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Section 5

Spatial and temporal vision in patients treated for bilateral congenital cataracts

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Abstract

Using the method of limits, we measured spatial and temporal vision in 13 children who had been deprived of patterned visual input during infancy until they were treated for dense central cataracts in both eyes. Spatial vision was assessed with vertical sine-wave gratings, and temporal vision was assessed with an unpatterned luminance field sinusoidally modulated over time. Under these testing conditions, spatial contrast sensitivity at low and medium spatial frequencies (0.33–2 c deg^{-1}) was within normal limits, but sensitivity at higher spatial frequencies and grating acuity were reduced on average by 1.3 and 0.5 log units, respectively. Temporal vision was affected less severely, with losses in sensitivity only for low temporal frequencies (5 and 10 Hz), which averaged 0.4 log units. Thus, spatial and temporal vision are likely mediated by different neural mechanisms, that are differentially affected by deprivation.

Keywords: Children; Visual deprivation; Binocular congenital cataract; Spatial contrast sensitivity; Grating acuity; Temporal contrast sensitivity; Critical flicker fusion frequency

Spatial and temporal vision mature at different rates and attain adult levels at different ages (Banks, 1982; Ellemberg, Lewis, Liu & Maurer, 1999). At 6 months of age, grating acuity falls short of that of adults by nearly a factor of 8 (e.g. van Hof-van Duin & Mohn, 1986), and it does not attain adult levels before 4–6 years of age (Mayer & Dobson, 1982; Ellemberg et al., 1999). In contrast, critical flicker fusion frequency is already quite mature in the 1-month old at 41 Hz, and improves rapidly to attain adult levels (51 Hz) by 3 months of age (Regal, 1981). Although the 3-month-old's spatial contrast sensitivity (Atkinson, Braddick & Moar, 1977; Banks & Salapatek, 1978, 1981; Peterzell, Werner & Kaplan, 1995) and temporal contrast sensitivity (Swanson & Birch, 1990; Hartmann & Banks, 1992; Teller, Lindsey, Mar, Succop & Mahal, 1992; Rasengane, Allen & Manny, 1997) are both 1.0 log lower than in the adult, spatial contrast sensitivity develops more slowly: at 4 years of age sensitivity across the visible range of spatial frequencies is still 0.5 log units lower than in adults whereas temporal contrast sensitivity is mature at higher temporal frequencies and is only 0.25 log units lower than adult values at lower temporal frequencies (Ellemberg et al., 1999).

Because spatial and temporal vision mature at different rates, we predicted that they might be differentially affected by early binocular deprivation (Maurer & Lewis, 1993). Although three previous studies of binocularly deprived humans have documented losses in spatial vision (Mioche & Perenin, 1986; Tytla, Maurer, Lewis & Brent, 1988; Birch, Stager, Leffler & Weakley, 1998), the only study to measure both their spatial and temporal vision included only four patients (Mioche & Perenin, 1986). Although the authors of that study made no comparison, inspection of their figures suggests that the losses in spatial contrast sensitivity were greater than the losses in temporal contrast sensitivity. However, Mioche and Perenin (1986) tested temporal contrast sensitivity with a temporally modulated 0.3 c

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deg^{-1} grating, a stimulus that would tap only the subset of neural mechanisms grossly tuned to that spatial frequency. To overcome this limitation, we measured temporal contrast sensitivity with a uniform luminance field in order to assess the overall temporal sensitivity of the visual system.

One study compared spatial and temporal vision in monkeys that had been binocularly deprived for varying durations beginning at 1 month of age (Harwerth, Smith, Paul, Crawford & von Noorden, 1991). Like humans binocularly deprived from birth (Mioche & Perenin, 1986; Tytla et al., 1988; Birch et al., 1998), the monkeys showed severe losses in spatial contrast sensitivity, especially at medium and high spatial frequencies. Interestingly, binocularly deprived monkeys had little if any deficit for temporal contrast sensitivity, as assessed with a uniform field flicker (Harwerth et al., 1991).

Overall, the findings indicate that early binocular deprivation in both humans and monkeys depresses spatial vision. In monkeys, such deprivation has little or no effect on the development of temporal contrast sensitivity, at least when deprivation begins at 1 month of age. The goal of our study was to determine whether spatial and temporal vision are affected similarly by binocular deprivation in humans. We measured spatial and temporal contrast sensitivity, grating acuity and critical flicker fusion frequency in 13 patients who had been binocularly deprived of patterned visual input during early infancy by dense central cataracts. The cataracts were treated during infancy by surgically removing the natural lens of the eye and replacing it with a contact lens to restore nearly normal visual input. We compared the results to those of 13 age-matched normal children and adults tested under the same conditions. To determine if there are any effects of aphakia (absence of a natural lens) and its optical correction, we also tested two patients who had normal vision until an eye injury that led to a traumatic cataract after 11 years of age.

