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Contrast sensitivity in the 'good eye' of adult patients with severe impairment in the other eye

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Abstract

Purpose: It is widely accepted that monocular deprivation results in improved visual performance in the non-pathological eye. The current study investigates the effect of deprivation due to severe impairment in one eye during late childhood or adulthood, on the spatial performance of the fellow 'good' eye.
Methods: Twenty patients (age: 29 ± 9 years) with severe visual impairment in one eye (visual acuity equal or worse than count fingers at 1 m), for a period longer than 2 years, participated in the study. Only patients with an age less than 50 years and monocular deprivation onset greater than 9 years were included. On the basis of the time of deprivation the patients were categorised into two subgroups: (i) long-past deprivation (N = 8, age 28 ± 8 years, 9–20 years of deprivation) and recent deprivation (N = 12, age 30 ± 11 years, 2–4 years of deprivation). Eighteen more participants (age: 28 ± 5 years) with normal binocular vision served as the control group. Best-corrected contrast sensitivity was evaluated using reversing (2 Hz) vertical sinusoidal gratings. Seven spatial frequencies (1, 2, 4, 8, 12, 16 and 24 c/deg) were tested. Performance of the control group was tested both monocularly (dominant eye) and binocularly.
Results: In normal subjects, binocular viewing improved contrast sensitivity on average by 4.2 dB (corresponding to a 70% improvement in contrast threshold) compared to monocular recordings. Average contrast sensitivity in subjects with impaired vision in one eye was found to be higher by 5.0 dB (corresponding to an 83% improvement in contrast threshold) compared with the dominant eye of the control group. The increase in sensitivity was independent of spatial frequency. No differences were observed between the two subgroups with recent and long-past deprivation.
Conclusions: Notable improvement in contrast sensitivity was found in the non-pathological eye of patients with severe impairment in the other eye at an age after the "critical" period of visual development. These findings are consistent with growing evidence supporting functional changes as a result of altered experience or injury in the adult vision system.

Introduction

It is well established that vision with two eyes is enhanced over what would be expected with just one eye, when conditions of binocular overlap and fusion are achieved. This phenomenon, called *binocular summation*, may be partly explained by probability factors, i.e. the use of two independent "detectors"[1,2] but is mainly attributed to the existence of neurons in the visual cortex that "summate" the signals from the two eyes.[3] Many psychophysical studies have shown that binocular facilitation enhances contrast sensitivity[4–7] and perceived suprathreshold contrast,[8,9] with the summation ratio, at and around threshold, for normal observers being higher than that predicted by probability summation (approximately 1.4).[8–10]

Table 1. Personal and clinical details of the one-eyed patients

Personal details			Affected eye			Healthy eye	
N	**Age (years)**	**Gender**	**Deprivation time (years)**	**Visual acuity**	**Ocular pathology**	**BCVA (logMAR)**	**Sph.Eq. (D)**
S01	46	M	2	LP	RD	−0.24	plano
S02	30	M	2	NLP	Melanoma	−0.22	plano
S03	42	M	2	NLP	RD	−0.08	plano
S04	42	M	3	LP	RD	0.00	plano
S05	13	F	3	FC at $\leq$20 cm	Strabismus Surgery	−0.26	−0.50
S06	13	M	4	LP	Trauma	0.00	−4.00
S07	29	M	4	NLP	Melanoma	−0.22	plano
S08	36	F	4	LP	RD	−0.22	plano
S09	38	M	4	FC at 1 m	Trauma	−0.12	plano
S10	22	M	4	NLP	Trauma	−0.22	plano
S11	24	F	4	FC at 1 m	Strabismus Surgery	−0.10	−1.75
S12	27	M	3	FC at 1 m	RD	−0.28	plano
S13	26	M	9	LP	Trauma	−0.18	plano
S14	23	M	10	LP	RD	−0.28	plano
S15	46	M	11	NLP	RD	−0.14	plano
S16	25	F	16	FC at $\leq$20 cm	Strabismus	−0.10	plano
S17	30	M	20	NLP	Trauma	−0.10	plano
S18	23	M	13	NLP	Melanoma	0.00	−1.13
S19	29	F	19	NLP	Trauma	0.00	−2.75
S20	25	F	16	FC at $\leq$20 cm	Trauma	−0.10	−4.00

FC, Finger counting; LP, Light perception; NLP, No light perception; RD, Retinal detachment.

