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Repeated measurements of contrast sensitivity reveal limits to visual plasticity after early binocular deprivation in humans

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Abstract

Contrast sensitivity improves in visually normal children until 7 years of age and is impaired in children who experienced early visual deprivation from bilateral congenital cataracts. Here, we investigated whether the deficits after early visual deprivation change during childhood by retesting the contrast sensitivity of seven patients treated for bilateral congenital cataract who had been first tested before 7.5 years of age, and of two patients first tested after 11 years of age. For the younger group, contrast sensitivity at low spatial frequencies improved after 1- and 2-year intervals, while their sensitivity at mid and high spatial frequencies did not change. There was no systematic change in the two older patients. The results indicate that early visual input sets up the neural substrate for later improvement in contrast sensitivity at mid and high spatial frequencies. However, there is sufficient plasticity during middle childhood to allow some recovery at low spatial frequencies. The results shed new light on the role of early visual experience and the nature of developmental plasticity.

Keywords: Visual deprivation; Binocular deprivation; Congenital cataract; Developmental plasticity; Contrast sensitivity

1. Introduction

Infants' vision is poor compared to that of adults; newborns can see a pattern of stripes only if the spatial frequency is less than 1.0 cpd, whereas visually normal adults can see spatial frequencies 40 times higher than that (reviewed in Maurer & Lewis, 2001a, 2001b). Even for those spatial frequencies, young infants respond to a grating only if it has at least 1.7 log units more contrast than is necessary for an adult with normal vision (Atkinson, Braddick, & Moar, 1977; Banks & Salapatek, 1978; reviewed in Maurer & Lewis, 2001a, 2001b). Contrast sensitivity improves during infancy but it takes 7 years for it to reach adult levels (Ellemberg, Lewis, Liu, & Maurer, 1999). Early visual deprivation prevents normal development: children who were deprived of patterned visual input from birth because of dense, central cataracts in both eyes later have abnormal contrast sensitivity, but the extent of the deficit varies with spatial frequency. Sensitivity is usually normal or nearly normal at low spatial frequencies (i.e., for wide stripes), impaired to varying extents at middle spatial frequencies, and severely impaired at high spatial frequencies (i.e., for thin stripes), including an abnormally low acuity limit (Birch, Stager, Leffler, & Weakley, 1998; Ellemberg, Lewis, Maurer, Lui, & Brent, 1999; Mioche & Perenin, 1986; Tytla, Maurer, Lewis, & Brent, 1988; reviewed in Maurer & Lewis, 2001a).

The studies establishing the deleterious effects of early binocular deprivation on later contrast sensitivity included children of a variety of ages, all of whom had suffered deprivation during part of the first year of life (Ellemberg, Lewis, Maurer et al., 1999; Mioche & Perenin, 1986; Tytla et al., 1988). Across studies, the age range extends from 5 years to more than 20 years, with the bulk of the data from younger patients: all 12 of the patients were 8 years of age or younger in the cohort studied by Birch et al. (1998), as were 88% of the 8 patients in an earlier study from our lab (Tytla et al., 1988), and 69% of the 13 patients in the study that forms the basis for the current report (Ellemberg, Lewis, Maurer et al., 1999). (The exception is the study by Mioche and Perenin (1986) of eight patients,

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all of whom were at least 12 years old.) The large percentage of children under 8 years of age in previous studies raises the possibility that some patients were tested when the visual system was still plastic and capable of recovering from the initial deprivation, and hence that the size of the deficit was overestimated. Indeed, in a study combining children treated for total and partial bilateral cataracts, there were improvements in acuity between measurements at 7–10 years and measurements at 10–12 years (Magnusson, Abrahamsson, & Sjöstrand, 2002). Alternatively, because the spatial contrast sensitivity of children with normal eyes continues to improve until 7 years of age (Ellemberg, Lewis, Liu et al., 1999), it is possible that the size of the deficit was underestimated in some of the younger patients because their sensitivity had ceased to improve, while that of their age mates was still changing. The purpose of the current study was to investigate these possibilities by retesting the contrast sensitivity of patients treated for bilateral congenital cataract after intervals of 1 and/or 2 years.

