

18 | Biological limits on visual development in primates

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The goal of this chapter is to explore the factors that limit the development of spatial visual performance in nonhuman primates. We first consider the factors that govern the development of normal visual performance, with a particular view to exploring those factors that are relatively inaccessible in the human subjects whose vision is the subject of the greater part of this volume. Second, we examine the limits on performance when abnormal visual development is used to induce experimental amblyopia in nonhuman primates. In each part, we first summarize the behavioral data that document the similarities between the development of vision in monkeys and humans, because it is these similarities that justify the use of the monkey model for human visual development. We then present neuroanatomical and neurophysiological evidence that allows us to deduce (or surmise) the particular sites in the visual system whose function changes during development, and the nature of those changes. We conclude that in normal development a great part of the change in visual performance from infancy to adulthood is attributable to improvements in the capacity of peripheral visual mechanisms, though perhaps not the photoreceptors themselves. Abnormal development produced by visual deprivation, anisometropia, or strabismus, however, appears to result in changes in the primary visual cortex, which ceases to be a faithful relay of visual signals from the periphery.

Many of the questions that arise when considering visual development in monkeys are, of course, the same as those that apply in humans. This volume contains thorough reviews of many of these topics, particularly the chapters by Banks and Crowell, Birch, Braddick, Hainline, Held, and Wilson. As we shall see, much interest centers on the role of retinal maturation, reviewed here by Hendrickson, and of central nervous system development.

DEVELOPMENT OF SPATIAL VISION

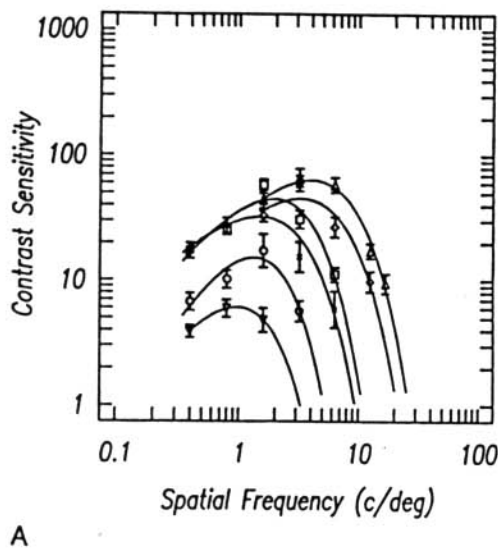
There are two distinct kinds of developmental change that contribute to changes in visual performance during

development, which we can term changes of *scale* and changes of *sensitivity*. By changes of scale we mean changes in the spatial filtering or integration properties of elements of the visual system. The visual system of infants operates on a much coarser spatial scale than that of adults; and it may be that if allowance is made for this point, the infant's visual capacity is otherwise more or less adult-like. We use the term sensitivity to describe the efficiency or accuracy with which the visual system can process targets adjusted for changes in spatial scale. Thus, the sensitivity of an immature observer cannot be properly estimated if performance is measured with targets selected for adults—it is often necessary to adapt the characteristics of the display to the scale of the system under study.

Changes in both scale and sensitivity can be simply documented by measuring the development of the *spatial contrast sensitivity function*, which describes the performance of the visual system in terms of the minimal detectable contrast for sinusoidal grating patterns of varying spatial frequency (Campbell and Green, 1965). Figure 18-1 shows the development of contrast sensitivity for two individual monkey infants (Boothe et al., 1988). At each age, these functions show the usual bandpass shape, with a range of intermediate spatial frequencies being detectable at lower contrasts than at either lower or higher frequencies. Contrast sensitivity functions from monkeys at the earliest test ages were considerably reduced in overall amplitude as well as in the range of resolvable spatial frequencies. Examination of the longitudinal development of contrast sensitivity in infant monkeys reveals that the observed changes in contrast sensitivity are the result of the function shifting upward in sensitivity and rightward toward higher spatial frequencies concurrently. A subsequent analysis of these data shows that the full developmental change in spatial contrast sensitivity can be accounted for by the hypothesis that the function does not change shape, but undergoes changes in *only* scale and sensitivity during development (Movshon and Kiorpes, 1988). Although behavioral contrast sensitivity data from human infants are sparse, human contrast sensitivity seems also to de-

velop according to the same rules as in monkeys, albeit more slowly (Banks and Salapatek, 1981; Movshon and Kiorpes, 1988). An analysis of human development using evoked potential measurements confirmed this impression (Norcia et al., 1990).

