The Role of Visual Input in Setting up Spatial Filters in the Human Visual System

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Abstract

At birth the visual system can detect only big objects that are high in contrast. The adult visual system can resolve, with high precision, small objects that are in low contrast. We present a series of studies that provide insight into the role of visual input in the development of the neural architecture underlying human spatial vision. We conclude that the limits to spatial vision during normal development appear to result primarily from retinal immaturities. However, in the absence of early visual experience, the limits to spatial vision appear to be mainly cortical in origin. The early visual input--from the low spatial frequencies to which the infant's vision is known to be limited--sets up the neural architecture that will eventually become fine-tuned to high spatial frequencies.

Introduction

Spatial vision is the aspect of visual processing that enables us to detect and perceive objects, patterns, and textures of different sizes against a background. Two main factors constrain the resolution of our spatial vision. First, the size of objects limits their detectability. That is, the smaller an object is, the more difficult it is to detect. For example, it is easier to detect the full moon in the sky than it is to detect the small adjacent stars. The second factor limiting spatial vision is contrast, the difference in luminance between objects and their background. High contrast objects are easier to detect than low contrast objects. For example, it is easier to detect the full moon that is bright, against the dark sky than it is to detect the new moon that is dark, against an equally dark sky. The minimum amount of contrast necessary to resolve an object varies with the size of the object. The human visual system is composed of overlapping spatial filters tuned to different bands of spatial frequency[1]. The spatial contrast sensitivity function provides a detailed characterization of the limits of spatial vision and its underlying spatial filters. Spatial contrast sensitivity is an index of the minimum amount of contrast required for one to detect sinusoidal gratings ranging from very low to very high spatial frequencies. The spatial contrast sensitivity function of an adult is characterized by a peak of sensitivity at around 3 –5 cycles per degree (cpd), with a sharp decline in contrast sensitivity at higher spatial frequencies and a gradual drop in contrast sensitivity at lower spatial frequencies. A typical function for normal adults is shown by the upper curve in Figure 1. To gain insight into the origins of the spatial filters underlying human spatial vision, the present report reviews a series of studies from our and other labs that investigated the influence of visual input on development of spatial contrast sensitivity. Specifically we document the final development of spatial contrast sensitivity during childhood, we describe the effects of monocular and binocular deprivation during early childhood on subsequent spatial contrast sensitivity, and we examine how the early deprivation affects the tuning of the spatial filters by visual input later during childhood.

Normal Development

Spatial contrast sensitivity is very immature during infancy.[2-5] One-month-olds (the youngest age tested with behavioral techniques) perform poorly and produce a contrast sensitivity function without the low-frequency fall-off typical of adults and older infants.[2,3] The contrast sensitivity function of the 2-month-old has the same overall shape as that of adults. However, they are about 20 times less sensitive than adults up to about 2 - 3 cpd and show no evidence of seeing higher spatial frequencies at even very high contrast. Even at visible spatial frequencies, the 3-month-old's contrast sensitivity is still reduced by over 1.0 log unit relative to that of the adult.[2,3,6-8] At 4 years of age, spatial contrast sensitivity is still approximately 0.5 log units lower than that of adults[8 - 13].

Studies of older children indicate that contrast sensitivity is not adult-like until middle childhood, with estimates of when it reaches adult levels ranging from 6 years to sometime after 15 years of age.[8,11,14-18] In the most comprehensive study, we measured the development of contrast sensitivity in 96 normal children ranging in age from 4-7 years and, for comparison, in 24 normal adults.[16] Participants monocularly viewed vertical sinusoidal gratings of different spatial frequencies and, for each spatial frequency, were asked to indicate when the stimulus just appeared as contrast was increased from subthreshold

values and to indicate when the stimulus first disappeared as contrast was reduced from suprathreshold levels. Spatial contrast sensitivity at each frequency was derived by taking the reciprocal of the geometric mean of the recorded contrast thresholds. For analysis and plotting, the thresholds were log transformed.

The mean spatial contrast sensitivity functions of the five age groups are plotted in Figure 1. The spatial contrast sensitivity of the 4- and 5-year-olds was lower than adult values by approximately a factor of 2 or 0.5 log units. These findings are similar to those reported previously for children of the same age.[8-13] Between 5 and 6 years of age, there was a significant improvement in contrast sensitivity at each spatial frequency; yet sensitivity was still significantly lower than in adults. By age 7, spatial contrast sensitivity attained adult values for all spatial frequencies tested. This finding is in close agreement with that of Bradley and Freeman[11] who found that asymptotic levels were reached by 8 years of age (but see[8,9,12,14]).