There is evidence that the visual cortical pathway remains plastic after infancy from studies of individuals who became blind at various times after birth. When visual input is absent from birth, the visual cortex responds to auditory and tactile input, and perhaps even aspects of language. However, these alterations are seen not only in the congenitally blind (e.g., Gizewski, Gasser, de Greiff, Boehm, & Forsting, 2003; Roder, Stock, Bien, Neville, & Rosler, 2002; Sadato et al., 1998; reviewed in Maurer, Lewis, & Mondloch, 2005), but also in children who became blind as late as adolescence (Cohen et al., 1999; Sadato, Okada, Honda, & Yonekura, 2002) and, to a lesser extent, even in adults who became blind after 18 years of age or who were simply blind-folded for 5 days in the laboratory (Burton, Diamond, & McDermott, 2003; Burton, Sinclair, & McLaren, 2004; Burton, Snyder, Conturo et al., 2002; Burton, Snyder, Diamond, & Raichle, 2002; Pascual-Leone & Hamilton, 2001). Similarly, in adults with strabismic amblyopia (reduced acuity in one eye because of childhood misalignment of the eyes), some types of training have been effective in inducing large improvements in vision even when begun in adulthood (Kupfer, 1957; Levi, Polat, & Hu, 1997; Polat, Ma-Naim, Belkin, & Sagi, 2004; Simmers & Gray, 1999; reviewed in Levi, 2005). Such evidence suggests that responses of visual cortical neurons can be refashioned even in adulthood.

To investigate the degree of plasticity at different ages after early visual deprivation, we retested the contrast sensitivity of nine of the children treated for bilateral congenital cataract who had been included in an earlier report (Ellemberg, Lewis, Maurer et al., 1999). Because we expected plasticity to be greater during, or shortly after, the period when contrast sensitivity is still improving in visually normal children (i.e., up to age 7 years), we selected for follow-up mainly patients who were less than 7.5 years old at the time of the first test. Retesting occurred at intervals of 1 and/or 2 years. Patients' data were compared to those from cross-sectional samples of visually normal controls tested previously with the same method (Ellemberg, Lewis, Liu et al., 1999).

2. Method

2.1. Participants

The patient group was comprised of nine children treated for bilateral congenital cataracts who had participated in our original studies of contrast sensitivity (Ellemberg, Lewis, Maurer et al., 1999), seven of whom were less than 7.5 years old at the time of the first test (range 5.0–7.4 years). Patients had been included in the original study only if they had dense central cataract(s) which had been diagnosed on the first eye exam and by 6 months of age, no abnormalities in the retina or ocular media, and no evidence of neurological or cognitive abnormality. The cataracts had been removed during infancy and the eyes given compensatory contact lenses to focus visual input. We define the end of deprivation as the time of first contact lenses, because it is only then that the retina receives focused patterned visual input. For the patients first tested before 7.5 years of age, deprivation ended at a *M* age of 4.3 months (range 3.0–6.7 months). The remaining two patients were 11.7 and 18 years old at the time of the first test and had deprivation lasting 5.5 and 8.8 months, respectively. For each bilateral patient, we randomly selected one eye for follow-up. Clinical details are given in Table 1.

2.2. Apparatus, stimuli, and procedure

The apparatus and stimuli for the follow-up tests were identical to those used for the patients' first test of contrast sensitivity (Ellemberg, Lewis, Maurer et al., 1999) and for our study of visually normal children (Ellemberg, Lewis, Liu et al., 1999). Briefly, participants viewed vertical sinusoidal gratings on a green phosphor Tektronics 5130 oscilloscope CRT display that was 13° wide by 10°

Table 1
Clinical details

Patient	Eye tested	Deprivation from birth until (days)	Age at test 1 (years)	Age at 1-year follow-up (years)	Age at 2-year follow-up (years)	Snellen acuity at test 1[a]	Nystagmus when viewing binocularly?	Additional details
K.C.	Right	144	5.0	5.9		20/125	Yes	Strabismus surgery at age 2 years
J.F.	Right	100	6.6	7.6	8.6	20/35	Yes	Eye muscle surgery at age 1.6 years
V.C.	Left	202	6.2	7.2	8.2	20/200	Yes	Strabismus surgery at age 6 years
A.A.	Left	134	6.6	7.5	8.5	20/70	Yes	Strabismus surgery at age 3 years
Al.B.	Right	106	5.3	6.6	7.4	20/120	Yes	Secondary membrane surgery at age 0.9 years
An.L.	Left	139	7.4		9.4	20/80	Yes	No other surgery or complications
Ag.L.	Right	165	11.7		13.7	20/50	No	No other surgery or complications
A.B.	Right	91	7.4	8.4		20/100	No	Secondary membrane surgery at age 7 years
I.W.	Left	264	18.0	19.0		20/50	Yes	Strabismus surgery at age 6 years

[a] Measurement closest to the time of the test.

high when viewed from 57 cm. Contrast sensitivity at 0.33, 0.5, 1.0, 2.0, 3.0, 5.0, 10.0 and 20.0 cycles per degree (cpd) was measured by varying contrast from a subthreshold value up to 52%, with contrast defined as the difference between the maximum and minimum luminance in the stimulus divided by their sum. The space-average luminance of the stimuli was 9 cd/m^2. All stimuli were within the range in which contrast was linearly related to the *Z*-axis voltage of the oscilloscope.

