Visual acuity: the role of visual input in inducing postnatal change

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Abstract

Some crude visual abilities are present at birth, and hence, do not depend on visual experience. However, there are substantial and rapid postnatal improvements. For example, the acuity of newborns is 40 times worse than that of normal adults, largely because of retinal immaturities. Between birth and 6 months of age, there is a five-fold increase in acuity, followed by slow improvement to adult levels by 6 years of age. This review examines the role of visual experience in inducing those improvements by comparing the visual development of normal children with that of children treated for congenital cataracts that blocked patterned visual input until the cataracts were removed surgically and the eyes were given a compensatory optical correction. The acuity of children treated for congenital cataracts does not improve before they receive patterned visual input, but then increases rapidly to reach normal limits by 1 year of age. However, the patients later show permanent deficits in acuity, presumably because the initial deprivation caused damage to the visual cortex. Studies of children who developed cataracts after birth indicate that visual input is also necessary to consolidate cortical connections. Moreover, the deleterious effects of visual deprivation are worse if there was also uneven competition between the eyes — because the deprivation was monocular and there was little patching of the non-deprived eye — but only at some points during development. Thus, the development of visual acuity is shaped by experience-dependent and competitive mechanisms that have different temporal parameters. © 2001 Association for Research in Nervous and Mental Disease. Published by Elsevier Science B.V. All rights reserved.

Keywords: Visual acuity; Newborn; Development; Congenital cataract; Visual deprivation; Infant

1. Introduction

No matter how it is measured, the visual acuity of newborn infants is poor, and improves rapidly during the first 6 months. However, it is not until later in childhood that acuity is as good as that of adults tested with the same methods. In this article, we review the role of visual input in driving the rapid postnatal change. We do so by contrasting the development of visual acuity in normal children and in children who missed early visual input because they were born with cataracts in one or both eyes. A cataract is an opacity in the lens of the eye which, in the patients we selected for study, was dense and central, and large enough to prevent any fixation or following. Treatment involves removal of the cataractous lens and fitting the eye with a compensatory contact lens to focus visual input. Visual acuity on the day of contact lens fitting, that is, at the time of the first focussed patterned visual input, indicates whether postnatal improvements are possible in the absence of visual input. Subsequent changes in acuity indicate whether there is recovery from the initial deprivation and allow deductions about the role of early visual input in molding the neural architecture for later changes.

2. Normal development

In adults, visual acuity is typically tested with a chart of letters of decreasing size and the measure of acuity is the smallest letters the adult can read accurately. For infants, acuity is measured with stripes of decreasing size and the goal is to determine the finest stripes the infant can resolve. One method, called preferential looking, takes advantage of infants' preference to look at something patterned over something plain [1]. The infant is shown black-and-white stripes paired with a gray stimulus of the same mean luminance, and the size of the stripes is varied across trials. On half the trials, the stripes are to the left of center and on the other half, they are to the right. An observer watches the baby's eye movements and the measure of an infant's acuity

is the smallest size of stripe for which there is longer looking to the stripes than to the plain gray. There are various methods for determining the threshold, ranging from long psychophysical procedures [2–6] to a more subjective test called the Teller Acuity Card Procedure [7,8], with good agreement across procedures in the estimated acuity at different points in normal development.

When tested with preferential looking, the acuity of newborns is typically about 40 times worse than that of normal adults tested under the same conditions [5,6,9–12]: newborns show preferences for stripes only if they are 30–60$'$ wide, whereas adults can resolve stripes less than 1$'$ wide (1$'$ is one-sixtieth of a degree of visual angle). Babies born prematurely perform even worse than full-term newborns [6,13,14], but not when matched for postconceptional age [6,13,15,16] (but see [14]). Such poor acuity limits newborns to seeing only large objects: mom's hair and the rails of the crib, but not the details of her face or the texture in the wood. Poor acuity may underlie young infants' limited reactions to objects, including faces. For example, they fixate mainly large features on the external contour of faces and geometric shapes [17,18], recognize mom only if her hair is visible [19,20], and orient preferentially towards a representation of a face with unrealistically large blobs representing the eyes and mouth [21].