More traditional measures of visual acuity, such as spatial resolution, are also captured by the contrast sensitivity function: The spatial frequency at which contrast sensitivity falls to 1 corresponds to the resolution limit for grating targets. It can readily be appreciated from Figure 18-1 that the development of spatial resolution depends jointly on changes in scale and sensitivity. Nonetheless, the value of spatial resolution succinctly captures an important limit on visual performance; because it is more easily measured than the entire contrast sensitivity function, it has been widely studied. The development of resolution shows a similar developmental profile in all species examined. Spatial resolution in newborns is at least 10-fold poorer than in adults; it improves rapidly over the early weeks or months of life, then continues to develop at a slower rate to adult levels. Adult levels are reached by the end of the first year in monkeys (Boothe et al., 1988) and between 3 and 5 years in humans (Mayer and Dobson, 1982; Birch et al., 1983). Comparison of the time courses for the development of human and monkey spatial resolution reveals several similarities. Spatial resolution is comparable in newborn human and monkey infants, both measuring between 1 and 2 cycles/degree (cpd). Adult levels of resolution are in the range of 30–50 cpd. The time courses for the development of resolution in monkeys and humans superimpose fairly well if human age is plotted in postnatal months and monkey age in postnatal weeks (Teller and Boothe, 1979).



The open circles in Figure 18-2 show the development of grating resolution in seven longitudinally tested infant monkeys, as measured behaviorally using a combination of preferential-looking and operant techniques (Kiorpes and Movshon, 1989a). Development proceeds smoothly and regularly until adult levels are approached after the age of 1 year.

The development of various other visual functions has been studied in human and monkey infants. Direct comparisons are possible only for the development of vernier acuity, which occurs during roughly the same period as the development of spatial resolution but which takes place at a substantially higher rate (Shimojo et al., 1984; Kiorpes and Movshon, 1989a).

LIMITS ON NORMAL VISUAL DEVELOPMENT

Analysis of the factors that limit the development of visual performance has attracted considerable attention. In humans, it is of course not possible to make physiological measurements of the properties of individual visual mechanisms during development, whereas such measurements can be, and have been, made in monkeys.

Physiological Optics

The first issue is whether development is limited by the mechanisms that lead to the formation of the clear, focused, stable retinal image needed for good vision. Much of the best evidence here comes from humans and is reviewed elsewhere in this volume (see Chapters 4, 6, 30, 32).

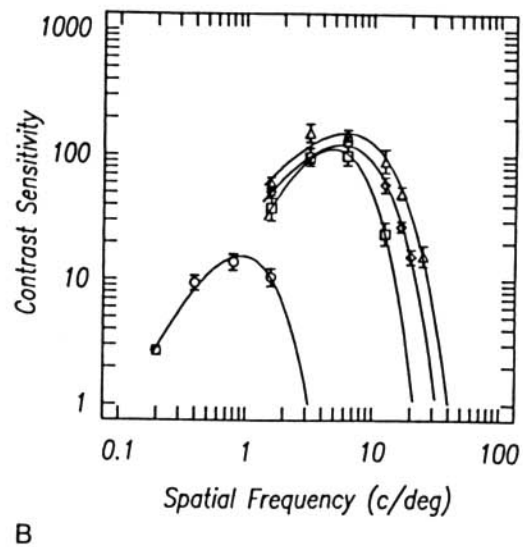


FIG. 18-1. Development of spatial contrast sensitivity in two macaque monkeys. Note the regular translation of the contrast sensitivity func-

tion upward and to the right during the course of development. (From Boothe et al., 1988. By permission.)