All tests were monocular and patients viewed the display through a 3.5 mm artificial pupil designed to minimize the effect of differences among patients in the shape and size of the pupil. The deprived eye of each patient was corrected optically for the viewing distance by the patient's own contact lens and, if necessary by an additional spectacle lens mounted in a trial frame in front of the eye. The research protocol and procedures were approved by the Research Ethics Boards of McMaster University and The Hospital for Sick Children. For patients less than 16 years old, a parent signed a consent form after the procedures were explained. In addition, patients between the ages of 7 and 15 years gave verbal assent after the investigator read to them a simplified explanation. Patients 16 and older provided their own written consent.

Participants sat 57 cm from the screen, except for tests of 20 cpd, for which the testing distance was doubled. They indicated when the stimulus just appeared as contrast was increased from subthreshold values – an ascending threshold – and when it first disappeared as contrast was reduced from suprathreshold levels—a descending threshold. For each spatial frequency, we collected three ascending and three descending thresholds, with the spatial frequencies tested in random order.

2.3. Data analysis

For each patient and test point, we computed the geometric mean of the six contrast thresholds for each spatial frequency, and then took its reciprocal as the measure of contrast sensitivity (CS). Fig. 1 presents the results for each patient. Because this is a study of a small number of patients from a rare population, our conclusions are based primarily on inspection of the graphs, which show consistent patterns across similar spatial frequencies.

For calculation of the change in contrast sensitivity across tests, the data were log transformed, such that improvement was calculated as log (CS at Test $x+1$/CS at Test x). Fig. 2 presents the mean change for the patients first tested before 7.5 years of age for the six spatial frequencies that were visible to all patients (from 0.33 to 5.0 cpd). In Fig. 2, stasis is represented by a value of zero, improved sensitivity by positive values, and decreased sensitivity by negative values. Performance relative to the visually normal groups was calculated as log (CS of patient at Test *x*/CS of norm group). At each test point, each patient was compared to the visually normal group of the next lower age (e.g., a child tested at 6.7 years was compared to the norms for 6-year-olds). Fig. 3 presents the calculated deficit for each patient at each test point. Fig. 4 presents the group mean for the change in deficit across test points, with reduced deficits