Two other methods have been used to determine the smallest stripes that newborns can resolve: optokinetic nystagmus (OKN) and visually evoked potentials (VEPs). OKN is a series of reflexive pursuit and saccadic eye movements elicited by a repetitive pattern moving through the visual field. The measure of acuity is the width of the smallest stripes that elicit OKN [1,22–25]. VEPs have also been used to measure acuity at birth, typically by showing infants phase-reversing stripes or checks and determining the smallest elements that a evoke a brain potential different from that evoked by a flashing gray field [22,26]. A comparison of the absolute acuity values obtained from the various methods is inappropriate because of differences in the typical stimuli (a small patch of stationary stripes for preferential looking acuity versus phase-reversing stripes or checks for VEP acuity versus a full field of moving stripes for OKN acuity). However, when stimulus factors are equated, estimates of acuity based on preferential looking, OKN, and VEP yield similar values for newborns [22].

Why is newborns' acuity so poor? Poor attention or a lack of motivation are unlikely explanations because different methods yield similar estimates, and VEP acuity, unlike preferential looking, is influenced little by attention or motivation [22]. Poor optics of the eye are also unlikely to play a major role. First, transmittance through the ocular media is better in newborns than in adults, especially at short wave lengths (reviewed in [27]). Moreover, differences between newborns and adults in pupil diameter, in the amount of diffraction, or in the amount of spherical and chromatic aberration are likely to have negligible effects on acuity (reviewed in [27]). Finally, inaccurate accommodation

does not appear to impose an important limitation because acuity at birth does not vary with viewing distance [28,29]. In contrast, immaturities in the retina appear to cause a major limitation on newborns' acuity. Rather than the densely packed cones with well-aligned, slender outer and inner segments that guide light in the adult fovea, newborns' cones are short, stumpy, and sparsely packed. Their outer segments have small diameters that do not line up well with their wide inner segments. Ideal observer models indicate that newborns' cones are 350 times less efficient at capturing light than those in the adult fovea [27]. However, the newborns' measured acuity is poorer than can be accounted for by the retinal limitations alone [27,30]; hence, there must be additional limitations between the retina and the visual cortex.

Visual acuity improves rapidly in the first 6 months after birth. Fig. 1 shows measurements based on preferential looking. By 6 months of age, acuity has improved from being about 40 times worse than that of normal adults to being only eight times worse. Over this same period, babies show increased sensitivity to the internal features of faces [17] and recognize a familiar face even when it is wearing a bathing cap or is presented in a novel point of view [31,32]. They also demonstrate sensitivity to small differences in direction of eye gaze [33–35]. Thereafter, acuity improves more gradually and reaches adult values at 4–6 years of age [5,6,10]. However, the developmental pattern differs across techniques. For example, Lewis et al. [36] report that preferential looking acuity develops more slowly than OKN
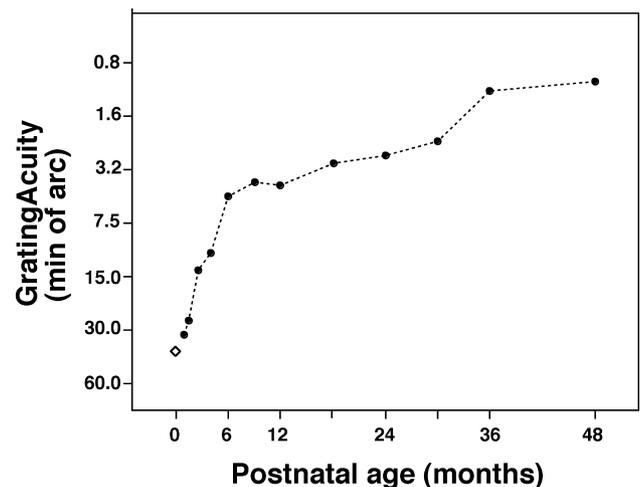
Fig. 1. Changes in grating acuity between birth and 48 months when measured by preferential looking. The *y*-axis shows the size of the smallest stripes to which subjects respond, plotted in minutes of arc, where 1$'$ is one-sixtieth of a degree of visual angle. Thus, the smaller the number, the better the grating acuity. Filled circles represent the mean acuity of 1–48-month-olds tested monocularly by Mayer et al. [102]. The open symbol represents the log mean grating acuity of newborns tested binocularly [5,6,9–12]. Monocular and binocular acuities do not differ prior to 6 months of age [103]. Grating acuity measured by preferential looking reaches adult values somewhere between 4 and 6 years of age, depending on the testing parameters. Adapted with permission from [98].