1. Methods

1.1. Subjects

1.1.1. Congenital cases

A total of 13 patients treated for bilateral congenital cataracts (mean age at test = 9.2 years, range = 5.0–18.8 years) participated in this study. The clinical details of the patients are provided in Table 1. To be included in the study, the patients had to meet all of the following criteria: dense central cataracts in both eyes diagnosed on the first eye exam and by 6 months of age; no abnormalities in the ocular media or the retina, including no evidence of persistent hyperplastic primary vitreous, and no ocular disease such as glaucoma. Patients who did not wear their optical correction regularly after treatment (at least 75% of the time) were also excluded. We did include patients with common associated abnormalities such as strabismus, nystagmus or microcornea.

We assumed that any child who had dense central cataracts diagnosed on the first eye exam before 6 months of age had been deprived from birth because it would be unusual to have dense cataracts develop rapidly between birth and 6 months. Consequently, we defined the duration of deprivation as the period extending from birth until the age of first optical correction following surgery to remove the cataract (i.e. the first time the infant received focused visual input onto the retina).

1.1.2. Aphakic controls

To assess the effects of aphakia on spatial and temporal vision, we also tested two patients who had a normal visual history until they developed a dense central traumatic cataract as a result of an eye injury. Like the congenital cases, the aphakic controls had the cataracts treated by surgically removing the natural lens of the eye and replacing it with a contact lens to restore nearly normal visual input. Clinical details of the aphakic controls are provided in Table 1.

1.1.3. Normal controls

The results from each of the 15 patients (13 congenital cases and two aphakic controls) were compared to those of age-matched normal controls tested under identical conditions. All had no history of eye problems and all met our criteria on a visual screening exam (for criteria, see Ellemberg et al., 1999).

1.2. Apparatus and stimuli

The apparatus and stimuli were identical to those reported previously for normal children and adults (Ellemberg et al., 1999). Briefly, spatial vision was measured with vertical sinusoidal gratings generated on a green phosphor Tektronix 5130 oscilloscope CRT display by Z-axis modulation. The gratings were 13 deg wide and 10 deg high when viewed from a distance of 57 cm. Spatial contrast sensitivity was measured at 0.33, 0.5, 1, 2, 3, 5, 10 and 20 c deg^{-1}. Grating acuity was assessed with a contrast level of 52%. The contrast of the stimuli was defined as the difference between maximum and minimum luminance divided by their sum.

Temporal vision was measured with a spatially unpatterned light display, the luminance of which was varied over time with a sinusoidal function generator. The unpatterned luminance field was 50° in diameter when viewed from a distance of 57 cm. Temporal

contrast sensitivity was measured at 5, 10, 20 and 30 Hz. Critical flicker fusion frequency was assessed at a contrast level of 65%.

The space- and time-average luminances of the test stimuli were 9 cd m^{-2}. The linearity of the stimuli was verified by using a Minolta LS-100 photometer. All stimuli were within the range in which contrast was linearly related to the Z-axis voltage (i.e. 52 and 65% for spatial and temporal contrast stimuli, respectively).

1.3. Procedure

The procedure was identical to that of Ellemberg et al. (1999) except that: (i) each eye of the patients was corrected optically for the viewing distance by mounting the appropriate corrective lens in a trial frame; and (ii) all participants viewed the display through a 3.5 mm artificial pupil in order to minimize the differences among patients in the shape and size of the pupil. Briefly, participants had one eye patched with 3M Micropore™ tape (half were tested first with the left eye and the remaining half were tested first with the right eye). The method of limits was used to measure both spatial and temporal thresholds. Spatial contrast sensitivity was assessed at a viewing distance of 57 cm for spatial frequencies ranging from.33 to 10 c deg^{-1}, and at twice that distance for spatial frequencies of 20 c deg^{-1}. Three ascending and three descending thresholds were recorded for each spatial frequency, with the ascending thresholds measured first. The frequencies were tested in a random order. Grating acuity was assessed at a viewing distance of 228 cm. Three ascending-descending thresholds were recorded, with ascending thresholds measured first.