It is plausible that binocular integration would be severely constrained in conditions of abnormal binocular visual experience, such as form deprivation in one eye early in life. Wiesel and Hubel's groundbreaking observations, showing that monocular deprivation in the cat and the monkey,[11–14] alters the morphological and functional representation of the two eyes in the cortex, lead to a plethora of experiments designed to reveal the underlying mechanisms and operational characteristics of this process (see for review[15–17]). For example, following studies demonstrated that an imbalanced ocular input caused by lid closure of one eye leads to a shift in the preference of cortical neurons for the non-deprived eye.[18–20] This is also accompanied by a shrinkage of ocular dominance columns representing the closed eye and an expansion of columns representing the open eye,[21] resulting in increased responses of the cortical neurons in the non-deprived eye.[22]

These physiological changes suggest that monocular deprivation in humans, resulting from eye injuries, pathologies or enucleation during the period that the visual system has considerable plasticity, would result in the non-deprived eye mediating better performance than either eye of a person with normal binocular vision. This has been initially shown in studies investigating alignment sensitivity (vernier acuity)[23,24] but was also evident for contrast sensitivity.[25] Nicholas and colleagues[25] found higher contrast sensitivity in subjects who had an eye removed very early during development because of a retinoblastoma compared to the better eye of control subjects, with the difference being more evident the earlier the eye was enucleated. The current study investigated the effect of monocular deprivation in late childhood or adulthood, due to ocular trauma/pathology, on the spatial performance of the fellow eye. For comparison, binocular summation was evaluated in aged-matched controls with normal binocular vision.

Methods

Study - Participants

Twenty patients with severe visual impairment in one eye[26] and an average age of 29 (SD 9, range 13–46) years were included in the study. Patients were recruited from the outpatient Retina Clinic of the University Hospital of Heraklion, Crete, Greece, in a prospective consecutive non-randomized fashion. Patients had been diagnosed with severe visual impairment in one eye due to an ocular pathology (e.g., retinal detachment, melanoma) or trauma at least 2 years prior to their visit. Only patients with an age less than 50 years and monocular deprivation onset greater than 9 years were selected for further analysis. The affected eye of all patients was measured with a visual acuity equal or worse than finger counting at 1 m (see *Table 1*). Average deprivation time (years of monocular vision) was 8 (SD 6, range 2–20) years. Average age of deprivation onset was 21 (SD 12, range 9–44) years. On the

basis of the time of deprivation due to severe visual impairment the patients were also allocated into two subgroups: (i) long-past deprivation (N = 8, age 28 ± 8 years, 9–20 years of deprivation) and recent deprivation (N = 12, age 30 ± 11 years, 2–4 years of deprivation). Exclusion criteria for the non-affected eye were any retinal/macular pathology, media opacities (cataract, corneal opacity and vitreous hemorrhage), ocular surgery, glaucoma, major systemic disease and neurological or any other disorders that may compromise successful study participation. Exclusion criteria for the control group were all the above-mentioned exclusion criteria including spectacle-corrected visual acuity worse than 0.00 logMAR (Snellen 6/6, 20/20), myopia or hyperopia >4.00 D and anisometropia >0.50 D. The average spherical equivalent was −0.59 ± 1.30 D for the patients with monocular impaired visual acuity and −0.97 ± 1.65 D for the control group.

Verbal consent was obtained from all participants after they had received an oral explanation of the nature of the study. The study was conducted in adherence to the tenets of the Declaration of Helsinki and followed a protocol approved by the University of Crete Research Board.

Experimental Procedure

Both visual acuity and contrast sensitivity measurements were performed at 4.0 m distance, monocularly (dominant eye for the control group) and binocularly (only for the control group), with best spectacle sphero-cylindrical correction and natural pupils. Eye dominance (in control normals) was determined by looking through a central hole in an A4 card, held by the participant in both hands away from the body. During the monocular measurements in both groups the other eye was covered with a non-translucent eye patch, while participants were asked to keep the eye closed underneath. The order of viewing testing (monocular vs. binocular) for the control group was counterbalanced.

