .49).

(b) Effect of location:

There was a significant effect of location (p = .0129), with Tukey's HSD tests revealing that the loss scores at 0° were greater than those in the near periphery (p < .05) but not elsewhere. There was no significant interaction of patching with location (p = .44).

(ii) Effect of patching on loss scores at 20° nasally and 30° temporally:

The 2-way ANOVA (see Table 55) contained a between factor of patching (three levels) and a within factor of location (two levels: 20° nasally, 30° temporally). The duration of deprivation was similar across groups being 6.8, 7.8, and 5.9 months for the excellent, fair, and poor patchers, respectively. However, the onset of deprivation varied across groups being 15.0, 8.0, and 111.1 months for the excellent, fair, and poor patchers, respectively. Given the small

sample, no subjects were eliminated from the analysis so as to match the groups for the age of onset of deprivation.

(a) Effect of patching:

There was no significant effect of patching (p = .54).

(b) Effect of location:

There was no significant effect of location (p = .20) and no significant interaction of patching with location (p = .77).

To summarize, these analyses indicated that there was no significant influence of patching of the nondeprived eye on the sensitivity of the deprived eye.

(6) Influence of visual acuity:

One possible explanation for a loss of sensitivity may be that the typically poorer visual acuity of a deprived eye, relative to a normal eye (see Maurer et al., 1989), is limiting peripheral detection. Note that both visual acuity and peripheral vision in a deprived eye are likely to be influenced by the same parameters; that is, by the timing and duration of deprivation and whether it is monocular or binocular (see Maurer et al., 1989). Although there were few significant correlations of visual acuity and the extent of the visual field in deprived subjects (see Experiment 4), the influence of acuity on peripheral sensitivity also needs to be evaluated.

To evaluate the influence of visual acuity, Pearson correlations were calculated between the log 2 value of the deprived eye's median visual acuity and its loss scores at 0° centrally, near, mid, and far periphery (n = 33 eyes), or at 20° nasally and 30° temporally (n = 46 eyes). Note that for patients who contributed data from two deprived eyes, only the acuity of the right eye was used in the correlation so as to avoid sampling acuities from two related eyes.

With alpha corrected for the number of tests, there were no significant correlations (2 tailed tests) between the log 2 value of the median Snellen acuities and the loss scores at 0° centrally, near, mid, and far periphery or at 20° nasally or 30° temporally (Table 56).

Discussion

This study indicated several important findings. First, relative to the fellow nondeprived eye or to normal volunteers, deprived eyes were less sensitive. Second, there were some effects of the timing of deprivation, whether the deprivation was monocular or binocular in nature, and the location in the field at which sensitivity was assessed. Third, the fellow nondeprived eye of monocularly deprived children exhibited normal sensitivity and thus was acceptable as a standard to evaluate the data from the fellow deprived eye.

The most important finding was that every deprived eye exhibited a loss of sensitivity across the visual field. This is the first

test of sensitivity on other than the horizontal meridian in deprived humans and parallels similar findings in monocularly deprived cats (van Hof-van Duin, 1977) and monocularly deprived monkeys (Sparks et al., 1986). The loss of sensitivity observed in the deprived eyes is unlikely to result from strabismus or poor visual acuity. First, there are no systematic effects of strabismus on sensitivity in humans (Aggarwal & Verma, 1980; Fusco et al., 1983; Inoue et al., 1985; Jacobson et al., 1983; Mehdorn, 1986; Sireteanu & Fronius, 1989). Second, there were no significant correlations of visual acuity with loss of sensitivity.

Moreover, this research found especially large losses of sensitivity at 0° centrally, particularly in children treated for developmental cataracts. Possibly, the central visual field is especially vulnerable to deprivation given its slow development in normal children (see Experiment 3). Other aspects of visual behavior occurring in the center of the visual field also are vulnerable to deprivation. For example, children treated for unilateral congenital cataracts, although not children treated for bilateral congenital or traumatic cataracts, exhibit larger losses of contrast sensitivity centrally compared to peripherally (Tytla, Lewis, Maurer, & Brent, 1991). As well, larger central rather than peripheral losses of sensitivity have been reported in one study of strabismic amblyopia (Sireteanu & Fronius, 1989; but see Mehdorn, 1986).

It was surprising to find that children treated for a cataract of later onset exhibited a poorer outcome at 0° centrally than did

children with congenital onset. This occurred despite the fact that the mean visual acuity of the children in the developmental group was higher. Nor can this difference be explained by the argument that the data arose from a biased sample. In Experiment 4, some children treated for a congenital cataract could not be tested with the Goldmann perimeter because of their nystagmus, which potentially created a biased sample. With tests of the Octopus perimeter, unlike those of the Goldmann perimeter, it is possible to test even children treated for a congenital cataract who have nystagmus and, as such, there was no loss of subjects.

Rather, there are three possible explanations of the difference in performance between the children treated for congenital versus for developmental cataracts. First, the difference in performance may reflect the limited and hence potentially unreliable sampling of thresholds at 0° centrally, relative to the more extensive sampling in the peripheral portions of the field. Note that Experiment 3, which used the same procedure and apparatus as in this experiment, revealed that there was poorer test-retest reliability at 0° centrally than elsewhere in the field for the 7-year-olds, but not for the adults. Moreover, one conclusion from Experiment 3 was that normal 8- and 9-year-olds had lower thresholds at 0° centrally compared to 7-year-olds and adults. It may be, then, that the fast bracketing procedure used by the Octopus perimeter is less accurate for assessment of performance at 0° centrally. Further research should be directed towards examining performance at 0° centrally in even

larger samples of normal and deprived children using more extensive normal bracketing procedures.

Second, as reviewed previously, the age of onset of deprivation is not always certain in children who develop a cataract (nontraumatic cases) after a period of normal visual experience. Some of the developmental cases might have had earlier and longer deprivation than the medical records indicate. If the patients treated for developmental cataracts had deprivation that started earlier in life than what their medical charts indicate, and their deprivation was longer than that of the children treated for congenital cataracts, then it may not be surprising to find that the fields of the children treated for developmental cataracts were even more insensitive than those of children treated for a congenital cataract.

Third, the difference in performance may be real. The central vision of the newborn infant is poor and improves dramatically with age (van Hof-van Duin & Mohn, 1986). As such, a short period of deprivation from birth may exert little effect upon the extremely poor central vision of the infant. In contrast, deprivation beginning at later ages, when the central vision of the infant is improving, may be more detrimental. Relative to the performance at 0° centrally, smaller losses of equal magnitude were found in the near, mid, and far periphery. That children treated for developmental cataracts do not have larger peripheral losses than do children treated for congenital cataracts may reflect the fact that peripheral sensitivity is always poor, at least relative to central sensitivity (see Experiment

3), and so may be less affected by deprivation or by when it begins. Results from tests using the Goldmann perimeter complement these findings: the loss of the extent of the field is similar for children treated for congenital or for developmental deprivation (see Experiment 4).

The present experiment also reported that children treated for unilateral congenital cataracts had larger losses of sensitivity at 20° nasally than at 30° temporally compared to other groups of deprived eyes. Experiment 4 found that children treated for unilateral cataracts exhibited more restricted fields than children treated for bilateral cataracts. Together, these results suggest that competition very early in life from the nondeprived fellow eye plays some part in influencing peripheral vision. However, if competition is influential in affecting development, it might be expected that the history of patching of the nondeprived eye would affect peripheral vision, which was not demonstrated in this experiment but was demonstrated in Experiment 4. Moreover, given that long-term monocular deprivation affects the central retina in monkeys (von Noorden, Crawford, & Middleditch, 1977), then interocular competition might be expected at least to affect sensitivity in the central visual field. There are four reasons why the effects of the removal of interocular competition through patching of the nondeprived eye may not have been observed in Experiment 5. First, possibly patching of the nondeprived eye truly does not affect sensitivity in the deprived eye. Yet, Experiment 4 revealed that

patching did affect the extent of the temporal field of the deprived eye. Possibly, competition has different effects upon sensitivity than upon the extent of the field. Second, the groups used in the analyses of patching in Experiment 5 were not well matched on the variable of age of onset of deprivation, which may bias the outcome. In contrast, the groups in Experiment 4 were well matched for both the age of onset and the duration of deprivation. Third, the groups in Experiment 4 tended to have an earlier onset of deprivation than the groups in Experiment 5. Patching beginning earlier, rather than later, in life might modify outcome more. Fourth, the sampling technique used in Experiment 5 was fast bracketing which, as outlined in detail previously, has some weaknesses. Using a normal bracketing strategy, future research should evaluate the influence of occlusion of the nondeprived eye on peripheral vision, especially in the central and far temporal fields, in groups of patients well matched for such variables as timing and duration of deprivation. Such research would constitute both an empirical test of the theory of competition and an assessment of the ophthalmological treatment of children with unilateral cataracts.

There exists another apparent difference in results between Experiment 4 and this experiment. Experiment 4 reported that the extent of the <u>temporal</u> field was most restricted. In contrast, the present experiment reported that the <u>central</u> field and the <u>nasal</u> field were most affected. There may be two reasons for this difference. First, nasal and temporal differences <u>near the center of the field</u>

were examined with static perimetry in the present experiment as compared to examination of the peripheral edges of the field with kinetic perimetry in Experiment 4. Deprivation may have its most deleterious effects upon the central, nasal, and temporal areas of the visual field. To confirm this possibility, we need to use static perimetry to systematically sample nasal and temporal thresholds across the horizontal meridian from 0° centrally to the furthermost edges of the field in deprived eyes, as has been done in normal children (see Experiment 3).

Second, it could be argued that the seemingly discrepant results from the present experiment and from Experiment 4 reflect a general pattern of development and vulnerability. Initially, in young babies, the near temporal field (e.g., 30° temporally) begins to develop before the near nasal visual field (e.g., 20° nasally) (Lewis et al., 1985; Maurer & Lewis, 1991). Development of both hemifields then progresses with the far temporal field (see Experiment 1) and perhaps 0° centrally (see Experiment 3) developing last, as assessed with small stimuli in school aged children. While development of the field is occurring, it should be vulnerable to abnormal visual experience. As such, it may not be surprising to find that there will be a general trend for deprivation to affect most the latest developing parts of the field (e.g., 0° centrally and the extreme temporal field). Moreover, because the development of the near nasal field lags behind that of the near temporal field early in life, certain types of experience, such as monocular deprivation beginning

at birth, may exert more deleterious effects at 20° nasally than 30° temporally.

Indeed, Experiment 5 found that children treated for unilateral congenital cataracts, but not other groups of deprived eyes, exhibited larger losses at 20° nasally than at 30° temporally. This finding parallels previous work with a few patients which indicated that the nasal visual field is particularly affected in children treated for unilateral congenital cataracts (Lewis et al., 1986; Moran & Gordon, 1982) but not in children treated for bilateral congenital or bilateral developmental cataracts (Mioche & Perenin, 1986). In general, children treated for unilateral congenital cataracts exhibit a more infantile pattern of behavior than do children treated for bilateral cataracts. For example, children treated for unilateral congenital cataracts have larger losses of contrast sensitivity in the nasal visual field than in the temporal field (Tytla et al., 1991). In contrast, children treated for bilateral congenital cataracts or for traumatic cataracts exhibit a constant loss of contrast sensitivity across eccentricity (Tytla et al., 1991). In addition, children treated for unilateral congenital cataracts exhibit decreased loss of contrast sensitivity as the flicker rate of gratings increases, except for the coarsest stripes (Maurer & Lewis, in press; Tytla, Maurer, Lewis, & Brent, 1988). In contrast, children treated for bilateral congenital cataracts exhibit a constant loss of contrast sensitivity as the flicker rate of gratings increases (Maurer & Lewis, in press; Tytla et al., 1988). Like children treated for unilateral congenital cataract and

unlike normal adults, three-month-old infants exhibit better sensitivity to flickering stripes than to stationary stripes (Atkinson, Braddick, & Moar, 1977; Maurer & Lewis, in press). My finding that children treated for unilateral congenital cataract, unlike other groups of deprived eyes, have especially large losses of sensitivity in the nasal field complements the suggestion that the visual behavior of children treated for unilateral congenital cataracts is like that of infants.

Note, however, that the literature on nasal versus temporal differences in deprived animals is not identical to that of deprived humans. In cats, either monocular <u>or</u> binocular deprivation from birth particularly affects the nasal visual field (Sherman, 1973). As well, in monocularly deprived monkeys, the nasal field is not always most vulnerable (Hendrickson et al., 1977; Sparks et al., 1986; Wilson et al., 1989; but see Joseph & Casagrande, 1980). Possibly, because of species differences, studies of animals are not directly comparable to those with humans. To summarize, then, the present experiment agrees with previous findings in deprived humans concerning the effects of monocular deprivation from birth on sensitivity at 20° nasally relative to 30° temporally but differs from findings in deprived animals.

Thus far, I have examined the factors found to be significant in the analysis, such as condition, timing, and location. Examination of the effects which were nonsignificant, such as the duration of deprivation, also can be illuminating. This experiment indicated that

children with short periods of deprivation were as impaired as children with longer deprivation. To date, there has been no published study of the effects of the duration of deprivation in deprived humans or in lid sutured cats. However, duration of deprivation has been shown to affect the extent of the field when monocularly lid sutured monkeys were tested with statically presented targets of a fixed luminance (Sparks et al., 1986) and in dark reared cats tested with kinetically presented spoons of cat food (Maire-Lepoivre et al., 1988). Moreover, Experiment 4 indicated that the patients with longer duration of deprivation, as assessed with kinetic perimetry on the Goldmann perimeter, exhibited more restricted visual fields. Why was this pattern not confirmed in the present experiment?

It might be expected that longer duration of deprivation would affect those parts of the field slowest to develop, such as 0° centrally or the edges of the field. Possibly, Experiment 5 might have revealed some effects of the duration of deprivation on sensitivity if the assessment at 0° centrally had been conducted using a normal bracketing method or if I had analyzed sensitivity at the extreme edges of the field, instead of collapsing data. Finally, although Experiment 4 indicated that the patients with longer duration of deprivation had more restricted visual fields, its method of assessment was kinetic perimetry on the Goldmann perimeter. Measurement of the extent of the field in deprived animals using a target of fixed luminance (Maire-Lepoivre et al., 1988; Sparks et al.,

1986) is more similar to tests with the Goldmann perimeter than with the Octopus perimeter, which was used in the present experiment.

These experiments in children treated for cataract document significant losses. Before it can be argued that the losses are neurally based, it is necessary to evaluate the potential role of optical variables. It is likely that optics can explain some of the losses in peripheral vision exhibited by the deprived eyes. Yet, given the magnitude of the losses in peripheral detection and the systematic effects of the parameters of deprivation upon peripheral vision, it is likely that there is also a neural basis to the losses. The next experiment will evaluate in detail the possible influence of optics upon peripheral vision in deprived eyes. The final chapter will consist of an examination of the underlying physiological basis of peripheral vision and the effects thereon of deprivation.

Experiment 6

Control experiments

As noted in Experiments 4 and 5, both the extent of the visual field and sensitivity across the field were abnormal in every deprived eye. Before attributing these abnormalities solely to the abnormal functioning of neural mechanisms underlying peripheral vision, it is necessary to appreciate the possible contribution of optical factors. Affecting the optics of the eye are the cornea, the iris

and pupil, the crystalline lens, and the eye's dimensions. In this section, I will examine briefly the possible influence of the aforementioned factors upon peripheral vision in a deprived eye. This description will be supplemented, where possible, by experimental work conducted with aphakic subjects.

Subject. In all cases other than where noted, the following experiments were conducted with a seventeen-year-old female, J.W., who had been treated for a unilateral congenital cataract and fitted with a contact lens at 5 months of age, which she then wore regularly. Throughout the first five years of life, J.W. was an excellent patcher, occluding her nondeprived eye 50% of the waking time. The acuity of her nondeprived eye was 20/20 and that of her deprived eye 20/40, as measured with the Snellen eye chart. The deprived eye also exhibited a variable exotropia of approximately 1° (in which the eye turns outwards) with right hypotropia of approximately 7° (in which the eye turns upwards). Other than the treatment for her cataract, J.W. had no other surgery or ocular problems.

With regard to peripheral vision, J.W. exhibited both a restricted and less sensitive visual field in her deprived eye but a field of normal extent and sensitivity in her fellow nondeprived eye. For example, her deprived eye was restricted 28° temporally for the test with the intense light (stimulus I4e) with the Goldmann perimeter and exhibited a loss of 10 db at 0° centrally for the test of

sensitivity with the Octopus perimeter. These losses were comparable to those from the group of deprived eyes, which exhibited a mean restriction of 30.3° temporally and a mean loss of 10.8 db centrally on the same tests.

J.W. was chosen for the following experimental work for three reasons. First, optical parameters are alterable by visual deprivation. For example, the axial length of the eye increases during childhood (Larsen, 1971) and is abnormally long in eyes treated for a cataract (Rasooly & BenEzra, 1988; but see von Noorden & Lewis, 1987). Given that optical parameters might be affected more in an eye deprived from birth than one which had a later onset of deprivation, it is wisest to work with a subject like J.W. who experienced congenital deprivation. Second, J.W. possessed a fellow nondeprived eye which would serve as a standard against which to evaluate optical properties in the deprived eye. Third, J.W. was unusually cooperative and had reasonably steady fixation with her deprived eye.

(A) <u>The influence of the cornea:</u>

This section will present background concerning how the structure of the cornea may moderate peripheral vision as well as measurements of J.W.'s corneas.

The cornea is a highly transparent structure measuring in diameter approximately 12 mm and in thickness approximately 0.5 mm centrally and 0.7 mm peripherally. It has an anterior and a

posterior surface, both of which refract light. The cornea is composed of several layers: the epithelium, Bowman's membrane, the stroma, Descemet's membrane, and the endothelium. It is unnecessary to consider the details of each of these layers except to note that the media are not homogeneous and differences in their refractive indexes affect the deviation of light as it passes from one medium to another of a different density. This contributes to the scattering of light and so reduces the quality of the retinal image (Charman, 1983).

The shape of the anterior surface of the typical cornea in adults is ellipsoidal (Charman, 1983). At the anterior pole of the adult's cornea, the radius of curvature is 7.8 mm with a standard deviation of 0.4 mm (LeGrand & El Hage, 1980). Moving laterally, the cornea flattens and the radius of curvature of the peripheral cornea increases to about 12 mm. The radius of curvature and the size and shape of the cornea likely will affect peripheral vision. Peripheral flattening of the cornea improves the sharpness of focus by reducing the effects of spherical aberration (Charman, 1983). Spherical aberration occurs when rays of light nearer the edge of the pupil are bent more than rays nearer the optical axis (the line connecting the centers of the anterior and posterior cornea and anterior and posterior lens). Yet, if the cornea is too flattened, then a principle ray of light likely will be unable to enter its periphery, thereby decreasing the size of, or sensitivity across, the visual field. In

addition to the flattening of the cornea, its diameter may affect peripheral vision as a smaller cornea may catch fewer rays of light.

The anterior surface of the cornea contributes most of the refracting power of the eye - approximately 48 of 60 diopters (D). (A diopter is the unit of focal power, with the focal length being expressed in meters. So, for example, a contact lens of +3 D focuses light at 33 cm in front of a nonaccommodating emmetropic eye.) If the central or peripheral dioptric powers of the cornea are abnormally low then this would affect the bending of the light and its ability to reach the retina or the blur circle it forms on the retina, potentially resulting in a smaller or less sensitive field because of the loss of light.

In summary, changes in the shape, radius of curvature, or dioptric power of the anterior surface of the cornea will be particularly important in affecting peripheral vision (Charman, 1983).

The radius of curvature of the center of the posterior surface of the cornea is less than that of the anterior surface, being approximately 6.5 mm (Charman, 1983). In contrast to the anterior surface, the power of the posterior surface is a negative or diverging power (approximately -5.9 D) because the light travels from the higher refractive index of the cornea to the lower refractive index of the aqueous humor, the fluid which fills the chamber posterior to the cornea (Bennett & Rabbetts, 1989). Changes of the posterior surface

are relatively less important than of the anterior surface for refraction and hence for peripheral vision.

There is no published information comparing an aphakic eye to a normal eye in terms of corneal parameters such as dioptric power, radius of curvature, and position of the apex, and little information on the deforming effects on the corneal surface of surgery to remove the crystalline lens (Bennett & Rabbetts, 1989) Although most normal eyes have astigmatism (LeGrand & El Hage, 1980), following surgery to remove a cataract, there usually is some further deformation of the cornea (Bennett & Rabbetts, 1989). Astigmatism is defined as a defective curvature of the cornea which prevents the eye from bringing lines of different orientations into simultaneous sharp focus. Because a stimulus presented along an angle in the field may appear blurred, astigmatism potentially may limit the extent of, or sensitivity across, the visual field.

The following experiment was designed so as to allow measurement of the corneal parameters of J.W.'s deprived and nondeprived eyes. If the corneal parameters of J.W.'s deprived eye are abnormal, then these parameters may explain why J.W. has restricted and less sensitive fields. Conversely, if there is little or no difference between the eyes, yet the extent of, or sensitivity across, J.W.'s field is dramatically different, then this argues either for the role of other optical factors or for abnormality of neural pathways as the causal mechanism. The issue of whether J.W.'s results can be generalized to other patients will be addressed in the Discussion.

Method

The following procedure was designed to measure the corneal parameters of J.W.'s nondeprived and deprived eyes. At the beginning of the procedure, J.W. was instructed by the ophthalmic technician to fixate the red light emitting diode (LED) of the Allergan Humphrey Auto Keratometer (Model 420). The keratometer then projected three beams of near infrared light onto J.W.'s cornea with the reflected rays captured by the machine's photosensors. For each eye, the dioptric power and the radius of curvature were measured along the apex of the cornea (apical K readings), at 13.5° in the right and left periphery (peripheral K readings), and at the visual axis (central K readings). The visual axis is defined as the line passing through the cornea which connects the fixation point in the visual field with the fovea. Also measured were the amount of astigmatism, shape of the cornea (rate of flattening from the apex in the horizontal meridian), and whether the apex was horizontally and/or vertically displaced from the optical axis, defined as that line which connects the four optical centers of the eye (anterior and posterior cornea and lens). Finally, an ophthalmologist measured the horizontal and vertical diameters of each cornea using corneal calipers (see Table 57).

Results and Discussion

The horizontal and vertical diameters of the cornea of the deprived eye were identical to those of the nondeprived eye (Table 57), and all values were within normal limits (Brent, 1990). This suggests that the corneas should be equally good in capturing rays of light. In the central cornea, J.W.'s deprived eye had a power of 41 D at 69° from the horizontal axis and a radius of curvature of approximately 8 mm. The values for the nondeprived eye were similar (Table 57), with all values within the normal range (Woolf, 1948). Also, the dioptric power and radius of curvature both apically and peripherally were similar in the deprived and nondeprived eyes (Table 57). This suggests that the ability of the cornea to bend light is similar in both eyes.

Two differences were apparent between J.W.'s eyes. First, although the deprived eye exhibited no more corneal astigmatism than is typically found following surgery to remove the crystalline lens (Bennett and Rabbetts, 1989), it nonetheless was more than that found in the nondeprived eye and present at an oblique angle (Table 57). Moreover, it was more than is typically found in a group of individuals who wear glasses: 14% of individuals wearing glasses have astigmatism as great as 1.25 to 2.00 D, with approximately 1/3 of such individuals having oblique astigmatism (Bennett & Rabbetts, 1989). The greater amount of oblique astigmatism found in the deprived eye, if uncorrected by her contact lens, likely would reduce

the sharpness of the image and limit the size of, or sensitivity across, the visual field, particularly for measurements along the oblique meridia. However, given that J.W. wore a contact lens prescribed by the ophthalmologist that was designed to optically correct the astigmatism present in her deprived eye, it is unlikely that the corneal astigmatism alone accounts for the abnormality of her peripheral vision.

A second interocular difference was the shape of the cornea. The cornea of the deprived eye was elliptical - the cornea was steepest at the apex where it had the smallest radius of curvature. In the periphery, the cornea was flatter and the radius of curvature was largest. In contrast, the cornea of the nondeprived eye was spherical. The apex was less well defined than in the deprived eye - there was little corneal flattening out from the apex where the radius of curvature was the largest. Although there were some differences in the corneal shape of each eye, overall, based on normative data established by the manufacturers of the Humphrey Auto Keratometer, both shapes are normal (see Manual for the Humphrey Auto Keratometer, Model 420).