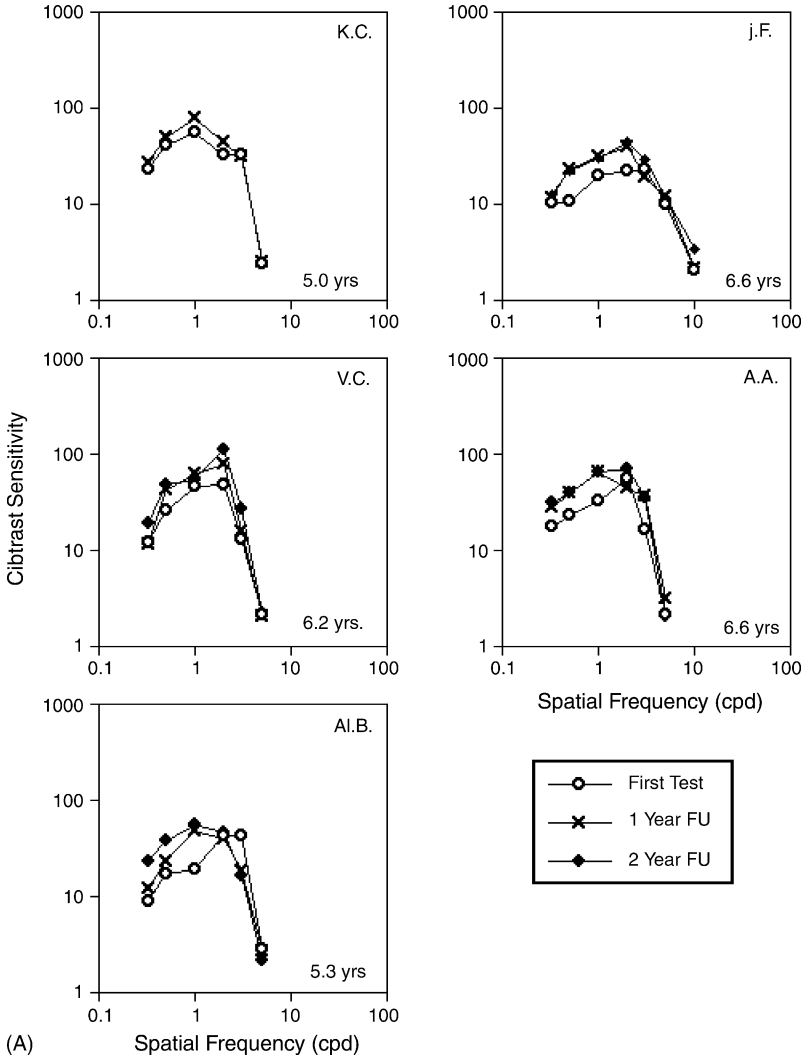

Fig. 1. The contrast sensitivity function for each patient for the first test (◯), the 1-year follow-up (×), and the 2-year follow-up (♦). Shown is the inverse of the contrast threshold for each spatial frequency, with higher values indicating better sensitivity. The number in the bottom right corner is the child's age at the first test.

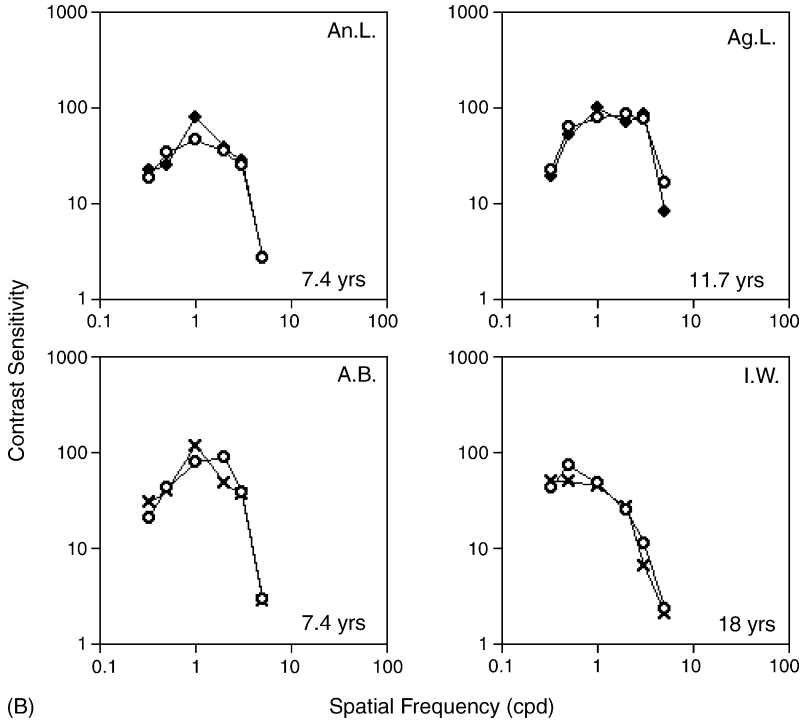

Fig. 1. (*Continued*).

represented by positive values and increased deficits represented by negative values.

3. Results

3.1. Patients first tested before 7.5 years of age

Fig. 1 indicates that, both at 1 and 2 years after the initial test, most patients first tested before 7.5 years of age improved at low spatial frequencies but did not change at higher spatial frequencies (≥5.0 cpd). Fig. 2 confirms this pattern in the mean amount of change at each spatial frequency. Fig. 3 shows that the improvement at low spatial frequencies was sufficiently large in many cases to reduce the size of the deficit compared to the control group. For example, every patient had a smaller deficit at 1 cpd when retested than at the original test. Because of the lack of improvement at higher spatial frequencies, patients' deficit tended to grow compared to the control group. For example, all but one patient had a larger deficit at 5 cpd at each retest than at the original test. Fig. 4 confirms these patterns in the group means.

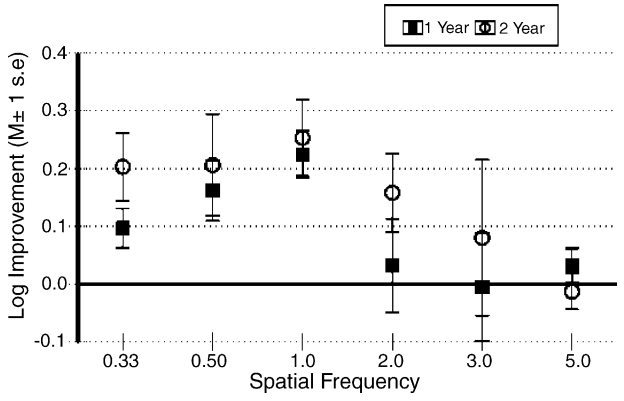

Fig. 2. The mean amount of change (±1S.E.) between the first test and the 1-year follow-up (■) and between the first test and the 2-year follow-up (◯) for the patients first tested before 7.5 years of age. Represented is the log change in contrast sensitivity between tests. Positive values represent an improvement in contrast sensitivity; negative values, a deterioration.