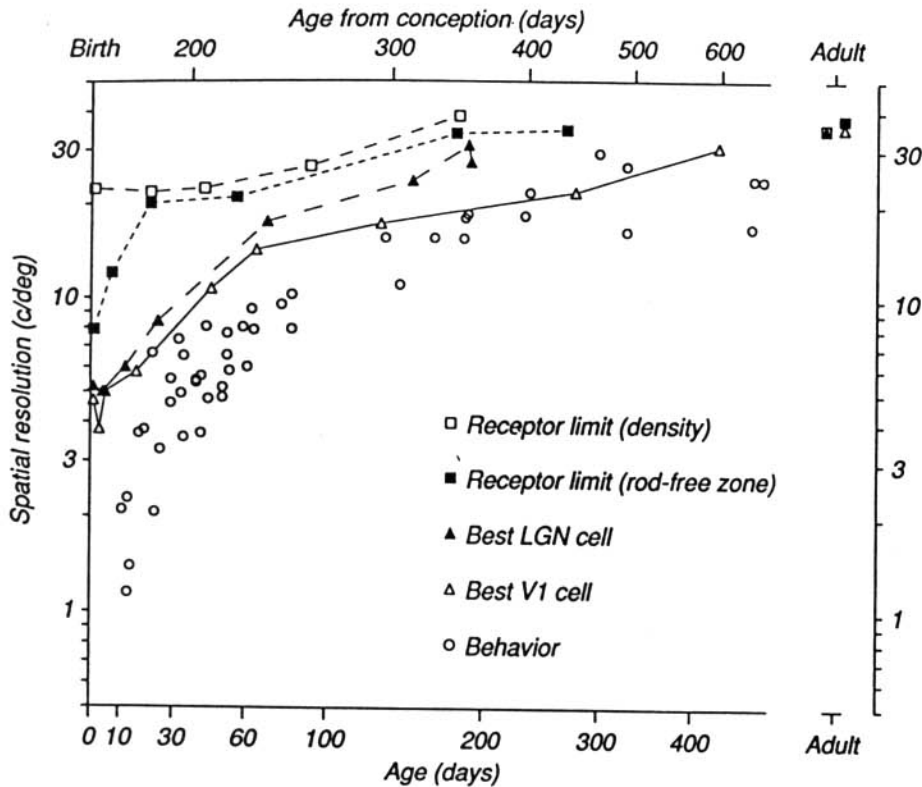


FIG. 18-2. Time course of the development of visual resolution in macaque monkeys, combining data on different functions from several sources. \square = Nyquist frequency for the retinal cone mosaic, calculated from the retinal density data of Packer et al. (1990). \blacksquare = Nyquist frequency for the cone mosaic, calculated by Jacobs and Blakemore (1988) from measurements of Hendrickson and Kupfer (1976) of the size of the rod-free zone. Both Nyquist calculations assume that the central cone mosaic is a perfect hexagonal lattice; the Nyquist frequency is calculated from the cone spacings in such a lattice. Growth of the eye and associated retinal magnification is assumed to follow the function suggested by Jacobs and Blakemore (1988). \blacktriangle = visual resolution of the best LGN neuron recorded from suitable populations in normally reared monkeys (Blakemore and Vital-Durand, 1986a). \triangle = visual resolution of the best neuron recorded from the striate cortex in normally reared monkeys of different ages (Blakemore and Vital-Durand, 1983a, b; Blakemore, 1990). \circ = behavioral measurements of visual resolution for seven macaque monkeys studied longitudinally (Kiorpes and Movshon, 1989a). The abscissa indicates the approximate postconceptional age at the top and conventionally at the bottom.

The quality of the retinal image in the infant monkey eye is remarkably good and quickly approaches adult levels (Williams and Boothe, 1981). Quantitatively, the developmental improvement in contrast modulation transfer is negligible over the range of frequencies visible to the infant monkey.

An aspect of optical development that is quantitatively important when considering the development of spatial properties in the visual system is the change in retinal image magnification that accompanies the growth of the eye. For example, the axial length of the monkey eye increases by about two-thirds between birth and adulthood (Blakemore and Vital-Durand, 1986a). This has no direct effect on the quality of the retinal image, but it alters the effect of that image on the subsequent neural elements of the visual system. All other things being equal, visual resolution should grow by about

two-thirds as the eye grows, simply because the size of the retinal image increases while the size of the neural elements used to analyze the image does not.

Although young infants often demonstrate the ability to accommodate correctly to a range of target distances, they do so less consistently than older infants. A similar trend is apparent in infant monkeys (Howland et al., 1982). Accommodative accuracy is poor in infant monkeys prior to about 5 postnatal weeks. Infants older than 5 weeks show consistently high accommodative accuracy over a range of target distances from 20 to 100 cm, corresponding to a 1–5 diopter (D) range of accommodative power. It appears that accommodative control in monkey infants is no worse than is needed to maintain an image of good apparent quality (cf. Green et al., 1980).

Data on the quality of oculomotor control in infant

monkeys are sparse but consistent with the available data on humans (Shupert and Fuchs, 1986). As in the case of accommodation, oculomotor performance is likely to be sufficiently good at all ages to impose no limit to visual performance.

In summary, it appears that neither the quality of the optics of the infant eye, the ability of infants to accommodate, nor their ability to fixate and track visual targets accurately imposes significant limits on performance. Indeed it appears that these physiological mechanisms develop at a pace that just comfortably *prevents* them from becoming an impediment to clear vision (Kiorpes and Movshon, 1990).

Photoreceptors

The next question is whether the retinal image, once formed, can be adequately captured and encoded by the photoreceptors (see Chapter 17 for a detailed review of retinal development). Yuodelis and Hendrickson (1986) provided evidence of two significant immaturities in the retinas of human infants. First, cones in the central retina of infants are abnormally shaped in a way that probably significantly impedes their light-gathering power; second, the density of cones in the infant's central retina is low, resulting in a limit to the resolution with which the retinal image can be adequately sampled. Based on an analysis of these data using an "ideal detector" model (Geisler, 1984), Banks and Bennett (1988) suggested that spatial visual development during the early postnatal months in human infants may largely be due to the maturation of foveal cone position and morphology (see Chapter 6 for a more recent recapitulation of this analysis). Their analysis readily accounts for a good part of the improvement in sensitivity seen during development, and modeling of the effects of cone migration by Wilson (see Chapter 32) suggests that a portion of the change in spatial scale can be attributed to retinal changes as well.

