



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.

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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 (Elleberg, 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; Elleberg, 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 (Elleberg, 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 (Elleberg, 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, Liu et al., 1999; 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.

139 high when viewed from 57 cm. Contrast sensitivity at 0.33, 0.5, 1.0, 2.0, 3.0,
 140 5.0, 10.0 and 20.0 cycles per degree (cpd) was measured by varying contrast
 141 from a subthreshold value up to 52%, with contrast defined as the difference
 142 between the maximum and minimum luminance in the stimulus divided by their
 143 sum. The space-average luminance of the stimuli was 9 cpd/m². All stimuli were
 144 within the range in which contrast was linearly related to the Z-axis voltage of
 145 the oscilloscope.

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

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

162 ascending and three descending thresholds, with the spatial frequencies tested
 163 in random order.

2.3. Data analysis 164

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

171 For calculation of the change in contrast sensitivity across tests, the data
 172 were log transformed, such that improvement was calculated as log (CS at Test
 173 $x + 1/CS$ at Test x). Fig. 2 presents the mean change for the patients first tested
 174 before 7.5 years of age for the six spatial frequencies that were visible to all
 175 patients (from 0.33 to 5.0 cpd). In Fig. 2, stasis is represented by a value of zero,
 176 improved sensitivity by positive values, and decreased sensitivity by negative
 177 values. Performance relative to the visually normal groups was calculated as
 178 log (CS of patient at Test x/CS of norm group). At each test point, each patient
 179 was compared to the visually normal group of the next lower age (e.g., a child
 180 tested at 6.7 years was compared to the norms for 6 years old). Fig. 3 presents
 181 the calculated deficit for each patient at each test point. Fig. 4 presents the
 182 group mean for the change in deficit across test points, with reduced deficits