3.2. Influence of age of first test

Fig. 1 indicates that the contrast sensitivity of the two patients who had their first test after 11 years of age did not change systematically between tests. Inspection of the graphs indicates that the improvements at low spatial frequencies were more likely in the five patients whose first test occurred before 7 years of age than in the four patients whose first test occurred later. Note, however, that there was substantial improvement at 1 cpd evident even in the two patients first tested at 7.4 years of age and in the patient first tested at age 11.7 years.

4. Discussion

Like previous studies (Birch et al., 1998; Mioche & Perenin, 1986; Tytla et al., 1988), the results indicate that early binocular deprivation prevents the development of normal contrast sensitivity, with larger deficits at mid and high spatial frequencies than at low spatial frequencies. In fact, most patients could not see any spatial frequency above 5.0 cpd, even at the highest contrast. However, by measuring contrast sensitivity repeatedly, we demonstrated for the first time that some but not all aspects of

contrast sensitivity are still plastic in visually deprived humans during the age range from 5 to 8 years.

4.1. Substantial improvements at low spatial frequencies

At low spatial frequencies (0.33, 0.50, and 1 cpd), patients' sensitivity improved significantly over intervals of 1–2 years, keeping pace with, or even exceeding, the increases in sensitivity that occur in visually normal children between 5 and 7 years of age (Ellemberg, Lewis, Liu et al., 1999). As a result, their deficits for low spatial frequencies remained constant or even decreased: the mean deficit at the last test was 0.19, 0.24, and 0.30 log units for 0.33, 0.50, and 1.0 cpd, respectively, with a range in deficit from 0 to 0.56 log units. The improvements over 1–2 years are likely to reflect changes in the visual nervous system, rather than a practice effect, or merely an indirect benefit of the improvements in spatial attention that occur during middle childhood (reviewed in Brodeur, Trick, & Enns, 1997). An explanation of the improvement based on immature attentional control or absence of practice at the first test cannot explain why the improvement was limited to low spatial frequencies nor why this cohort performed normally or nearly normally on the first test when the same methods were used to measure sensitivity to high rates of flicker (Ellemberg, Lewis, Maurer et al., 1999).

Interestingly, the improvements occurred in the spatial frequency range that is visible to the visually normal newborn, namely spatial frequencies up to 1 cpd (reviewed in Maurer & Lewis, 2001a, 2001b). The changes at 2 and 3 cpd, spatial frequencies to which newborns do not respond, were smaller, less consistent (note standard error bars in Figs. 2 and 4), and slower (i.e., evident over the 2-year but not the 1-year interval). Changes at still higher spatial frequencies (≥5.0 cpd) were nonexistent.