Development of the monkey retina has been studied by Hendrickson and Kupfer (1976) and Packer et al. (1990). Qualitatively, the changes in retinal organization are similar to those seen in humans, but quantitatively they seem to be somewhat less pronounced. The upper two curves in Figure 18-2 plot the development of the theoretical resolution limit for the cones of the central retina in monkeys, assuming that the limit of vision corresponds to the limit of retinal sampling (in theory, undersampled "aliased" images of frequencies higher than the resolution limit could be seen because the capacity of the optics exceeds the capacity of the receptors). The uppermost curve (open squares) is calculated from the cone densities measured by Packer et al. (1990); the subadjacent curve (filled squares) is based on densities calculated by Jacobs and Blakemore (1988)

from the size of the central rod-free zone reported by Hendrickson and Kupfer (1976). The change in the size of the rod-free zone should predict density under the assumption that all the cones within that zone remain within it as they migrate across the retina; this assumption seems to be consistent with the values reported by Packer et al. (1990). The wide variation between the two curves at ages less than 20 days seems to reflect real differences in the maturity of different monkeys at birth and makes it difficult to draw straightforward conclusions about the limits imposed by the receptors. It appears, however, that beyond the age of 20 days the resolution of the retinal mosaic significantly exceeds the behavioral resolution (open circles); for example, behavioral resolution between 20 and 30 days does not exceed 4–6 cpd, yet retinal resolution is four times greater.

The other important component of photoreceptor development is the change in sensitivity attributable to increased light capture. As noted above, increasing sensitivity ought to increase spatial resolution simply by raising the contrast sensitivity function and exposing more of its "shoulder" above the limiting value of 1. We are aware of no calculations comparable to those of Banks and Bennett that are based on Packer et al.'s monkey data, but the relatively modest postnatal maturation of the outer segment morphology of monkey cones suggests that the contribution of this change to monkey visual development is small.

Neural Mechanisms

In monkeys it is possible to study the spatial resolution of the neural elements that intervene between phototransduction and behavior. It is technically difficult to record retinal activity in infant monkeys, and we are not aware of physiological data on monkey retinal development. Ample data exist, however, for more central neural processing. The two last curves (filled and open triangles) in Figure 18-2 show data of this kind collected by Blakemore and Vital-Durand (1983a,b, 1986a; Blakemore, 1990), reporting the highest spatial resolution encountered in single-unit recordings from the lateral geniculate nucleus (LGN) and primary visual cortex of normally reared monkeys of various ages. It is evident from these data that the visual resolution of neurons in these structures barely, but reliably, exceeds behavioral resolution throughout most of development. Like behavioral resolution, it appears that the resolution of central neurons does not begin to approach the photoreceptor sampling limit until about 6 months of age. Although not pictured in Figure 18-2, Blakemore and Vital-Durand (1983a, 1986a; Blakemore and Hawken, 1985) also reported that the responsiveness and sensitivity of LGN and V1 neurons in young monkeys is

reduced in comparison with those in adults; the improvement in neuronal responsiveness takes place over the period during which behavioral sensitivity also improves.

It may be that the performance of LGN neurons, at least, is better in young monkeys than reported by Blakemore and Vital-Durand (1986a). Blakemore and Hawken (1985, unpublished observations) studied the resolution and contrast sensitivity of neurons in infant monkey LGN, and showed that even near the time of birth resolution may approach 10 cpd, and contrast sensitivity (defined by a statistical criterion) may exceed 10. It is possible that a more quantitative examination of cortical unit properties would reveal similar subtle improvements over the data reproduced in Figure 18-2. It appears unlikely, however, that new measurements of the resolution of central neurons would alter the conclusion that they do not approach the sampling limit set by the retinal mosaic.

In summary, it appears that peripheral optical and receptor mechanisms account for only a modest part of the improvement in visual resolution seen during development in monkeys. It seems that the greatest part of the developmental change is captured by considering the fidelity with which LGN neurons approach the retinal limits. Two important unexplored processing stages could account for this improvement. First, it might be that signal transmission within the retina is impeded early in development, and that the retina's functional properties develop importantly over the first 2 months of life. Alternatively, it might be that retinal processing is normal in young monkeys, but that the relay of signals from retinal ganglion cells to the LGN is of low fidelity. This question could be resolved with data on the function of retinal ganglion cells, but no data are presently available for monkeys (but see Tootle and Friedlander (1989) for data on kittens). It is possible to conclude that the development of resolution in visual cortex and the development of behavioral resolution are only slightly worse than the signals presented by the LGN permit.

ENVIRONMENTAL INFLUENCES ON VISUAL DEVELOPMENT

The development of the visual system is not a simple maturation; rather, it is susceptible to modification by the visual environment within which the system grows. Such modification can be produced within an early *sensitive period* whose duration varies from species to species and possibly from function to function within a species (Movshon and Van Sluyters, 1981; Harwerth et al., 1986). In humans, the sensitive period lasts between 2 and 5 years from birth; in monkeys, it is complete within a year, perhaps less.