acuity between 4 and 18 months of age. Similarly, Sokol and colleagues [37] report that preferential looking acuity develops more slowly than VEP acuity between 2 and 12 months of age even when the same stimuli are used for the two measurements. These differences are not surprising in light of the fact that the three methods differ in the responses required to register a response (voluntary looking for preferential looking versus evoked brain waves for VEP versus automatically elicited eye movements for OKN), in the part of the retina likely to be involved, at least during the first year of life (the peripheral retina for preferential looking acuity and the central retina for OKN and VEP acuity), and in the areas of the brain involved (the geniculo-striate pathway for preferential looking acuity and VEP acuity versus subcortical centers for optokinetic eye movements) [36,38,39].

The rapid improvement in the first 6 months, in part, reflects the development of foveal cones so that they filter out less information and allow finer and finer detail through to tune cells in the visual cortex [27,40,41]. The more gradual changes through the ages of 4–6 years are likely to reflect further maturation of the retinal and cortical architecture [40–46] (reviewed in [47]).

3. Early binocular deprivation

Patterned visual input is necessary for the postnatal development of acuity. The evidence for that conclusion comes from comparisons of infants with normal eyes with infants who were deprived of early visual input because of dense central cataracts in both eyes. The cataractous lenses were removed surgically and 1–3 weeks later, after the eye had healed from the surgery, the eye was given a compensatory contact lens to provide the first focussed patterned visual input. We measured the acuity of 12 patients treated for bilateral congenital cataract within 10 min of that first patterned input [48]. Despite a variation in the age at treatment from 1 week to 9 months, the acuity was, on average, like that of normal newborns, with no sign that improvement had occurred with increasing age in the absence of patterned visual input. As a consequence, the acuity fell farther below the norm for the patient's age, the later treatment occurred during the first year.

After 1 h of patterned visual input, the acuity of treated eyes improved to the level of a normal 6-week-old [48]. The improvement occurred in 20 of the 24 eyes tested and reduced the gap compared with age-matched normals from about 2 to 1.4 octaves (an octave is a halving or a doubling of a value). There was additional improvement over the next month that reduced the deficit to about one octave (see Fig. 2). The improvements over the first hour and the next month were not related to the age at treatment. A control experiment showed that the improvement after the first hour of patterned visual input was, in fact, a consequence of visual experience: when we patched one eye of

eight bilateral cases between the first test and the retest after 1 h of waking time, acuity in the unpatched eyes improved, but acuity in the patched eyes did not.