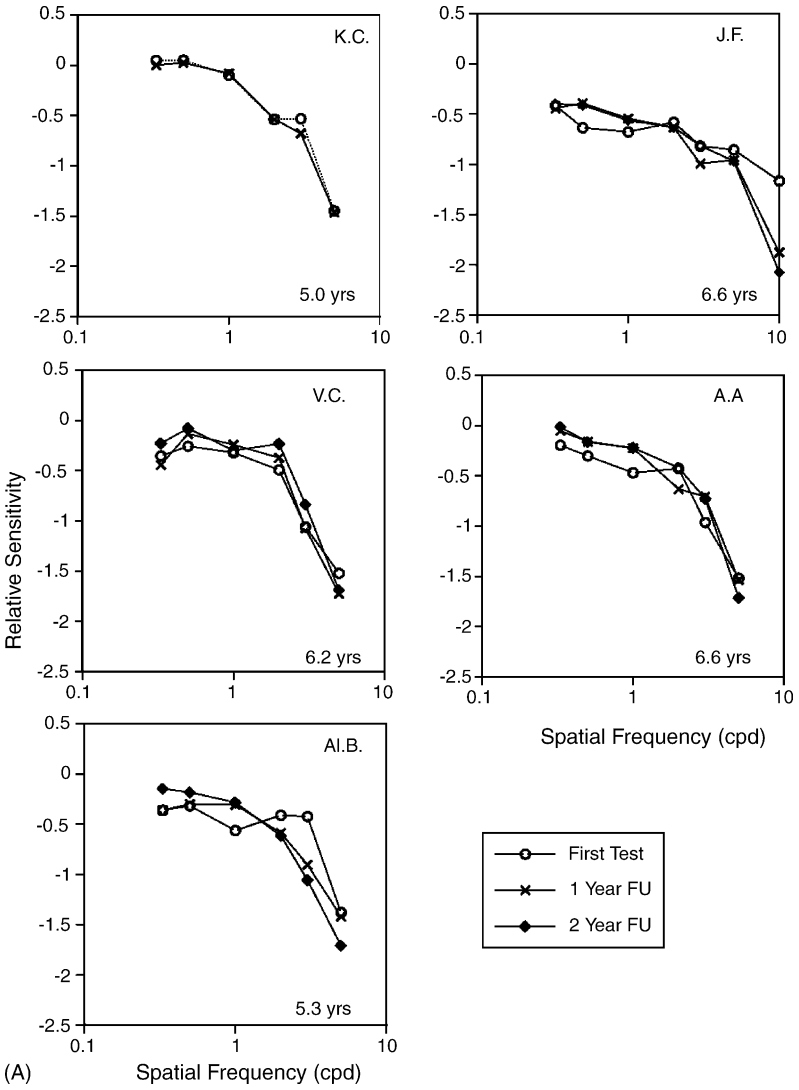

Fig. 3. The reduction in contrast sensitivity for each patient relative to the normative age group for the first test (◯), the 1-year follow-up (×), and the 2-year follow-up (♦). Shown is the ratio of the patient's contrast sensitivity to that of the visually normal group of the next lower age, plotted in log units, such that negative values indicate deficits.

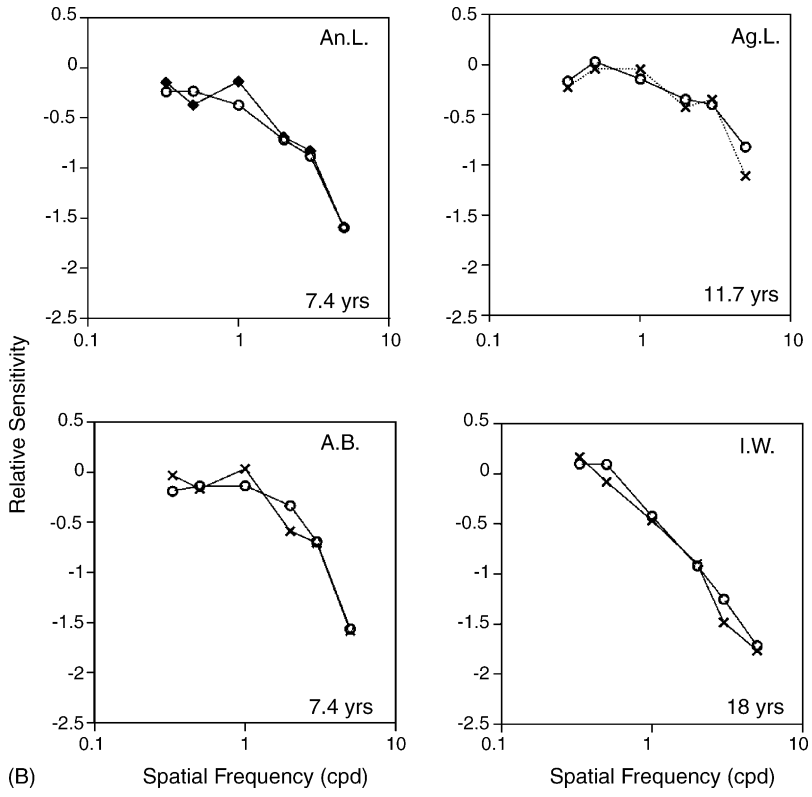

Fig. 3. (*Continued*).

The improvements at low spatial frequencies suggest that visual neurons tuned to low spatial frequencies are able to develop nearly normal sensitivity despite missing functional input during the first few months after birth. Longitudinal results from infancy on a similar cohort shed light on the mechanisms likely to be involved in this recovery: at the time of first patterned visual input, eyes treated for bilateral congenital cataract have acuity within the range of typical newborns (i.e., 0.5–1.0 cpd) rather than age mates, but after just 1 h of visual input, their acuity is significantly better (Maurer, Lewis, Brent, & Levin, 1999). At 5–7.4 years of age, the contrast sensitivity of the bilateral patients in the current study was well below normal levels for low spatial frequencies ($\leq$1.0 cpd); yet contrast sensitivity was above the level of newborns with normal eyes and the deficit shrank by the later tests (Fig. 4). Together, the results suggest that intrinsic processes not only preserve the neural architecture underlying sensitivity to low spatial frequencies during the initial period of deprivation (so that the deprived patients do not lose the newborn level of sensitivity to gratings $\leq$1.0 cpd), but also induce changes allowing accelerated change immediately after treatment and, perhaps, the changes later during childhood that are documented here. Although the data indicate that such plasticity is still apparent at age 7 years and perhaps even later, Fig. 1 suggests that it is stronger before 7 years of age than later. The sample did not include the distribution of ages necessary to determine when the plasticity wanes or when it ceases and it is possible that improvements at low spatial frequencies continue past 7.5 years of age.