Table 1
Clinical details of the patients. Congenital cases are in order of increased deprivation

Patient (age at test-years)	Refraction[a]		Age of diagnosis/ contact lenses (days)	Snellen acuity[a]	Nystagmus	Additional details
Congenital cases						
M.M. (5.3)	OD[b]	+13.50	12/48	20/70	Manifest	No other surgery or complications
	OS	+18.00		20/100	OU	
A.B. (7.5)	OD	+14.75	61/91	20/100	Latent	Secondary membrane surgery at age 7
	OS	+16.00		20/100	OU	years
J.S. (5.0)	OD	+27.00	61/92	20/70	Manifest	Strabismus surgery for LET/RET at
	OS	+30.00		20/70	OU	age 3 years
J.F. (6.7)	OD	+22.00	77/100	20/35	Manifest	Microcornea OU; ocular muscle surgery
	OS	+20.00		20/200	OU	OU at age 1.6 years
Al.B. (5.5)	OD	+23.50	63/106	20/120	Manifest	Microcornea OU; Secondary membrane
	OS	+28.00		20/140	OU	surgery at age 0.9 year
A.A. (6.7)	OD	+15.50	104/134	20/100	Manifest	Microcornea OU; strabismus surgery
	OS	+13.50		20/70	OU	for RET at age 3 years
An.L. (7.5)	OD	+16.25	Birth/139	20/100	Manifest	No other surgery or complications
	OS	+16.00		20/80	OU	
K.C. (5.0)	OD	+31.00	100/144	20/125	Manifest	Microcornea OU; strabismus surgery
	OS	+32.00		20/140	OU	for LET at age 2 years
Ag.L. (11.7)	OD	+16.50	61/165	20/50	Latent	No other surgery or complications
	OS	+15.50		20/100	OS	
A.C. (18.8)	OD	+11.25	123/196	20/40	Manifest	No other surgery or complications
	OS	+12.25	123/161	20/50	OU	
C.P. (17.2)	OD	+13.00	143/187	20/50	Latent	Strabismus surgery for LET at age 1.8
	OS	+14.50		20/30	OU	years
V.C. (5.3)	OD	+19.00	158/202	20/100	Manifest	Microcornea OU; strabismus surgery
	OS	+20.00		20/200	OU	for LET at age 6.0 years
I.W. (18)	OD	+11.75	92/151	20/70	Manifest	Strabismus surgery for LET/RET at
	OS	+12.75	92/264	20/50	OU	age 6.0 years
Aphakic controls						
M.B. (15)	OD	+12.00	11 years of age	20/30	None	No other surgery or complications
W.C. (17.8)	OS	+13.00	14 years of age	20/20	None	No other surgery or complications

[a] Measurement closest to the time of the test.
[b] OD, right eye; OS, left eye; OU, both eyes; RET, right esotropia; LET, left esotropia.

Temporal contrast sensitivity was assessed from a viewing distance of 57 cm. Three ascending thresholds were taken for each temporal frequency, with the frequencies tested in a random order (we measured only ascending thresholds because afterimages cause unpatterned flicker to persist after the flicker has stopped). Critical flicker fusion frequency was also assessed at a viewing distance of 57 cm, but both ascending and descending thresholds were measured, in accordance with the classical literature (De Lange, 1952, 1954). Three ascending and three descending thresholds were recorded, with the ascending thresholds always measured first.

Half of the subjects first received the tests for grating acuity and contrast sensitivity, with grating acuity always measured first. The remaining half first received the tests for critical flicker fusion frequency and temporal contrast sensitivity, with critical flicker fusion frequency always measured first. Age-matched normal controls received the same testing and eye order as did the patient with whom they were matched.

1.4. Data analysis

For analysis, the thresholds were log transformed. Spatial and temporal contrast sensitivity at each frequency were derived by taking the reciprocal of the geometric mean of the recorded contrast thresholds.

The data for each patient are plotted as relative sensitivities, which is defined in the following way:

$$\text{Relative sensitivity} = \log\left(\frac{\text{patient's sensitivity}}{\text{mean normal sensitivity}}\right)$$

To assess any effect of duration of the deprivation, we used regression analyses to compute its effect on acuity, critical flicker fusion frequency, spatial contrast sensitivity at 5 c deg^{-1} (the highest spatial frequency to which all deprived eyes were sensitive), and temporal contrast sensitivity at 5 Hz (where losses were greatest). We calculated each regression twice, once using each patient's better eye (as determined by Snellen acuity), and once using the worse eye. When acuity was equal, we selected the eyes randomly for these analyses.

2. Results

2.1. Congenital cases

2.1.1. Spatial vision

Figure 1 presents the losses in spatial contrast sensitivity of the 26 eyes from 13 patients treated for bilateral congenital cataracts. The ordinate value of zero on each panel indicates that the patient's sensitivity is equal to that of the age-matched control. Negative values, in turn, indicate that the patient's sensitivity was lower than that of the control subject. All subsequent data from both spatial and temporal contrast sensitivity will be presented in this way.

There are several similarities across patients. Most had little if any loss in sensitivity at low and medium spatial frequencies (from 0.33 to 2 c deg^{-1}). The losses in sensitivity increased monotonically with progressively higher spatial frequencies, with the maximum reduction in sensitivity ranging from 1.0 to 1.5 log across eyes. There was no relationship between duration of deprivation and spatial contrast sensitivity at 5 c deg^{-1} ($r = 0.10$, $P > 0.1$).