To summarize, these data indicate that, with the exception of astigmatism which was corrected by the contact lens, the corneas of J.W.'s nondeprived and deprived eyes were similar, and, moreover, like those of normal individuals. Therefore, the corneal parameters alone cannot account for the abnormal peripheral vision of J.W.'s deprived eye.

(B) Influence of the iris and the pupil:

Following surgery for the extraction of a cataract, the size and the shape of the pupil in an aphakic eye can be noticeably different than in a normal eye. In this section, the possible influences of the size and of the shape of the pupil upon peripheral vision will be evaluated.

(1) Size of the pupil:

The size of the pupil may influence peripheral vision because of its effects on the geometrical projection of the visual field, on the clarity of the retinal image, on the level of retinal illumination, and on the relation between the brightness of the object and its background.

First, a larger pupil will allow more of the visual field to project onto the retina (Ferree, Rand, & Sloan, 1934), which may increase the likelihood of detection of a target presented further in the periphery. However, a larger pupil also leads to the Stiles-Crawford effect. That is, light rays entering the periphery of the pupil reach the receptors at more oblique angles and are less effective in stimulating a response than rays entering the center of the pupil (Micheals, 1980). This suggests that larger pupils may be less effective visually than smaller pupils, whether the experimenter uses kinetic perimetry to measure the extent of the field or static perimetry to measure sensitivity. Overall, then, evidence indicates that the effect of a larger pupil may not necessarily be a larger or more sensitive visual field.

Second, the size of the pupil will affect the clarity of the retinal image, with a clearer image likely improving the ability of the eye to detect more peripheral and/or dimmer targets. The diameter of the pupil will affect the clarity of the retinal image via its effects on refraction, spherical aberration, and diffraction (Ferree et al., 1934; Mikelberg et al., 1987).

Decreasing the size of the pupil will cause a refractive effect which serves to sharpen the retinal image. After rays of light pass through a pinhole pupil, they proceed to an image point on the retina. This image is composed of little patches of light that duplicate the shape of the aperture or pupil. Because the pupil typically is round, the retinal images are made up of blur circles. Smaller apertures will constrict these blur circles, which therefore overlap less, producing sharper images. As well, decrease in the size of the pupil also decreases the effects of spherical aberration present in the peripheral parts of the crystalline lens and the cornea (Ferree et al., 1934). In large pupils, the rays of light near its edge are bent more than rays nearer the optical axis (the line connecting the centers of the anterior and posterior cornea and anterior and posterior lens), which will form a blurred retinal image. In smaller pupils, there will be a decreased effect of spherical aberration.

Although a small pupil can lead to a refractive effect and to reduced spherical aberration, both of which will create sharper retinal images, a small pupil also can lead to more diffraction, which produces greater blur (Forbes, 1966). Diffraction is produced when a

wave front is cut off by some obstacle, such as by the edge of a pupil. The effect of diffraction is to cause the rays near the edge of the pupil to form bands about each point in the retinal image, thereby degrading the retinal image. There must be, then, some optimal pupil size which provides a balance between the effects of refraction, spherical aberration, and diffraction, and leads to the sharpest retinal image and, consequently, to improved peripheral vision.

Another variable to consider is the interplay of pupil size and retinal illuminance. Retinal illuminance depends not only upon stimulus luminance and the posterior nodal distance (defined as the distance from the center of curvature of the optical surfaces of the eye through which light rays will pass undeflected to the back of the eyeball) but also upon the square of pupil size (Brown, Dobson, & Maier, 1987). As such, relative to a a larger pupil, a smaller pupil allows less illumination of the retina. This may act so as to shift the level of the subject's retinal adaptation from photopic to mesopic conditions or from mesopic to scotopic conditions (Mikelberg et al., 1987). Heuer, Gressel, Anderson, Knighton, & Fantes (1985) have shown, using the Octopus perimeter, that less retinal illumination decreases sensitivity.

Finally, detection of an object depends not only upon its intensity but also upon the difference in intensity of the object and its background, which may be affected by pupil size. Wood, Wild, Bullimore, and Gilmartin (1988) assessed perimetric sensitivity under two adaptation levels: 10 asb (mesopic conditions) and 45 asb

(photopic conditions). They found that peripheral sensitivity increased with increased pupil size, with the effect similar at both levels of adaptation. Other reports agree that individuals with pharmacologically enlarged pupils have larger fields and individuals with pharmacologically decreased pupils have smaller fields than normal (Bedwell & Davies, 1977; Day & Scheie, 1953; Lindstrom et al., 1968; Lyne & Phillips, 1969; McCluskey et al., 1986; Mikelberg et al., 1987; but see Radzikhousky, 1972). In one study, the loss of field extent was striking - McCluskey et al. (1986) reported that after application of pilocarpine, a miotic drug, field size decreased by 24% for tests with the Goldmann I4e target (intense light), 33% for tests with the I3e stimulus (light of intermediate intensity), and 65% for tests with the I2e stimulus (dim light). Possibly, the restrictions of the field were greatest with the dimmest stimuli because this stimulus transmitted insufficient energy through the tiny pupil to optimally stimulate the photoreceptors. As well, the observed field loss is usually not localized to a particular area of the field. When it is, the loss is most marked in the periphery of the field (Wood et al., 1988), especially in the superior temporal field (Lindstrom et al., 1968).

Although Wood et al. (1988) reported that peripheral sensitivity increases with pupil size, they also cautioned that changes of pupil size within the normal range are relatively unimportant for peripheral sensitivity. The finding that interindividual differences in pupil size in normal eyes have negligible effects on field size has

been confirmed in tests with kinetic perimetry (Aspinall, 1967; Drance, Berry, & Hughes, 1967) and with static perimetry (Brenton & Phelphs, 1986).

There are several implications from the foregoing discussion for the research presented in this dissertation. First, from the theoretical work, it is difficult to predict what will be the effects of pupil size upon peripheral vision. For example, though decreased pupil size will increase the clarity of the retinal image, it also may lead to less retinal illumination and to a smaller projection of the visual field onto the retina. As well, the implication from the study by Wood et al. (1988) is that small changes in pupil size within the normal range will have little effect on the visibility of the stimulus, at least for tests with the Goldmann perimeter which has a background level of 31 asb. It is unknown whether this statement holds for tests with the Octopus perimeter because the background conditions of the Octopus perimeter (4 asb) are dimmer than those tested by Wood et al. (1988). However, Heuer et al. (1985) indicate that changes in pupil size will influence thresholds measured by the Octopus perimeter.

Second, the foregoing research suggests that extremely small pupils outside the normal range will lead to reduced sensitivity. This finding is especially pertinent given that the pupil of the deprived eye in the patients assessed in Experiments 4 and 5 often was smaller (i.e., 2 to 7 mm) than in the nondeprived eye and sometimes outside the normal range. In the following experiment, the potential

effect upon peripheral vision of pupil size was evaluated by measuring the extent of the visual field when J.W.'s pupils were of typical size, and also when enlarged via mydriatic drugs and constricted via miotic drugs.

Method

Subject. Both of J.W.'s pupils were reactive to light. The pupil of J.W.'s nondeprived eye was round and centered while the pupil in her deprived eye was slightly elongated vertically and was slightly displaced nasally. When light adapted, the horizontal diameter of the pupil in J.W.'s nondeprived eye was 4.5 mm and in the deprived eye 3.5 mm, with only the former value being similar to those of normal subjects. [When light adapted, the typical pupillary diameter is 4.8 mm in 10-year-olds, 4.0 mm in 45-year-olds, and 3.4 mm in 80-year-olds (Bennett & Rabbetts, 1989)].

Stimuli and apparatus. The Goldmann perimeter was used (see Experiment 1) with stimuli I4e, I3e, and I2e, which are each 0.107° in size but 318, 100, and 31.8 cd/m^2 in intensity, respectively.

Procedure. The assessment of the extent of J.W.'s visual field using kinetic perimetry with the Goldmann perimeter was similar to the procedure described in Experiment 4. The visual fields were measured under four sets of pupillary conditions: when the pupils

were of typical size, when equal in size, when enlarged, and when constricted.

First, the extent of the visual field for each eye was measured using the intense, intermediate, and dim stimuli when each of J.W.'s pupils was of typical size.

Second, to determine whether the visual field of the deprived eye is restricted even when its pupil is of the same size as that of the fellow nondeprived eye, a mydriatic drug (Mydriacil 1% plus Cyclogyl 1%) was administered by the ophthalmologist to the pupil of J.W.'s deprived eye. The horizontal diameter of the pupil subsequently enlarged from 3.5 to 4.5 mm, which was the same size as that of the nondeprived eye under baseline conditions. The visual field for the deprived eye then was measured with the intense, intermediate, and dim stimuli.

Third, the pupil of J.W.'s nondeprived eye was enlarged from 4.5 to 7 mm through the administration of a mydriatic drug (Phenylephrine 2.5%) by the ophthalmologist. The extent of the field then was assessed with the intense, intermediate, and dim stimuli.

Fourth, to avoid using both mydriatic and miotic drugs in the pupil on the same day and to avoid fatigue of the subject, the following condition was conducted on a different day from the previous measurements. Following administration of a miotic drug (Pilocarpine Nitrate 1%) to each pupil by the ophthalmologist, the horizontal diameter of the pupil of the nondeprived eye decreased from 4.5 to 2 mm and that of the deprived eye from 3.5 to 2.5 mm.

Results and Discussion

Under typical pupillary conditions, the extent of the field of J.W.'s nondeprived eye was normal and that of her deprived eye restricted. For tests with the intense light (I4e), intermediate light (I3e), and the dim light (I2e), the amount of the restriction of the field for the deprived eye compared to the fellow eye varied from 8° to 28°, 9° to 36°, and from 22° to 39°, respectively, depending upon the meridian. For tests with each stimulus, the largest restriction was in the temporal field (see Table 58).

When the size of the pupil of the deprived eye was enlarged to equal that of the nondeprived eye, the field for the deprived eye still was restricted. The amount of restriction of the field for the deprived eye for tests with the intense, intermediate, and dim lights ranged from 11° to 33°, 22° to 34°, and from 22° to 52°, respectively, depending upon the meridian (see Table 58). Even when both pupils were matched for diameter of the aperture, then, the field for the nondeprived eye was still much larger under all testing conditions and particularly so for tests with dim stimuli. In conclusion, though the size of the pupil may be important optically, it alone does not account for the huge losses of the field in J.W.'s deprived eye.

When each pupil was enlarged, the extent of the field changed only slightly relative to the field size under typical pupillary conditions, for tests of both the nondeprived and the deprived eye. For tests with the intense, intermediate, and dim lights, the field for

the nondeprived eye decreased an average 1.6°, 1.4°, and 4.0°, respectively. Corresponding losses for the deprived eye were, on average, 4.4°, 5.3°, and 4.3° (see Table 59). It is unknown why the losses were larger in the deprived eye.

When each pupil is constricted, the extent of the field changed only slightly relative to the field size under typical pupillary conditions, for tests of both the nondeprived and the deprived eye. For tests with the intense, intermediate, and dim lights, the field for the nondeprived eye decreased an average of 0.5°, 0°, and 1.5°, respectively. Corresponding losses for the deprived eye were, on average, 3.5°, 3.3°, and 4.5° (see Table 59). It is unknown why the losses were larger in the deprived eye.

To summarize, these experiments in a single deprived subject indicated that even when the pupil in the deprived eye is matched in size to that of the nondeprived eye, the field for the deprived eye still exhibits large losses. Moreover, increasing or decreasing the pupil size does not change the extent of the field by more than a few degrees. The finding that decreased pupil size has a fairly trivial effect upon peripheral vision is particularly important given that a small pupil is typical of the patients assessed in Experiments 4 and 5. Overall, the effects of changes in pupil size are smaller than would be predicted from the literature, possibly because fatigue of the subject was avoided in the present experiment, which would minimize large losses of the visual field seen in other experimental work. This experiment indicates, then, that pupil size plays a limited role in

explaining the losses of the visual field, at least in one deprived subject.

(2) Shape of the pupil:

The sole study in the literature on peripheral vision which mentions pupil shape in aphakic individuals reports that adults treated for senile cataracts who had normally shaped (i.e., round) pupils of average size nonetheless exhibited fields restricted by 10° to 15° (Beasley, 1965). This indicates that even under normal pupillary conditions, fields may be abnormal.

Because pupil shape cannot be manipulated easily, and pupillary location not at all, there was no experimental investigation of these variables. Yet, two pieces of evidence suggest that the shape and location of the pupil may not alter peripheral vision. First, the previous experiment demonstrated that differences of several millimeters in the size of the pupil have little effect on peripheral vision. Therefore, smaller differences in the shape of the pupil should not be important. Second, Experiments 4 and 5 demonstrated that deprived subjects exhibit especially large losses in the temporal extent of the field and in sensitivity at 0° centrally. Yet, observation of the deprived subjects indicated that the pupil shape and location within the iris varied widely across patients. There is no correlational evidence, then, to suggest that the often abnormal shape and location of the pupil in the deprived eye contributed to the abnormal fields.

(C) Influence of aphakia and subsequent optical correction:

The crystalline lens is onion-like, being composed of layers of fibers which form the nucleus and surrounding cortex. The periphery of the lens, particularly the anterior portion, displays marked flattening, which, like the cornea, helps to decrease spherical aberration (Bennett & Rabbetts, 1989). In this section, I will examine the impact upon peripheral vision of the removal of the crystalline lens (aphakia) and subsequent optical correction. As part of my examination, I will present measurements of the extent of the visual field in adults treated for senile cataracts. I also will present experiments designed to evaluate the role of variables such as the size of the optical zone of the contact lens, possible blockage of light by the edge of the contact lens, and the correction of refractive errors in the periphery of an aphakic eye.

First, with the loss of accommodation and the use of an optical device, the aphakic eye becomes focussed for some particular distance, with objects located at other distances not seen clearly. As such, the visual input to a deprived eye continues to be mildly abnormal. Note that aphakia per se at the time of testing does not explain the abnormal peripheral vision observed in deprived subjects because during testing each deprived eye was properly corrected for the testing distance.

Second, because of replacement of the natural crystalline lens with an optical device located in front of the eye, optical correction of an aphakic eye will increase the size of the retinal image relative to

that seen by a normal eye (aniseikonia). The amount of magnification depends upon the power of the optical device and upon its distance from the entrance pupil. For example, Micheals (1980) estimates that a +10.50 D spectacle lens located 15 mm in front of the eye will induce approximately 34% magnification of the image and, with a contact lens, the magnification will be less. Aniseikonia might affect peripheral vision in two ways. First, because of additional interocular competition, aniseikonia may affect the neural pathways mediating peripheral vision.

Second, because aniseikonia increases the size of the retinal image in the aphakic eye relative to that seen by the nondeprived eye, it may, in one of two ways, affect test measurement. One possibility is that aniseikonia may improve sensitivity because the increased retinal image size is spread over a larger retinal area, thereby stimulating more receptors (see Banks & Bennett, 1988). A second possibility is that aniseikonia may worsen sensitivity. If the number of receptors remains constant with less light falling on each receptor, then there may be insufficient stimulation of the receptors. Therefore, the aphakic eye will exhibit impaired detection of the peripheral stimulus. This prediction of worsened sensitivity is unlikely to be true if Weber's law applies because any changes in stimulation will equally affect the stimulus and background, hence having no effect on sensitivity. In fact, Wilson (1970) argues that Weber's fraction remains constant from 5° to 55° in the nasal field under photopic testing conditions. Although Wilson's parameters are

not directly comparable to those used in my research, they suggest that Weber's law may apply for the conditions used in my research. Therefore, the second possibility, that aniseikonia may worsen sensitivity, is less likely to be viable than the first possibility, that aniseikonia may improve sensitivity for the test measurements.

In summary, it is difficult to predict the overall effects of aniseikonia given that aniseikonia may impair the neural pathways but may aid retinal stimulation. Nonetheless, its possible role in peripheral vision cannot be discounted.

Further evidence for the role of aphakia in peripheral vision arises from behavioral work. Beasley (1965) found a 10° to 15° narrowing of the visual field in adults with previously normal vision who subsequently were treated for senile cataracts and corrected with spectacle lenses. This finding indicates, then, that some optical component likely underlies the abnormal fields seen in the deprived children (Experiments 4 & 5). Beasley (1965) suggests that this optical mechanism may be the prismatic effect.

Spectacle lens and, to some extent, contact lens placed in front of an aphakic eye will cause a prismatic effect in the periphery. The prismatic effect occurs when a ray of light passes through the correcting lens at other than its optical center and then is deviated by some amount. Prentice's rule specifies that the amount of the prismatic effect is equal to the power of the lens (in D) multiplied by the distance (in cm) from the optical center of the lens to where the ray of light entered (Micheals, 1980). For example, a ray of light

which enters 1.2 cm from the optical center of a contact lens with a power of +15 D will create a prismatic effect which then will be detected as a narrowing of the visual field by 10°.

The foregoing discussion indicates how peripheral vision may be altered by magnification and the prismatic effect, which result from the wearing of optical correction in an eye treated for a cataract. To examine this issue, in one of the following experiments, I measured the extent of the visual field in adults treated for senile cataracts. However, there are other ways in which aphakia and optical correction may alter the visual field in patients. For example, the size of the optical zone of the contact lens may be small. As a result, only a portion of the patient's eye, and hence their true visual field, is being optically corrected. Lack of optical correction may lead to abnormal findings on tests of peripheral vision. Alternatively, the edge of the contact lens may block light rays entering from the periphery and therefore restrict the detection of peripheral lights. As well, patients may exhibit refractive errors in the periphery of their eye which are not being corrected by the contact lens. As such, these uncorrected peripheral errors may lead to abnormal peripheral vision. In the remaining experiments, I evaluated the effects upon peripheral vision of the size of the optical zone of the contact lens, possible blockage of light by the edge of the contact lens, and the correction of refractive errors in the periphery of an eye treated for a congenital cataract.

(a) Measurement of the field in adults treated for senile cataracts:

As discussed, Beasley (1965) reported that even adults with previously normal vision who subsequently are treated for a cataract exhibit restrictions of the visual field. These restrictions in adults have been attributed to the prismatic effect and not to neural factors. Given that adults treated for cataracts exhibit losses in the extent of their field, then some of the losses in children may arise for non-neural reasons. Because the study by Beasley (1965) was conducted on adults corrected with spectacles and my study was carried out with children who wore a contact lens, I measured the extent of the visual field in two adults treated for senile cataracts who were corrected for testing with a combination of a spectacle lens and contact lens and subsequently with only a contact lens.

Subjects.

(1) P. V. had normal vision until at 40 years of age he began to experience blurry vision in his right eye. A dense cataract subsequently was removed and the eye fitted with a contact lens when P. V. was 48 years old. The power of the contact lens (+11.0 D) was similar to those worn by the patients assessed in Experiments 4 and 5. At the time of the test of the visual field, P. V. was 51 years old. The acuity in each eye, measured at far with a Snellen eye chart and at near with a Near Number chart, was 20/20. Each eye was properly corrected for the tests of visual acuity.

(2) G. C. had normal vision until at 46 years of age he began to experience blurry vision in his left eye. A dense cataract subsequently was removed and the eye fitted with a contact lens when G. C. was 52 years old. The power of the contact lens (+11.25 D) was similar to those worn by the patients assessed in Experiments 4 and 5. At the time of the test of the visual field, G. C. was 57 years old. As measured with a Snellen eye chart, the vision at far in the normal and aphakic eyes was 20/15 and 20/20, respectively. As measured with a Near Number chart, the vision at near in the normal and aphakic eyes was 20/20 and 20/30, respectively. All measurements of visual acuity were with appropriate optical correction.

Method

The normal eye and then the aphakic eye of P. V. and G. C. were tested monocularly with stimuli I4e (intense light) and I2e (dim light) of the Goldmann perimeter. The procedure was the same as described in Experiment 4 with the following exception.

Because the normal eye of each subject exhibited limited ability to accommodate, it was corrected for the testing distance by a +3 D spectacle add sitting in a lens holder 5 mm in front of the eye. The aphakic eye then was tested twice using different combinations of correcting devices. So as to make the testing conditions of the aphakic eye as similar as possible to that of the fellow eye, the

aphakic eye was corrected for the testing distance with a contact lens and a +3 D spectacle add. So as to allow comparison of the fields of the aphakic eye of P. V. and G. C. to that of the deprived subjects reported in Experiment 4, the aphakic eyes of G. C. and P. V. also were tested when they were corrected for the testing distance with a contact lens of the appropriate power.

Results and Discussion

In comparison to the visual field of the normal eye, the field for the aphakic eye of P. V. was restricted. When corrected with a contact lens and spectacle add, the field for tests with the intense light and with the dim light showed an average restriction of 9.5° and 3.6°. When corrected with only a contact lens, the field for tests with the intense light and with the dim light showed an average restriction of 11.1° and 4.4°.

In comparison to the visual field of the normal eye, the field for the aphakic eye of G. C. typically was restricted. When corrected with a contact lens and spectacle add, the field for tests with the intense light and with the dim light showed an average restriction of 4.4° and 5.1°. When corrected with only a contact lens, the field for the test with the dim light showed an average restriction of 6.6°. In contrast, when tested with the intense light, the field of the aphakic eye was an average of 1.0° larger than in the normal fellow eye.

These results indicate that the visual field for the aphakic eye of two individuals each treated for a unilateral senile cataract typically was restricted no more than 11°. Moreover, the magnitude of the restriction was similar to that reported by Beasley (1965) in his study of adults with a similar visual history who were tested with spectacle corrections and under similar lighting conditions. Because these two individuals had decades of normal visual experience prior to the onset of a senile cataract, presumably both the optics and the underlying neural pathways of the normal and the aphakic eyes developed normally. The only differences between the normal and the aphakic eye, then, should be that the correcting device of the aphakic eye is a contact lens plus spectacle add which sat anteriorly to the location of the crystalline lens, as well as any surgically induced effects on the cornea and pupil. The poorer performance of the aphakic eye relative to that of the normal fellow eye presumably reflects the contribution of these optical factors.

The losses of the visual field of the children treated for cataracts were much larger than those found in the adults (see Figure 9 for an example). The implication of this finding, then, is that the losses of the children not only have an optical component but also likely have some neural basis. The physiology underlying the losses found in the children will be explored in the final chapter.

(b) Size of the optical zone of the contact lens:

One explanation for an abnormal field may be that the optical zone of the contact lens worn by a deprived eye may be small and therefore limit the transmittance of peripheral rays of light through the pupil. Outside the optical zone, rays either may not be transmitted or not be focussed clearly upon the retina. Reduced fields are reported in normal adults who wear colored contact lens with small optical zones of 5 mm (Insler, Hendricks, & George, 1988) but there are no reports of the fields of adults wearing lens with larger optical zones. In the data reported in Experiments 4 and 5, the optical zones of the contact lens worn by the deprived subjects were large, ranging from 7 mm to 8 mm. For example, the size of the optical zone of J.W.'s contact lens was 7.97 mm. Given the large size, it is unlikely that the optical zone of the lens is able to explain the restricted, or less sensitive, visual fields in the deprived eyes.

(c) Blockage of peripheral rays of light by the contact lens:

A second possibility is that the edge of the contact lens blocks the most peripheral rays of light from entering the pupil, thereby restricting the extent of, or sensitivity across, the field. To evaluate this possibility, the extent of the visual field of J.W. was assessed when her deprived eye was optically corrected and wore no correction.

Method

J.W.'s visual field was measured using small intense, intermediate, and dim Goldmann stimuli (Objects I4e, I3e, I2e) and a larger intense stimulus (Object III4e), both when the deprived eye wore a contact lens of the appropriate power and when it was without optical correction. Object III4e is a 0.3° light of 318 cd/m^2 and was chosen because of the possibility that the uncorrected deprived eye would be unable to see smaller or dimmer stimuli.

Results and Discussion

Without appropriate optical correction, the deprived eye was unable to see the small dim (I2e) or the small intermediate (I3e) stimulus. For the small intense (I4e) and the large intense (III4e) stimulus, the extent of the visual field was the same when the deprived eye was tested either with the appropriate optical correction or without any optical correction. Given that the field remained the same size, it can be argued that the contact lens is not blocking detection of stimuli nor does magnification of the retinal image or the prismatic effect caused by the contact lens affect measurement. Alternatively, it can be argued that the contact lens is correcting the central but not the peripheral portion of the eye. If this is true, then the periphery of J.W.'s eye was uncorrected both with and without optical correction. As such, it would not be

surprising that the extent of the visual field would be the same under both testing conditions and less than in a normal eye.