Although visual acuity does not improve postnatally in the absence of patterned visual input, the cortical connections necessary for neonatal levels of acuity are not lost: even when deprivation lasted up to 9 months of age, acuity was no worse than that of normal newborns. Moreover, results after the first hour of patterned visual input indicate that the cortical system of patients was not dormant during the period of deprivation. Rather, it changed in order to be able to respond more quickly than normal to visual input. Perhaps, spontaneous retinal activity, which has been shown to play an important role in sculpting cortical connections in the cat before eye opening (reviewed in [49]), is sufficient to maintain or even further those cortical connections in humans. The rapid improvement in our patients upon the first visual input is consistent with studies of monocularly
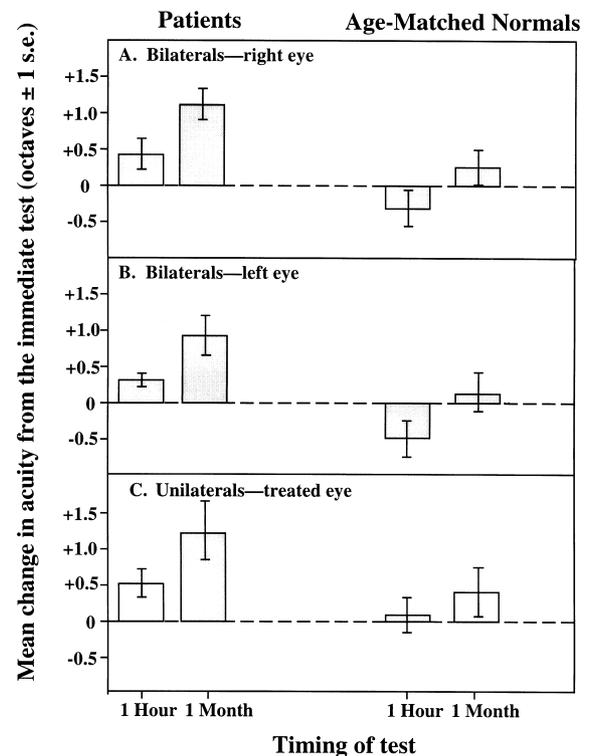

Fig. 2. Mean change in acuity (±1 SE) after first exposure to focussed patterned visual input in children treated for congenital cataracts. Shown are the change in acuity between the immediate test and the test after the first hour of visual input and between the immediate test and the test 1 month later. The *y*-axis plots the amount of change in octaves, where one octave is a doubling or a halving of a value. Values above zero represent an improvement from the immediate test; values below zero represent a decline. The left half of each panel shows the results for infants treated for congenital cataracts; the right half of each panel shows the results for age-matched control groups. Panels (A) and (B) show the results for bilateral cases and their comparison groups. Panel (C) shows the results for the treated eye of unilateral cases and their comparison groups. In every comparison, patient groups improved more than the age-matched controls. Adapted with permission from Figure 3 of reference [48]. Copyright 1999, American Association for the Advancement of Science.

deprived and dark-reared cats which, upon the first exposure to light, show rapid changes in measured acuity and in markers of plasticity in the visual cortex [50–53].

By 1 year of age, the acuity of eyes treated for bilateral congenital cataract has improved further and the acuity of most eyes falls within normal limits [36,54–60]. The implication is that acuity improved faster than normal as it changed from the newborn level immediately after treatment (well below normal if the patient was older than 2 months) to normal values at 1 year of age. Like the results during the first hour and month after treatment, these findings indicate a readiness of cortical neurons to respond to visually-driven activity.

Despite the improvement to normal by 1 year of age, continued assessment of the patients treated for bilateral congenital cataracts reveals later deficits in acuity. Fig. 3 shows the loss in grating acuity relative to age-matched normals for 13 patients ranging in age from 5 to 19 years at the time of the test. All 26 eyes had a deficit in grating acuity, usually on the order of 0.5 log units, or 1.5 octaves, even when the deprivation lasted for only the first 7 weeks of life [61]. The deficits were not related to the age of treatment, at least when treatment occurred between 7 weeks and 9 months of age. Thus, binocular deprivation during the early postnatal period, when poor acuity limits normal infants to seeing wide stripes, prevents the later development of sensitivity to fine detail.