Figure 2 shows the loss in grating acuity for each of the 13 congenital cases relative to age-matched normals, plotted as a function of the duration of deprivation. All patients suffered reductions in grating acuity which averaged approximately 0.5 log units, or approximately 1.5 octaves. There was no relationship between the losses in grating acuity and the duration of deprivation ($r = 0.04$, $P > 0.1$).

2.1.2. Temporal vision

Losses in temporal contrast sensitivity for the 13 congenital cases are shown in Fig. 3. All patients were able to detect the entire range of temporal frequencies tested, and about half performed within normal limits at every temporal frequency. For all patients, the losses in sensitivity increased monotonically with progressively lower temporal frequencies, with little if any loss at medium and high temporal frequencies (20–30 Hz), and mild reductions at lower temporal frequencies, which averaged about 0.4 log units. The regression analyses indicated no relationship between the duration of deprivation and the amount of loss at the lowest temporal frequency ($r = 0.10$, $P > 0.1$).

The mean critical flicker fusion frequencies of each eye of the congenital cases are presented in Fig. 4. Critical flicker fusion frequency appears to be normal in all patients and was not related to the duration of deprivation ($r = 0.30$, $P > 0.1$).

2.2. Aphakic controls

Figure 5 indicates that the two aphakic control subjects performed like their age-matched normal subjects on spatial and temporal contrast sensitivity. Further, losses in acuity were trivial (-0.05 and -0.01 log units for M.B. and W.C., respectively) and critical flicker fusion frequency was normal ($+0.03$ and $+0.01$ log units for M.B. and W.C., respectively).

3. Discussion

The findings indicate that binocular deprivation during early infancy affects some aspects of spatial and