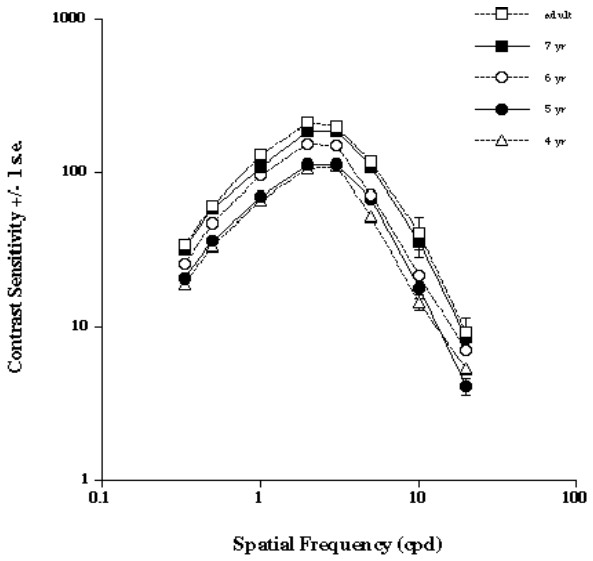

Figure 1. Mean contrast sensitivity (+/-1 S.E.) as a function of spatial frequency for adults and four groups of children. When not shown, standard error bars are smaller than the data points. Reprinted with permission from Ellemberg, D., Lewis, T.L., Liu, C.H., & Maurer, D. The development of spatial and temporal vision during childhood. Vision Research, 1999, 39, 2325-2333,

Neuronal Influences on the Development of Spatial Vision

The normal development of spatial vision is likely limited by slow retinal development, with some additional limitations from immaturities in the geniculostriate pathway. Compared to the adult's fovea, in the newborn's fovea, the length of the outer segments of cones is 16-fold shorter and cone-packing density is 4-fold less.[19] Short outer segments of foveal cones make the cones less efficient in producing isomerization for a given quantum of light. The role of the retina in limiting the functioning of the geniculostriate pathway, at least during early infancy, is also indicated by the finding that cortical spatial contrast sensitivity and acuity, as measured by visually evoked potentials, mature no faster than contrast sensitivity and acuity measured by electroretinograms.[20,21] Further, post-receptoral immaturities likely impose additional limitations on the contrast sensitivity of the young infant.[22] Although considerable foveal maturation occurs between birth and early childhood, measurements from a 45-month-old indicate that the length of the outer segments of foveal cones is still 30-50% shorter than in adults.[19] A model on the front-end limits (optical and receptoral) of spatial vision during development[23,24] predicts that the 45-month-old's shorter cone outer segments should cause a reduction in contrast sensitivity by a factor of 1.1. Assuming that the data from the one 4-year-old retina provided by Yuodelis and Hendrickson[19] lie within the normal range for that age group, the difference between Wilson's[24] predictions for the reduction in contrast sensitivity at age 4 (reduction of a factor of 1.1) and our findings (a reduction of a factor of 2) could then be attributed to post-receptoral immaturities.

Some aspects of the geniculostriate pathway have already matured by 4 years of age, and hence would not contribute to the limitations we observed at that age. However, there is evidence of changes in connectivity and responsivity within the geniculostriate pathway that extend past infancy. Within the primary visual cortex, there is an increase in synaptic density followed by an about 50% decrease that is not complete until 11 years of age.[25-27] These cortical changes may contribute to the increase in spatial contrast sensitivity that occurs during childhood.

The Role of Visual Input

Children treated for congenital cataracts provide the opportunity to examine the role of visual input in driving development of spatial vision. A cataract is an opacity in the lens of the eye which, in the children we selected for study, was sufficiently dense to block visual input to the retina and prevent fixation and following. The cataractous lens was removed surgically and the eye was given an optical correction, usually a contact lens, to provide nearly normal visual input. Studies of children treated for bilateral cataracts allow inferences about the role of visual deprivation per se and hence the role that visual input plays in normal visual development. Comparisons to children treated for unilateral cataract allow additional inferences about the effects of uneven competition between the eyes for cortical connections.

To measure the role of visual input on the development of spatial vision, we measured spatial contrast sensitivity of 13 children treated for bilateral congenital cataract and 15 children treated for unilateral congenital cataract,[28,29] using the same method as in our tests of normal development describe above (for more details see Ellemberg, et al.[16]). Deprivation had lasted from birth until 1.6 - 8.8 months of age (mean = 4.8 months) in bilateral cases and until 1.3 - 10. 4 months of age (mean = 5.0 months) in unilateral

cases. Patients were at least 4 years of age at the time of the first test (range 4 to 28 years) and their results were compared to those of age-matched normals.