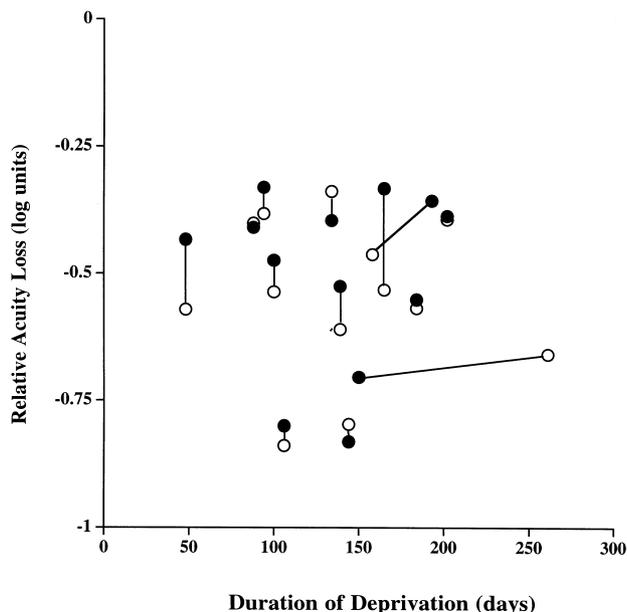Our longitudinal study of contrast sensitivity has begun to

Fig. 3. Asymptotic deficit in grating acuity as a function of age of onset of binocular deprivation. The *y*-axis plots the deficit in log units, where zero is normal and larger negative values indicate larger deficits. Each dot represents the result for one deprived eye. A solid line joins the right (filled circles) and left (open circles) eyes from the same patient. Adapted with permission from the American Association for the Advancement of Science and Maurer et al. [48]. (Reprinted from [61], page 3485, Copyright 1999, with permission from Elsevier Science).

reveal the origin of this sleeper effect [62]. Contrast sensitivity is a measure of the minimum contrast necessary to see sine waves of different frequencies. When first tested at 4–7 years of age, patients treated for bilateral congenital cataracts needed more contrast than age-matched normals to see sine waves of all spatial frequencies, except for a few patients who were normal at the lowest spatial frequencies (the widest stripes). When retested 1 and/or 2 years later, patients had improved, often more than normals, at low and medium spatial frequencies (wide and medium stripes), but not at high spatial frequencies (narrow stripes). Together, the results indicate that patients treated for bilateral congenital cataracts had hit an asymptote in sensitivity to high spatial frequencies by the age of 4 years. Age-matched normals improved at all spatial frequencies until the age of 7 years [47]. The consequence is that patients' deficits at high spatial frequencies increased after the age of 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 narrow stripes after the age of 4. Thus, the benefits of the poor quality visual input infants receive during the first few months, or the damage from its absence, are not evident until later in development. In related work, we have shown that the same is true for the infant's early postnatal exposure to faces [63]: infants treated for bilateral congenital cataracts fail to develop the expert configural processing of faces that emerges in normal children after the age of 10 [64]. The sleeper effects may be related to abnormal pruning of cortical synapses, pruning that is normally evident in the visual cortex after 1 year of age and in higher visual cortical areas after 2 years of age [65–67].

Evidence for permanent deficits in asymptotic acuity have also been reported in other human cohorts [68,69] and in binocularly deprived monkeys [70]. There are similar deficits in asymptotic letter acuity [38,68]. However, if the binocular deprivation is extremely short — less than 10 days after birth — some, but not all children are able to achieve a normal linear letter acuity of 20/20 [71]. Thus, the visual system appears to be less sensitive to visual input (or its absence) before 10 days of age.