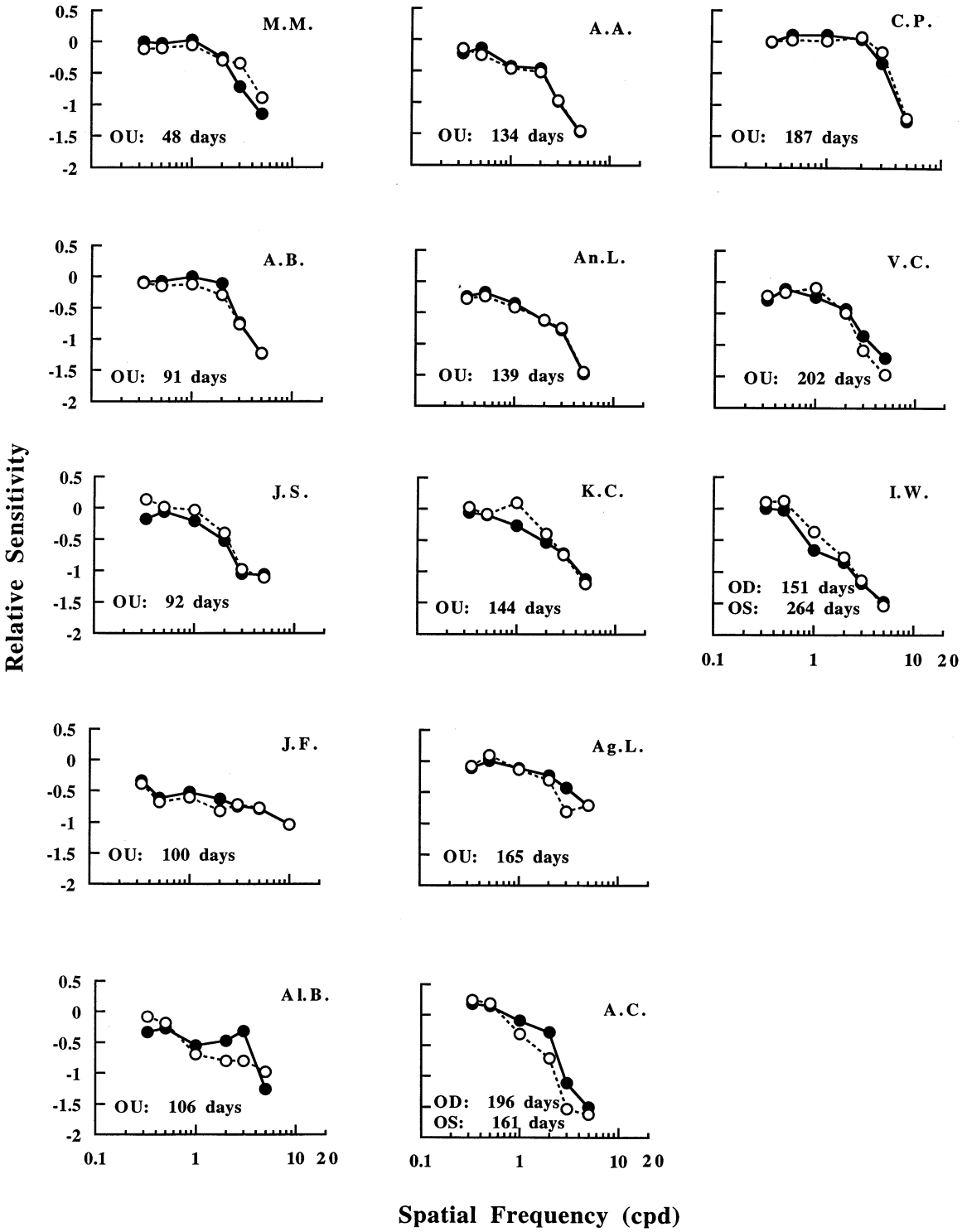

Fig. 1. Relative loss in spatial contrast sensitivity in the right eyes (●) and the left eyes (○) of the 13 patients treated for bilateral congenital cataracts. Each point represents the log ratio of the patient's sensitivity to the sensitivity of the age-matched normal subject. The figures are arranged in order of increasing period of deprivation.

temporal vision more severely than others. Spatial contrast sensitivity at low and medium frequencies (0.33 to 2 c deg^{-1}) was relatively unaffected by deprivation. In contrast, there were severe reductions in spatial contrast sensitivity at high frequencies that exceeded 1.0 log units in every eye and extended up to 1.5 log units. Similarly, there were reductions in grating acuity, reductions that averaged 0.5 log units (1.5 octaves).

Deprivation during early infancy had very different effects on temporal contrast sensitivity. Critical flicker

fusion frequency was normal and most patients had little if any loss at medium and high temporal frequencies (20–30 Hz). Although losses in sensitivity increased monotonically with progressively lower temporal frequencies, these reductions were typically quite small and exceeded 1.0 log units in only two of the patients. In fact, even at low temporal frequencies, performance was within normal limits for about half the patients.

Our findings of a large reduction in grating acuity and spatial contrast sensitivity at high spatial frequencies following early binocular deprivation agree with previous studies of human and non-human primates (Mioche & Perenin, 1986; Tytla et al., 1988; Harwerth et al., 1991; Lewis, Maurer & Brent, 1995). However, some of our findings for temporal contrast sensitivity differ from those of Harwerth et al. (1991). Some of our patients performed abnormally at low temporal frequencies, while Harwerth et al. (1991) found that the temporal contrast sensitivity of binocularly deprived monkeys was within normal limits at all temporal frequencies, perhaps because all of those monkeys had received normal visual input during the first month of life. Nonetheless, our findings agree with their pattern of greater losses in spatial than in temporal contrast sensitivity and their report of no losses at medium and high temporal frequencies.

Our data do not demonstrate any relation between duration of deprivation and the severity of the deficits. However, we assumed that any child who had dense central cataracts diagnosed on the first eye exam before 6 months of age had been deprived from birth. There is a possibility that some of these children had a short period of patterned visual experience prior to the onset of dense cataracts. In those cases, we would have overestimated the duration of deprivation and added noise to the data relating duration of deprivation to visual outcome.

We have attributed the losses in spatial and temporal vision to early visual deprivation caused by dense central cataracts. The results from the two aphakic control patients indicate that aphakia per se (the absence of a natural crystalline lens) did not contribute to the losses in children treated for bilateral congenital cataract. Moreover, the associated visual disorders of strabismus and horizontal nystagmus, which were present in many of the patients, may have contributed to the losses, but are not likely to have caused the overall pattern of results.

Seven of the patients (see Table 1) had no known misalignment of the eyes; yet, their pattern of deficits for both spatial and temporal vision was similar to that of the patients with strabismus. Further, losses in patients with strabismic amblyopia are typically less severe than those shown by our patients (Hess & Howell, 1977). Horizontal nystagmus found in many of our patients could possibly blur the high spatial frequencies