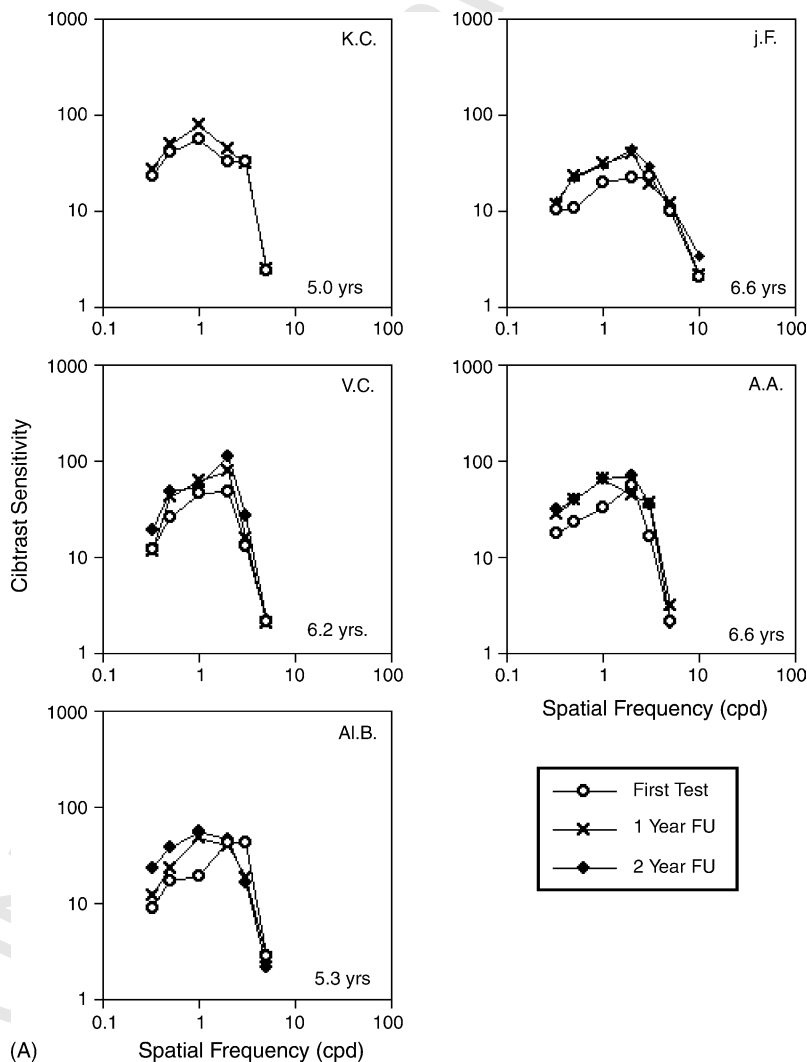


Fig. 1. The contrast sensitivity function for each patient for the first test (○), the 1-year follow-up (×), and the 2-year follow-up (◆). Shown is the inverse of the contrast threshold for each spatial frequency, with higher values indicating better sensitivity. The number in the bottom right corner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

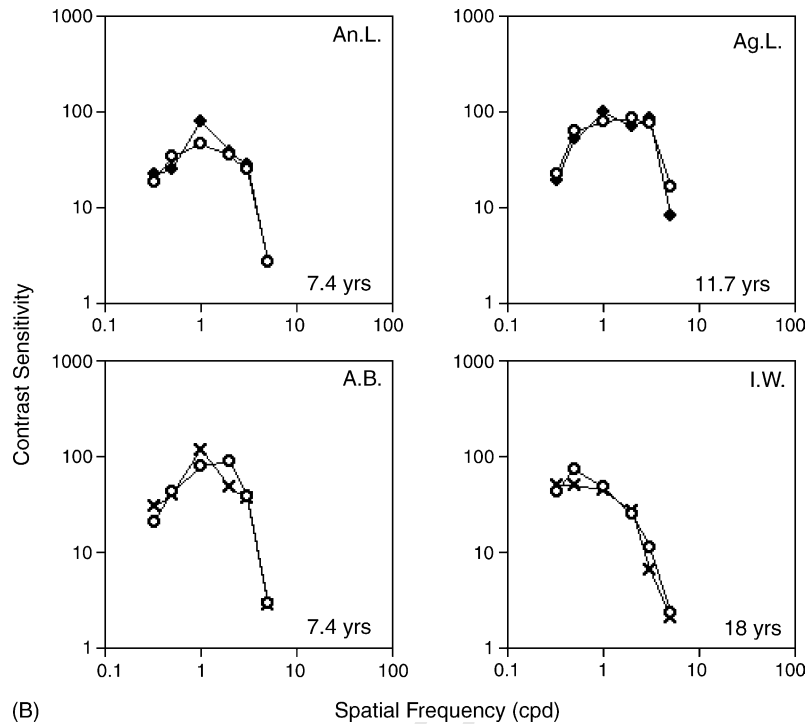


Fig. 1. (Continued).

183 represented by positive values and increased deficits represented by negative values.
184

185 3. Results

186 3.1. Patients first tested before 7.5 years of age

187 Fig. 1 indicates that, both at 1 and 2 years after the initial
188 test, most patients first tested before 7.5 years of age improved
189 at low spatial frequencies but did not change at higher spatial
190 frequencies (≥ 5.0 cpd). Fig. 2 confirms this pattern in the mean
191 amount of change at each spatial frequency. Fig. 3 shows that
192 the improvement at low spatial frequencies was sufficiently large

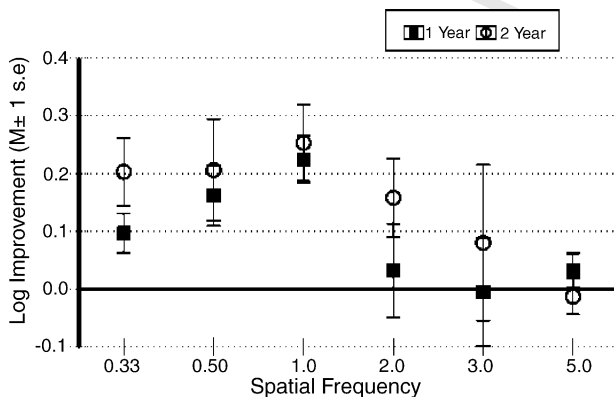


Fig. 2. The mean amount of change (± 1 s.e.) between the first test and the 1-year follow-up (■) and between the first test and the 2-year follow-up (○) for the patients first tested before 7.5 years of age. Represented is the log change in contrast sensitivity between tests. Positive values represent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