There is evidence that blur resulting from defocus decreases sensitivity, at least in the central visual field (Atchinson, 1987; Benedetto & Cyrlin, 1985; Fankhauser & Enoch, 1962; Serra, 1983). Yet, two facts argue against the view that the poor performance of the deprived eye results from an uncorrected periphery. First, there is more oblique astigmatism in nondeprived than in aphakic eyes of adults of the same age (Millodot, 1974). Second, the central and peripheral optics of a normal eye differ: With increasing eccentricity, there is increasing refractive error (Jennings & Charman, 1978; Leibowitz, Johnson, & Isabelle, 1972; Millodot & Lamont, 1974; Rempt, Hoogerheide, & Hoogenboom, 1976). Therefore, it can be argued that the periphery of neither the deprived eyes nor the normal eyes was properly optically corrected during tests of the visual field and yet the nondeprived eyes still exhibited larger visual fields. This suggests, then, that peripheral optics may not be contributing to the abnormal fields demonstrated by the deprived eyes. The following experiment was designed to test the possibility that the peripheral optics of the deprived eye degraded visual performance.

(d) Correction of refractive errors in the periphery of a deprived eye:

Method

The temporal field of the deprived eye of J.W. was measured with the intense stimulus (I4e) when its periphery was optically corrected by holding a spectacle lens against the eye. Because of constraints of time in the clinic, peripheral refraction was not conducted and instead a variety of spectacle lens of differing powers were used: + and - (1, 2, 3, 4, 5, 7, 10, and 20 D).

Results and Discussion

Regardless of the correction used, the extent of the the field of J.W.'s deprived eye did not. change. This indicates that the peripheral optics found in a deprived eye presumably have little influence on peripheral vision and are unable to explain the restrictions of the field.

Summary of studies examining the influence of aphakia and subsequent optical correction:

To summarize, these experiments indicate that the abnormal peripheral vision of the deprived eye is not explainable by the size of the optical zone of the contact lens, blockage of peripheral rays of light by the contact lens itself, or by refractive errors in the

periphery. This does not mean that optical factors are unimportant as evidenced by the small restrictions of the visual field found in adults treated for senile cataracts. Together, these pieces of evidence indicate that aphakia per se may play some limited role in restricting the extent of the field or in reducing sensitivity across the field, likely because of magnification and the prismatic effect.

(D) Influence of the length of the eye:

There is agreement that early abnormal experience affects the overall growth of the eye both in animals and in humans, although disagreement about the direction of the effect (Mitchell & Timney, 1984). For example, Wilson, Fernandes, Chandler, Tigges, Boothe, & Gammon (1987) reported that the eye of rhesus monkeys rendered aphakic shortly after birth is shorter than the fellow nondeprived eye. This was true regardless of whether the aphakic eye wore a contact lens or whether the nondeprived eye was occluded. In contrast, Wiesel and Raviola (1977) reported that unilateral and bilateral lid suture in infant macaque monkeys lead to elongation of the deprived eye but that other ocular structures are unaffected.

As in animals, the results in children are discrepant. von Noorden and Lewis (1987) found that there is not a consistent elongation of the abnormal eye in children diagnosed during the first year of life with a unilateral dense cataract. In contrast, Rasooly and BenEzra (1988) found that, relative to the nondeprived eye, there is an average elongation of the deprived eye of 1.5 mm in children

unilaterally deprived from birth and 0.7 mm in those subjects unilaterally deprived later in childhood. In children treated for bilateral congenital cataract, there is little interocular difference in axial length. Although these eyes are shorter than those of children treated for unilateral congenital cataract, their length has not been compared to that of normal eyes (Rasooly & BenEzra, 1988)

The ophthalmologist assessed the axial length of the eye when fitting the contact lens of the patients assessed in Experiments 4 and 5. Presumably, axial length should not affect the patient's peripheral vision. The following experiment was designed to assess the effects of deprivation on growth of the eye in one deprived subject. J.W. was chosen as the subject both to complete my knowledge regarding optical factors in one subject and because her deprivation was unilateral and beginning at birth, a condition in which it may be more likely to find alterations in the growth of the eye than in cases of deprivation of late onset.

Method

An ophthalmic technician conducted A-scans of each of J.W.'s eyes using the A-2000 Ultrasonographic Scanning Machine. Briefly, this technique assesses the axial length of the eye by measuring the time necessary for an ultrasonic wave reflected from an ocular surface to return to its point of origin.

Results and Discussion

The axial lengths of J.W.'s nondeprived and deprived eyes were 26.1 mm and 25.1 mm, respectively. This magnitude of interocular difference is within the range of values for aphakic versus normal eyes found by von Noorden and Lewis (1987). More importantly, both values for axial length are within the normal range (Sorsby, Benjamin, Davey, Sheridan, & Tanner, 1957). This suggests that axial length does not account for the abnormal peripheral vision of J.W.'s deprived eye.

Summary and conclusions

In conclusion, this experimental work has demonstrated that each of the measurements of J.W.'s cornea (with the exception of astigmatism), pupil, and axial length was within the normal range. This suggests that none of these optical factors individually can account for the large losses in the extent of, or sensitivity across, the field shown by J.W.'s deprived eye. A more likely candidate to have influenced peripheral vision is the prismatic effect although, again, losses were larger than can be accounted by it alone. Similarly, the role of magnification cannot be discounted. It also is important to acknowledge that should the optical factors operate in the <u>same</u> direction, then the <u>sum</u> of these factors may be able to account for some of the losses in peripheral vision.

One concern is the typicality of J.W.'s measurements for other deprived subjects. It should be noted that J.W. possesses a slight nystagmus and good acuity in her deprived eye (Snellen value of 20/40) and, as such, is atypical of children treated for a unilateral congenital cataract. Yet, if visual deprivation does alter the optics of the eye, then these changes are most likely to occur in a patient with unilateral deprivation from birth. As such, J.W. was an ideal subject to assess. Moreover, many of the measurements of J.W.'s deprived eye, such as the keratometer readings and refractive error, appeared typical of measurements from other deprived eyes available in the medical records. It appears, then, that the measurements from J.W.'s deprived eye were likely to be representative of the group of deprived subjects. If the alteration of optical factors is as undramatic in other deprived eyes as observed in J.W., optical factors may have a small role in explaining their abnormal peripheral vision.

To more precisely delineate the role of optics in peripheral vision, a three-fold multidisciplinary approach involving specialists in Ophthalmology, Optics, and Psychology is advised. The first arm of the study should consist of ray tracing programs to evaluate how optical parameters such as the cornea, pupil, crystalline lens, contact lens, axial length of the eye, and so forth would bend and scatter light. Such modelling would be of assistance in delineating the relative role of optical versus neural mechanisms in peripheral vision. The second arm would involve examination of the development of optical parameters, relative to age-matched normal

children, in deprived patients with differing visual histories. The third arm of the study would involve examination of the peripheral vision of deprived patients followed by an attempt to relate the individual's visual outcome to the optical indices. Such research would allow increased understanding of the potential role of optics in peripheral vision.

Because the foregoing experiments indicate that optical factors are unlikely to explain all of the observed abnormalities in peripheral vision, the final chapter evaluates an alternative evaluation - that the abnormal peripheral vision is mediated in part by abnormal neural functioning.

CHAPTER 4

GENERAL DISCUSSION

The results demonstrate that the development of peripheral vision continues throughout early childhood, at least for tests using a small stimulus, and is adversely affected by deprivation. Specifically, when normal 5-, 6-, and 7-year-old children and adults are tested with a small stimulus, then the extent of the visual field is adultlike approximately by six years of age, except for the inferior temporal field. In addition, reliability of responding is excellent and interocular differences small (Experiment 1). When the 5-year-old children are retested at 7 years of age, they demonstrate increased visual fields as large as those of the original sample of 7-year-old children (Experiment 2). These age-related differences in school-aged children are unlikely to result from improvements in attentional and cognitive mechanisms because even the youngest children have blind spots in the same location and of the same size as the adults (see Experiment 1). Experiment 3 demonstrated that there are age-related changes not only in the extent of the visual field but also in thresholds across the visual field. When normal 7-, 8-, and 9-year-olds and adults are tested with a small stimulus, then sensitivity across the peripheral visual field is adultlike by seven years of age, with later development at 0° centrally. In addition, reliability of

responding is excellent and interocular differences small (Experiment 3).

The literature contains limited and contradictory information on the normal development of peripheral vision in school-aged children. My research adds to the literature by assessing peripheral vision in a large sample of normal children. Moreover, it examines reliability of responding in a larger sample of children than assessed in previous studies and is the first report of interocular differences. Finally, the normative standards collected in Experiments 1 and 3 have clinical significance. The normative data were used to evaluate the peripheral vision of children treated for cataracts (see Experiments 4 and 5) and, potentially, may prove useful for the assessment of children with other types of eye disorders, such as retinopathy of prematurity or strabismus.

My research also indicated that deprivation from dense and central cataracts adversely affected peripheral vision in every deprived eye. Assessment of the extent of the visual field in patients revealed that the temporal field is most restricted (Experiment 4). Assessment of sensitivity across the visual field and, in more detail at 20° nasally and 30° temporally, indicated that the largest losses of sensitivity were at 0° centrally and 20° nasally (Experiment 5). Furthermore, there are some effects of the duration and the timing of visual deprivation and whether it is monocular or binocular. First, I found that children with longer deprivation have more restricted visual fields than do children with shorter deprivation, except in the

superior visual field. Second, children with monocular deprivation have more restricted fields than do children with binocular deprivation, at least for tests with the dim light. Third, children treated for developmental cataracts have larger losses of sensitivity at 0° centrally than elsewhere across the visual field. Moreover, the losses at 0° centrally are larger than in children treated for congenital cataracts. Finally, children treated for unilateral congenital cataracts, but not other groups of deprived children, have larger losses of sensitivity at 20° nasally than at 30° temporally.

My results indicate that the visual fields of some groups of patients, namely children with unilateral congenital deprivation, children with developmental deprivation, and children with long deprivation, are especially vulnerable (see Experiments 4 & 5). The data from children with unilateral congenital deprivation or deprivation of long duration argue for early and fast ophthalmological treatment following the diagnosis of a dense and central cataract. Moreover, the results from children with developmental deprivation indicate that even later onset deprivation can have deleterious influence. As well, my findings parallel conclusions from studies of deprived cats and monkeys. For example, earlier onset of deprivation in monocularly deprived cats and monkeys leads to poorer peripheral vision (Bisti & Carmignoto, 1988; Joseph & Casagrande, 1980; Sparks et al., 1986; Wilson et al., 1989). Longer deprivation in monocularly deprived monkeys and in dark

reared cats leads to more restricted fields (Kalil, 1978; Maire-Lepoivre et al., 1988; Sparks et al., 1986).

Yet, some results were surprising, such as the finding that children treated for developmental cataracts have larger losses of sensitivity at 0° centrally than do children treated for congenital cataracts (Experiment 5). This is especially surprising given that there was no effect of the timing of deprivation on the extent of the visual field (Experiment 4). As discussed in Experiment 5, there are three possible explanations why children treated for developmental cataracts may have larger losses at 0° centrally than do children treated for congenital cataracts.

First, the difference in performance may reflect the limited and hence potentially unreliable sampling of thresholds at 0° centrally, relative to the more extensive sampling in the peripheral portions of the field. Note that Experiment 3, which used the same procedure and apparatus as Experiment 5, revealed that there was poorer test-retest reliability at 0° centrally than elsewhere in the field for the 7-year-olds, but not for the adults. Moreover, one conclusion from Experiment 3 was that normal 8- and 9-year-olds had lower thresholds at 0° centrally compared to 7-year-olds and adults. It may be, then, that the fast bracketing procedure used by the Octopus perimeter is less accurate for assessment of performance at 0° centrally.

Second, the age of onset of deprivation is not always certain in children who develop a cataract (nontraumatic cases) after a period

of normal visual experience. Some of the developmental cases might have had earlier and much longer deprivation than the medical records indicate. In fact, in some cases, deprivation may have been longer than that of the children treated for congenital cataracts. If the patients treated for developmental cataracts truly had early and long deprivation, then it may not be surprising to find that their fields were even more insensitive than those of children treated for a congenital cataract.

Third, the difference in performance may be real. The central vision of the newborn infant is poor and improves dramatically with age (van Hof-van Duin & Mohn, 1986). As such, a short period of deprivation from birth may exert little effect upon the extremely poor central vision of the infant. In contrast, deprivation beginning at later ages, when the central vision of the infant is improving, may be more detrimental. Relative to the performance at 0° centrally, smaller losses of equal magnitude were found in the near, mid, and far periphery. That children treated for developmental cataracts have smaller losses peripherally than they do centrally and, moreover, do not have larger peripheral losses than do children treated for congenital cataracts may reflect the fact that peripheral sensitivity is always poor, at least relative to central sensitivity (see Experiment 3). As such, peripheral sensitivity may be less affected by deprivation or by when it begins. Results from tests using the Goldmann perimeter complement these findings: the loss of the

extent of the field is similar for children treated for congenital or for developmental deprivation (see Experiment 4).

Comparison across experiments also reveals that the effects of the duration of deprivation were not identical. For the test of the extent of the visual field, I found that children with longer deprivation have more restricted visual fields than do children with shorter deprivation, except in the superior visual field (Experiment 4) but there were no significant effects of the duration of deprivation upon sensitivity (Experiment 5). How can these findings be explained?

First, differences in outcome across experiments are unlikely to reflect differences in the samples of patients assessed as these samples were relatively similar (see Tables 43, 48, and 49).

Second, with regard to the effects of duration, it might be expected that longer duration of deprivation would affect those parts of the field slowest to develop, such as 0° centrally or the edges of the field. Note that for the test of the extent of the field, the data were collapsed so as to allow examination of the edges of the nasal, temporal, superior, and inferior fields while for the test of sensitivity, the data were collapsed so as to allow examination of the effects of deprivation upon the central field, near, mid, and far periphery. When the edges of the field were examined, as in Experiment 4, I did find that longer duration of deprivation led to more restricted visual fields. Possibly, Experiment 5 also might have revealed some effects of the duration of deprivation on sensitivity if

the assessment at 0° centrally had been conducted using a normal bracketing method or if I had analyzed sensitivity at the extreme edges of the field, instead of collapsing data.

In an attempt to determine the basis for the abnormal peripheral vision in the deprived eyes, several control experiments were conducted on subjects treated for a cataract (see Experiment 6). This work indicated that optical factors, such as corneal astigmatism, corneal shape, and dioptric power, a misshapen pupil, absence of the crystalline lens, contact lens, and increased axial length of the eye, explain only some of the losses of peripheral vision following visual deprivation and subsequent aphakia. Of these factors, aphakia per se may be the most important contributor to the observed losses of peripheral vision in the patients while the contact lens likely exerts little or no effect. These conclusions must be tempered by the fact that most of the experimental investigations were conducted in only one patient. These experiments indicate that optical factors account for about 10° of the loss of the extent of the field and an unquantified amount for losses of sensitivity across the field. Another explanation for the observed losses is that visual deprivation from cataracts affects the pathways mediating peripheral vision. This possibility will be addressed further later in this chapter.

The literature contains references to the peripheral vision of only a few deprived patients. My research has enlarged substantially the sample of tested patients and confirmed the typicality of the

finding that deprivation indeed does affect peripheral vision. Moreover, I have assessed not only the extent of the visual field and sensitivity along the horizontal meridian, as other studies have done, but also assessed sensitivity across the entire field. I found that, under the testing conditions used, children treated for a cataract have impaired peripheral vision at all assessed locations. As discussed above, my study also is the first to consider the influence of variables upon peripheral vision in humans such as the timing and the duration of deprivation.

My research also is the first to examine the effects upon peripheral vision in humans of whether the deprivation was monocular or binocular, and the effects of patching of the nondeprived eye in children treated for unilateral cataracts. As such, this research allows an evaluation of the mechanism of interocular competition versus of deprivation. My finding that eyes treated for dense and central cataracts have abnormal peripheral vision suggests that deprivation is altering visual functioning. The further finding that the deprived eye of children treated for unilateral cataracts has a smaller visual field than the deprived eyes of children treated for bilateral cataracts suggests that the nondeprived eye in unilateral cases has a competitive advantage relative to the fellow deprived eye. Moreover, the observation that better compliance with the patching regime leads to a larger visual field, at least along one meridian, in the deprived eye suggests that the reversal of the competitive advantage of the nondeprived eye is beneficial. These

data suggest that ophthalmologists need to be especially vigilant in monitoring patients with unilateral deprivation. My findings complement those from deprived animals. For example, monocularly deprived cats have more restricted fields than do binocularly deprived cats (Sherman, 1973). Moreover, removal of competition via retinal lesions or enucleation of the nondeprived eye increases peripheral vision in the deprived eye (see Hendrickson et al., 1977; Joseph & Casagrande, 1980; Smith et al, 1982; but see Heitlander & Hoffmann, 1978) although reverse suturing of the nondeprived eye has little effect (Joseph & Casagrande, 1980; Sherman, 1973, 1974b; Smith et al., 1982; Wilson et al., 1989; but see Heitlander & Hoffmann, 1978; van Hof-van Duin, 1977). My research is the first to suggest that both the mechanisms of deprivation and of interocular competition operate for some aspects of peripheral vision in deprived humans.

As well, my research has allowed testing of a general developmental principle that a relationship exists between the rate of development of an ability and the effects thereon of deprivation. This research has provided further evidence that deprivation especially affects abilities which are initially immature and develop slowly. For example, deprivation affected the extent of the temporal field more than the extent of the nasal, superior, and inferior fields. As well, deprivation affected sensitivity more at 0° centrally than peripherally, and sensitivity at 20° nasally more than at 30° temporally. The most affected locations are the parts of the visual

field slowest to develop (see this research and Lewis et al., 1985). These results parallel findings regarding contrast sensitivity and visual acuity and contrast with findings regarding color vision in the same sample of patients. Like peripheral vision, contrast sensitivity and visual acuity are slow to develop and vulnerable to deprivation from dense and central cataracts (Maurer et al., 1989). In contrast to peripheral vision, color vision develops early in life and is little affected by deprivation, except in patients treated for monocular deprivation of long duration (Maurer et al., 1989).

Moreover, my research indicates that some groups of deprived eyes have especially infant-like behavior. Early in infancy, there is better sensitivity in the near temporal field (e.g., 30° temporally) than in the near nasal field (20° nasally) (Lewis et al., 1985). My research revealed that children treated for a unilateral congenital cataract, unlike other groups of deprived eyes,. have especially large losses of sensitivity at 20° in the nasal field. As such, my finding of larger losses in the nasal field complements previous reports that children treated for a unilateral congenital cataract show a more infantile pattern of visual behavior. For example, children treated for unilateral congenital cataracts have larger losses of contrast sensitivity in the nasal visual field than in the temporal field (Tytla et al., 1991). In contrast, children treated for bilateral congenital cataracts or for traumatic cataracts exhibit a constant loss of contrast sensitivity across eccentricity (Tytla et al., 1991). In addition, children treated for unilateral congenital cataracts exhibit decreased

loss of contrast sensitivity as the flicker rate of gratings increases, except for the coarsest stripes (Maurer & Lewis, in press). In contrast, children treated for bilateral congenital cataracts exhibit a constant loss of contrast sensitivity as the flicker rate of gratings increases (Maurer & Lewis, in press). Like children treated for unilateral congenital cataract and unlike normal adults, three-month-old infants exhibit better sensitivity to flickering stripes than to stationary stripes (Atkinson et al., 1977; Maurer & Lewis, in press).

For different aspects of peripheral vision, there are different effects of the duration and timing of deprivation, whether the deprivation was monocular or binocular, and of patching. Similarly, these variables have different influences upon other aspects of vision (Maurer & Lewis, in press). For example, each of these variables affects visual acuity. Poorer visual acuity occurs following early rather than later deprivation, following longer rather than shorter deprivation, following monocular rather than binocular deprivation, and with less patching of the nondeprived eye in monocularly deprived children. In contrast, only the timing of deprivation affects optokinetic nystagmus: Early rather than later deprivation leads to asymmetrical optokinetic nystagmus. There are no effects of the duration of deprivation, whether deprivation was monocular or binocular, or of patching.

This research also found that even children diagnosed after the age at which adult-like behavior is seen suffered some losses of their peripheral vision and more than could be explained by aphakia per

se (see Chapter 3). This suggests that deprivation can affect abilities after the age at which adult-like behavior is evidenced. There exists a precedent for this finding. The optokinetic nystagmus of infants is symmetrical by 6 months of age when subjects are tested with large stripes (Smith, Lewis, Maurer, & Haslip, 1991). Yet, visual deprivation from cataracts beginning as late as 18 months after birth can affect optokinetic nystagmus to such stripes, rendering it asymmetrical (Lewis, Maurer, & Brent, 1989; Maurer et al., 1989). If optokinetic nystagmus is affected even long after the adult-like behavior is shown, then presumably the pathways mediating symmetrical optokinetic nystagmus continue to be plastic long after birth. The story of peripheral vision may be analogous to that of optokinetic nystagmus with deprivation altering the neural mediation of peripheral vision even during childhood.

The final portion of this chapter will examine the likely physiological basis for the abnormal peripheral vision in the deprived children. Peripheral vision develops similarly in humans and in cats (Maurer & Lewis, 1991; Sireteanu & Maurer, 1982). Although unstudied, the development of peripheral vision also may be similar in monkeys, given the similarity of the visual system of monkeys and humans (Rakic, 1976). Given the similarity of the development of peripheral vision in cats and in humans, and perhaps in monkeys, it is reasonable to assume that its neural basis also may be similar. Drawing upon anatomical, physiological, and behavioral studies of normal, visually deprived, and/or lesioned cats and

monkeys, I next will explore the probable physiological mediation of peripheral vision in normal and visually deprived humans.

Physiological basis of peripheral vision

On the basis of electrophysiological and/or lesioning studies in monkeys and cats, pathways from the retina to the lateral geniculate nucleus, visual cortex, frontal eye fields, parietal cortex, inferotemporal cortex, and superior colliculus have been implicated in peripheral vision. This section will present the evidence for the role of the superior colliculus and cortical areas in peripheral vision and then, in the next section, I will examine the pathways interconnecting these structures.

(1) Role of the superior colliculus in peripheral vision

Lesioning studies suggest that the superior colliculus is important for peripheral vision in cats, monkeys, and in humans, as loss of the superior colliculus leads to loss of vision in the far periphery and abnormal eye movements. Following bilateral collicular lesions, monkeys tested binocularly localize flashes presented on the horizontal meridian between 6° and 43° but not between 43° and 80° peripherally (Butter, Weinstein, Bender, & Gross, 1978). Moreover, even their central vision is affected as they exhibit transient deficits in sensitivity for lights fixated by the fovea (Latto, 1977). Following bilateral lesions of the superior colliculus,

monocular testing revealed that cats are initially blind and then orient to stimuli presented no further than 30° to 60° from the midline (Sprague & Meikle, 1965). Following unilateral lesions of the superior colliculus, monocular testing of cats reveals homonymous field losses (defined as losses on the same side of visual space for each eye; e.g., on the right side of the field for both the right and the left eyes). These cats neglect stimuli positioned 60° or more peripherally in the field contralateral to the lesion (Sprague & Meikle, 1965). Following unilateral malformation of the superior colliculus, humans have a slightly elevated brightness threshold at 30° temporally, the only location tested, in the contralateral field (Zihl & von Cramon, 1979). Moreover, following unilateral or bilateral collicular lesions, cats are slow to orient their eyes to a target and exhibit difficulty in localizing and pursuing a target (Sprague & Meikle, 1965). Following unilateral collicular lesions, monkeys exhibit increased latency to initiate a saccade and undershoot their target (Mohler & Wurtz, 1977).

Evidence from electrophysiological studies in monkeys and cats suggests that the superior colliculus is able to signal information about the appearance, the location, and the direction of movement of a peripheral stimulus and also may be involved with shifts of gaze. In contrast, the superior colliculus does not signal information about the size, shape, or orientation of a stimulus.