Studies of monkeys that were deprived by lid suture or dark-rearing indicate that early visual deprivation damages the visual cortex. Like the children treated for bilateral congenital cataract after 10 days of age, such monkeys have permanent deficits in grating acuity [70]. The retina appears normal, with no changes in the electroretinogram [72], the topography of photoreceptors [73,74], or the morphology of retinal ganglion cells (reviewed in [75]), unless the deprivation lasted past 2 years of age. Although cells in the monkey's lateral geniculate nucleus are smaller than normal, they have normal electrophysiological properties, including normal sensitivity to high spatial frequencies (narrow stripes) [76], and presumably send normal signals to the primary visual cortex. It is at the level of the primary visual cortex that damage from early visual deprivation

becomes apparent: most cells respond more sluggishly than normal, have abnormally large receptive fields, and are poorly tuned to orientation and spatial frequency [72,77–79]. Across the population of cells in the primary visual cortex of binocularly deprived monkeys, there is a large reduction in acuity [77,78]. Thus, reductions in acuity after early binocular deprivation in humans are likely to reflect abnormalities in neurons in the primary visual cortex and their projections. After early binocular deprivation in humans, the visual abilities that remain may be mediated by the few remaining normal cells in the primary visual cortex, by the many cells in the primary visual cortex that respond abnormally, or by cells in extrageniculo-striate pathways that appear to play a greater role than normal in mediating some visual functions after binocular deprivation, at least in cats [80–82].

4. Early monocular deprivation

Studies of children treated for bilateral congenital cataracts allow measurement of the effects of visual deprivation per se. Studies of children treated for unilateral congenital cataracts allow assessment of the additional adverse effect of uneven competition between the eyes for cortical connections. Evidence for such uneven competition lies in the finding of larger deficits in a treated eye after monocular than after binocular deprivation of the same duration and/or the finding that the size of the deficit in an eye treated for unilateral congenital cataract is related inversely to the number of hours/day that the non-deprived eye was patched to lessen the competition and force usage of the previously deprived eye. As in our studies of bilateral congenital cataracts, we included only children with cataracts that were sufficiently large and dense to block all patterned visual input and with no other abnormalities likely to interfere with visual development. Treatment involved surgical removal of the cataractous lens and fitting the eye with a contact lens to focus patterned visual input. After treatment, parents were instructed to patch the non-deprived eye 50–90% of the waking time until at least age 5. The recommended amount of patching varied among ophthalmologists and decades, but was independent of the severity of the deficit. There was even more variability in the actual amount of patching because compliance with the recommended amount varied among parents. This variability in a sufficiently large sample allows statistical assessment of the independent influences of age of treatment and amount of patching.

During the first month after treatment, the results were similar for children treated for unilateral and for bilateral congenital cataracts, and there was no evidence of adverse effects from uneven competition between the eyes during the period of monocular deprivation or immediately thereafter [48]. On the first test 10 min after the first focussed patterned visual input, the mean acuity of the 16 children treated for unilateral congenital cataracts was at the newborn level and there was no sign of better (or worse) acuity in children treated at later ages. After the first hour of visual input and over the next month, there was a significant improvement of a magnitude similar to that after treatment for bilateral congenital cataract (see the bottom panel of Fig. 2). Moreover, the amount of improvement over the first month was not related to the amount of patching of the non-deprived eye which, in this cohort, varied from none to 9 waking hours/day. In other words, despite the large differences in the signals reaching the visual cortex from a normally developing eye versus an eye receiving no pattern before treatment, or from an eye with much lower acuity than the fellow eye after treatment, there is no evidence that the uneven signals result in competitive suppression of the deprived eye. Visual activity is sufficient to drive the initial recovery from deprivation.