During the sensitive period, deprivation of form or light experience has a globally disruptive effect on visual performance; various aspects of vision can be disrupted in a more specific fashion by arranging partial deprivation of specific kinds (for reviews see Movshon and Van Sluyters, 1981; Movshon and Kiorpes, 1990). Vivid evidence of the visual consequences of this deprivation is shown in Figure 18-3, which shows the effect of monocular eye closure for varying periods on the spatial contrast sensitivity of four monkeys (Harwerth et al., 1983). It is evident that the deprivation (which in case D was only of 2 weeks' duration) almost totally abolished the visual resolution and sensitivity in the deprived eye; in fact, the "contrast sensitivity functions" measured in the deprived eye may reflect nothing more than the monkey's ability to discriminate the brightness of diffuse lights when viewed through that eye.

The consequences of less radical forms of visual deprivation on visual development can also be substantial. We have explored the consequences of unilateral blur and strabismus—two forms of abnormal early visual experience designed to mimic the anisometropia and strabismus that often lead to amblyopia in human subjects. We created early unilateral blur using one of two methods: (1) continuous instillation of atropine in one eye for the first 6 months of life—the resulting dilation of the pupil and paralysis of accommodation substantially degrade the retinal image (Kiorpes et al., 1987); and (2) extended-wear soft contact lenses of -10 D

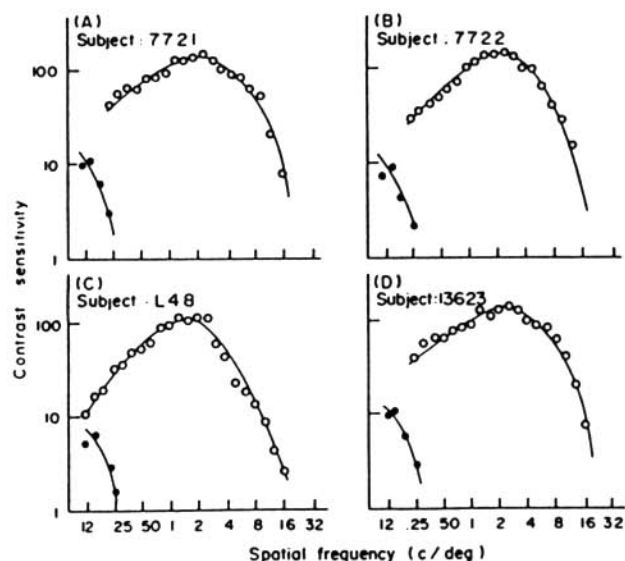


FIG. 18-3. Measurements of the spatial contrast sensitivity of four monkeys deprived of vision in one eye from birth to the ages of 22 months (A), 18 months (B), 8 weeks (C), and 2 weeks (D). At the end of the deprivation the lids were opened and visual experience permitted until the animals were fully mature. ● = data obtained using the deprived eye; ○ = data obtained using the nondeprived eye. (From Harwerth et al., 1989. By permission.)

(Kiorpes and Movshon, 1989b). We have also explored the effect of early strabismus, produced by either surgical alteration of the extraocular muscles (Kiorpes and Boothe, 1980; Kiorpes et al., 1989) or injection of a neurotoxin into a muscle (Kiorpes and Movshon, 1989b). Behaviorally, strabismus and anisometropia produce similar effects: loss of visual resolution and contrast sensitivity in the treated eye compared to its normal fellow. The loss, however, is always much more modest than that produced by total deprivation. Figure 18-4 shows behavioral contrast sensitivity data for two monkeys that developed amblyopia as the result of abnormal experience. Figure 18-4A shows data from an animal raised with blurred vision induced by contact lenses; Figure 18-4B shows data from an animal made strabismic by injection of botulinum A neurotoxin into the lateral rectus muscle. In both cases, the treated eye shows reliable losses of resolution and sensitivity compared to the fellow eye, but it retains a sizable fraction of its sensitivity and resolving power.

These results and others from other laboratories show that the visual performance measures obtained in animal models of deprivation are in substantial agreement with those from amblyopic humans (see Chapter 24 for a review). As in the course of normal development, it is therefore reasonable to seek explanations for the neural foundations of human visual function in neurobiological data from an animal model.

LIMITS ON VISUAL PERFORMANCE AFTER VISUAL DEPRIVATION

It is possible to draw an unambiguous conclusion from the available data on the effects of visual deprivation on central visual processing in primates. This conclusion—that the only substantial effects of visual deprivation are expressed in the visual cortex—is wholly different from our conclusion that the cortex does *not* limit normal visual development.