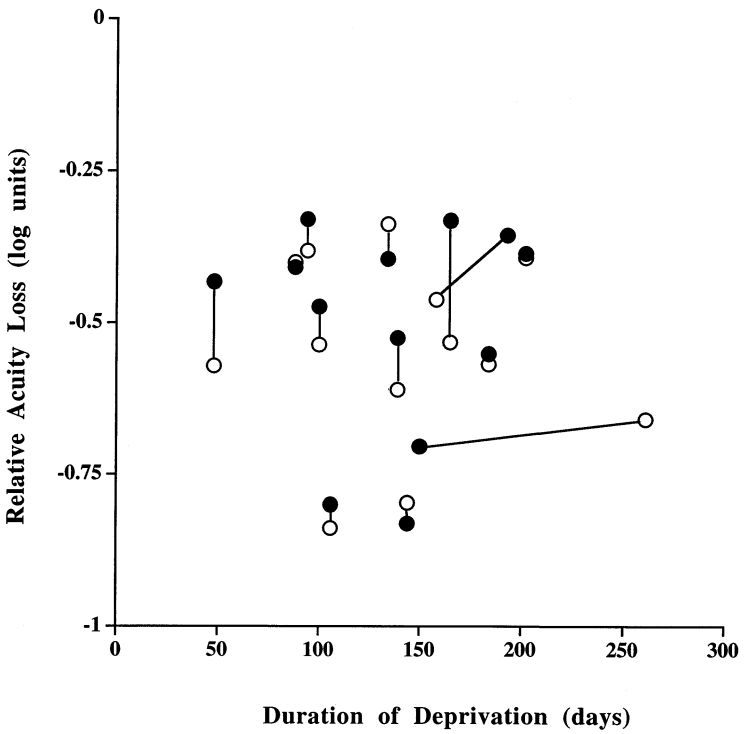

Fig. 2. Relative loss in grating acuity of the right eyes (●) and the left eyes (○) of patients treated for bilateral congenital cataracts. Each point represents the log ratio of the patient's acuity to the acuity of the age-matched normal subject, plotted as a function of duration of deprivation. A solid line joins the eyes from the same patient.