193 in many cases to reduce the size of the deficit compared to the
194 control group. For example, every patient had a smaller deficit at
195 1 cpd when retested than at the original test. Because of the lack of
196 improvement at higher spatial frequencies, patients' deficit
197 tended to grow compared to the control group. For example, all
198 but one patient had a larger deficit at 5 cpd at each retest than
199 at the original test. Fig. 4 confirms these patterns in the group
200 means.

201 3.2. Influence of age of first test

202 Fig. 1 indicates that the contrast sensitivity of the two patients
203 who had their first test after 11 years of age did not change sys-
204 tematically between tests. Inspection of the graphs indicates that
205 the improvements at low spatial frequencies were more likely
206 in the five patients whose first test occurred before 7 years of
207 age than in the four patients whose first test occurred later. Note,
208 however, that there was substantial improvement at 1 cpd evi-
209 dent even in the two patients first tested at 7.4 years of age
210 and in the patient first tested at age 11.7 years.

211 4. Discussion

212 Like previous studies (Birch et al., 1998; Mioche & Perenin,
213 1986; Tytla et al., 1988), the results indicate that early binocular
214 deprivation prevents the development of normal contrast sensi-
215 tivity, with larger deficits at mid and high spatial frequencies
216 than at low spatial frequencies. In fact, most patients could not
217 see any spatial frequency above 5.0 cpd, even at the highest con-
218 trast. However, by measuring contrast sensitivity repeatedly, we
219 demonstrated for the first time that some but not all aspects of

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

222 4.1. Substantial improvements at low spatial frequencies

223 At low spatial frequencies (0.33, 0.50, and 1 cpd), patients’
 224 sensitivity improved significantly over intervals of 1–2 years,
 225 keeping pace with, or even exceeding, the increases in sensitivi-
 226 ty that occur in visually normal children between 5 and 7 years
 227 of age (Ellemberg, Lewis, Liu et al., 1999). As a result, their
 228 deficits for low spatial frequencies remained constant or even
 229 decreased: the mean deficit at the last test was 0.19, 0.24, and
 230 0.30 log units for 0.33, 0.50, and 1.0 cpd, respectively, with a
 231 range in deficit from 0 to 0.56 log units. The improvements over
 232 1–2 years are likely to reflect changes in the visual nervous sys-
 233 tem, rather than a practice effect, or merely an indirect benefit
 234 of the improvements in spatial attention that occur during mid-

235 dle childhood (reviewed in Brodeur, Trick, & Enns, 1997). An
 236 explanation of the improvement based on immature attentional
 237 control or absence of practice at the first test cannot explain
 238 why the improvement was limited to low spatial frequencies nor
 239 why this cohort performed normally or nearly normally on the
 240 first test when the same methods were used to measure sensi-
 241 tivity to high rates of flicker (Ellemberg, Lewis, Maurer et al.,
 242 1999).

243 Interestingly, the improvements occurred in the spatial fre-
 244 quency range that is visible to the visually normal newborn,
 245 namely spatial frequencies up to 1 cpd (reviewed in Maurer
 246 & Lewis, 2001a, 2001b). The changes at 2 and 3 cpd, spatial
 247 frequencies to which newborns do not respond, were smaller,
 248 less consistent (note standard error bars in Figs. 2–3), and
 249 slower (i.e., evident over the 2-year but not the 1-year interval).
 250 Changes at still higher spatial frequencies (≥ 5.0 cpd) were non-
 251 existent.