Figure 2 shows the results for the better eye (determined by alignment history and Snellen acuity) of a typical binocularly deprived patient. The patient suffered 3.5 months of deprivation from birth and the open circles represent the results for the first test, when he was 5.5 years of age. Panel A shows the contrast sensitivity function and panel B shows the losses relative to an age-matched normal subject where 0 means that performance was normal and increasingly negative values represent increasingly larger deficits. For this patient and for all patients tested, deficits increased with spatial frequency, exceeding half a log unit at higher spatial frequencies. This pattern of results is similar to that reported in other studies.[30-32] Within our small sample, there was no effect of the duration of binocular deprivation on the size of the deficit in contrast sensitivity at 5 c/deg, even though the duration of deprivation had varied from 1-8 months. This result resembles a similar finding in binocularly deprived monkeys[33] and previous findings that the duration of binocular deprivation from cataracts does not affect the size of the ultimate deficit in acuity.[30,34] Thus, binocular deprivation for as little as the first month of life--a period during which normal infants can see only low spatial frequencies--prevents the later development of normal sensitivity to high spatial frequencies.

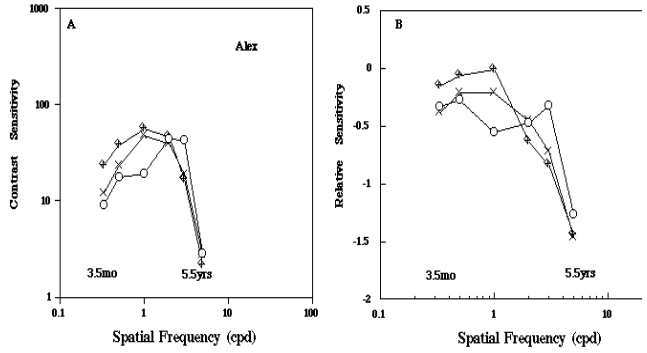

Figure 2. Contrast sensitivity (Panel A) and relative loss (Panel B) of a typical patient who suffered 3.5 months of binocular deprivation at birth and was tested at 5.5 years of age. Open circles represent the data for the first test, Xs represent the data for the followup after 1 year and filled diamonds represent the data followup after 2 years.

Figure 3 shows the results for the treated eye of a monocularly deprived patient who had suffered 6.1 months of deprivation from birth and then had patched the nondeprived eye a mean of 2.5 hours per day throughout early childhood in order to reduce uneven competition between the eyes for cortical connections. As in Figure 2, the open circles represent the results for the first test, when he was 5.8 years of age. Panel A shows the contrast sensitivity function and panel B shows the losses relative to an age-matched normal. The pattern of results is similar to that of our other patients who suffered from monocular deprivation except that the deficits are worse in children who had later treatment and little patching of the nondeprived eye, a finding that agrees with previous studies of monocularly deprived patients.[30,35,36] Among the poor patchers, the deficits exceed 1.0 log unit and are larger than any deficit we observed in children treated for bilateral congenital cataracts.[29]

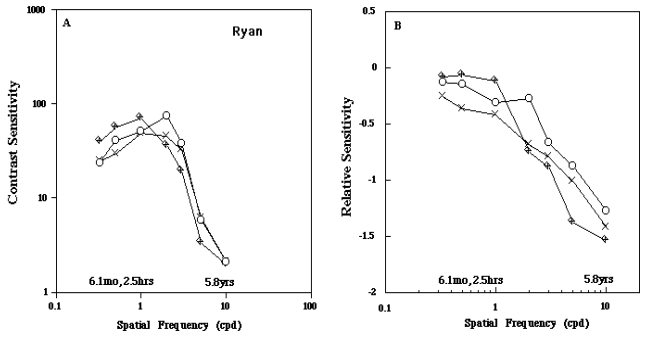

Figure 3. Contrast sensitivity (Panel A) and relative loss (Panel B) of a typical patient who suffered 6.1 months of monocular deprivation at birth, patched the nondeprived eye an average of 2.5 hrs per day, and was tested at 5.8 years of age. Open circles represent the data for the first test, Xs represent the data for the followup after 1 year and filled diamonds represent the data followup after 2 years.