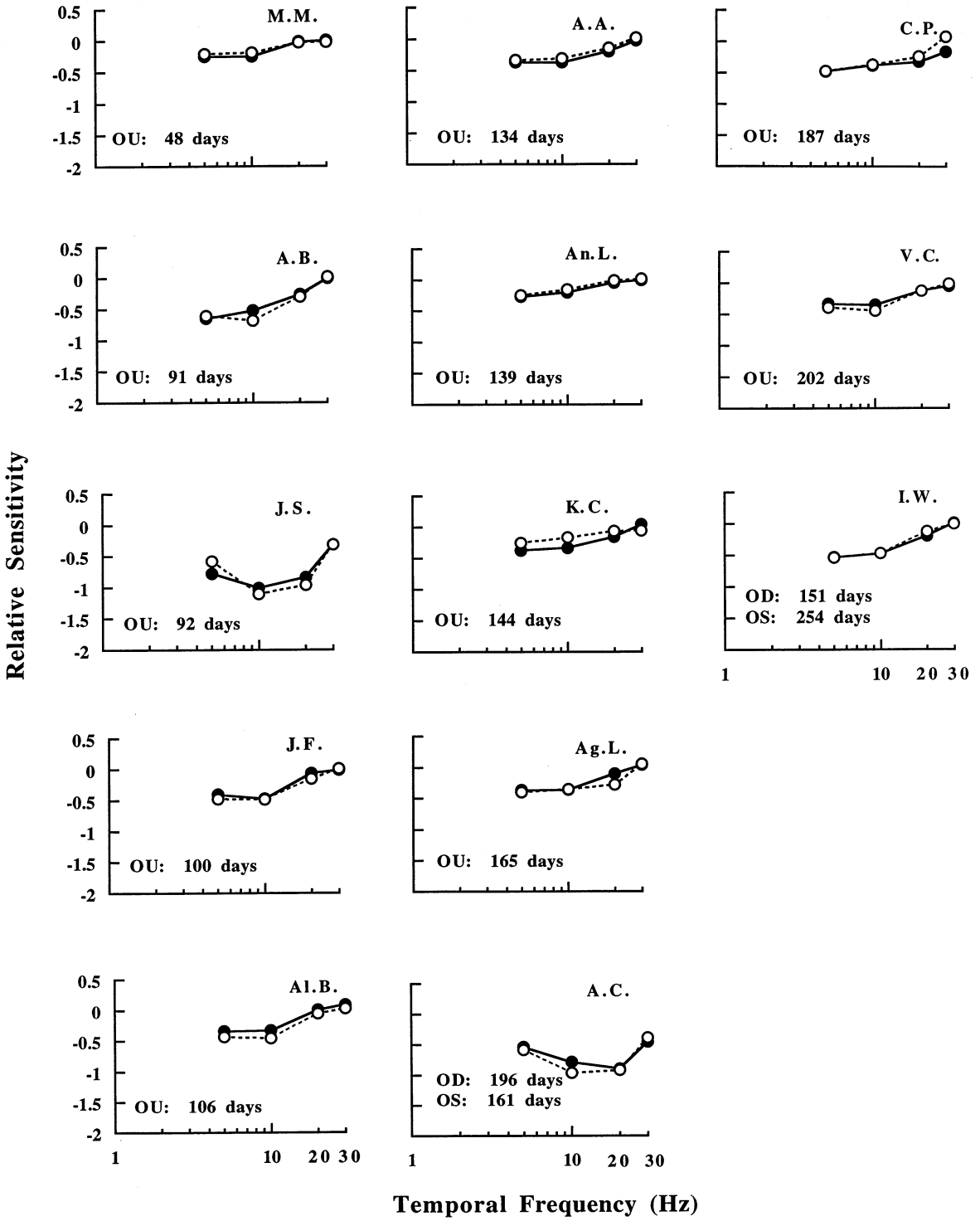

Fig. 3. Relative loss in temporal contrast sensitivity of the right eyes (●) and the left eyes (○) of the 13 patients treated for bilateral congenital cataracts. Each point represents the log ratio of the patient's sensitivity to the sensitivity of the age-matched normal subject. The figures are arranged in order of increasing period of deprivation.

of gratings oriented orthogonally to its direction. However, nystagmus is unlikely to account for the losses at low spatial frequencies found in some patients. Nor can it account for the losses in temporal contrast sensitivity because the temporal modulation always stimulated all parts of the retina. Moreover, it may not contribute to the losses even at high spatial frequencies because Higgins, Daugman and Mansifield (1982) found that stabilizing a grating on the retina of an amblyope with nystagmus did not improve sensitivity, and that jiggling

a grating did not decrease sensitivity of a normal eye. In summary, it seems that the pattern of losses in spatial and temporal vision resulted from early pattern deprivation and not from associated conditions.

Our findings support the theory that the effects of deprivation are related to the normal pattern of development and that deprivation affects least the aspects of vision that are relatively mature during early infancy (Maurer & Lewis, 1993). Specifically, critical flicker fusion frequency, which matures in early infancy, is less affected by deprivation than are spatial vision and temporal contrast sensitivity, both of which are immature at birth and develop gradually. Even within a visual function, the aspects that are most affected by deprivation are the ones that are slowest to mature. Specifically, the losses in spatial contrast sensitivity were greater at higher than at lower spatial frequencies, and previous studies have shown that sensitivity to higher spatial frequencies matures more slowly during infancy than does sensitivity to lower spatial frequencies (Atkinson et al., 1977; Banks & Salapatek, 1978, 1981). Similarly, the losses in temporal contrast sensitivity were greater at lower than at higher temporal frequencies, and sensitivity to the lower temporal frequencies is the last to mature (Ellemberg et al., 1999). This is similar to findings for peripheral vision reported by Bowering, Maurer, Lewis and Brent (1997): peripheral sensitivity in the far temporal visual field is the slowest to develop and is most severely affected by deprivation; sensitivity in the superior visual field is first to mature and is least affected by deprivation.

A more detailed comparison of our results to those from normal infants indicates that early deprivation did not arrest development. The cataracts had always been diagnosed by 6 months of age. Yet on every part of both the spatial and temporal contrast sensitivity curves, all of the patients performed better than normal 6-month-olds (Peterzell et al., 1995; Regal, 1981; van Hof-van Duin & Mohn, 1986; Swanson & Birch, 1990). Moreover, our findings indicate that subsequent development must have been greater for the parts of the curves that normally develop more quickly (lower spatial frequencies and higher temporal frequencies). Thus, early visual deprivation does not prevent some subsequent development of both spatial and temporal vision—at least after deprivation lasting no longer than 264 days and after periods of recovery of more than 4 years, as was true for our patients.

The losses in spatial and temporal vision are likely caused at the level of the striate cortex and beyond because in the monkey model there are no changes in photoreceptor properties (Hendrickson & Boothe, 1976), electroretinograms (Crawford, Blake, Cool & von Noorden, 1975), or physiological properties of lateral geniculate nucleus neurons (Blakemore & Vital-Durand, 1986; Levitt, Movshon, Sherman & Spear, 1989) following early bilateral visual deprivation. In contrast, at the level of the striate cortex, there is a