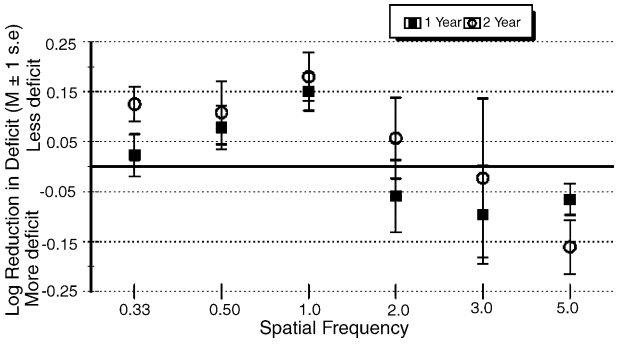

Fig. 4. The mean reduction (±1S.E.) in the size of the patients' deficit between the first test and the 1-year follow-up (■) and between the first test and the 2-year follow-up (◯) for patients first tested before 7.5 years of age. Represented is the log reduction in deficit compared to visually normal controls. Positive values indicate a reduction in the size of the deficit on retest; negative values indicate an increase in the size of the deficit on the retest.

4.2. Stasis at high spatial frequencies

Unlike visually normal children, patients' sensitivity for spatial frequencies of 5.0 cpd and above did not change during this period, thereby leading to greater deficits.

The patients' failure to improve substantially at mid and high spatial frequencies after 5 years of age might be related to the continued mild visual deprivation that they experience: treatment involved removing the natural lens from the eye, rendering it *aphakic*, and the compensatory correction by contact

lenses, combined with bifocal glasses, provide only two planes of focus, one at far and one at near. Objects at other distances are blurred—and hence do not provide optimally focused patterned visual input to the retina. One possibility is that this continuing mild deprivation prevents improvements in contrast sensitivity after age 5, especially for high spatial frequencies. Although theoretically plausible, empirical evidence argues against this explanation: there are reports of patients who were aphakic during this age period, because of earlier treatment for partial or total cataract, who achieved 20/20 letter acuity (i.e., normal sensitivity to high spatial frequencies) (Kugelberg, 1992; Magnusson et al., 2002). Thus, the continuing mild deprivation from aphakia does not prevent normal development of sensitivity to high spatial frequencies. We cannot rule out a contribution from the nystagmus – short, jerky eye movements that prevent stable fixation – that is present in most of the bilateral patients (see Table 1), except to note that the two patients with a first test at 7.4 years of age performed similarly even though one had nystagmus (An.L.) and the other did not (A.B.) when viewing binocularly. Thus, the lack of improvement at high and mid spatial frequencies is likely to have been caused by the initial visual deprivation, rather than visual abnormalities in the age range from 5 to 8 years.

For spatial frequencies of 10 cpd and above, most patients were unable to detect the gratings even at maximum contrast at any test point. In other words, neurons tuned to high spatial frequencies appear not to develop when patterned visual input is missing during the first 3–9 months of life. The visually normal child first shows behavioural evidence of sensitivity to spatial frequencies of 10 cpd at 18–24 months of age (reviewed in Maurer & Lewis, 2001a, 2001b). Thus, visual input in the first 6 months of life is necessary to set up, or preserve, the neural substrate that will later mediate sensitivity to high spatial frequencies. Studies of children who had a period of postnatal visual deprivation because of developmental or traumatic cataracts, indicate that the fine-tuning of that neural substrate also depends on patterned visual input: children do not develop normal sensitivity to high spatial frequencies if there is visual deprivation during any part of the period of normal development of contrast sensitivity (up to 7 years of age) or for a few years beyond (up to about 10 years of age) (Lewis & Maurer, 2005; Vaegan & Taylor, 1979).