By the first birthday, evidence of adverse effects from uneven competition has emerged. Although the majority of eyes have improved to be within the normal limits, more than 20% are outside the normal limits, and the acuity is worse in patients who did less patching of the non-deprived eye [36,60]. In fact, children who had patched the non-deprived eye for less than 3 h/day have acuity in the deprived eye that is significantly worse than the acuity of children treated at the same age for bilateral congenital cataracts [36]. Unlike the case for children treated for bilateral congenital cataracts, there is no effect of the age of treatment, that is, no effect of the number of months before the first birthday during which the child's visual cortex received patterned visual input through the formerly deprived eye [36,60]. These results imply that by 1 year of age, competitive interactions have begun to limit recovery from deprivation. Note, however, that although more patching of the non-deprived eye was correlated with better acuity in the treated eye, we cannot be certain that the extra patching caused the better acuity. It is possible that those with better acuity in the treated eye were more likely to be compliant with the prescribed patching regimen. Support for a causal influence from occlusion comes from studies of cats in which compliance is not an issue. Mitchell and Gingras [52] obtained results similar to ours from monocularly deprived kittens: the initial recovery of acuity from deprivation was similar in kittens with or without reverse suture (permanent closure of the initially non-deprived eye to eliminate the competitive disadvantage of the initially deprived eye). Only later did the kittens with reverse suture show greater recovery. Mitchell and Gingras concluded that the initial recovery after monocular deprivation is driven solely by visually-driven cortical activity and only later, after a scaffold of connections from the deprived eye has been established in the primary visual cortex, does uneven competition between the eyes limit the amount of recovery. Similarly, in humans, the initial recovery appears to be driven exclusively by visual activity, perhaps taking advantage of the normal postnatal exuberant proliferation of

synapses in the visual cortex [44,67]. Once the visual activity has induced functional changes, presumably by strengthening responses in the primary visual cortex driven by the previously deprived eye, competitive interactions emerge and quickly become the strongest determinant of recovery.

As would be expected, children treated for unilateral congenital cataracts show deficits in asymptotic acuity and the deficits are worse in children who had later treatment and little patching of the non-deprived eye [56,60,68,83]. Among the poor patchers, the deficits exceed 1 log unit and are larger than any deficit we observed in children treated for bilateral congenital cataracts [61]. Our longitudinal study of contrast sensitivity beginning when patients were 4–7 years of age revealed that some of the children treated for unilateral congenital cataracts actually lost sensitivity to higher spatial frequencies when retested 1 and/or 2 years later [62]. Thus, a history of uneven competition between the eyes can cause a later loss of acuity, presumably reflecting the loss of functional cortical connections that had been formed in the initial recovery from deprivation. Such losses of cortical connections after monocular deprivation are also implicated in monkeys in which the visual resolution of cells in primary visual cortex driven by the deprived eye is sometimes less than that of normal newborn monkeys [84].

Moreover, uneven competition between a formerly deprived eye and a non-deprived eye leads to subtle deficits in the non-deprived eye [83,85–87]. Group analyses of vision in the non-deprived eyes reveal small but statistically significant losses in grating acuity, in contrast sensitivity for high spatial frequencies (narrow stripes), and in letter acuity, losses which are not related to the amount of time that the non-deprived eye had been patched throughout early childhood. The implication is that optimal development of acuity for both eyes requires balanced visual activity. When there is an initial period of advantage for one eye, until the acuity of the deprived eye approaches that of the non-deprived eye at about 1 year of age, the ultimate acuity of both eyes will be compromised, with major deficits in the deprived eye and subtle deficits in the non-deprived eye. The only exceptions reported in the literature are when the non-deprived eye is paired with a non-functioning eye that remains untreated [87] or when the imbalance is minimized because of very early treatment for unilateral congenital cataract (before 6 weeks of age) followed by very aggressive patching (at least 75% of waking time) [56].

The initial recovery after the end of deprivation from unilateral congenital cataract might reflect mainly the relative sparing of neurons representing the peripheral visual field, which appear to dominate measurements of visual acuity with preferential looking during infancy (reviewed in [36]). This hypothesis is consistent with evidence of faster development of peripheral than central visual acuity in normal infants between 3 and 8 months of age [39], as well as evidence that the primary visual cortex of monocularly deprived kittens exhibits a substantial loss of NMDA receptors, which have been shown to be involved in cortical plasticity, except for preservation of the normal complement of NMDA receptors in areas representing the peripheral visual field [88]. In contrast, the visual cortex of binocularly deprived kittens exhibits a substantial increase in NMDA receptors for both the central and peripheral visual fields [89]. It also fits with evidence that both the light sensitivity and contrast sensitivity of older children with a history of early monocular deprivation are degraded less in the periphery than in the central visual field [38,90]. Measurements during infancy with techniques that appear to be influenced more by central vision than preferential looking — OKN [36] and VEPs [86] — indicate much less recovery during the first months following treatment for unilateral congenital cataract.