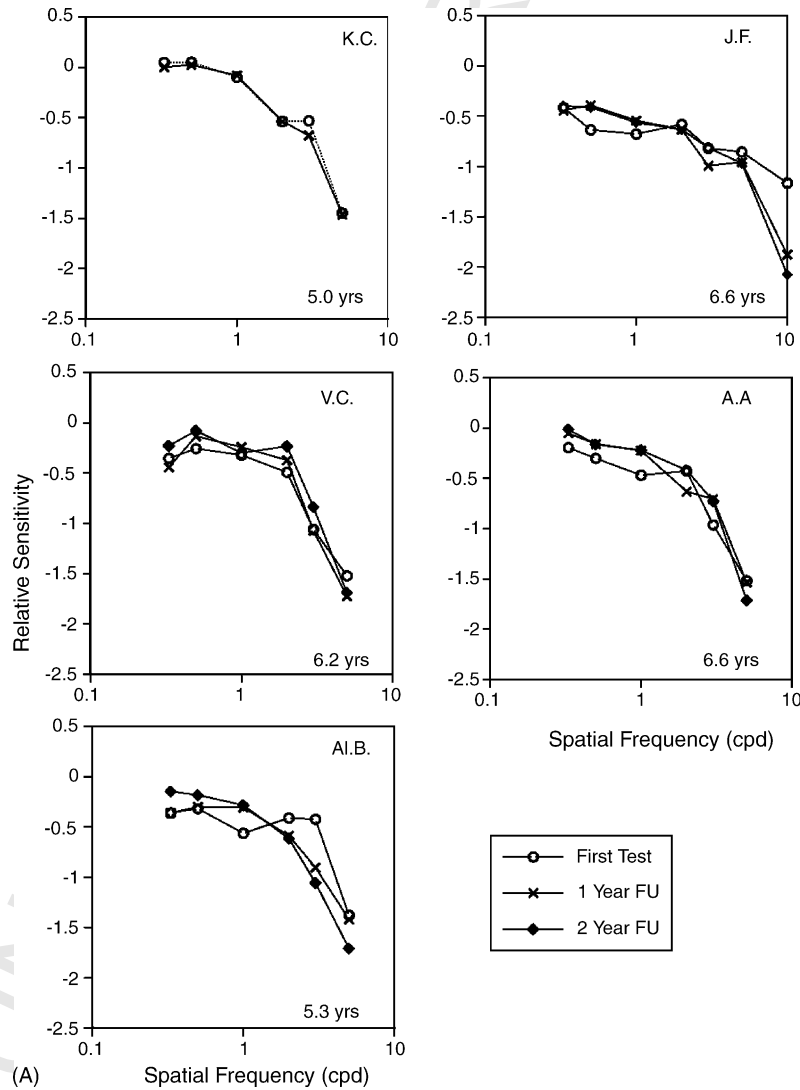


Fig. 3. The reduction in contrast sensitivity for each patient relative to the normative age group for the first test (○), the 1-year follow-up (×), and the 2-year follow-up (◆). Shown is the ratio of the patient’s contrast sensitivity to that of the visually normal group of the next lower age, plotted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