4.3. Small changes at mid spatial frequencies

Unlike high spatial frequencies, every patient was able to detect spatial frequencies near the peak of the normal contrast sensitivity function (2–3 cpd), a range of frequencies to which visually normal infants first become sensitive between 3 and 6 months of age (reviewed in Maurer & Lewis, 2001a, 2001b). However, patients' sensitivity in this range improved less dramatically and consistently than at lower spatial frequencies. This was true despite the fact that most of the patients were still in the period of continuing normal development (5–7 years of age; Ellemberg, Lewis, Liu et al., 1999). As a result, their deficit did not shrink with increasing age, as was true at lower spatial frequencies, and, in fact, in some cases increased (see Fig. 3). This sleeper effect implies that early visual input sets up the conditions for the development of peak contrast sensitivity later in life. When that input is missing, the brain will still have neurons tuned to mid spatial frequencies, but they will have higher-than-normal thresholds. Because of the distribution of cases in our sample, we cannot rule out the possibility that that there is some improvement at mid spatial frequencies – albeit not normalization (Mioche & Perenin, 1986) – after 7.5 years of age either because the recovery processes are slower to develop for mid than for low spatial frequencies or because they depend on the earlier changes at low spatial frequencies.

4.4. Developmental mechanisms

The monkey model for deprivation amblyopia indicates that the failure to improve after early visual deprivation is likely to reflect changes at the cortical level. In monkeys reared with one or both eyelids sutured shut during early infancy, as in visually deprived humans, there are reductions in contrast sensitivity, especially at medium and high spatial frequencies (Harwerth, Smith, Paul, Crawford, & von Noorden, 1991). Despite the early deprivation, there are no observable anatomical changes in photoreceptors or retinal ganglion cells (Boothe, Dobson, & Teller, 1985; Clark, Hendrickson, & Curcio, 1988; Hendrickson & Boothe, 1976) or in the electrophysiological properties of neurons in the lateral geniculate nucleus, including the distribution of optimal spatial frequencies to drive each cell (Blakemore & Vital-Durant, 1986; Levitt, Movshon, Sherman, & Spear, 1989). Instead changes are evident at the level of the visual cortex: cortical neurons respond sluggishly to visual stimulation and the population of cells has reduced sensitivity to high spatial frequencies and to low contrast (Blakemore, 1990; Blakemore & Vital-Durand, 1983; Crawford, Blake, Cool, & von Noorden, 1975; reviewed in Movshon & Kiorpes, 1993). One possibility is that early visual input induces changes in visual cortical neurons, thereby creating the neural substrate underlying later improvements for mid and high spatial frequencies. It is more likely that it preserves and enhances a neural substrate that developed prenatally based on genetic coding and spontaneous retinal activity (reviewed in Katz & Shatz, 1996). Recent evidence from a variety of species indicates that, before any visual input, visual cortical neurons have some adult-like properties and have an adult-like arrangement of clusters responding preferentially to input from each eye (Crair, 1999; Crair, Gillespie, & Stryker, 1998; Crair, Horton, Antonini, & Stryker, 2001; Crowley & Katz, 1999; Ruthazer, 2005). Early visual deprivation may prevent normal development by altering the intrinsically specified substrate. It could do so by allowing the substrate to be used for processing non-visual inputs, as occurs in the congenitally blind (reviewed in Maurer et al., 2005)—through Hebbian competition among inputs from different sensory modalities that causes the unused visual synapses to be pruned and/or inhibited (Pascual-Leone & Hamilton, 2001; reviewed in Maurer & Mondloch, in press). Alternatively, or in addition, early visual deprivation could induce the use of pathways that bypass the intrinsically specified substrate in the primary visual cortex, as occurs in hood-reared cats (Zablocka & Zernicki, 1996; Zablocka, Zernicki, & Kosmal, 1976; Zablocka, Zernicki, &

Kosmal, 1980). In such cats, vision appears to be mediated by pathways that bypass the primary visual cortex and send inputs to higher visual areas through the superior colliculus and pretectum. Neurons in those alternative pathways are likely to have lower limits on the acuity and contrast sensitivity that they can mediate.

5. Conclusions

In summary, the results indicate that early visual input is not necessary for the later development of nearly normal sensitivity to low spatial frequencies, despite the fact that these are the spatial frequencies to which the visually normal infant responds. For those low spatial frequencies, there is recovery from early visual deprivation that continues into middle childhood, until at least age 7 years of age. Instead, early visual input is necessary to set up the neural substrate underlying the later development of any sensitivity to high spatial frequencies and the later development of normal sensitivity to mid spatial frequencies. Despite the fact that the visually normal newborn cannot yet detect high spatial frequencies at any contrast, early visual input is necessary if later development is to proceed normally. Greater recovery might be possible—if treatment was even earlier, or if children received visual training with feedback in addition to exposure to the normal visual world. Nevertheless, our results indicate the importance of early visual input in creating the conditions for later plasticity.

Acknowledgements

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