Physiological Optics

It is relatively easy to rule out the peripheral factors that influence normal development because it seems that the optical quality of the eye and the maturation of the morphology and distribution of photoreceptors (Hendrickson and Boothe, 1976) are unaffected by any of the forms of visual deprivation discussed here. Visual deprivation, either complete or partial, can alter the normal development of the refractive state of the eye (Wiesel and Raviola, 1977; Kiorpes et al., 1987). This change, however, has no practical consequence for vision once corrected with suitable lenses.

Neural Mechanisms

Figure 18-5 presents four sets of data obtained by Blakemore and Vital-Durand (1983a, 1986a,b; Blakemore,

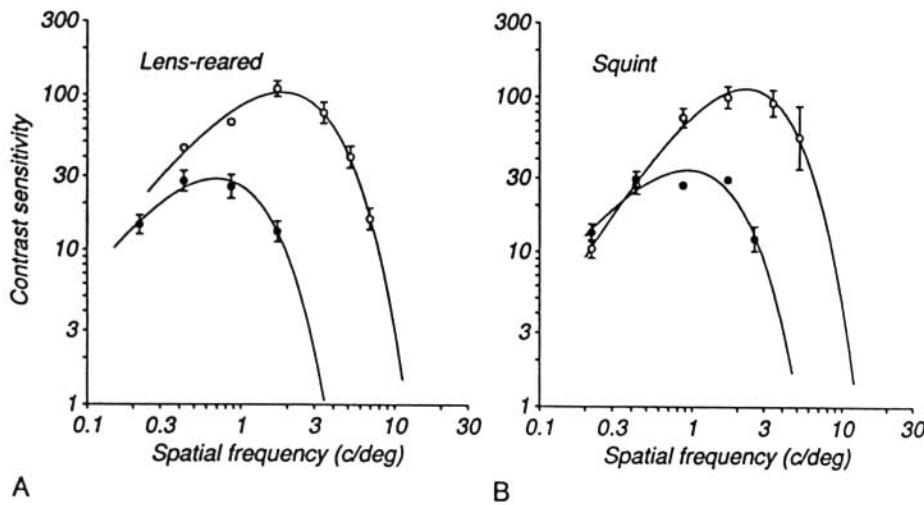


FIG. 18-4. Spatial contrast sensitivity of two macaque monkeys subjected to mild forms of visual deprivation. (A) Data from a monkey raised from birth to the age of 6 months with unilaterally blurred vision resulting from wearing extended-wear contact lenses of -10 D in one eye and zero power in the other. (B) Data from a monkey made strabismic by injection of botulinum A neurotoxin into the lateral rectus muscle of one eye at the age of 26 days. ● = data obtained from the treated eye; ○ = data obtained from the untreated eye.