The cells of the superficial layers of the superior colliculus are topographically organized, with greater magnification of

representation from the central versus the more peripheral portion of the visual field in both the monkey and the cat (Stein & Gordon, 1981). Cells in the caudal, rostral, medial, and lateral parts of the superior colliculus respond to stimuli in the temporal, nasal, superior, and inferior visual field, respectively (Robinson & McClurkin, 1989; Stein & Gordon, 1981). The topographic organization suggests that the collicular cells can identify the location of the stimulus within the visual field. Moreover, because cells are directionally selective, preferring horizontal movement towards the periphery of the field, they are able to gather information about the direction of movement of peripheral stimuli. In contrast, cells do not have precise requirements for stimulus shape, size, or orientation, information that is less crucial in alerting the animal to the presence of peripheral objects (Robinson & McClurkin, 1989; Stein, 1984; Stein & Gordon, 1981). In addition to providing information about the appearance of a peripheral stimulus, the cells also may facilitate a shift in the animal's gaze to that stimulus. For example, cells in the superficial layers of the monkey's superior colliculus increase their firing rate prior to an eye movement to a target (Goldberg & Wurtz, 1972). Moreover, there is a motor map in the deep layers that is linked to the sensory map in the superficial layers as stimulation of the deep layers of the superior colliculus in the cat produces an eye movement that centers the area centralis on the point in the visual field that is represented in the sensory map (Stein, 1984).

(2) Role of cortical areas in peripheral vision:

Electrophysiological studies indicate that there exists a topographic map in Area 17 (V1 or striate cortex) of the cat and monkey, with neighboring areas of the visual field represented next to each other in the cortex (Daniel & Whitteridge, 1961; Tusa, Palmer, & Rosenquist, 1978). The central, inferior, and superior visual fields, and the far periphery are represented respectively in the lateral, anterior, posterior, and medial portions of the striate cortex. This map may allow the cortex to detect the location of a stimulus within the visual field. Second, electrical stimulation of the visual cortex elicits an eye movement (Schiller, 1972, 1977). This fact suggests that the cortex may be involved with the programming of eye movements.

Following bilateral lesions of Area 17 in a monkey or bilateral lesions of Areas 17, 18, and 19 with subsequent degeneration of the lateral geniculate nucleus in cats, binocular testing indicates that the visual fields are of normal extent (Humphrey, 1974; Sherman, 1974c, 1977b). Following bilateral lesions of either the posterior parietal cortex or of the frontal eye fields in monkeys reveals, respectively, no effect or a transient increase in the brightness thresholds of stimuli fixated foveally (Latto, 1977). Rather than mediating detection in the center of the field, the posterior parietal cortex may direct shifts of gaze within the visual field as some neurons respond only during an eye movement or during fixation (Lynch, Mountcastle, Talbot, & Yin, 1977).

In contrast, monocular testing of monkeys or cats following lesions of Area 17 or of higher cortical areas reveal deficits in peripheral vision. Following bilateral lesions of the visual cortex (Areas 17, 18, and 19) with degeneration of the lateral geniculate nucleus, cats detect stimuli from the midline throughout the temporal visual field but give no evidence of detecting stimuli in the nasal visual field (Sherman, 1974c, 1977b). Following unilateral or bilateral lesions of the frontal eye fields or of the striate cortex, but not bilateral lesions of the inferotemporal cortex, monkeys have higher thresholds for lights located up to 30° in the periphery (Cowey, 1967; Cowey & Weiskrantz, 1963, 1967; Latto & Cowey, 1971; Weiskrantz & Cowey, 1970). There also is evidence that the corpus callosum, which connects the two cortical hemispheres, may be important for peripheral vision. Sectioning of the posterior corpus callosum in young kittens, but not in adult cats, leads to divergent strabismus and loss of the nasal visual field of each eye (Elberger, 1979).

After unilateral lesions of the occipital lobes, patients exhibit a contralateral homonymous hemianopia (loss of the visual field contralateral to the lesion but on the same side of space for each eye. For example, a patient with a right sided lesion would have loss of the left field for the right and for the left eyes.) Nonetheless, they are able to localize large or intense stimuli in the hemianopic field (Perenin & Jeannerod, 1975, 1976; Weiskrantz, Warrington, Sanders, & Marshall, 1974). Following gunshot wounds which cause unilateral

or bilateral lesions of any one or more of the cerebral lobes, patients exhibit larger losses in the nasal field than in the temporal field (Koerner & Teuber, 1973; Teuber, Battersby, & Bender, 1960).

To summarize, lesioning studies suggest that cortical areas may play a role in detection in the nasal, temporal, superior, and inferior fields as shown by deficits across the fields. Lesioning studies of the superior colliculus suggest that it primarily mediates detection in the far periphery although its loss does lead to transient losses of detection in the center of the field.

In cats, loss of the visual cortex and of information traveling through the commissure of the superior colliculi reveals that structures interact in mediating peripheral vision. To date, there has been study of the effects of unilateral and of bilateral occipitotemporal lesions combined with a split of the collicular commissure. There also has been study of unilateral occipitotemporal lesions combined with lesion of either the contralateral substantia nigra pars reticulata or of the contralateral superior colliculus (Sherman, 1974c, 1977b; Sprague, 1966; Wallace et al., 1989, 1990). There has been no study of bilateral lesions of both the occipitotemporal cortex and superior colliculus.

Cats with a unilateral lesion of the occipitotemporal cortex have a contralateral hemianopic field (Sherman, 1977b; Sprague, 1966; Wallace et al., 1989, 1990). However, there is restoration of vision in the previously hemianopic field following either damage to the contralateral substantia nigra pars reticulata or of its pathways

through the collicular commissure (Wallace et al.,1990). Wallace et al. (1990) attribute the restoration of peripheral vision to the removal of inhibition to the ipsilateral superior colliculus from the contralateral substantia nigra pars reticulata, thus allowing the ipsilateral superior colliculus to mediate detection.

Wallace and coauthors do not, however, indicate which visual structures are mediating the restored detection. One possibility is that the projection from the temporal retinal ipsilateral to the lesioned visual cortex to the contralateral lateral geniculate nucleus and visual cortex may mediate detection in the nasal field. Supporting evidence for the role of the remaining visual cortex is the observation that bilateral lesions of the occipitotemporal cortex leave a cat blind and, following transection of the collicular commissure, each eye is able to see only from the midline to 90° ipsilaterally (Sherman, 1974c, 1977b).

In conclusion, then, the superior colliculus, the cortex, and the substantia nigra pars reticulata are involved in peripheral vision, with lesions of these structures leading to dramatic changes in peripheral vision.

Pathways serving peripheral vision

The previous section has established the importance of the superior colliculus and cortical areas for peripheral vision. Now, I will present an overview of the pathways linking these structures to the

retina and then examine, in more detail, the pathways to the cortical areas and the pathways to the superior colliculus.

Several projections to the superior colliculus and cortex arise from the retina in the monkey and cat and likely in the human (see Figure 12). First, there are direct pathways from the retina to the contralateral superior colliculus. Second, there are indirect pathways from the retina to the contralateral superior colliculus via the contralateral LGN and visual cortex. Third, there is an indirect pathway from the temporal retina through the ipsilateral lateral geniculate nucleus and visual cortices to the superior colliculus. Finally, there are connections between the superior colliculi (Sprague pathways) that carry information from the substantia nigra pars reticulata, and perhaps from other structures to which the substantia nigra pars reticulata projects, as well as connections between the visual cortices (corpus callosum) (Garey & Powell, 1968; Hoffmann, 1972, 1973; Ikeda, Plant, & Tremain, 1977; Kirk, Levick, & Cleland, 1976; Schiller, Stryker, Cynader, & Berman, 1974; Stone, 1966; Stone & Fukuda, 1974; Wallace et al., 1989, 1990, Wassle & Illing, 1980).

These pathways mediate detection in different portions of the visual field. Studies of cats with cortical lesions indicate that the direct projection from the nasal retina to the contralateral superior colliculus can mediate detection in the temporal field and the projection from the temporal retina through the ipsilateral lateral geniculate nucleus and visual cortex can mediate detection in the nasal field (Sherman, 1974c, 1977b) although, under some

circumstances, the ipsilateral visual cortex is not necessary for detection in the nasal field (see Wallace et al., 1989, 1990). Possibly, the mediation of peripheral vision by these pathways is similar in humans and in cats: Following an unilateral cortical lesion and transection of the collicular commissure, cats show better responding in the temporal than in the nasal field. Humans with cortical lesions, like cortically lesioned cats, also have larger losses in the nasal than in the temporal field (Koerner & Teuber, 1973). To date, however, there has been no study of the peripheral vision of humans with loss of the substantia nigra pars reticulata.

Several researchers have argued that the pathways from the retina through the lateral geniculate nucleus in the monkey, and likely in the human, operate as parallel processing systems and are known as the magnocellular and parvocellular pathways (Livingston & Hubel, 1988; Schiller, 1986). Supposedly, each pathway is specialized for a different function with cells in the parvocellular pathway sensitive to fine detail, color, and texture and cells in the magnocellular pathway sensitive to low contrast, depth, and direction of motion (Livingston & Hubel, 1988; Schiller, 1986; Schiller, Logothetis, & Charles, 1990). In contrast, other researchers have argued that considerable interactions exist between the pathways with regard to the mediation of abilities such as depth, form, and motion (DeYoe & VanEssen, 1988; Papathomas, Gorea, & Julesz, 1991). At present, the debate over interactive versus parallel processing streams is unresolved.

The function of peripheral vision is to alert the animal to the presence of an object in the periphery of the visual field. Cells able to detect a peripheral object need to be responsive to stimuli of low contrast, and to information about depth and motion. Once the stimulus is detected with peripheral vision, the animal then can orient to the object. The animal's central vision will perform a more detailed analysis of the characteristics of the object, such as its color and texture. The differing functions of peripheral and central vision suggest that peripheral vision is likely to be mediated by cells in the magnocellular pathway. However, if the interactive view of processing is accurate, there also may be some contribution to the magnocellular pathway by the parvocellular pathway.

The magnocellular and parvocellular pathways have different projections through the visual cortex. From the LGN, the magnocellular and parvocellular pathways project independently to different areas of V1, also known as Area 17, striate cortex, or the primary visual cortex. The magnocellular pathway from the LGN projects to layer 4b of V1, to a thick striped region of V2, and to V3, V4, V5, and the posterior parietal cortex. Other connections of the posterior parietal cortex are to the pulvinar of the thalamus, a structure in which there is convergence of pathways from the superior colliculus and the visual cortex with relay back to the visual cortex (Levine & Shefner, 1991; Schiller, 1986), and to the frontal eye fields (Hyvarinen, 1982). The cells of this pathway (V1, the thick stripes of V2, V3, V5, and the posterior parietal cortex) respond to

motion, orientation, and binocular disparity. As reviewed previously, lesions of the frontal eye fields, and hence of the magnocellular pathway, affect peripheral vision.

In contrast to the magnocellular system, once the parvocellular pathway leaves the LGN, it splits into a system of blobs and interblobs in cortical area V1. Blobs are areas of cortex sensitive to cytochrome oxidase (an enzyme which indicates the energy level of the structure) and cells within them are responsive to information about wavelength but not about orientation or motion. Interblobs, or the areas between the blobs, contain cells sensitive to orientation, wavelength, and binocular disparity. The blob regions of layers 2 and 3 of V1 project to the thin stripes of V2, then to V4 and V5 and to the inferotemporal cortex and the posterior parietal cortex. Note that the posterior parietal cortex receives input from both the magnocellular and parvocellular pathways, suggesting that there is considerable interaction between the two systems. The interblob regions of Layers 2 and 3 project to the pale stripes of V2, then to V4, and to the inferotemporal cortex and V5. Unlike the magnocellular system, the parvocellular system may be responsible for indicating information about the details about pattern (Schiller, 1986) but may not be involved in peripheral vision. As reviewed earlier, lesions of the inferotemporal cortex in monkeys, a structure which is part of the parvocellular pathway, do not affect peripheral vision but, to date, there are no studies of the effects of lesions of V4 and V5.

Not only do the magnocellular and parvocellular pathways have different projections to the cortex, they also develop at different rates. Anatomically, the magnocellular pathway through the LGN develops more slowly than does the parvocellular pathway. In the monkey, although the cells from the parvocellular and the magnocellular layers in the LGN are visually responsive on the first day of life (Blakemore & Vital-Durand, 1986), the stabilization of features such as the growth of cell lamina takes longer in the magnocellular layers (Pasik & Pasik, 1984). In the human, the magnocellular and parvocellular cells of the LGN reach adult size by 24 and 12 months of age, respectively (Hickey, 1977; Hickey & Peduzzi, 1987). Although the relative speed of anatomical development of the magnocellular and parvocellular pathways through the retina and the cortex is unknown, the general development of both the retina and the visual cortex continues for several years after birth in humans (Abramov et al., 1982; Conel, 1941, 1947, 1951, 1955, 1967; Yuodelis & Hendrickson, 1986).

In addition to the retinal projections to the visual cortex, there also are numerous projections to the superior colliculus. In the monkey and in the cat, the superior colliculus is laminated, being composed of several superficial and deep layers (Sparks & Hartwich-Young, 1989). The deep layers (layers 4 to 7) receive inputs from auditory, somatosensory, vestibular, motor, and visual structures. In contrast to the multimodal responsitivity of the deep layers, the superficial layers (layers 1 to 3) respond only to visual information,

which may come from more than one visual structure (Stein & Gordon, 1981). In both cats and monkeys, the superficial layers of the superior colliculus receive afferents primarily from the retina and the visual cortex (Robinson & McClurkin, 1989; Stein & Gordon, 1981) and send efferents to the visual cortex via such thalamic areas as the pulvinar and dorsal lateral geniculate nucleus (Benevento & Fallon, 1975; Graham, 1977; Molotchnikoff, Casanova, & Cerat, 1988; Robinson & McClurkin, 1989; Stein & Gordon, 1981). The deep layers of the superior colliculus in both cats and monkeys receive afferents from the corticotectal cells of the visual cortex (Schiller et al., 1974; Stein & Gordon, 1981), the fellow superior colliculus (Sparks & Hartwich-Young, 1989), parietal cortex (Sparks & Hartwich-Young, 1989), frontal eye fields (Sparks & Hartwich-Young, 1989), and from brainstem structures such as the mesencephalic reticular formation (Sparks & Hartwich-Young, 1989) and then project to the thalamus, basal ganglia, spinal cord, and numerous areas of the cortex. In monkeys, the projection from Area 17 (corticotectal cells) to the superior colliculus is a magnocellular pathway (Schiller, Malpeli, & Schein, 1979). As reviewed earlier, lesions of the superior colliculus, and hence of the magnocellular pathway, lead to abnormal eye movements and losses of the far periphery.

In conclusion, there are numerous connections between the retina, the lateral geniculate nucleus, the cortical areas, and the superior colliculus. This suggests that there is likely to be much redundancy among structures in the mediation of peripheral vision.

As such, it is impossible to speculate on which particular structures are most likely to be responsible for the slow development of peripheral vision in normal children. However, the limited evidence available in monkeys implicates the magnocellular pathway in peripheral vision. This pathway is both slow to develop and connects those structures identified as being involved in peripheral vision. The next section will evaluate the effects of visual deprivation upon visual structures.

Effects of visual deprivation on structures subserving peripheral vision

This section will examine the effects of deprivation upon the retina, lateral geniculate nucleus, cortical areas, and superior colliculus and how deprivation of the structure affects peripheral vision.

In monkeys, long-term unilateral lid suture beginning at 2 weeks of age for 24 months leads to decreased size and density of parafoveal retinal ganglion cells, unlike shorter periods of either unilateral or bilateral lid suture (von Noorden et al., 1977). Following either monocular or binocular lid suture in cats, the retinal ganglion cells are normal in size and frequency and also display normal receptive fields and axonal conduction velocities (Sherman & Stone,

1973). Following monocular deprivation, both X and Y[3] cells exhibit normal contrast sensitivity functions (Kratz, Mangel, Lehmkuhle, & Sherman, 1979). In summary, losses of peripheral vision following lid suture in cats or in monkeys, at least for short periods, are unlikely to be related to changes in retinal functioning.

Once past the level of the retina, the physiological effects of deprivation are different in the cat and in the monkey (Sherman & Spear, 1982; Mitchell & Timney, 1984). Given that findings in the monkey are more relevant to understanding deprived humans, this section will concentrate on the effects in the monkey. In monkeys, there are some anatomical effects, but no physiological effects, of deprivation upon the LGN. In cats, there are more severe morphological and physiological effects of deprivation on the LGN.

Following short-term binocular deprivation of 63 days from birth in monkeys, there is a decrease in the size of the cells of the magnocellular layers but no effect on cells in the parvocellular layers (Headon & Powell, 1978). In contrast, following long-term binocular deprivation of 376 days from birth, there is decreased size of cells in all layers, even of those cells representing the monocular crescent of

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[3] X, Y, and W are classes of retinal ganglion cells in the cat. X cells are those cells which sum information from different parts of the receptive field linearly and have slow conducting axons. They also typically are encountered frequently near the area centralis and have small receptive field centers. In contrast, Y cells are cells which sum information from different parts of the receptive field nonlinearly and have fast conducting axons. They tend to have large receptive field centers. W cells have large receptive field centers, and very slow conducting axons. X cells likely serve high resolution pattern vision and Y cells detect movement of objects but the function of W cells is unknown (Stone, 1983).

the visual field (Headon & Powell, 1978). Crawford, Pesch, von Noorden, Harwerth, and Smith (1991) also report that cells in all layers are reduced in size following binocular deprivation ranging from 14 to 112 days, with the amount of shrinkage unrelated to the duration of deprivation. Note, however, that their deprivation, unlike that of Headon & Powell (1978), began one month after birth.

Following short-term monocular deprivation of 12 to 64 days beginning shortly after birth, there is hypertrophy of cells in the nondeprived parvocellular layers. After opening of the deprived eye, there is shrinkage of cells in both the deprived and the undeprived magnocellular layers (Headon, Sloper, Hiorns, & Powell, 1985a). The end result is that the deprived cells are shrunken and the undeprived cells normal in size in both the magnocellular and parvocellular layers (Headon et al., 1985a). As well, deprivation from birth leads to greater effects on the ipsilateral than on the contralateral parvocellular cells in both deprived and undeprived laminae (Headon, Sloper, Hiorns, & Powell, 1985b).

Moreover, the size of cells in the parvocellular layers are equally reduced following either short-term monocular deprivation lasting 1 to 2 weeks or for an unspecified period of long-term deprivation, each of which began 2 weeks after birth (Gurski, Hickey, & Sparks, 1985). Relative to long-term deprivation, short-term deprivation leads to smaller reductions in the size of cells of the central areas of the deprived magnocellular lamina and increased size in the peripheral regions of all magnocellular lamina (Gurski et

al., 1985). Following extremely long-term monocular deprivation of 400 days from birth in monkeys, cells in all layers subserving the deprived eye are decreased in size, except those cells representing the monocular crescent (Headon & Powell, 1978; but see Sloper, Headon, & Powell, 1987).

When monocular deprivation begins even later after birth, either at 58 or 103 days, and lasts for 20 or 239 days, respectively, there are minimal effects, at most, on the size of cells (Headon & Powell, 1978).

The above results in monocularly deprived monkeys are extended by Blakemore and Vital-Durand (1986). These latter authors agree that monocular deprivation from birth leads to smaller cells in the lamina serving the deprived eye (Blakemore & Vital-Durand, 1986; Garey & Vital-Durand, 1981). Moreover, they argue that early implementation of reverse suturing of the previously deprived eye leads to normal sized cells for the previously deprived eye (Garey & Vital-Durand, 1981). The mechanism has been elaborated upon by Sloper, Headon, & Powell (1984). When the previously deprived eye is opened after 3 weeks of monocular deprivation from birth and the previously open eye closed for several weeks (reverse suturing), then there is hypertrophy of the cells in the deprived and nondeprived parvocellular layers. Subsequently, there is a decrease in the size of cells in the undeprived parvocellular layers (Sloper et al., 1984). In the magnocellular layers, reverse suturing leads to reversal of the

shrinkage of the deprived cells and hypertrophy of the undeprived cells (Sloper et al., 1984).

Finally, Blakemore & Vital-Durand (1986) indicate that there is no suggestion of a selective loss of Y cells in deprived laminae, and no difference in density of cells in deprived or nondeprived layers, whether magnocellular or parvocellular (Blakemore & Vital-Durand, 1986).

Following monocular deprivation from birth or binocular deprivation beginning soon after birth in monkeys, there are no physiological effects on either X or Y cells of the magnocellular or parvocellular layers, with the exception that Y cells in the magnocellular layers were not studied after binocular deprivation. Following binocular deprivation beginning at birth, the spatial properties of X cells of the magnocellular and parvocellular layers are normal but Y cells have not been studied (Blakemore & Vital-Durand, 1986). Unlike in the cat, the monkey's LGN does not possess W cells (Blakemore & Vital-Durand, 1986).

In an investigation of the number and size of X and Y cells following monocular deprivation in the cat, Sherman, Hoffmann, and Stone (1972) reported that there is a selective effect of deprivation upon Y cells. In the binocular segment of layer A of the LGN contralateral to the deprived eye, cells are shrunken and only 9% of the Y cells remain. In the monocular segment contralateral to the deprived eye, cells are normal in size with a normal complement of Y cells (also see Guillery & Stelzner, 1970; Lehmkuhle, Kratz, Mangel, &

Sherman, 1978; Sherman, Wilson, & Guillery, 1975). The differential effects upon the monocular versus the binocular segments suggests that binocular competition had occurred. That is, there was competition between the nondeprived and the deprived eyes for synaptic space in layers receiving binocular input. Finally, Hickey (1980) reported that W cells of layers C1 and C2 serving the deprived eye are not significantly smaller than cells in the nondeprived laminae.

In the cat's LGN, the effects of binocular lid suturing upon Y cells are less severe than the effects of monocular suturing, except in the monocular segment. This suggests that unfair interocular competition occurs during monocular deprivation. Following binocular deprivation from lid suture, nearly 30% of Y cells remained, with similar losses in the binocular and monocular segments (Sherman et al., 1972). This suggests that the mechanism at work was deprivation per se. Moreover, there were no apparent effects on cell size (Sherman et al., 1972; but see Stone, 1983). The comparable losses in the binocular and monocular segments of the lateral geniculate nucleus in binocularly sutured cats, but not in monocularly sutured cats, suggests that the effects of binocular suture are worse than that of monocular suture but only in the monocular segment. It is unknown why this occurs. Finally, following binocular deprivation arising from dark rearing, there again was a decrease of Y but not of X cells (Kratz, 1982; Kratz, Sherman, & Kalil, 1979).

Physiologically, both Y and X cells of the LGN in cats were reported to respond normally to visual stimulation following monocular or binocular lid suture (Sherman et al, 1972). To date, there has been no study of responsiveness in W cells following either monocular or binocular deprivation.

To summarize, deprivation from lid suture in the monkey leads to smaller cell sizes but no physiological changes in the LGN. In contrast, deprivation in the cat leads to a selective loss of Y cells, particularly following monocular deprivation, but minimal physiological effects. The findings in deprived humans are likely to be most similar to those from the monkey. This suggests that, except for the size of cells, the LGN of children treated for dense and central cataracts may be normal.

Lid suture has been shown to affect cortical properties both in monkeys and cats, with binocular deprivation in monkeys exerting more severe effects than in cats (see Crawford, Blake, Cool, & von Noorden, 1975).

To date, there has been no assessment of the effects of lid suture during infancy on cytochrome oxidase staining. Following monocular deprivation from lid suture in the monkey, there is a decrease in the proportion of cells in layer 4 representing the deprived eye and an increase in the representation of the nondeprived eye. Outside of layer 4C, nearly all cells are driven by the nondeprived eye (reviewed in Mitchell & Timney, 1984; Wiesel, 1982). The results of reverse suturing of the eyes, which involves

closing the nondeprived eye and opening the deprived eye, reveal different effects upon the magnocellular and the parvocellular pathways. If reverse suturing is done early in life, then the previously deprived eye now controls more synaptic space in layer 4C and the previously nondeprived eye controls less, with the cortical cells driven by the previously deprived eye (reviewed in Wiesel, 1982). Moreover, the period of modifiability of the visual cortex differs for layers receiving input from the magnocellular layers of the LGN (layer 4C-alpha of the cortex) versus from the parvocellular layers (layer 4C-beta), with the critical period for the magnocellular layers ending by 3 weeks while that of the parvocellular layers lasting until about 6 weeks (reviewed in Wiesel, 1982).