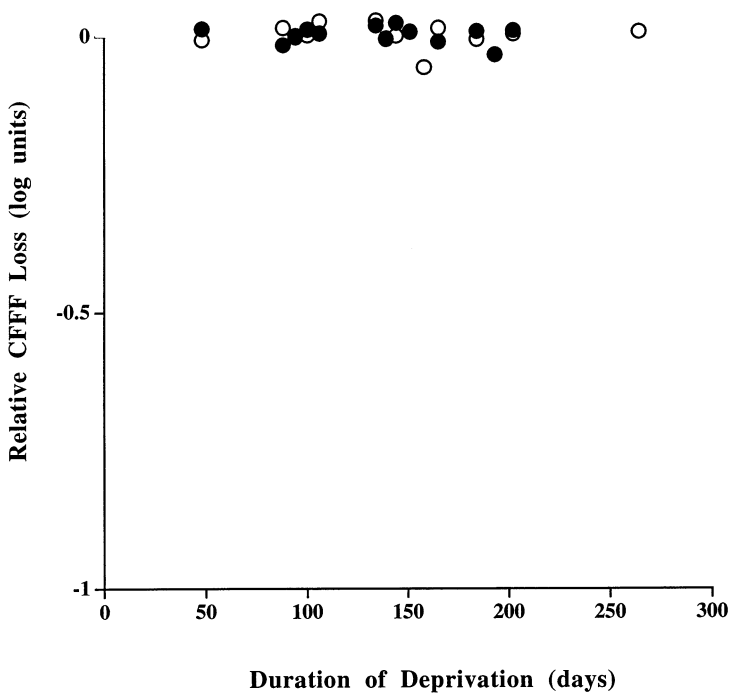

Fig. 4. Relative loss in critical flicker fusion frequency of the of the right eyes (●) and the left eyes (○) of patients treated for bilateral congenital cataracts. Each point represents the log ratio of the patient's critical flicker fusion frequency to the critical flicker fusion frequency of the age-matched normal subject, plotted as a function of duration of deprivation.

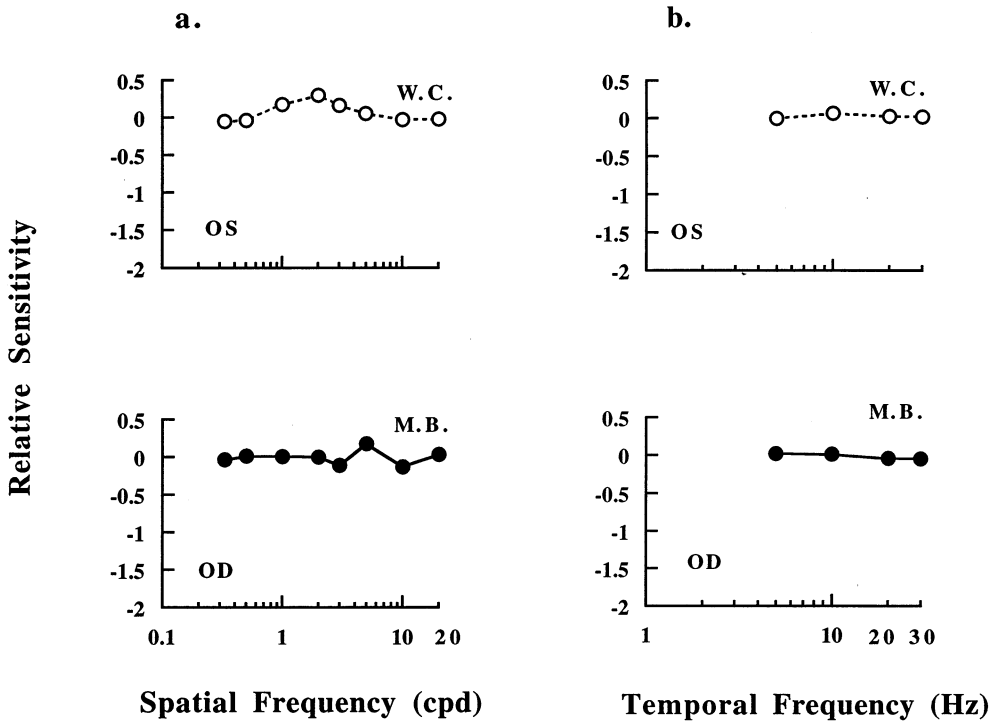

Fig. 5. (a) Relative spatial contrast sensitivity of the aphakic eyes of the two traumatic cases. Each point represents the log ratio of the patient's sensitivity to the sensitivity of the age-matched normal subject. (b) Relative temporal contrast sensitivity of the aphakic eyes of the two traumatic cases. Each point represents the log ratio of the patient's sensitivity to the sensitivity of the age-matched normal subject.

nearly 4-fold reduction in binocularly driven cells (Crawford et al., 1975; Crawford, Pesch, von Noorden, Harwerth, & Smith, 1991). Further, striate cortex neurons respond more sluggishly and have marked reductions in both their spatial resolution and contrast sensitivity (Blakemore & Vital-Durand, 1983; Blakemore, 1990; reviewed in Movshon & Kiorpes, 1993). Their receptive fields are also abnormally large and poorly tuned. Thus, the reductions in visual performance measured in our binocularly deprived patients are likely to reflect abnormalities in the striate cortex and its projections.

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