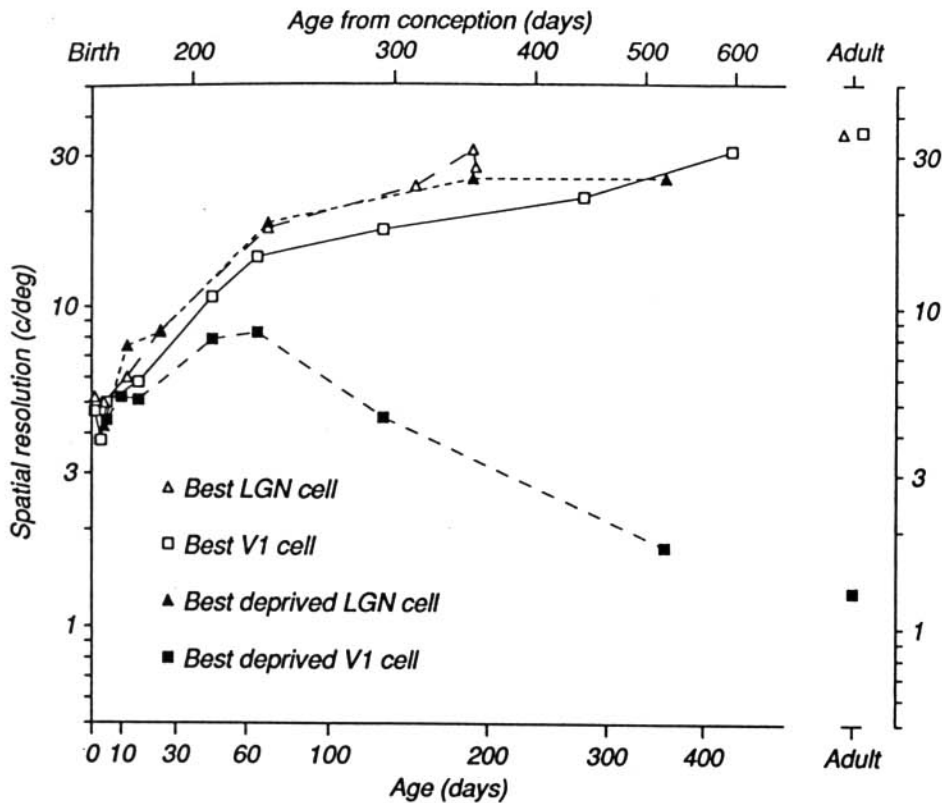


FIG. 18-5. Development of neural visual resolution in normally reared and deprived monkeys. Δ = visual resolution of the best LGN neuron recorded from suitable populations in normally reared monkeys (Blakemore and Vital-Durand, 1986a). \square = visual resolution of the best neuron recorded from the striate cortex in normally reared monkeys of different ages (Blakemore and Vital-Durand, 1983a; Blakemore, 1990). \blacktriangle = visual resolution of the best LGN neuron recorded from layers innervated by the deprived eye in monkeys subjected to unilateral eye closure from birth to the time of recording (Blakemore and Vital-Durand, 1986b). \blacksquare = visual resolution of the best neurons recorded from the striate cortex of monkeys binocularly deprived from birth to the age of recording by bilateral eyelid suture (Blakemore and Vital-Durand, 1983b, unpublished observations; Blakemore, 1990).

1990, unpublished observations). The upper two curves (open symbols) are transferred from Figure 18-2 and show the development of visual resolution in the LGN and primary visual cortex, as measured by the resolution of the best cell encountered in animals of particular ages. The data represented by filled triangles show the development of visual resolution of cells in the deprived layers of the LGN of animals subjected to monocular eyelid suture from birth to the age of recording. Comparison of these data with data from normals (filled triangles) or from the nondeprived layers of the LGN in the same animals (not shown) (Blakemore and Vital-Durand, 1986b) shows that deprivation appears to be without effect on the resolution of these cells. In a more exhaustive analysis, Levitt et al. (1989) showed that the quantitatively measured spatial and temporal properties of LGN neurons in long-term deprived monkeys are almost indistinguishable from those of normal or nondeprived cells. This lack of effect of monocular deprivation on properties in the LGN is in stark contrast to

the devastating behavioral consequences of this deprivation (Fig. 18-3).

The bottom curve in Figure 18-5 shows the data of Blakemore and Vital-Durand on the development of visual resolution in deprived cortical neurons. In this case, the monkeys were binocularly deprived of vision because monocular deprivation removes almost all detectable influence of the deprived eye from the visual cortex (LeVay et al., 1980). It is evident that, although neuronal properties develop roughly normally for the first month or so, neuronal performance falls dramatically thereafter. The best neuronal resolution is deprived animals older than a year or so is actually worse than it is at birth.

The results in Figure 18-5 speak largely for themselves. In animals deprived of patterned visual experience, the relay of neuronal signals up to the LGN appears unimpeded. These signals do not transfer effectively to the cortex. It is thus safe to conclude that the failure in these cases arises at the geniculocortical synapse or

perhaps among the earliest synaptic relays within the cortex itself.

Although the previous results strongly implicate cortical mechanisms in the visual effects of deprivation, it may be that these "blockbuster" environmental manipulations are too overwhelming to reveal more subtle changes that might be produced under less extreme conditions. We therefore also explored the effects of early blur-rearing and strabismus on the properties of neurons in the LGN and V1. Figure 18-6 shows summary scatter diagrams of the joint distribution of optimal spatial frequency and peak contrast sensitivity for populations of neurons from four monkeys raised with unilateral blur by means of atropinization (Fig. 18-6A) (Movshon et al., 1987) and from three monkeys raised with esotropic strabismus induced surgically (Fig. 18-6B) (Eggers et al., 1984). In each part of the figure, data represented as points are for neurons dominated by and tested through the untreated eye, and data represented as squares are for neurons tested through the blurred or deviated eye. Inspection reveals that in both cases neurons driven through the treated eye have poorer contrast sensitivity and spatial resolution than neurons driven through the fellow eye; the shifts in sensitivity and scale are comparable to those seen behaviorally in similarly reared animals (e.g., Fig. 18-4). In the case of the blur-reared monkeys, control experiments show that, as in the case of monocular visual deprivation, the res-

olution and sensitivity of neurons in the LGN is unaffected by the early rearing conditions. We conclude that the effects of these two modest forms of visual penalization, like those of complete deprivation, are expressed in the cortex, and that the peripheral optical and neural mechanisms of the visual system are unaffected.