One implication for children treated for cataracts is that damage to the magnocellular pathways, which are believed to be involved in peripheral vision, is less modifiable than damage to the parvocellular pathways. As such, patching in cases of monocular deprivation may have less benefit for peripheral vision than for abilities that appear more dependent upon the parvocellular pathway, such as visual acuity. A second implication is that deprivation of late onset should affect parvocellular pathways more than magnocellular pathways. Yet, the present research reported abnormal peripheral vision even in children with unilateral deprivation of late onset. This suggests, then, that the magnocellular pathway in the human may be vulnerable for longer than that of the

monkey. Alternatively, losses of peripheral vision in these patients may reflect damage to other parts of the visual system. A caveat to either of these conclusions is as follows. Possibly, predictions should be made by looking at the developmental pattern and not by examining studies of reverse suturing as alteration of pathways that have already formed may be different than modifying developing pathways.

Following binocular deprivation in the monkey, many cells in Area 17 (Crawford et al., 1975; Crawford et al., 1991; Wiesel & Hubel, 1974; reviewed in Wiesel, 1982; but see Hyvarinen, Hyvarinen, & Linnankoski, 1981), Area 19 (Hyvarinen, Carlson, & Hyvarinen, 1981; but see Hyvarinen, et al., 1981) and in the posterior parietal cortex (Hyvarinen et al., 1981) are unresponsive. Of the responsive cells in Area 17, few are binocular (Crawford et al., 1975; Crawford et al., 1991; Wiesel & Hubel, 1974; reviewed in Wiesel, 1982). There are no studies of the differential effects of binocular deprivation on the magnocellular and parvocellular pathways through the cortex in monkeys.

Following monocular deprivation in cats, the deprived eye is able to drive only about 10% of cells in the binocular segment of Area 17 and these cells typically have weak responses and lack the normal selectivity for direction and orientation (Sherman & Spear, 1982). The effects on the monocular segment of Area 17 are less severe, with about 67% of cells driven by the deprived eye and about one-third of cells having nonspecific receptive fields. Moreover, the

distribution of afferents from Y cells of the LGN is more reduced than from X cells. The functioning of W cells in the cortex remains unstudied in deprived cats.

In the cat, Area 18 receives input from the Y and W cells but not from the X cells of the LGN, which only project to Area 17. Following monocular deprivation, there continues to be a projection from the LGN to Area 18 although the size of the cells of the LGN projecting to Area 18 are more diminished than those cells projecting to Area 17. This again suggests that there is a selective effect of deprivation on the Y geniculocortical projection.

Following binocular deprivation in the cat, both the monocular and the binocular segments of Area 17 are affected, with few responsive cells. Of the responsive cells, most are unselective for direction or orientation. As occurs for the monocular segment of LGN, binocular deprivation affects the monocular segment of Area 17 more than does monocular deprivation. Many of the cells in Area 17 continue to receive input from the X and the remaining Y cells of the LGN. The results from study of Area 18 are essentially similar to those of Area 17 (Sherman & Spear, 1982).

Like the LGN and visual cortex, the corpus callosum is developing early in life and is affected by visual experience. Until three months of age, normal kittens have more callosally projecting neurons in Areas 17 and 18 than do adult cats (Innocenti & Frost, 1979). Following monocular deprivation, monocular enucleation, or strabismus, the projections of the callosal neurons are even more

widespread than in normal adult cats (Innocenti & Frost, 1979). In contrast, following binocular deprivation, binocular enucleation, or dark-rearing, the projections of the callosal neurons are decreased compared to adult cats (Innocenti & Frost, 1979, 1980; Innocenti, Frost, & Illes, 1985; Lund & Mitchell, 1979). These results suggest that the corpus callosum, and hence peripheral vision, may be affected in children with cataracts.

To summarize, the effects of monocular or binocular deprivation upon the cortex in monkeys and in cats are severe, with many cells unresponsive. As well, the callosally projecting neurons of Areas 17 and 18 in deprived cats are either more or less numerous than in normal cats. Moreover, studies of reverse suturing in monkeys revealed that the magnocellular layers projecting to the visual cortex have a shorter period for recovery than do the parvocellular layers. It is unknown whether a similar finding would hold for binocularly deprived monkeys. In contrast, at the level of the LGN, both parvocellular and magnocellular layers appear vulnerable, at least to prolonged periods of deprivation beginning at birth. With regard to children treated for dense and central cataracts, it is likely that deprivation, whether monocular or binocular, renders many cortical cells unresponsive with the magnocellular pathways from the retina through the cortex affected by abnormal visual experience.

The effects of lid suture upon the superior colliculus have been studied in cats (Hoffmann & Sherman, 1974, 1975) but not in

monkeys. Although the development of the superior colliculus is unstudied in the primate, the adult-like properties are similar in primates and cats (Stein & Gordon, 1981) and, possibly, so may be the effects of deprivation.

Following monocular deprivation in the cat, the direct retinotectal Y and W pathways are normal while the indirect Y cell pathway from the retina through the ipsilateral LGN and visual cortex is abnormal. Within the superior colliculus, the deprived eye dominates fewer cells and drives fewer directionally selective cells within the binocular segment than does the nondeprived eye but there is no effect of deprivation upon selectivity for the velocity of the stimulus. The monocular segment of the superior colliculus, which responds to stimuli in the far temporal periphery of the visual field, appears unaffected (Hoffmann & Sherman, 1974). This physiological finding agrees with the behavioral observation that monocularly deprived cats see only in their far temporal periphery (Sherman, 1973, 1974a, 1974b; Sherman & Sprague, 1979; but see Heitlander & Hoffmann, 1978; van Hof-van Duin, 1977). Furthermore, it appears that the abnormal corticotectal pathways of the monocularly deprived cats suppress the mediation of detection in the near temporal field by the normal retinotectal pathways, although such suppression can be removed by decortication (Sherman, 1974a; Sherman & Sprague, 1979). Following destruction of the geniculocortical pathways via bilateral lesions of the visual cortex, there is an expansion of the field for the deprived eye and a loss of

the field for the nondeprived eye. Like binocularly deprived cats (Sherman, 1973) and normally reared cats with bilateral cortical lesions (Sherman, 1974c), each eye of the monocularly deprived cat now sees from the midline throughout the temporal hemifield. The effect of cortical lesions is to remove the suppression by the geniculocortical pathways, allowing full expression of the normal direct Y and direct W pathways projecting to the superior colliculus. Thus, the cat now detects objects throughout the entire temporal hemifield.

Following binocular deprivation in cats, there is a severe loss of the indirect Y pathway, a moderate loss of the direct Y pathway, and no effect on the direct W pathway (Hoffmann & Sherman, 1975). This indicates that binocular deprivation disturbs more pathways than does monocular deprivation. Because electrical stimulation of the visual cortex drives the normal number of cells in the superior colliculus, the loss of the indirect Y pathway must be in the geniculocortical portion, which also correlates with the loss of Y cells in the LGN of binocularly deprived cats (see Sherman et al., 1972). That all Y cells are susceptible to abnormal experience is shown by the moderate loss of Y cells in the direct pathway. Compared to normal cats, in binocularly deprived cats, fewer cells can be driven by either eye, there is a reduction in the number of directionally selective cells, and an abnormality in selectivity for the speed of the stimulus, with losses evident in both the monocular and binocular segments. Given that there are comparable losses in the binocular

and monocular segments of the superior colliculus in binocularly but not in monocularly sutured cats, this suggests that the effects of binocular suture are worse than that of monocular suture, at least in the monocular segment.

The behavior of binocularly deprived cats is consistent with the physiological findings that suggest that the binocularly deprived animal uses direct retinotectal pathways for peripheral vision. On tests of visual perimetry, binocularly deprived cats (Sherman, 1973, 1974b, 1977a), like normally reared cats with bilateral occipitocortical lesions combined with split of the tectal commissure (to remove intercollicular suppression, which does not develop in binocularly deprived cats) (Sherman, 1974c, 1977b), do not give evidence of detecting stimuli in the nasal visual field (but see Kossut et al., 1978; Maire-Lepoivre et al., 1988; van Hof-van Duin, cited in Zablocka, 1983; Zablocka, 1983). Moreover, unilateral or bilateral lesions of the occipitotemporal cortex have no effect on the extent of the visual field in binocularly deprived cats (Sherman, 1977a). In contrast, lesions of the superior colliculus lead to blindness in the contralateral hemifield (Sherman, 1977a). Together, these pieces of evidence suggest that binocularly deprived cats, like cortically lesioned cats, rely for peripheral orientation not on the geniculocortical Y pathway but only on the retinotectal Y and/or W pathways.

To summarize, lesioning studies indicate that the abnormal corticotectal pathways in monocularly deprived cats suppress the

mediation of detection in the near temporal field by the normal retinotectal pathways. In contrast, binocularly deprived cats rely on retinotectal pathways. Using the results of further lesioning studies, conducted primarily in cats, I next will examine the likely physiological mediation of detection in the temporal, nasal, inferior, and superior fields.

Evidence for the role of the direct W pathway in temporal detection arises from studies of cats with immunological lesions of 70% of Y cells in the direct Y pathway and 82% of Y cells in the indirect Y pathway but a normal direct W pathway. (The X pathway likely mediates information about pattern vision and not about peripheral vision.) These cats exhibit normal responsiveness to stimuli presented in the temporal hemifield but not in the nasal hemifield (Spear, Miller, Vielhuber, & Kornguth, 1986). This suggests, then, that the retinotectal W pathway may be <u>sufficient</u> to mediate detection in the temporal hemifield. Although the technique is not yet developed, immunological lesioning of the direct W pathway would provide another test of its role in peripheral vision.

In contrast to cats, the evidence regarding the underlying mechanisms of detection in the temporal hemifield is less conclusive in monkeys. To date, the peripheral vision of only monocularly deprived monkeys has been studied with these animals, in the worst cases, exhibiting deficits throughout the field. Although the LGN of deprived monkeys appears normal physiologically and the superior colliculus remains unstudied, there are serious losses in the visual

cortex. At this time, the evidence does not conclusively suggest that deprivation affects the magnocellular pathway in monkeys (see Headon & Powell, 1978; Wiesel, 1982). Suggested work in monkeys would include examination of the visual field in normal infant monkeys, binocularly deprived monkeys, cortically lesioned monkeys (provided that intercollicular suppression is avoided), and in immunologically lesioned monkeys, with relation of the exhibited behavior under each condition to the physiological status of the underlying pathways.

Several pieces of evidence indicate that detection in the nasal field is mediated by the indirect pathway through the visual cortex. First, partial loss of detection in the nasal field occurs in decorticated cats and in humans with loss of parts of the cortex (Koerner & Teuber, 1973; Sherman, 1974c, 1977b; Teuber et al., 1960), in cats with immunologically induced loss of the indirect Y pathway through the cortex (Spear et al., 1986), and in deprived cats with severe losses of Y cells in the LGN (see Sherman et al., 1972). Second, the full development of the nasal field in kittens may depend upon the development of the indirect Y pathway to the superior colliculus. At about eight weeks of age, the corticotectal cells of layer 5 of the visual cortex achieve adultlike properties. These corticotectal cells project to the superior colliculus and, as seen from studies of decortication and cortical cooling, affect the development of adult-like properties of the collicular cells, which also occurs at eight weeks of age (reviewed in Stein, 1984; Tsumoto, Suda, & Sato, 1984). Over

this time period, kittens develop full nasal fields (Sireteanu & Maurer, 1982). Although the temporal field also is expanding during this time (Sireteanu & Maurer, 1982), its development likely is not dependent upon the development of the corticotectal pathway, as lesioning of the visual cortex in cats only affects the nasal field (Sherman, 1974c, 1977b). Together, these pieces of data suggest that the development of the nasal field in cats is dependent upon the postnatal development of the Y cell projection from corticotectal cells to the superior colliculus. Similar correlational data in the monkey are unavailable as there have been no studies of the development of its peripheral vision. However, note that in the cat the visual cortex may not be <u>necessary</u> for detection in the contralateral nasal field. As demonstrated by Wallace et al. (1990), cats are able to detect stimuli in the previously hemianopic contralateral field with both eyes following unilateral lesions of the visual cortex and contralateral substantia nigra pars reticulata. The visual structures mediating such detection are unknown.

These studies of deprived or lesioned cats suggest, then, that the indirect Y pathway may be necessary for nasal detection and W cells sufficient for temporal detection. Whether Y or W cells mediate detection in the superior and inferior visual fields is unknown. Given that all parts of the visual fields are represented topographically in the visual cortex (Daniel & Whitteridge, 1961; Tusa et al., 1978) and superior colliculus (Robinson & McClurkin, 1989; Stein & Gordon, 1981), then damage to these structures could impair functioning not

only nasally and temporally but also inferiorly, superiorly, and centrally. The effect of the lesion will depend upon the degree of redundancy among structures for the mediation of peripheral vision.

Although the evidence suggests that development of peripheral vision and the effects of cortical lesioning are similar in cats and in humans, it is important to acknowledge that some behavioral differences are apparent across species. Typically, deprived humans exhibit impaired sensitivity across the visual field. At least in some groups of deprived eyes, there are especially large losses of sensitivity centrally compared to peripherally and at 20° nasally compared to 30° temporally (Experiment 5). As well, both monocular and binocular deprivation in humans lead to losses in the extent of the nasal, superior, and inferior fields, and especially in the temporal field (Experiment 4). Although deprived cats exhibit loss of the extent of the field, losses are more pronounced nasally (see Chapter 1). These differences in outcome between cats and humans suggest several possibilities. First, the pathways may be different between cats and humans. This possibility is feasible given that the visual system of cats and monkeys differs and that humans are most similar to monkeys. For example, projections of the LGN cells go primarily to Area 17 in the monkey but to many cortical areas in the cat (Stone, 1983). Second, the pathways may be similar between cats and humans but affected differently by deprivation. Given that deprivation can affect the anatomical and physiological development of structures differently in monkeys and cats (see this chapter), so

too may there be differences between cats and humans. Third, because cats have not been tested with small stimuli, there may be undiscovered deficits in the peripheral vision of deprived cats.

To summarize, a number of different structures have been implicated in peripheral vision. These include the retina, lateral geniculate nucleus, superior colliculus, and cortical areas. Given the numerous connections linking these areas, there is likely to be much redundancy among structures with regard to the mediation of peripheral vision. As such, it is difficult to speculate on which particular structures are most likely to be responsible for the slow development of peripheral vision in normal children. Physiological examination of deprived monkeys and cats suggest that in deprived humans the lateral geniculate nucleus may be little affected but that there will be severe effects upon the cortical areas and the projections to the superior colliculus, with the magnocellular pathway likely to be affected. Moreover, the Y indirect pathway may be necessary for nasal detection and W cells sufficient for temporal detection, at least in cats. At this time, the physiological mediation of detection in the superior, inferior, and central fields is less certain. Studies from cats are less likely to be relevant for understanding of humans than studies of monkeys. Systematic elimination of each pathway via immunological lesioning, if methodologically possible for Y and W cells, and preferably carried out in primates, may help to resolve which cell types and which pathways are necessary versus sufficient for detection in different parts of the visual field. Until

further work is conducted in primates, the physiological basis of peripheral vision in humans will remain uncertain, other than to say that the functioning of the magnocellular pathway may be responsible for peripheral vision.

Suggestions for future research

The examination of peripheral vision in young infants requires many trials and, consequently, conclusions are limited to what the group of infants is able to detect. To date, no study has examined the normal development of peripheral vision or the effects thereon of deprivation in individual infants. This study is important because it would allow the tracing of peripheral vision within individuals over time, which is an extremely sensitive index of development. As well, a study of individual aphakic infants might reveal whether the mild but continued deprivation resulting from aphakia per se exerts any deleterious effects upon the development of peripheral vision. A first suggestion for future research, then, is to attempt to develop methods to allow such study.

Second, although this dissertation has explored sensitivity across the field, it has done so using a fast bracketing procedure, except at 20° nasally and 30° temporally. To precisely measure the normal development and the effects thereon of deprivation, sensitivity at selected intervals along the horizontal and the vertical meridia should be measured in normal children and in children

treated for cataracts, with subsequent comparison of data from the center of the field versus in the nasal, temporal, superior, and inferior hemifields. This procedure is important for several reasons. It would allow further assessment of the principle that slowly developing behaviors, such as occurs at the center of the field, in the near nasal field, and at the edges of the field, are most vulnerable to deprivation. Moreover, by assessing sensitivity at the edges of the visual field, these data then can be compared to the data from the extent of the field to determine if the temporal field is indeed most vulnerable, as was found in Experiment 4. Also, this investigation would allow independent confirmation of two surprising findings from the fast bracketing procedure: that normal 7-year-olds are more sensitive at 0° centrally than are 8- and 9-year-olds and that children with developmental deprivation have less sensitive fields at 0° centrally than do children with congenital deprivation.

Third, a weakness of the research was that there were limited numbers of some types of patients. For example, research might further investigate the effects of occlusion of the fellow nondeprived eye using a larger sample of children with a range of patching histories. Such a study would provide information about how interocular competition affects the nervous system and also might influence ophthalmological treatment of both the deprived and the nondeprived eyes of patients. As well, there were a limited number of children with early onset developmental cataracts. In order to evaluate the effects of such deprivation, it is necessary to carefully

document outcome in a larger sample with variability in the age of onset and the duration of deprivation. This brings up a larger concern present in clinical research. We conceptualize variables such as the timing and the duration of deprivation as being independent factors possessing unlimited and naturally occurring variability. In fact, factors of clinical interest may not operate independently of each other and there may be limitations to their variability. For example, children with traumatic cataracts, at least in my sample, usually were males at least at the toddler stage who, because of the traumatic nature of their injury, generally received prompt treatment. In contrast, these same variables did not necessarily define other groups of deprived eyes. In other groups of deprived eyes, treatment was not always prompt and there tended to be an equal number of males and females. Moreover, in children who developed cataracts after birth, the cataract could have been diagnosed early in infancy or much later during childhood. As such, it is important to be aware of differences that characterize and hence may potentially limit clinical research.

Fourth, the literature suggests that strabismus in humans and in monkeys has no systematic effect upon the visual field. Yet, the effect upon peripheral vision of the age of onset and duration of the strabismus, whether it is esotropic (the eye turns inwards) or exotropic (the eye turns outwards), the angle of deviation of the eye, and of any associated amblyopia has not been carefully evaluated. Understanding of the effects of abnormal experience from strabismus

is of importance as strabismus constitutes a different clinical condition than cataracts and hence may exert different effects upon the developing nervous system. Study of the effects of strabismus upon peripheral vision is of importance also for interpreting results from children treated for cataracts, many of whom have strabismus.

Fifth, the contribution of optical factors following treatment for a dense and central cataract to the observed losses of peripheral vision requires further investigation. As suggested in Chapter 3, it is important to examine whether an abnormal visual environment alters the development of optical parameters. Possibly, deprivation has little effect upon optics and any optically induced losses of peripheral vision in deprived children are no larger than those present in adults treated for senile cataracts. Alternatively, deprivation early in life may alter optics, subsequently leading to large losses of peripheral vision. Possible alterations in optical development are best assessed by comparing the optical parameters of deprived children with differing visual histories to those parameters present in adults treated for senile cataracts and in age-matched normal children. As well, ray tracings of the propagation of light in an aphakic eye versus in a normal eye could evaluate precisely how individual factors such as a misshapen pupil, abnormal axial length, or an unusually shaped cornea contribute to the abnormal peripheral vision. These pieces of knowledge are important clinically because they provide an index of how much of the loss of peripheral vision results from abnormal optical parameters rather

than from neural factors. If most of the loss of peripheral vision in deprived eyes is shown to result from the effects of deprivation upon neural pathways, then this provides impetus to the clinician to seek out the most effective treatment regime to minimize abnormal neural effects. If most of the loss of peripheral vision instead results from optical factors, then this forces reconsideration of current thinking regarding the importance of early ophthalmological treatment and provides incentive for the design of the most optimal optical correction.

Finally, Sprague (1991) has suggested that the superior colliculus may help to direct attention to objects in the visual field. If true, some of the observed losses of peripheral vision in the patients may be explainable by impairments in the attentional mechanisms directed by the superior colliculus, rather than by losses in peripheral sensitivity. Yet, it is unlikely that attentional problems explain all of the observed losses of peripheral vision: Should attentional mechanisms be impaired in patients, it is likely that there would be increasingly greater difficulty of detection for objects presented further and further in the periphery and, hence, increasingly larger losses in the periphery. Yet, my data from tests with the Octopus perimeter suggest that larger losses of sensitivity were observed centrally rather than peripherally and, moreover, there were equal losses of sensitivity in the near, mid, and far periphery.

Nonetheless, the question of the role of attention in peripheral vision is of interest. One method to examine the role of attention is to measure the time needed for subjects to process and respond to peripheral stimuli. If impairments in attentional mechanisms do contribute to some of the observed losses in the peripheral vision of patients, then a patient likely will exhibit a longer latency to detect and then to respond to a peripheral stimulus compared to an age matched normal subject. In order to ensure that longer response latencies are not due to a greater difficulty in detection by the patients, stimuli should be comparably above threshold for all subjects. Measurement of the latency to respond across age in normal children also is of valuable for examining the development of normal attentional mechanisms and their possible contribution to peripheral detection.

To summarize, this research suggests that peripheral vision develops slowly throughout early childhood, depending upon the stimulus used in testing, and is adversely affected by deprivation from dense and central cataracts. The physiological and optical bases for the observed effects were explored. This research has provided a further successful test of a basic principle of development, that slowly developing behaviors are vulnerable to abnormal experience. The findings of this dissertation may be used to guide other explorations of functioning of the brain and investigations of the mechanisms by which a child develops and adapts to its environment.

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APPENDIX A:
TABLES

Table 1.

Results of the 4-way ANOVA; Age, Test, Intensity, and Meridian

Effect	*F*	*df*	*p*
Age	**8.58**	**3,84**	**= .00015**
Test	1.20	2,168	= .30
Intensity	**2780.55**	**1,84**	**< .000001**
Meridian	**893.90**	**7,588**	**< .000001**
Age X Test	0.91	6,168	= .49
Age X Intensity	1.86	3,84	= .14
Age X Meridian	**13.33**	**21,588**	**< .000001**
Age X Test X Intensity	0.73	6,168	= .63
Age X Test X Meridian	1.07	42,1176	= .36
Age X Intensity X Meridian	1.25	21,588	= .20
Age X Test X Intensity X Meridian	**1.91**	**42,1176**	**= .00071**
Test X Intensity X Meridian	0.92	14,1176	= .53
Test X Intensity	**8.69**	**2,168**	**= .00049**
Test X Meridian	1.12	14,1176	= .34
Intensity X Meridian	**172.80**	**7,588**	**< .000001**

Table 2.

Mean Extent of the Field in Visual Degrees (Upper Row) and Standard Error (Lower Row) for the Eight Meridia on Test 1 with Target Intensity 31.8 cd/m^2 (Object 12c)

Age	Temporal 0°	Temporal 45°	Superior 90°	Superior 135°	Nasal 180°	Nasal 225°	Inferior 270°	Inferior 315°
5 yrs	43.5	34.0	28.8	31.0	32.7	30.4	32.3	41.0
	1.4	1.4	1.3	1.2	1.3	0.9	1.0	1.3
6 yrs	50.0	37.8	32.0	35.2	37.2	35.6	38.8	47.6
	1.6	1.4	1.2	1.2	1.4	1.2	1.3	1.7
7 yrs	51.3	41.0	33.0	35.3	37.3	34.9	38.1	47.5
	1.6	1.8	1.5	1.4	1.3	1.2	1.6	2.0
Adults	57.1	36.8	27.9	30.7	38.9	38.8	41.8	54.3
	1.7	1.7	1.1	1.2	1.2	1.3	1.5	1.9

Table 3.

Mean Extent of the Field in Visual Degrees (Upper Row) and Standard Error (Lower Row) for the Eight Meridia on Test 1 with Target Intensity 318 cd/m^2 (Object 14c)

	Temporal		Superior		Nasal		Inferior	
Age	0°	45°	90°	135°	180°	225°	270°	315°
5 yrs	70.3	56.6	45.6	46.4	48.5	45.6	53.1	68.4
	1.6	1.7	1.2	1.1	1.1	0.9	1.0	1.2
6 yrs	73.6	59.4	47.5	50.5	51.6	48.5	55.5	70.2
	1.7	1.2	1.2	0.9	0.8	1.1	1.1	1.2
7 yrs	74.9	60.4	48.2	52.7	54.1	50.0	59.2	71.2
	1.9	1.6	0.9	1.1	0.9	1.2	1.2	1.7
Adults	82.8	57.9	44.1	46.8	54.1	51.1	62.6	79.2
	1.3	2.3	1.9	1.6	1.2	0.8	1.1	1.5

Table 4.