Studies of monocularly deprived monkeys indicate that, as in binocularly deprived monkeys, the retina appears normal and cells in the lateral geniculate nucleus, although smaller than normal, have normal receptive field properties and, as a population, normal acuity [76,91]. It is at the level of the primary visual cortex that the greater impact of monocular than binocular deprivation is first evident. Most cells are driven only by the non-deprived eye, some cells are completely unresponsive, and the few cells drivable by the previously deprived eye have a much greater reduction in acuity than those in the primary visual cortex of binocularly deprived monkeys [79,84,92–94]. Reverse-suturing increases the proportion of cells that can be driven by the originally deprived eye [84,94–97]. Thus, uneven competition appears to have its impact at the level of the primary visual cortex.

5. The sensitive period for damage

Abnormal visual input during a sensitive period causes permanent deficits in acuity. As discussed previously, this sensitive period for the development of normal acuity is likely to begin shortly after birth. How long does it last? Studies of children who began with putatively normal eyes, but later developed dense, central cataracts that blocked patterned visual input to one or both eyes allow estimation of the end of the sensitive period for the development of normal acuity. When the cataract was caused by an eye injury, we can be confident about the age at which the blockage of patterned visual input began. We cannot be so confident when the cataract was caused by a metabolic or genetic disorder because these cataracts usually develop gradually and block more and more visual input as they become larger. Despite the resultant uncertainty in some cases about how much visual input was blocked for how long, it is clear that cataracts that become dense and central before the age of about 10 years cause permanent deficits in linear letter acuity, whether the deprivation had been monocular or binocular [98]. Thus, visual input is necessary for the refinements of visual acuity throughout the 5–7 years of

normal development of letter acuity, and it is necessary to consolidate or maintain synaptic connections for approximately 3–5 years thereafter. Only after the age of about 10 years are the connections for the development of normal visual acuity 'hard-wired'.

6. Conclusions

In summary, during early infancy, acuity is limited largely by the immaturity of the photoreceptors in the retina, with additional limitations in the geniculo-striate pathway. Nevertheless, the system is expectant and the first visual input sets up the architecture in the visual cortex that will be refined later to support fine visual acuity. These initial stages appear to be driven by visual activity, which remains critical until around 10 years of age, several years past the achievement of adult-like acuity. By 1 year of age, the adverse effects of uneven competition between the eyes become evident and indicate that visual activity is effective only if it is balanced between the two eyes.

It is important to note that the conclusions in this review should not be generalized to aspects of vision other than acuity. For example, the sensitive period for the development of normal peripheral light sensitivity lasts longer than the sensitive period for acuity; past 14 years of age [90,99,100]. However, as for acuity, the sensitive period for peripheral light sensitivity lasts many years longer than its period of normal development, and there are adverse effects of both visual deprivation and uneven competition between the eyes [90,99,100]. In contrast, the sensitive period for the development of normal sensitivity to the direction of global motion is very short: it is over before 1 year of age and likely to be over before its period of normal development is complete [101]. Furthermore, deficits in the perception of the direction of global motion in treated eyes are smaller after monocular deprivation than after binocular deprivation, a pattern that suggests collaborative interactions between the eyes as visual information is integrated in higher cortical areas beyond the primary visual cortex [101]. In each case, the limited vision of the newborn shapes the later development of the nervous system, but the details of the developmental mechanism depend on the aspect of vision and the neural circuitry that normally mediates it.

Acknowledgements

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