CONCLUSIONS

We explored the limits on visual performance in two domains: during early development and following abnormal early experience. Our conclusions from these two sets of studies are wholly different from each other. In agreement with others considering the human literature, we believe that the main limits to visual performance in young developing animals are peripheral. Perhaps because they are accessible to anatomical investigation, much attention has been focused in the human literature on the development of retinal photoreceptors. Our consideration of the data from nonhuman primates suggests that although their photoreceptor development in some respects mirrors that of humans, it does not account in quantitative terms for the improvement in visual performance seen during early development. Instead, it appears that peripheral neural processing of photoreceptor signals—either in the retina or between the retina

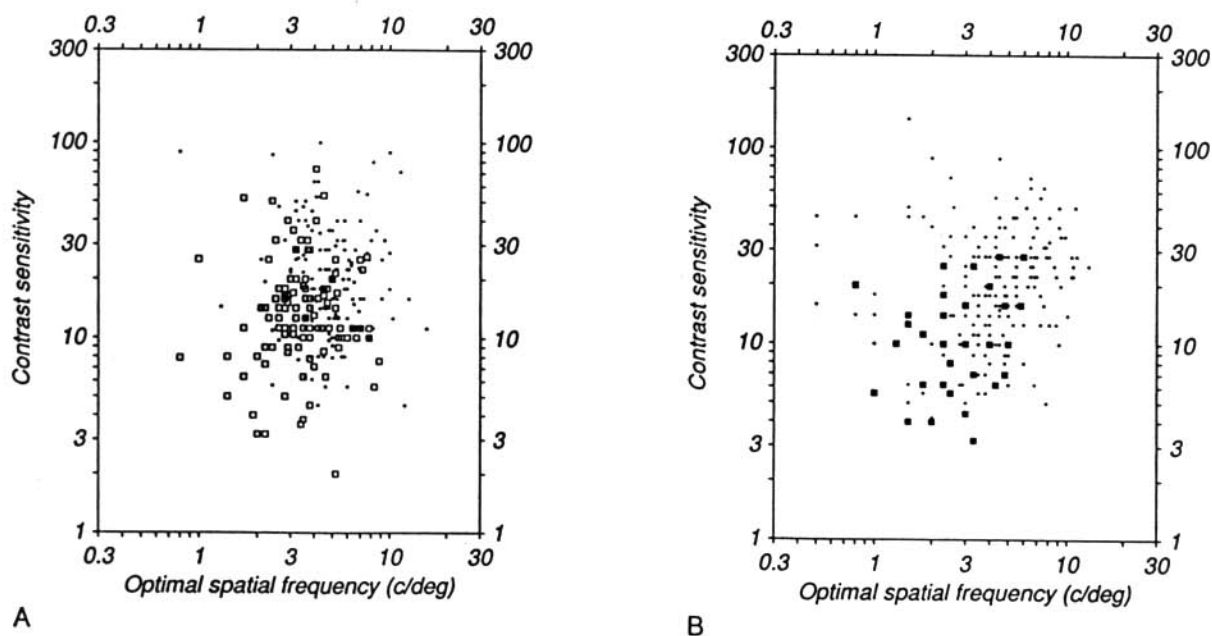


FIG. 18-6. Spatial properties of neurons recorded from the primary visual cortex of monkeys with experimentally induced amblyopia. (A) Data from 256 neurons from four monkeys raised with blurred vision produced by unilateral instillation of atropine from birth to the age of 6 months (Movshon et al., 1987). (B) Data from 234 neurons from three monkeys made strabismic by surgical alteration

of the medial and lateral rectus muscles before the age of 3 weeks (Eggers et al., 1984). (A,B) Each datum represents the optimal spatial frequency and contrast sensitivity of a single neuron. Points = data for neurons dominated by (and measured through) the untreated eye; squares = data for neurons dominated by the treated eye.

and the LGN—is the stage of the visual process that limits performance in young monkeys. It seems clear that once signals reach the LGN they are faithfully relayed and processed by the primary visual cortex and subsequent centers.

When development is disrupted by abnormal early experience, however, a different picture emerges. There is little evidence in the literature for significant functional changes in the image-forming machinery of the eye or for changes in the peripheral components of the visual nervous system. Indeed, it is striking that the visual periphery is so utterly indifferent to its early visual input. On the other hand, with deprivation, the cortex, which responds properly to its input in the young animal, now fails to process the rich array of visual information forwarded to it from the LGN. Evidence from several kinds of abnormal visual experience discussed here, and many others not mentioned, points with regularity to the cortex as the site of the deficit. Indeed, because *all* signals afferent to the cortex seem normal and *no* signals within the cortex survive deprivation with a semblance of normality, it may be that the developmental anomaly can be pinned down in many cases to be a single set of synapses—those that link geniculate afferents to their cortical target neurons.

It should be noted that significant differences may exist between species; considerable evidence (not all of it uncontested) suggests that abnormalities may develop in the peripheral visual system of cats following deprivation (Movshon and Van Sluyters, 1981; Friedlander and Tootle, 1990). One must assume, however, that the factors acting in humans are more similar to those in monkeys than in cats. On this basis we conclude that the factors limiting human visual development are in the peripheral visual pathway, whereas those affected by abnormal experience are within the central nervous system.

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