Average Interocular Difference Scores in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensity 31.8 cd/m^2 (Object I2e)

Age	Temporal 0°	45°	Superior 90°	135°	Nasal 180°	225°	Inferior 270°	315°
5 yrs	0	-1.5	-1.1	-0.18	-2.4	-1.9	-0.46	0.86
	1.2	1.5	1.5	1.0	1.5	0.94	1.0	1.3
6 yrs	3.9	0.21	-0.58	-2.1	-2.2	-1.9	-0.46	1.9
	1.5	0.98	0.74	1.1	0.84	0.78	1.1	1.2
7 yrs	1.6	-0.06	-1.3	-2.8	-3.5	-1.6	0.45	2.0
	1.1	1.0	0.85	1.1	0.93	1.0	1.0	1.4
Adults	0.50	-1.4	-0.63	-0.13	-2.6	-1.4	-0.44	2.9
	1.1	1.4	0.86	1.1	0.90	0.86	0.72	1.2

Note. A positive sign (not indicated) means that the measured extent of the field for the right eye is larger than for the left eye while a negative sign indicates the opposite finding.

Table 5.

Average Interocular Difference Scores in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensity 318 cd/m^2 (Object I4c)

	Temporal		Superior		Nasal		Inferior	
Age	0°	45°	90°	135°	180°	225°	270°	315°
5 yrs	1.1	0.14	0.71	-0.21	-1.1	-0.93	0.57	-0.68
	1.2	1.2	1.2	1.4	1.2	1.1	1.0	1.2
6 yrs	-0.50	-1.0	-0.21	-0.33	-0.62	-1.1	0.50	-0.50
	1.0	1.4	0.87	1.0	1.0	1.4	0.93	1.3
7 yrs	2.1	2.1	0.95	-0.15	-0.85	1.4	-0.15	-1.1
	1.3	1.4	0.89	0.99	1.2	1.2	1.4	1.1
Adults	2.9	-2.3	-0.81	0.69	-2.4	0.13	0.31	-0.06
	1.0	1.4	1.1	1.1	1.1	0.77	0.94	0.92

Note. A positive sign (not indicated) means that the measured extent of the field for the right eye is larger than for the left eye while a negative sign indicates the opposite finding.

Table 6.

Largest Interocular Difference Scores in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensity 31.8 cd/m^2 (Object I2c)

	Temporal		Superior		Nasal		Inferior	
Age	0°	45°	90°	135°	180°	225°	270°	315°
5 yrs	14	-17	-20	-11	-19	-10	-10	-14
	1.2	1.5	1.5	1.0	1.5	0.94	1.0	1.3
6 yrs	18	-9	-6	-10	-8	-9	-9	11
	1.5	0.98	0.74	1.1	0.84	0.78	1.1	1.2
7 yrs	9	9	-7	-9	-8	-8	8	13
	1.1	1.0	0.85	1.1	0.93	1.0	1.0	1.4
Adults	8	10	-6	-7	-8	-6	5	8
	1.1	1.4	0.86	1.1	0.90	0.86	0.72	1.2

Note. A positive sign (not indicated) means that the measured extent of the field for the right eye is larger than for the left eye while a negative sign indicates the opposite finding.

Table 7.

Largest Interocular Difference Scores in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensity 318 cd/m^2 (Object I4e)

	Temporal		Superior		Nasal		Inferior	
Age	0°	45°	90°	135°	180°	225°	270°	315°
5 yrs	-11	11	13	-14	-12	-13	11	14
	1.2	1.2	1.1	1.4	1.2	1.1	1.0	1.2
6 yrs	9	-12	-7	8	-8	-14	8	-11
	1.0	1.4	0.87	1.0	1.0	1.4	0.93	-0.50
7 yrs	9	10	7	-7	-11	9	12	-8
	1.3	1.4	0.99	1.2	1.2	1.4	1.4	1.1
Adults	7	-11	-6	8	7	5	-6	-7
	1.0	1.4	1.1	1.1	1.1	0.77	0.94	0.92

Note. A positive sign (not indicated) means that the measured extent of the field for the right eye is larger than for the left eye while a negative sign indicates the opposite finding.

Table 8.

Average Absolute Interocular Difference Scores in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensity 31.8 cd/m^2 (Object I2e)

Age	Temporal 0°	45°	Superior 90°	135°	Nasal 180°	225°	Inferior 270°	315°
5 yrs	4.9	6.0	5.3	3.7	6.0	4.1	4.2	5.1
	0.81	0.96	1.1	0.76	1.1	0.62	0.60	0.88
6 yrs	5.8	4.0	2.8	4.3	3.1	3.3	4.3	4.8
	1.1	0.49	0.49	0.74	0.71	0.54	0.61	0.76
7 yrs	3.9	3.3	3.4	4.8	4.5	3.3	3.5	4.4
	0.70	0.70	0.46	0.59	0.67	0.72	0.58	1.0
Adults	3.1	4.6	2.6	3.6	3.5	3.0	2.4	4.4
	0.72	0.80	0.55	0.59	0.68	0.51	0.38	0.87

Table 9.

Average Absolute Interocular Difference Scores in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensity 318 cd/m^2 (Object 14c)

	Temporal		Superior		Nasal		Inferior	
Age	0°	45°	90°	135°	180°	225°	270°	315°
5 yrs	4.8	5.4	4.4	5.8	4.5	4.2	4.1	4.7
	0.76	0.62	0.74	0.83	0.80	0.72	0.64	0.75
6 yrs	4.3	5.8	3.6	4.1	4.0	5.0	3.7	5.5
	0.53	0.80	0.43	0.61	0.54	0.98	0.54	0.66
7 yrs	4.6	5.2	3.1	3.5	4.1	4.2	5.2	4.3
	0.88	0.80	0.60	0.59	0.78	0.79	0.82	0.58
Adults	4.2	4.8	3.8	3.2	4.3	2.4	2.6	2.6
	0.65	0.95	0.57	0.73	0.69	0.47	0.68	0.65

Table 10.

Results of the 3-way ANOVA: Age, Intensity, and Meridian

Effect	*F*	*df*	*p*
Age	**4.83**	**3,84**	**= .0038**
Intensity	.61	1,84	= .44
Meridian	**2.58**	**7,588**	**= .0127**
Age X Intensity	.40	3,84	= .75
Age X Meridian	.58	21,588	= .93
Intensity X Meridian	.63	7,588	= .73
Age X Intensity X Meridian	1.33	21,588	= .15

Table 11.

Range of the Extent of the Field in Visual Degrees (Upper Row) and Standard Error (Lower Row) Along Eight Meridia During Test 1 with Target Intensity 31.8 cd/m^2 (Object 12c)

	Temporal		Superior		Nasal		Inferior	
Age	0°	45°	90°	135°	180°	225°	270°	315°
5 yrs	28	25	26	25	26	18	17	28
	1.4	1.4	1.3	1.2	1.3	0.85	1.0	1.3
6 yrs	25	29	23	24	25	25	24	27
	1.6	1.4	1.2	1.2	1.4	1.2	1.3	1.7
7 yrs	24	29	22	21	17	16	26	25
	1.6	1.8	1.5	1.4	1.3	1.2	1.6	2.0
Adults	20	20	14	15	15	16	13	28
	1.7	1.7	1.1	1.2	1.2	1.3	1.5	1.9

Table 12.

Range of the Extent of the Field in Visual Degrees (Upper Row) and Standard Error (Lower Row) Along Eight Meridia During Test 1 with Target Intensity 318 cd/m^2 (Object 14c)

	Temporal		Superior		Nasal		Inferior	
Age	0°	45°	90°	135°	180°	225°	270°	315°
5 yrs	26	32	21	19	21	20	17	22
	1.6	1.7	1.2	1.1	1.1	0.89	1.0	1.2
6 yrs	22	17	21	16	15	24	22	20
	1.7	1.2	1.2	0.91	0.81	1.1	1.1	1.2
7 yrs	30	22	14	17	14	20	16	25
	1.9	1.6	0.93	1.1	0.88	1.2	1.2	1.7
Adults	17	28	24	20	15	9	13	16
	1.3	2.3	1.9	1.6	1.2	0.81	1.1	1.5

Table 13.

Test-retest Reliability in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensity 31.8 cd/m^2 (Object I2e)

Age	Temporal 0°	45°	Superior 90°	135°	Nasal 180°	Inferior 225°	270°	315°
5 yrs	-0.96	1.9	2.5	1.4	2.7	0.36	-1.0	-1.0
	1.4	1.7	1.7	1.4	0.94	1.0	1.2	1.2
6 yrs	1.3	1.3	0.13	1.1	1.6	1.3	-0.33	1.0
	1.8	1.3	0.98	0.97	1.3	0.88	1.0	1.1
7 yrs	1.1	1.4	1.9	0.20	0.25	-1.0	-1.7	0.95
	1.5	1.4	1.2	1.0	0.99	1.2	1.1	1.8
Adults	1.4	0.56	0.06	1.3	1.2	3.7	2.3	2.4
	1.5	1.1	0.91	1.3	0.91	1.1	1.0	1.2

Note. A positive sign (not indicated) means that the measured extent of the field for the first test is larger than for the repeat test while a negative sign indicates the opposite finding.

Table 14.

Test-retest Reliability in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensity 318 cd/m^2 (Object I4c)

Age	Temporal 0°	45°	Superior 90°	135°	Nasal 180°	Inferior 225°	270°	315°
5 yrs	-1.8	-0.50	-0.71	-1.6	-0.89	-1.2	-0.96	-1.3
	1.4	1.6	1.4	1.3	1.2	1.2	1.2	1.6
6 yrs	-1.2	1.3	-0.25	-2.1	-1.7	1.3	-2.1	-3.7
	1.3	1.9	1.0	1.2	0.84	1.1	0.94	1.2
7 yrs	-1.1	-0.10	0.75	-0.05	0.60	-0.50	-3.1	-3.3
	1.1	1.3	0.97	1.2	1.1	1.0	0.92	1.8
Adults	1.8	-1.0	0.31	-0.50	1.0	-0.25	-0.38	-1.7
	0.82	1.5	1.3	1.5	1.2	1.0	1.0	0.98

Note. A positive sign (not indicated) means that the measured extent of the field for the first test is larger than for the repeat test while a negative sign indicates the opposite finding.

Table 15.

Mean Extent of the Field at 5 and at 7 Years (n = 27) in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia on Test 1 with Target Intensities 31.8 cd/m^2 (Object I2c) and 318 cd/m^2 (Object I4e)

	Temporal		Superior		Nasal		Inferior	
Intensity/Age	0°	45°	90°	135°	180°	225°	270°	315°
Dim								
5 yrs	43.6	34.0	28.8	31.1	32.8	30.4	32.4	41.1
	1.5	1.4	1.3	1.2	1.3	0.89	1.1	1.3
7 yrs	50.6	37.1	30.9	33.8	37.6	35.7	37.5	47.0
	1.2	1.1	0.75	0.79	0.83	0.96	1.0	1.3
Intense								
5 yrs	70.2	56.7	45.5	46.3	48.7	45.8	53.3	68.3
	1.7	1.8	1.2	1.1	1.2	0.90	1.0	1.2
7 yrs	81.4	60.0	49.3	52.0	54.9	49.7	60.6	75.6
	0.67	0.98	0.74	0.79	0.60	1.0	0.96	0.90

Table 16.

Results of the 4-way ANOVA: Age, Test, Intensity, and Meridian

Effect	*F*	*df*	*p*
Age	**44.57**	**1,26**	**< .00001**
Test	**10.32**	**1,26**	**= .0035**
Intensity	**2126.55**	**1,26**	**< .00001**
Meridian	**346.93**	**7,182**	**< .00001**
Age X Test	0.46	1,26	= .50
Age X Intensity	0.17	1,26	= .68
Age X Meridian	**8.65**	**7,182**	**< .00001**
Age X Test X Intensity	3.51	1,26	= .07
Age X Test X Meridian	1.00	7,182	= .43
Age X Intensity X Meridian	**2.65**	**7,182**	**= .0124**
Age X Test X Intensity X Meridian	1.62	7,182	= .13
Test X Intensity X Meridian	1.19	7,182	= .31
Test X Intensity	0.07	1,26	= .80
Test X Meridian	0.68	7,182	= .69
Intensity X Meridian	**74.45**	**7,182**	**< .00001**

Table 17.

Results of the 4-way ANOVA: Age, Test, Intensity, and Meridian

Effect	*F*	*df*	*p*
Age	0.46	2,60	= .63
Test	**5.50**	**1,60**	**= .0224**
Intensity	**2046.70**	**1,60**	**< .00001**
Meridian	**655.17**	**7,420**	**< .00001**
Age X Test	0.56	2,60	= .57
Age X Intensity	2.70	2,60	= .08
Age X Meridian	**15.10**	**14,420**	**< .00001**
Age X Test X Intensity	1.21	2,60	= .31
Age X Test X Meridian	1.49	14,420	= .11
Age X Intensity X Meridian	**1.91**	**14,420**	**= .0242**
Age X Test X Intensity X Meridian	**1.76**	**14,420**	**= .0417**
Test X Intensity X Meridian	0.96	7,420	= .46
Test X Intensity	2.76	1,60	= .10
Test X Meridian	1.13	7,420	= .35
Intensity X Meridian	**118.22**	**7,420**	**< .00001**

Table 18.

Average Interocular Difference Scores of the Experienced and Inexperienced 7-Year-Olds in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensities 31.8 cd/m^2 (Object I2e) and 318 cd/m^2 (Object I4e)

	Temporal		Superior		Nasal		Inferior	
Intensity	0°	45°	90°	135°	180°	225°	270°	315°
Dim								
Exp.	-0.52	0.63	-1.1	-1.9	-1.4	-2.0	-0.93	-1.3
	1.4	1.1	0.73	0.80	1.2	1.0	0.80	1.1
Inexp.	1.6	-0.06	-1.3	-2.8	-3.5	-1.6	0.45	2.0
	1.1	1.0	0.85	1.1	0.93	1.0	4.0	1.4
Intense								
Exp.	-0.07	1.1	-1.4	-2.6	-0.48	-0.30	0.63	-1.5
	0.62	1.2	0.80	1.2	0.91	1.2	0.90	0.96
Inexp.	2.1	2.1	0.95	-0.15	-0.85	1.4	-0.15	-1.1
	1.3	1.4	0.89	0.99	1.2	1.2	1.4	1.1

Note. A positive sign (not indicated) means that the measured extent of the field for the right eye is larger than for the left eye while a negative sign indicates the opposite finding.

Table 19.

Largest Interocular Difference Scores for 7-Year-Olds in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensities 31.8 cd/m^2 (Object I2c) and 318 cd/m^2 (Object I4c)

	Temporal		Superior		Nasal		Inferior	
Intensity	0°	45°	90°	135°	180°	225°	270°	315°
Dim								
Exp.	-12	11	-9	-9	-11	11	-8	-10
	1.4	1.1	0.73	0.80	1.2	1.0	0.80	1.1
Inexp.	9	9	-7	-9	-8	-8	8	13
	1.1	1.0	0.85	1.1	0.93	1.0	1.0	1.4
Intense								
Exp.	7	-13	-9	-13	-9	-13	-9	-10
	0.62	1.2	0.80	1.2	0.91	1.2	0.90	0.96
Inexp.	9	10	7	-7	-11	9	12	-8
	1.3	1.4	0.99	1.2	1.2	1.4	1.4	1.1

Note. A positive sign (not indicated) means that the measured extent of the field for the right eye is larger than for the left eye while a negative sign indicates the opposite finding.

Table 20.

Average Absolute Interocular Difference Scores for 7-Year-Olds in Visual Degrees (Upper Row) and Standard Error (Lower Row) for Eight Meridia with Target Intensities 31.8 cd/m^2 (Object I2c) and 318 cd/m^2 (Object I4c)

Intensity	Temporal 0°	45°	Superior 90°	135°	Nasal 180°	225°	Inferior 270°	315°
Dim								
Exp.	5.6	4.6	3.0	3.2	4.9	3.7	2.9	4.7
	0.80	0.65	0.49	0.62	0.72	0.83	0.59	0.64
Inexp.	3.9	3.3	3.4	4.8	4.5	3.3	3.5	4.4
	0.70	0.70	0.46	0.59	0.67	0.72	0.58	1.0
Intense								
Exp.	2.5	4.7	3.4	5.7	3.5	4.9	3.8	3.9
	0.37	0.79	0.52	0.70	0.61	0.71	0.52	0.64
Inexp.	4.6	5.2	3.1	3.5	4.1	4.2	5.2	4.3
	0.88	0.80	0.60	0.59	0.78	0.79	0.82	0.58

Table 21.

Range of the Extent of the Field in Visual Degrees (Upper Row) and Standard Error (Lower Row) Along Eight Meridia During Test 1 for 7-Year-Olds with Target Intensities 31.8 cd/m^2 (Object I2e) and 318 cd/m^2 (Object I4e)

	Temporal		Superior		Nasal		Inferior	
Intensity	0°	45°	90°	135°	180°	225°	270°	315°
Dim								
Exp.	24	20	14	15	14	17	18	24
	1.2	1.1	0.75	0.79	0.83	0.96	1.0	1.3
Inexp.	24	29	22	21	17	16	26	25
	1.6	1.8	1.5	1.4	1.3	1.2	1.6	2.0
Intense								
Exp.	12	17	17	17	13	19	20	16
	0.67	0.98	0.74	0.79	0.60	1.0	0.96	0.90
Inexp.	30	22	14	17	14	20	16	25
	1.9	1.6	0.93	1.1	0.88	1.2	1.2	1.7

Table 22.

Conversion Table for Decibels (db) to Apostilbs (asb) as Used Conventionally with the Octopus Perimeter

db	log units	asb/db conversion asb (10 to n)
0	3.0	1000
10	2.0	100
20	1.0	10
30	0	1.0
40	-1.0	0.10
50	-2.0	0.01
51	-2.1	0.008

Table 23.

Results of the 3-way ANOVA: Age (7-, 8-, 9-Year-Olds, and Adults), Test, and Location

Effect	*F*	*df*	*p*
Age	0.24	3,68	= .87
Test	2.06	2,136	= .13
Location	**6.84**	**1,68**	**= .0110**
Age X Test	0.25	6,136	= .96
Age X Location	0.54	3,68	= .65
Test X Location	0.41	2,136	= .67
Age X Test X Location	0.88	6,136	= .51

Table 24.

Average Signed Interocular Difference Scores in db (Upper Row) and Standard Error (Lower Row) for 20° Nasally and 30° Temporally

Age	Sensitivity	
	20° Nasally	30° Temporally
7 yrs	0.37	-0.10
	0.48	0.92
8 yrs	-0.09	-0.90
	0.44	0.53
9 yrs	-0.30	0.03
	0.27	0.70
Adults	-0.03	0.10
	0.29	0.53

Note. A positive sign (not indicated) means that measured sensitivity was better for the right eye than for the left eye while a negative sign indicates the opposite finding.

Table 25.

Results of the 2-way ANOVA: Age (7-, 8-, 9-Year-Olds and Adults) and Location

Effect	*F*	*df*	*p*
Age	0.50	3,68	= .68
Location	0.22	1,68	= .64
Age X Location	0.37	3,68	= .78

Table 26.

Largest Interocular Difference in Sensitivity in db (Upper Row) and Standard Error (Lower Row) for 20° Nasally and for 30° Temporally

Age	Sensitivity	
	20° Nasally	30° Temporally
7 yrs	-4.4	-8.4
	0.41	0.67
8 yrs	-3.8	-3.2
	0.37	0.33
9 yrs	-1.6	2.8
	0.27	0.57
Adults	2.0	5.6
	0.29	0.40

Note. A positive sign (not indicated) means that the right eye was more sensitive than the left eye while a negative sign indicates the opposite finding.

Table 27.

Average Absolute Interocular Difference Scores in db (Upper Row) and Standard Error (Lower Row) for 20° Nasally and 30° Temporally

Age	Sensitivity	
	20° Nasally	30° Temporally
7 yrs	1.5	2.6
	0.34	0.70
8 yrs	1.5	1.6
	0.29	0.43
9 yrs	0.77	1.9
	0.17	0.41
Adults	1.2	1.4
	0.13	0.41

Table 28.

Range of Sensitivity at 20° Nasally and at 30° Temporally in db (Upper Row) and Standard Error (Lower Row) During Test 1

Age	Sensitivity	
	20° Nasally	30° Temporally
7 yrs	7.6	16.0
	0.47	0.74
8 yrs	10.6	6.4
	0.50	0.38
9 yrs	4.4	7.6
	0.47	0.67
Adults	5.2	5.4
	0.29	0.37

Table 29.

Test-retest Reliability at 20° Nasally and at 30° Temporally in db (Upper Row) and Standard Error (Lower Row)

Age	Sensitivity	
	20° Nasally	30° Temporally
7 yrs	0.19	-1.08
	0.51	0.94
8 yrs	-0.26	-0.51
	0.48	0.42
9 yrs	-0.27	-0.30
	0.44	0.74
Adults	-0.87	-0.21
	0.31	0.22

Note. A positive score indicates that the right eye was more sensitive than the left eye while a negative score indicates the opposite finding.

Table 30.

Results of the 2-way ANOVA: Age (7-Year-Olds and Adults) and Location

Effect	*F*	*df*	*p*
Age	0.33	1,38	= .57
Location	114.53	6,228	< .00001
Age X Location	15.14	6,228	< .00001

Table 31.

Range (db) and Percentage of Normal Thresholds Compared to Adults Along the Horizontal Meridian for Test 1 for 7-, 8-, and 9-Year-Olds, and Adults and for Test 2 for 7-Year-Olds and Adults

	Horizontal Meridian										
Age	Nasal				Center		Temporal				
	60°	45°	30°	15°	0°	15°	30°	45°	60°	75°	90°
Test 1											
7 yrs											
	0-N	14-N	22-N	N	3-N	2-N	N	20-N	N	13-N	3-N
	21%	79%	90%	100%	21%	37%	100%	90%	100%	79%	53%
8 yrs											
	0-N	14-N	N	N	4-N	0-N	22-N	20-N	17-N	13-N	7-N
	42%	79%	100%	100%	0%	21%	95%	79%	79%	68%	26%
9 yrs											
	0-N	15-N	N	N	3-N	4-N	N	23-N	N	15-N	14-N
	18%	82%	100%	100%	0%	9%	100%	91%	100%	91%	73%
Adults											
	0-N	18-N	N	N	24-N	0-N	N	N	N	16-N	0-N
	32%	95%	100%	100%	68%	5%	100%	100%	100%	95%	63%

Test 2

7 yrs

0-N	0-N	17-N	20-N	7-N	0-N	24-N	N	15-N	0-N	0-N
42%	90%	95%	95%	21%	16%	90%	100%	85%	68%	37%

Adults

0-N	17-N	N	N	24-N	0-22	N	N	N	17-N	0-N
58%	90%	100%	100%	68%	0%	100%	100%	100%	95%	53%

Note. N = normal threshold

Table 32.

Range (db) and Percentage of Normal Thresholds Compared to Adults Along the Vertical Meridian for Test 1 for 7-, 8-, and 9-Year-Olds, and Adults and for Test 2 for 7-Year-Olds and Adults

	Vertical Meridian								
Age	Superior				Inferior				
	60°	45°	30°	15°	15°	30°	45°	60°	75°
Test 1									
7 yrs									
	0-N	0-N	7-N	22-N	24-N	22-N	16-N	0-N	0-N
	11%	5%	37%	95%	95%	90%	58%	11%	16%]
8 yrs									
	0-N	0-N	1-N	21-N	22-N	18-N	15-N	3-N	0-N
	16%	21%	63%	84%	90%	90%	79%	47%	5%
9 yrs									
	0-N	0-N	8-N	21-N	24-N	N	19-N	1-N	0-N
	9%	46%	64%	91%	82%	100%	82%	73%	28%
Adults									
	0-N	3-N	9-N	24-N	26-N	N	N	12-N	0-N
	11%	53%	79%	95%	95%	100%	100%	84%	21%

Test 2

7 yrs									
	0-N	0-N	7-N	22-N	22-N	18-N	13-N	0-N	0-N
	5%	11%	32%	84%	74%	84%	53%	11%	11%
Adults									
	0-N	7-N	N	N	N	N	N	13-N	0-N
	21%	68%	100%	100%	100%	100%	100%	95%	21%

Note. N = normal threshold.