A comparison of our results from patients to those from normal infants indicates that early deprivation did not arrest development. The cataracts had always been diagnosed on the first eye exam and always by 6 months of age. Yet on every part of the spatial contrast sensitivity curves, all of the patients performed better than normal 6-month-olds.[7] Thus, early visual deprivation does not prevent some subsequent development of spatial vision -- at least after deprivation lasting no longer than 10 months and after periods of recovery of more than 4 years, as was true for our patients.

Neuronal Mechanisms of Visual Deprivation

Deficits in spatial vision after deprivation are likely caused at the level of the striate cortex and beyond because visual deprivation in monkeys causes no change in photoreceptor topography,[37] electroretinogram,[38] or the physiological properties of LGN neurons.[39,40] In contrast, at the level of the striate cortex, there is a nearly 4-fold reduction in binocularly driven cells.[38,41] Further, striate cortex neurons respond more sluggishly and have marked reductions in both their spatial resolution and contrast sensitivity.[42,43] Their receptive fields are also abnormally large and poorly tuned.

Visual Recovery After Deprivation

The behavioural findings presented above indicate that early visual deprivation, whether binocular or monocular, compromises spatial vision at mid- to high spatial

frequencies and that the deficits are evident as early as 4 years of age. Secondly, in normal children, spatial vision continues to improve between 4 and 7 years of age. To determine whether the spatial filters of the deprived visual system also profit from visual input after age 4, we retested the contrast sensitivity of nine bilateral and eight unilateral patients one and/or two years later. Like normals, patients who were at least 7 years old at the first test (four binocular cases and three monocular cases) showed no change in sensitivity over the next 1 to 2 years. Typical results for patients less than 7 years old at the time of the first test are shown in Figures 2 and 3 where Xs represent the data for the followup after 1 year and filled diamonds represent the data followup after 2 years. As in these examples, most binocularly and monocularly deprived patients who were between 4 and 7 years old at the time of the initial test (n = 5 per group) showed improvements in sensitivity at low spatial frequencies, with the amount of improvement at least as great as in normals. Thus, after early deprivation, the low spatial frequency filters appear able to profit normally from visual input until 7 years of age.

The pattern of results was very different at high spatial frequencies for these 10 patients who were less than 7 years old at the time of the first test. At high spatial frequencies, the contrast threshold of children treated for bilateral congenital cataracts remained the same or improved, but less than normal. For 80% of the children treated for unilateral cataract, the contrast threshold actually got worse. Figures 2 and 3 show examples for typical patients. Together, the results indicate that patients treated for congenital cataract had reached an asymptote in sensitivity to high spatial frequencies by age 4. The consequence is that the patients' deficits at high spatial frequencies increased after age 4. In other words, visual input during the first few months after birth is necessary to set up the cortical neural architecture that will become fine-tuned to resolve high spatial frequencies. The fact that most of the patients treated for unilateral congenital cataract actually *lost* sensitivity to higher spatial frequencies when retested 1 and/or 2 years later indicates that a history of uneven competition between the eyes may induce the later loss of acuity. This loss presumably reflects the loss of functional cortical connections that had been formed in the initial recovery from deprivation. Such losses of cortical connections may be akin to those implicated in monkeys in which the visual resolution of cells in primary visual cortex driven by a formerly deprived eye is sometimes less than that of normal newborn monkeys.[44]

Conclusions

The limits to spatial vision during normal development appear to be mainly peripheral and likely result from retinal immaturities as well as immature processing of photoreceptor signals by the retino-geniculate pathway. In contrast, the limits of spatial vision following early visual deprivation appear to be mainly central and likely result from a failure of the striate cortex to correctly process the neuronal signals it receives from the retino-geniculate pathway. After visual deprivation, visual development is not arrested. Rather, lower spatial frequency filters appear to profit from normal visual input and develop normally. However, over the same age range, higher spatial frequency filters profit less from visual input and are affected adversely by uneven competition between the deprived and nondeprived eyes. Taken together these comparisons suggest that in normal infants, the early input--from the low spatial frequencies to which the infant's vision is known to be limited--sets up the neural architecture that will eventually become fine-tuned to high spatial frequencies.

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Biography

Dave Ellemberg received his B.A. degree in Psychology from McGill University in Montreal Québec in 1996, a M.Sc. degree in Psychology from McMaster University in Hamilton Ontario in 1997 and is completing a Ph.D. in Neuropsychology at l'Université de Montréal. His work has primarily focused on the study of human visual perception, including spatial and temporal vision and motion perception. He is a member of the Association for Research in Vision and Ophthalmology, the Society for Neuroscience, and the IS&T. Dave Ellemberg is supported by a fellowship from the Medical Research Council of Canada (E. A. Baker Doctoral Research Award).