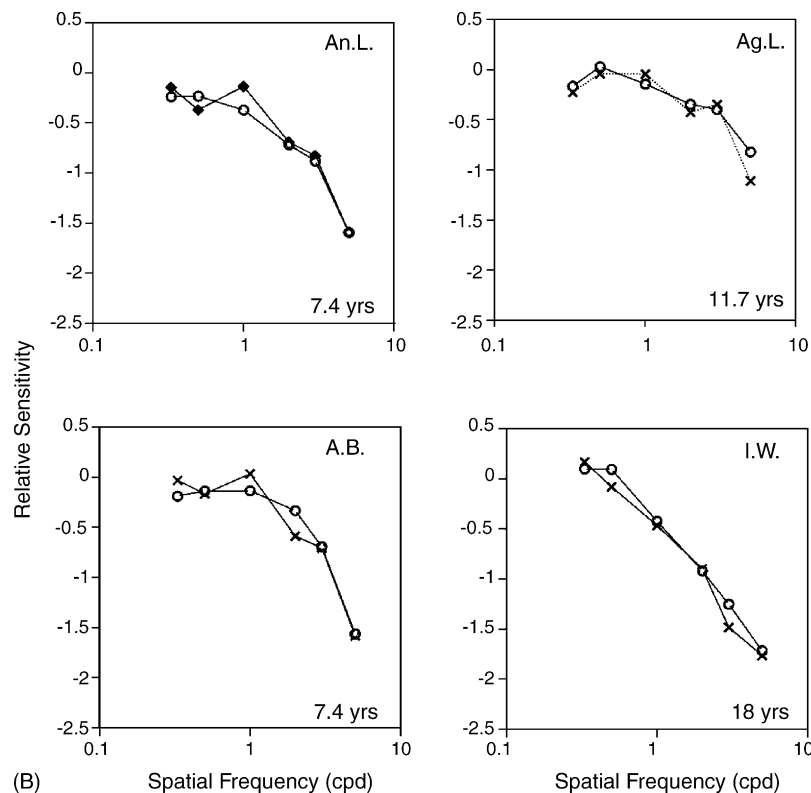


Fig. 3. (Continued).

252 The improvements at low spatial frequencies suggest that
 253 visual neurons tuned to low spatial frequencies are able to
 254 develop nearly normal sensitivity despite missing functional
 255 input during the first few months after birth. Longitudinal results
 256 from infancy on a similar cohort shed light on the mechanisms
 257 likely to be involved in this recovery: at the time of first patterned
 258 visual input, eyes treated for bilateral congenital cataract have
 259 acuity within the range of typical newborns (i.e., 0.5–1.0 cpd)
 260 rather than age mates, but after just 1 h of visual input, their
 261 acuity is significantly better (Maurer, Lewis, Brent, & Levin,
 262 1999). At 5–7.4 years of age, the contrast sensitivity of the bilateral
 263 patients in the current study was well below normal levels
 264 for low spatial frequencies (≤ 1.0 cpd); yet contrast sensitivity
 265 was above the level of newborns with normal eyes and the deficit
 266 shrank by the later tests (Fig. 4). Together, the results suggest
 267 that intrinsic processes not only preserve the neural architecture
 268 underlying sensitivity to low spatial frequencies during the initial
 269 period of deprivation (so that the deprived patients do not
 270 lose the newborn level of sensitivity to gratings ≤ 1.0 cpd), but
 271 also induce changes allowing accelerated change immediately
 272 after treatment and, perhaps, the changes later during childhood
 273 that are documented here. Although the data indicate that such
 274 plasticity is still apparent at age 7 years and perhaps even later,
 275 Fig. 1 suggests that it is stronger before 7 years of age than later.
 276 The sample did not include the distribution of ages necessary to
 277 determine when the plasticity wanes or when it ceases and it is
 278 possible that improvements at low spatial frequencies continue
 past 7.5 years of age.

4.2. Stasis at high spatial frequencies

279 Unlike visually normal children, patients' sensitivity for spa-
 280 tial frequencies of 5.0 cpd and above did not change during this
 281 period, thereby leading to greater deficits. 282

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

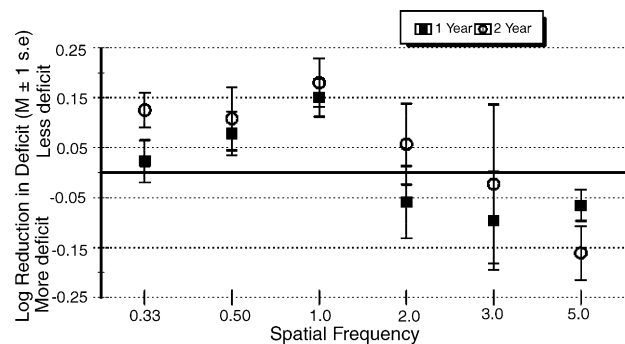


Fig. 4. The mean reduction (± 1 s.e.) in the size of the patients' deficit between the first test and the 1-year follow-up (■) and between the first test and the 2-year follow-up (○) for patients first tested before 7.5 years of age. Represented is the log reduction in deficit compared to visually normal 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.

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 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-Durant, 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.

Uncited reference

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