Table 33.

Range (db) and Percentage of Normal Thresholds Compared to Adults Along a Diagonal Meridian for Test 1 for 7-, 8-, and 9-Year-Olds, and Adults and for Test 2 for 7-Year-Olds and Adults

	Diagonal Meridian					
Age	Superior/Nasal			Inferior/Temporal		
	30°/30°	15°/15°	15°/15°	30°/30°	45°/45°	60°/60°
Test 1						
7 yrs	15-N 74%	N 100%	N 100%	20-N 90%	N 100%	14-N 84%
8 yrs	16-N 90%	23-N 95%	N 100%	17-N 90%	18-N 95%	13-N 79%
9 yrs	N 100%	N 100%	N 100%	N 100%	N 100%	14-N 91%
Adults	N 100%	N 100%	N 100%	N 100%	N 100%	12-N 95%

Test 2

7 yrs	8-N 74%	22-N 90%	N 100%	20-N 95%	11-N 90%	0-N 79%
Adults	N 100%	N 100%	N 100%	N 100%	N 100%	N 100%

Note. N = normal threshold.

Table 34.

Range (db) and Percentage of Normal Thresholds Compared to Adults Along a Diagonal Meridian for Test 1 for 7-, 8-, and 9-Year-Olds, and Adults and for Test 2 for 7-Year-Olds and Adults

	Diagonal Meridian				
Age	Inferior/Nasal		Superior/Temporal		
	30°/30°	15°/15°	15°/15°	30°/30°	45°/45°
Test 1					
7 yrs	1-N 63%	24-N 95%	N 100%	17-N 90%	0-N 53%
8 yrs	N 100%	N 100%	N 100%	14-N 95%	0-N 58%
9 yrs	N 100%	N 100%	N 100%	N 100%	8-N 91%
Adults	0-N 84%	N 100%	N 100%	N 100%	0-N 53%

Test 2

7 yrs	0-N	24-N	21-N	16-N	0-N
	74%	95%	84%	90%	37%
Adults	0-N	N	N	N	0-N
	95%	100%	100%	100%	74%

Note. N = normal threshold.

Table 35.

Results of the 3-way ANOVA: Age (7-Year-Olds and Adults), Test, and Location

Effect	*F*	*df*	*p*
Age	**11.81**	**1,38**	**= .0014**
Test	0.09	1,38	= .77
Location	**328.29**	**3,114**	**< .00001**
Age X Test	0.43	1,38	= .52
Age X Location	**11.12**	**3,114**	**< .00001**
Test X Location	0.73	3,114	= .54
Age X Test X Location	1.01	3,114	= .39

Table 36.

Results of the 2-way ANOVA: Age (7-, 8-, 9-Year-Olds, and Adults) and Location

Effect	*F*	*df*	*p*
Age	6.38	3,68	= .0007
Location	162.51	3,204	< .00001
Age X Location	12.07	9,204	< .00001

Table 37.

Test-retest Reliability in db (Upper Row) and Standard Error (Lower Row) for 0°, Near Periphery, Mid Periphery, and Far Periphery

Age	Peripheral Sensitivity			
	0°	Near	Mid	Far
7 yrs	-2.6	-0.35	-0.13	0.76
	3.1	0.29	0.58	0.74
Adults	0.20	-0.12	-0.17	-0.42
	0.85	0.09	0.19	0.28

Note. A positive sign (not indicated) means that the first test was more sensitive than the second test while a negative sign indicates the opposite finding.

Table 38.

Average Signed Interocular Difference Scores in db (Upper Row) and Standard Error (Lower Row) for 0°, Near Periphery, Mid Periphery, and Far Periphery

Age	Peripheral Sensitivity			
	0°	Near	Mid	Far
7 yrs	-1.9	1.3	-0.59	0.12
	3.4	1.0	1.1	1.1
Adults	2.2	0.16	0.63	1.6
	0.93	0.17	0.36	0.74

Note. A positive sign (not indicated) means that sensitivity was better for the right eye than for the left eye while a negative sign indicates the opposite finding.

Table 39.

Results of the 2-way ANOVA: Age (7-, 8-, 9-Year-Olds, and Adults) and Location

Effect	*F*	*df*	*p*
Age	1.27	1,18	= .28
Location	0.21	3,54	= .89
Age X Location	1.37	3,54	= .26

Table 40.

Largest Interocular Difference in Sensitivity in db (Upper Row) and Standard Error (Lower Row) for 0°, Near Periphery, Mid Periphery, and Far Periphery

Age	Peripheral Sensitivity			
	0°	Near	Mid	Far
7 yrs	13.0	2.7	4.1	-3.9
	2.7	0.41	0.87	0.64
Adults	6.0	-0.09	1.9	2.3
	0.85	0.01	0.29	0.40

Note. A positive sign (not indicated) means that measured sensitivity was better for the right eye than for the left eye while a negative sign indicates the opposite finding.

Table 41.

Average Absolute Interocular Difference Scores in db (Upper Row) and Standard Error (Lower Row) for 0°, Near Periphery, Mid Periphery, and Far Periphery

Age	Peripheral Sensitivity			
	0°	Near	Mid	Far
7 yrs	8.3	1.8	2.6	2.2
	2.1	0.94	0.41	0.80
Adults	2.2	0.17	0.79	1.9
	0.93	0.16	0.33	0.65

Table 42.

Range of Sensitivity in db (Upper Row) and Standard Error (Lower Row) for 0°, Near Periphery, Mid Periphery, and Far Periphery During Test 1

Age	Peripheral Sensitivity			
	0°	Near	Mid	Far
7 yrs	28	1.6	3.3	4.3
	1.8	0.14	0.24	1.6
8 yrs	26	2.0	2.8	4.3
	1.6	0.13	0.21	0.25
9 yrs	25	1.7	4.2	7.8
	2.6	0.15	0.40	0.60
Adults	7.0	2.0	3.1	3.8
	0.51	0.12	0.21	0.26

Table 43.

Characteristics of the Deprived Eyes Tested in Experiment 4

Condition	Age of onset (m) Mean Range	Duration (m) Mean Range	Deprived Eyes, Children
Unilateral Congenital Short Duration	0	4.8 3.2-5.8	8, 8
Unilateral Congenital Long Duration	0	12.2 8.2-16.0	6, 6
Unilateral Developmental Short Duration	38.2 20.4-52.9	3.0 1.0-5.6	8, 8
Unilateral Developmental Long Duration	33.4 11.3-71.0	12.4 10.3-15.2	3, 3
Bilateral Congenital Short Duration	0	3.7 1.5-6.1	8, 5
Bilateral Congenital Long Duration	0	13.4 6.9-17.8	5, 5
Bilateral Developmental Short Duration	34.2 7.5-66.9	4.1 1.4-6.2	14, 10

Bilateral Developmental	18.8	24.5	4, 3
Long Duration	12.7-36.4	8.0-45.0	

Table 44.

Results of the 3-way ANOVA: Group, Intensity, and Meridian

Effect	*F*	*df*	*p*
Group	2.09	3,25	= .13
Intensity	569.61	1,25	< .00001
Group X Intensity	0.20	3,25	= .90
Meridian	158.47	7,175	< .00001
Group X Meridian	1.71	21,175	= .03
Intensity X Meridian	28.76	7,175	< .00001
Group X Intensity X Meridian	0.55	21,175	= .95

Table 45.

Results of the 5-way ANOVA: Condition (Binocular, Monocular Deprivation), Timing (Birth, 18-72 Months), Duration (Short, Long), Intensity, and Meridian

Effect	*F*	*df*	*p*
Condition	1.13	1,48	= .29
Timing	0.81	1,48	= .37
Duration	**8.66**	**1,48**	**= .005**
Intensity	**36.39**	**1,48**	**< .00001**
Meridian	**76.31**	**7,336**	**< .00001**
Condition X Timing	2.85	1,48	= .10
Condition X Duration	2.03	1,48	= .16
Timing X Duration	3.74	1,48	=.06
Condition X Timing X Duration	0.12	1,48	= .73
Condition X Intensity	**7.75**	**1,48**	**=.0077**
Timing X Intensity	0.83	1,48	= .37
Condition X Timing X Intensity	0.02	1,48	= .88
Duration X Intensity	0.20	1,48	= .66
Condition X Duration X Intensity	0.54	1,48	= .47
Timing X Duration X Intensity	0.06	1,48	= .80
Condition Timing X Duration X Intensity	1.85	1,48	= .18
Condition X Meridian	0.62	7,336	= .74
Timing X Meridian	0.36	7,336	= .92
Condition X Timing X Meridian	0.03	7,336	= .41
Duration X Meridian	**2.90**	**7,336**	**=.0058**
Condition X Duration X Meridian	1.82	7,336	= .08
Timing X Duration X Meridian	0.19	7,336	= .99
Condition X Timing X Duration X Meridian	0.73	7,336	= .65

Intensity X Meridian	**5.87**	**7,336**	**< .0001**
Condition X Intensity X Meridian	0.55	7,336	=.80
Timing X Intensity X Meridian	0.45	7,336	=.87
Condition X Timing X Intensity X Meridian	1.23	7,336	=.26
Duration X Intensity X Meridian	1.35	7,336	=.22
Condition X Duration X Intensity X Meridian	0.60	7,336	=.76
Timing X Duration X Intensity X Meridian	1.84	7,336	=.08
Condition X Timing X Duration X Intensity X Meridian	0.67	7,336	=.69

Table 46.

Patching of the Nondeprived Eye and Extent of the Visual Field of the Deprived Eye

Effect	*F*	*df*	*p*
Patching	0.40	2,15	= .68
Intensity	22.15	1,15	= .0003
Patching X Intensity	1.72	2,15	= .21
Meridian	24.39	7,105	< .00001
Patching X Meridian	2.24	14,105	= .0105
Intensity X Meridian	4.23	7,105	= .0004
Patching X Intensity X Meridian	1.47	14,105	= .14

Table 47.

Pearson Correlations of Visual Acuity and Extent of the Visual Field for the Deprived Eye

Location (df = 44)	Intense Light r	Dim Light r
0°	.27	.46
45°	.12	.45
90°	.26	.45
135°	.34	.43
180°	.27	.43
225°	.09	.50 *
270°	.10	.52 *
315°	.28	.46

Note. * $p < .001$

Table 48.

Characteristics of the Deprived Eyes Assessed Across the Visual Field

Condition	Age of onset (m) Mean Range	Duration (m) Mean Range	Deprived Eyes, Children
Unilateral Congenital Short Duration	0	4.7 3.2-5.8	7, 7
Unilateral Congenital Long Duration	0	14.7 10.3-19.0	2, 2
Unilateral Developmental Short Duration	38.6 20.4-52.9	2.9 1.9-3.9	3, 3
Unilateral Developmental Long Duration	44.5 18.0-71.0	13.4 11.6-15.2	2, 2
Bilateral Congenital Short Duration	0	5.1 4.2-5.9	7, 6
Bilateral Congenital Long Duration	0	11.4 6.4-17.8	11, 9
Bilateral Developmental Short Duration	33.8 7.5-66.9	3.8 1.4-6.2	10, 7

Bilateral	Developmental	18.8	24.5	4, 3
Long	Duration	12.7-36.4	8.0-45.0	

Table 49.

Characteristics of the Deprived Eyes Assessed at 20° Nasally and 30° Temporally

Condition	Age of onset (m) Mean Range	Duration (m) Mean Range	Deprived Eyes, Children
Unilateral Congenital Short Duration	0	4.9 4.1-5.8	7, 7
Unilateral Congenital Long Duration	0	13.1 8.2-19.0	5, 5
Unilateral Developmental Short Duration	91.6 35.4-144.8	3.2 1.6-5.1	7, 7
Unilateral Developmental Long Duration	87.0 18.0-155.9	13.4 11.5-15.2	2, 2
Bilateral Congenital Short Duration	0	5.5 4.2-6.1	11, 8
Bilateral Congenital Long Duration	0	11.0 6.4-17.8	12, 9
Bilateral Developmental Short Duration	48.6 7.5-122.1	3.9 1.4-6.2	13,10

Bilateral Developmental	50.2	29.4	5, 5
Long Duration	12.7-105.0	8.0-74.1	

Table 50.

Results of the 2-way ANOVA: Visual History and Location

Effect	*F*	*df*	*p*
Visual History	0.44	2,25	= .65
Location	298.72	3,75	< .00001
Visual History X Location	0.39	6,75	= .88

Table 51.

Results of the 4-way ANOVA: Condition (Binocular, Monocular Deprivation), Timing (Birth, 18-72 Months), Duration, and Location

Effect	*F*	*df*	*p*
Condition	0.008	1,38	= .93
Timing	2.57	1,38	= .12
Condition X Timing	0.02	1,38	= .89
Duration	1.45	1,38	= .24
Condition X Duration	0.76	1,38	= .39
Timing X Duration	1.37	1,38	= .25
Condition X Timing X Duration	0.31	1,38	= .58
Location	**8.12**	**3,114**	**= .0001**
Location X Condition	1.64	3,114	= .19
Location X Timing	**8.83**	**3,114**	**< .00001**
Location X Timing X Condition	1.87	3,114	= .14
Location X Duration	0.22	3,114	= .88
Location X Duration X Condition	0.05	3,114	= .99
Location X Duration X Timing	1.23	3,114	= .30
Condition X Timing X Duration X Location	0.70	3,114	= .56

Table 52.

Results of the 2-way ANOVA: Group and Location

Effect	*F*	*df*	*p*
Group	0.61	3,29	= .62
Location	0.15	1,29	= .70
Group X Location	1.22	3,29	= .32

Table 53.

Results of the 4-way ANOVA: Condition (Binocular, Monocular Deprivation), Timing (Birth, 6-156 Months), Duration, and Location

Effect	*F*	*df*	*p*
Condition	0.56	1,54	= .46
Timing	0.09	1,54	= .77
Condition X Timing	1.50	1,54	= .23
Duration	1.69	1,54	= .20
Condition X Duration	0.02	1,54	= .88
Timing X Duration	1.02	1,54	= .32
Condition X Timing X Duration	2.18	1,54	= .15
Location	3.16	1,54	= .08
Location X Condition	2.98	1,54	= .09
Location X Timing	1.87	1,54	= .18
Location X Timing X Condition	**9.63**	**1,54**	**= .0030**
Location X Timing (unilaterals)	**7.28**	**1,54**	**= .0093**
Location X Timing (bilaterals)	2.40	1,54	= .13
Location (unilateral congenitals)	**16.65**	**1,54**	**= .0001**
Location (unilateral developmentals)	0.13	1,54	= .72
Location X Duration	3.20	1,54	= .08
Location X Duration X Condition	1.37	1,54	= .25
Location X Duration X Timing	3.11	1,54	= .08
Condition X Timing X Duration X Location	1.10	1,54	= .30

Table 54.

Results of the 2-way ANOVA: Patching and Location Across the Field

Effect	*F*	*df*	*p*
Patching	0.76	2,11	= .49
Location	4.19	3, 33	= .0129
Patching X Location	1.00	6, 33	= .44

Table 55.

Results of the 2-way ANOVA: Patching and Location (20° Nasally and 30° Temporally)

Effect	*F*	*df*	*p*
Patching	0.63	2,18	= .54
Location	1.77	1,18	= .20
Patching X Location	0.27	2,18	= .77

Table 56.

Pearson Correlations of Visual Acuity with Loss Scores of Peripheral Sensitivity

Location	*df*	*r*
0°	31	-.13
Near Periphery	31	.38
Mid Periphery	31	.32
Far Periphery	31	-.08
20° Nasally	44	.26
30° Temporally	44	-.21

Table 57.

Parameters of the Nondeprived and Deprived Eyes of J.W. and of Normal Subjects

	J.W.'s Nondeprived Eye	J.W.'s Deprived Eye	Normal Eyes
Dioptric power (D)			
Central	41.38 X 69°	42.37 X 121°	39-48[1]
Apical	41.56 X 84°	42.62 X 118°	
Right periphery	40.88 X 98°	41.37 X 107°	
Left periphery	41.06 X 85°	41.56 X 103°	
Radius of curvature (mm)			
Central	8.16	7.98	7.0-8.65[1]
Apical	8.12	7.92	
Cornea			
Shape	Spherical	Elliptical	--
Shape Factor	+.20	+.38	(-.08) - (+.40)[2]
Astigmatism (D)	+1.25 X 69°	+2.00 X 121°	0[3]
Corneal diameter (mm)			
Horizontal	11.75	11.75	--
Vertical	11.0	11.0	--
Axial length (mm)	26.1	25.1	21-26[4]

Pupil diameter (mm)	4.5	3.5	light adapted: 4.8 at 10 yrs 4.0 at 45 yrs 3.4 at 80 yrs[3]

1 = Woolf (1948)
2 = Manual for the Humphrey Auto Keratometer, Model 420
3 = Bennett and Rabbetts (1989)
4 = Sorsby, Benjamin, Davey, Sheridan, & Tanner (1957)

Table 58.

Loss of the Visual Field for J.W.'s Deprived Eye Under Different Pupillary Conditions

Intensity of Light	Loss scores for deprived eye (°)							
	0°	45°	90°	135°	180°	225°	270°	315°
	Temporal		Superior		Nasal		Inferior	
(1) Typical pupillary size:								
Intense (I4c)	27	14	8	9	15	15	15	28
Intermediate (I3c)	31	18	24	9	20	25	36	25
Dim (I2c)	39	34	22	28	30	33	28	36
(2) Pupils of equal size:								
Intense (I4c)	29	22	11	11	15	15	30	33
Intermediate (I3c)	33	28	25	22	31	29	34	28
Dim (I2c)	46	37	22	31	32	33	31	52

Table 59.

Loss of the Visual Field for J.W.'s Eyes Under Different Pupillary Conditions Relative to Typical Pupillary Conditions

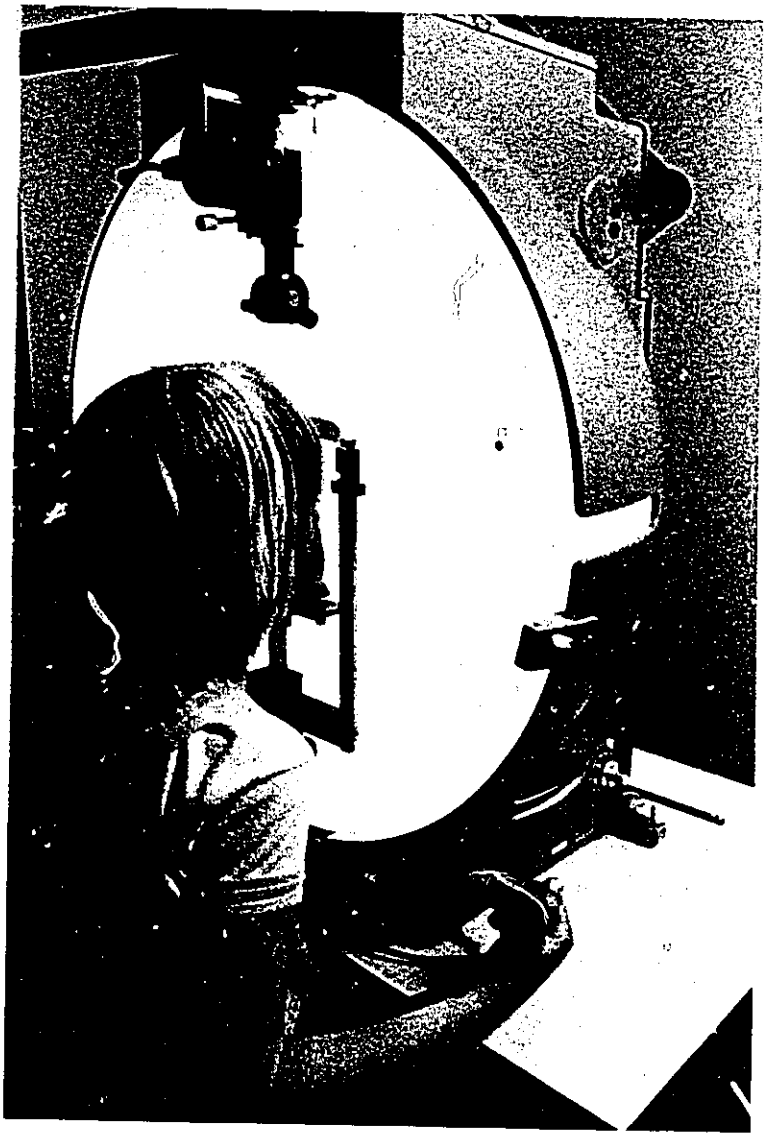
Intensity of Light	Loss scores (°)							
	0°	45°	90°	135°	180°	225°	270°	315°
	Temporal		Superior		Nasal		Inferior	
(1) Nondeprived eye under enlarged pupillary size:								
Intense (14c)	2	-1	-5	-6	-2	3	-2	-2
Intermediate (13c)	-3	-3	-2	0	-4	0	2	-1
Dim (12c)	-7	-7	-2	-10	-2	1	-1	-4
(2) Deprived eye under enlarged pupillary size:								
Intense (14c)	-2	-8	-3	-2	0	0	-15	-5
Intermediate (13c)	-2	-10	-1	-13	-11	-4	2	-3
Dim (12c)	-7	-3	0	-3	-2	0	-3	-16
(3) Nondeprived eye under constricted pupillary size:								
Intense (14c)	4	0	2	-1	-2	-5	-3	1
Intermediate (13c)	0	0	-1	-1	1	-4	4	1
Dim (12c)	-3	-7	3	-6	4	-1	0	-2

(4) Deprived eye under constricted pupillary size:

Intense (I4c)	-3	-12	0	0	0	0	-9	-4
Intermediate (I3c)	0	-12	2	-3	0	4	2	-19
Dim (I2c)	-14	-3	1	-1	3	-2	-6	-14

Note. A - sign indicates constriction of the field relative to baseline conditions and a + sign (not specified) the opposite.

APPENDIX B:
FIGURES



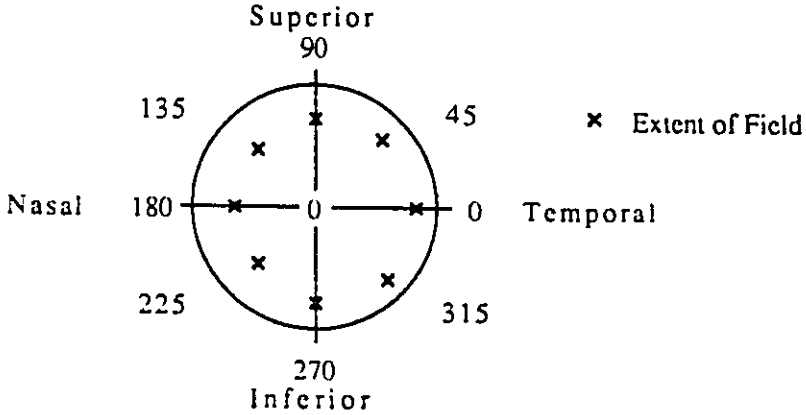


Figure 1. The Goldmann Perimeter and Extent of the Field

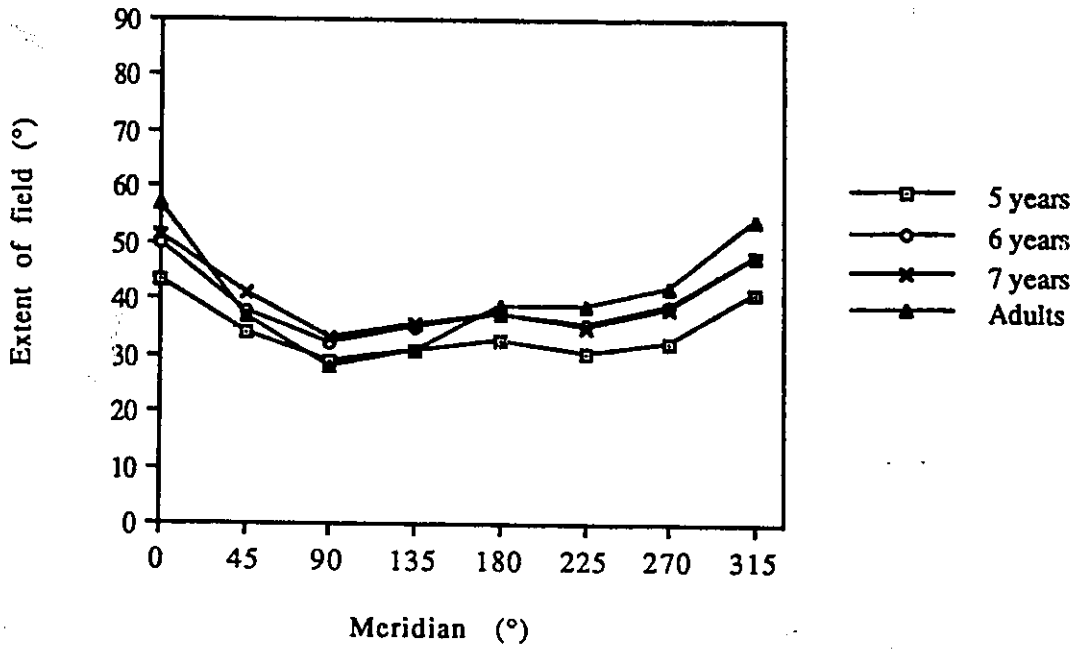


Figure 2. Extent of the Field for Test 1 with Dim Light

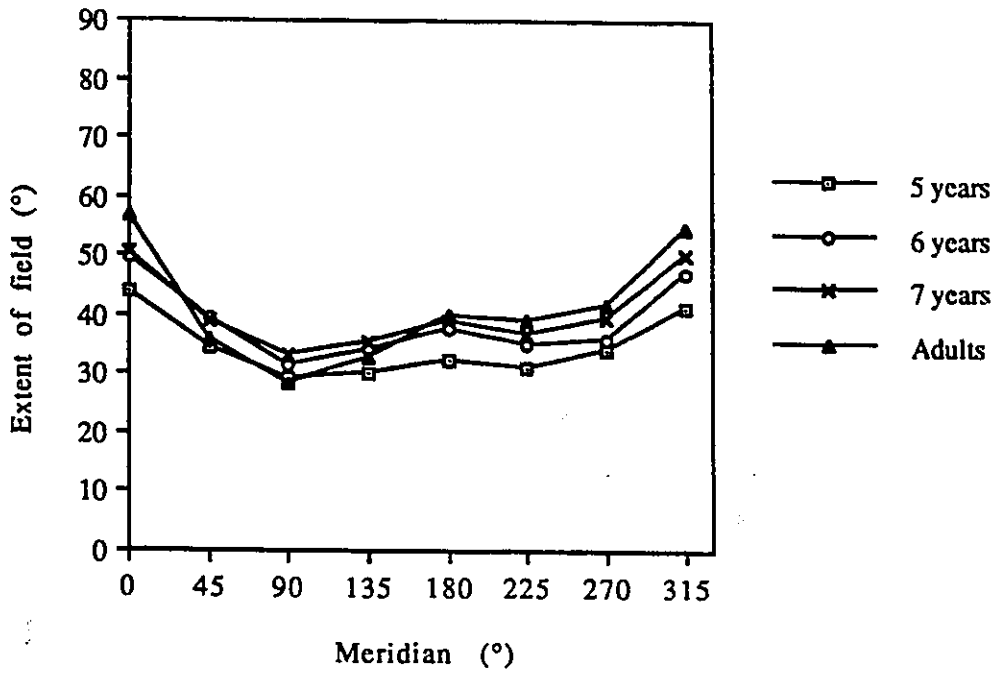


Figure 3. Extent of the Field for Test 2 with Dim Light

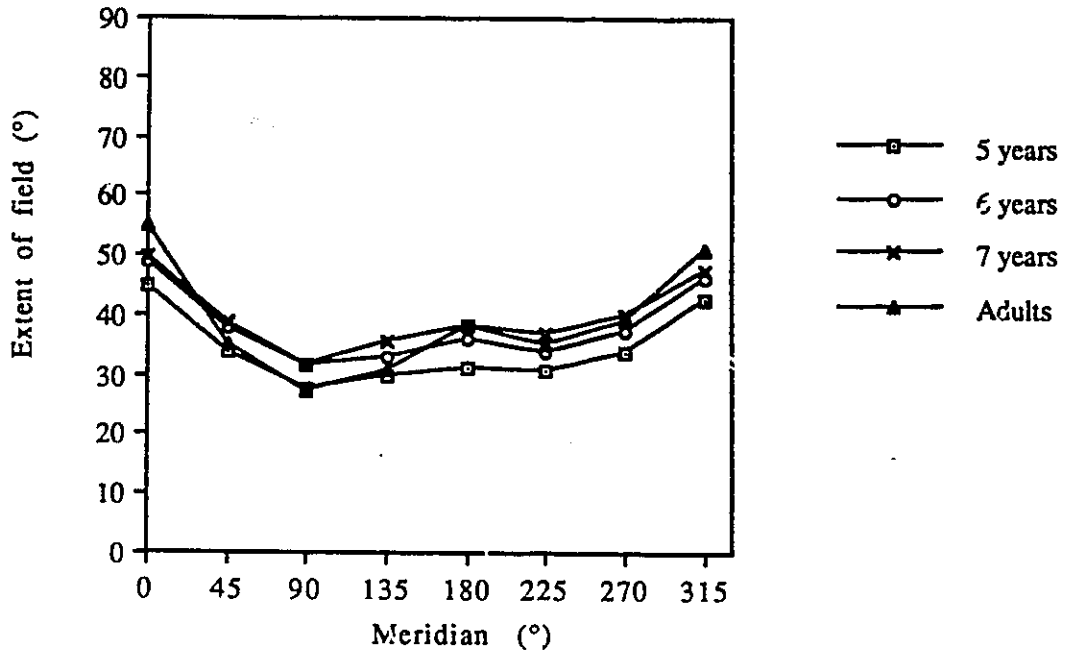


Figure 4. Extent of the Field for Test 3 with Dim Light

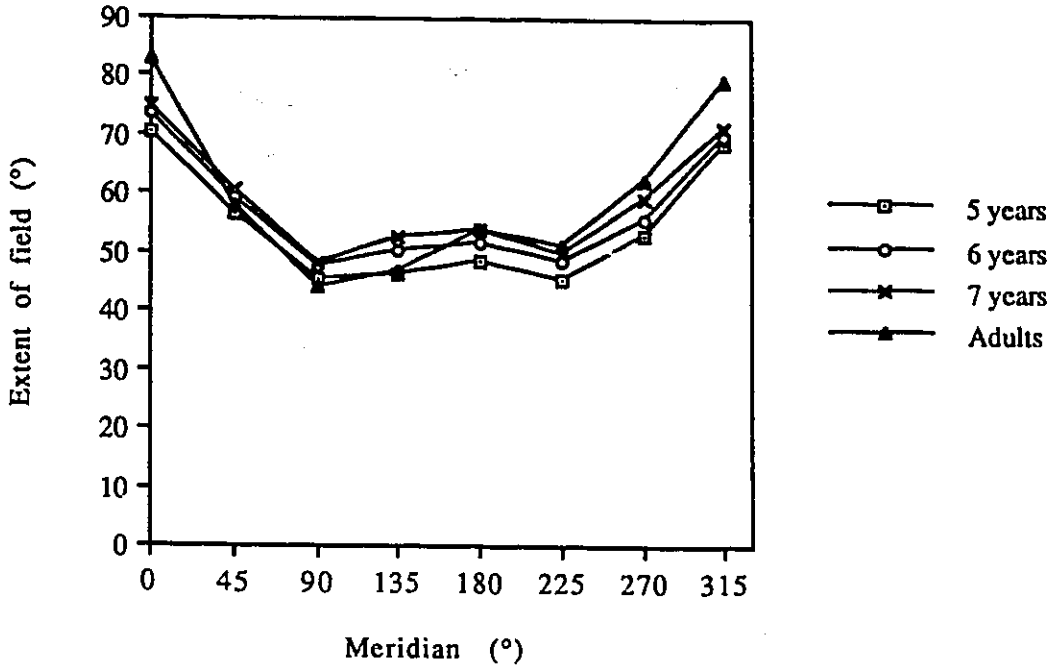


Figure 5. Extent of the Field for Test 1 with Intense Light

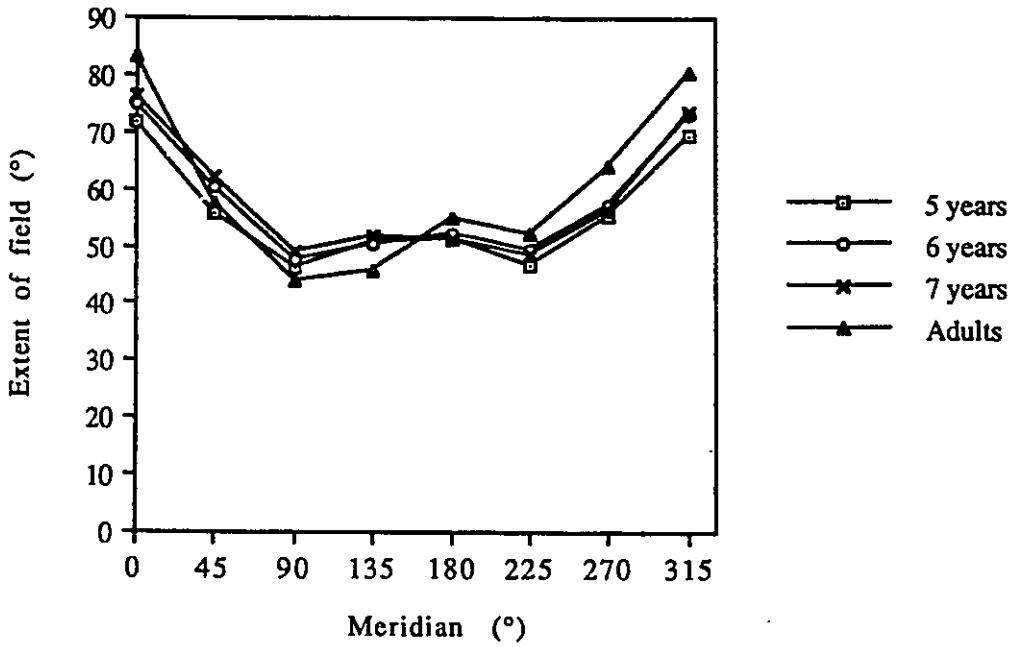


Figure 6. Extent of the Field for Test 2 with Intense Light

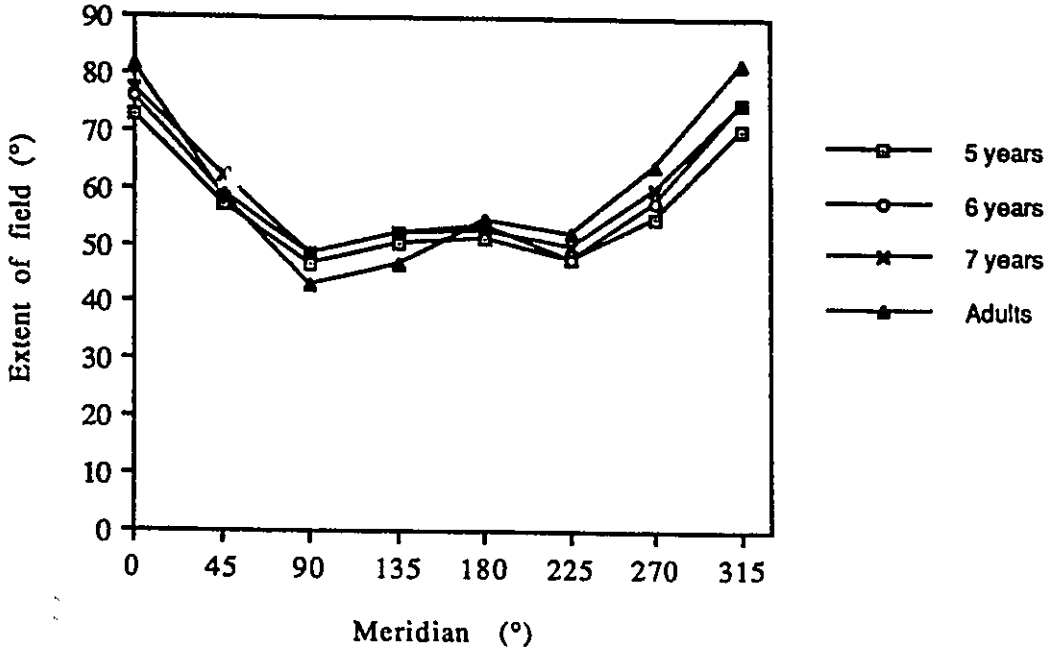


Figure 7. Extent of the Field for Test 3 with Intense Light

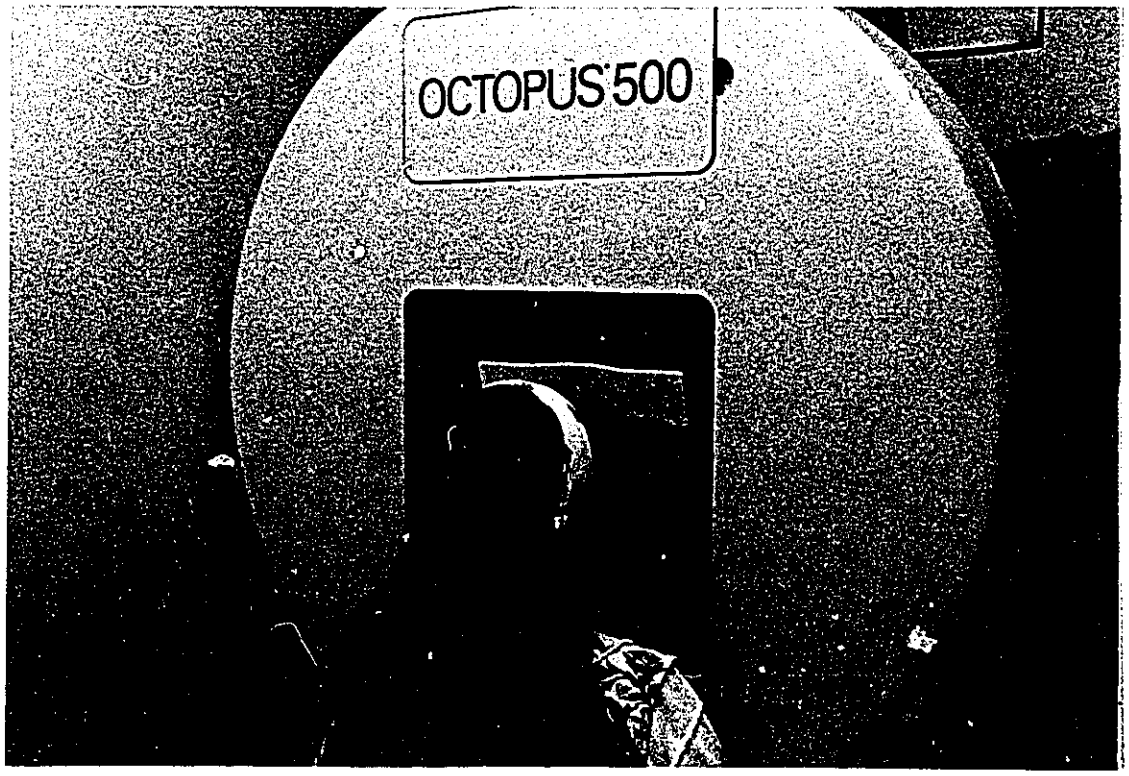


Figure 8. The Octopus Perimeter

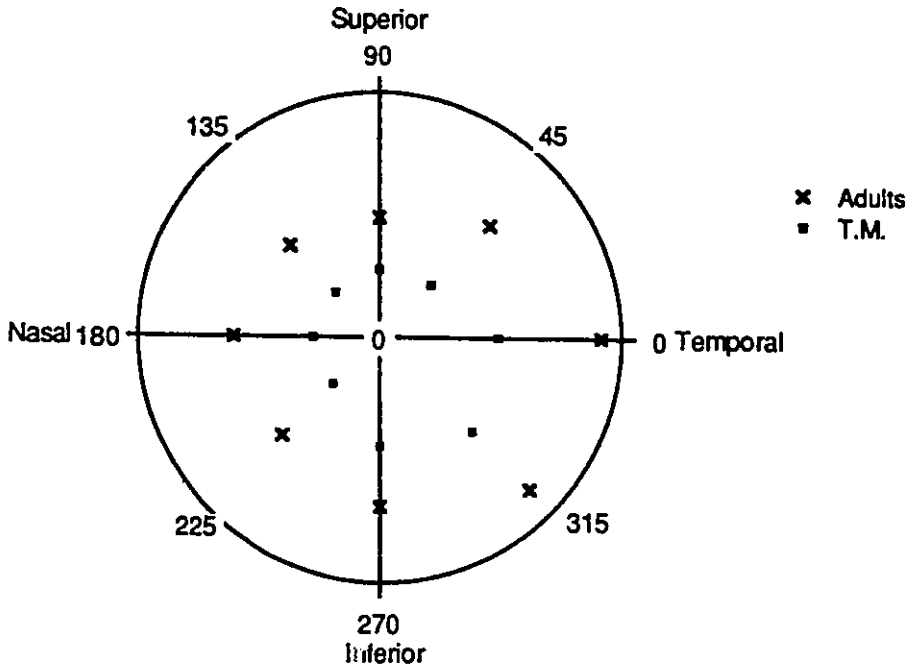


Figure 9. Extent of the Field for T. M. and Normal Adults Assessed with Intense Stimulus

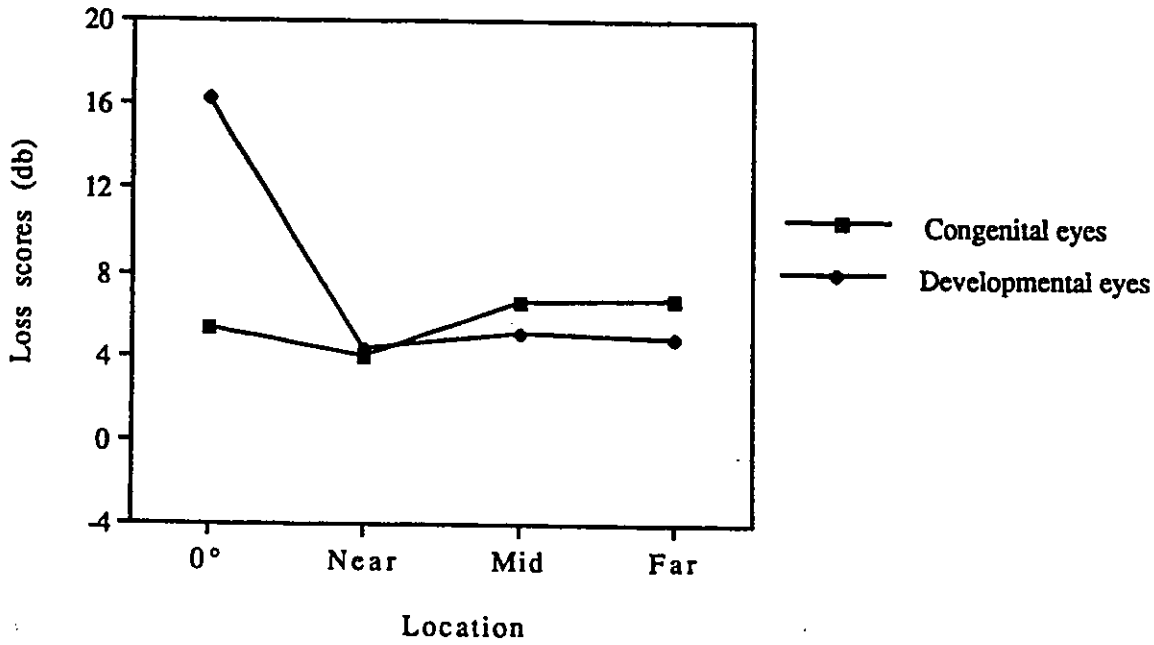


Figure 10. Loss Compared to Normal Subjects for Congenital Versus Developmental Cases

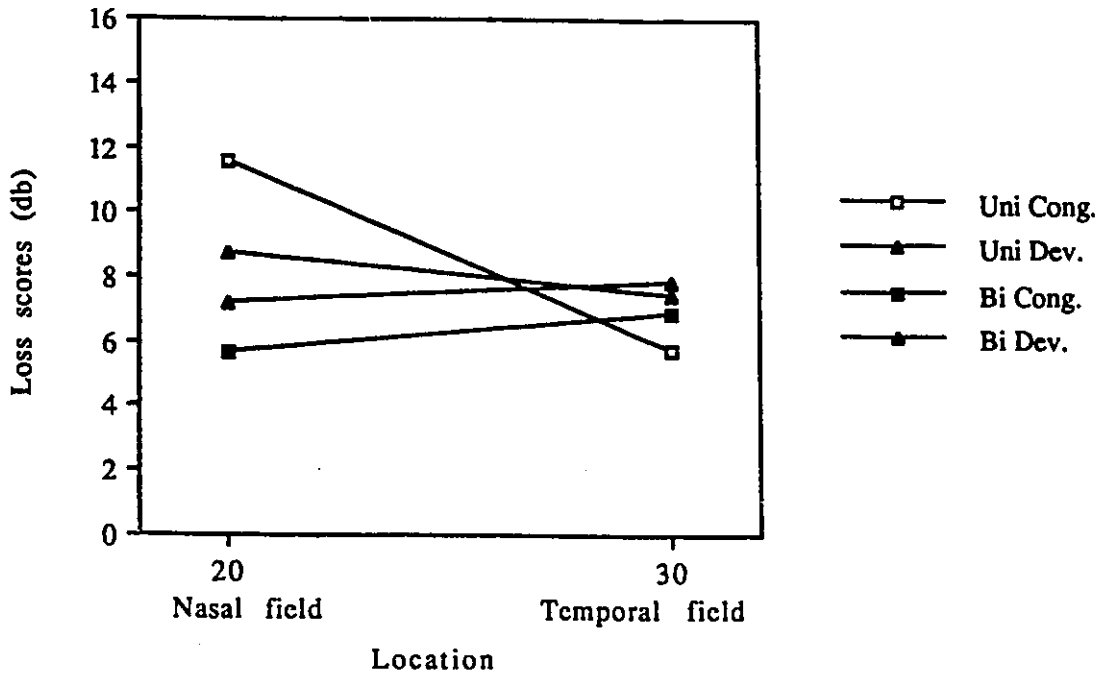


Figure 11. Loss Scores at 20° Nasally and 30° Temporally

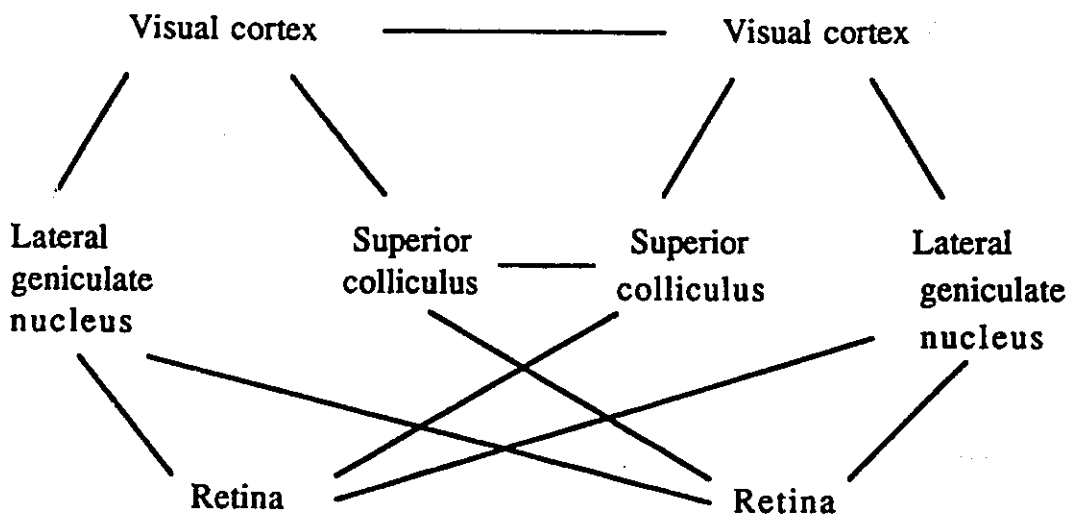


Figure 12. Visual Pathways