Visual acuity recordings

Visual acuity was assessed using the UoC European-wide logMAR charts (www.precision-vision.com).[27,28] A back-illuminated slim stand (www.sussexvision.co.uk) was used to hold the charts. Chart 1 and chart 2 were used for recording visual acuity of the dominant eye and of both eyes (in control normals) respectively. All subjects were asked to identify each letter one by one in each line starting from the upper left-hand letter, and to proceed by row until they could no longer name correctly at least one letter in a line. They were instructed to read slowly and guess the letters when they were unsure. Visual acuity was derived from the calculation of missed letters up to the last readable line.

Contrast sensitivity recordings and analysis

Contrast sensitivity was evaluated using a vertical (90°) sinusoidal grating (size: 3 deg), modulated at a frequency of 2 Hz in a square-wave reversal mode. The grating was displayed on a 21-inch Sony GDM F-520 CRT monitor, by means of a VSG 2/5 stimulus generator card (www.crsltd.com) until the subject gave a verbal "yes" or "no" response at each contrast level. Seven spatial frequencies (1, 2, 4, 8, 12, 16 and 24 c/deg) were tested. Mean screen luminance was 30 cd/m^2. The gamma functions of the red, green and blue guns of the monitor were calibrated with a PR-650 spectro-radiometer (www.photoresearch.com). Contrast (C) was defined in terms of Michelson, i.e. L_{max}-L_{min}\L-$_{max+Lmin}$., where L_{max} and L_{min} are the maximum and minimum screen luminances, respectively. Contrast sensitivity was measured in decibels (dB), where dB = 20 $\log_{10}$ C^{-1}(0 dB corresponds to 100% C and 0.01 CS, 6 dB to 50% C). Observers were asked to fixate on a cross, located in the centre of the screen, while room lights were extinguished during the experiment. Threshold was determined using a binary-search staircase with a contrast resolution of 1 dB (0.05 log units of contrast). Gratings were initially presented with a relatively high contrast (16 dB). If seen, contrast was decreased by 16 dB. If not, it was increased by 16 dB. Successive increments were halved until the increment was less than 1 dB. The average of three measurements was taken as a threshold for each spatial frequency. Subjects were given a 15-min practice session to get familiarised with the threshold procedure.

Contrast sensitivity functions (CSF) were plotted in a linear-log scale and were fitted with second-order polynomials. The spatial frequency cut-off (spatial frequency for CS = 0) was calculated from the linear slope of the linear-linear CSF plots (for spatial frequencies >2.5 c/deg). Furthermore, in order to evaluate any bandwidth-specific loss, the area under the contrast sensitivity function (AUCSF) was calculated[29, 30] for the following spatial frequency *(f)* ranges: from 0.0 to 1.2 log c/deg ($AUCSF_{full}$), from 0.0 to 0.5 log c/deg ($AUCSF_{low}$) and from 0.5 to 1.2 log c/deg ($AUCSF_{high}$) (see Equation 1).

$$\begin{aligned} AUCSF_{full} &= \int_0^{1.2} f(x)dx, AUCS_{Flow} \\ &= \int_0^{0.5} f(x)dx, AUCSF_{high} \\ &= \int_{0.5}^{1.2} f(x)dx(1) \end{aligned}$$

Statistical analysis

Post-hoc power calculation, performed using the G*Power version 3.1.3 (www.psycho.uni-duesseldorf.de/aap/projects/

Table 2. Mean (SD) visual acuity and contrast sensitivity values, for the two groups (one-eyed vs control) and the two subgroups of one-eyed patients (recent vs long past). AUCSF: area under the contrast sensitivity function

Group	Visual acuity (logMAR)	Contrast sensitivity			
		AUCSF low	AUCSF high (dB*c/deg)	AUCSF full	Cut-off frequency (c/deg)
Control					
Monocular	−0.11 (0.09)	28.6 (1.3)	22.8 (1.6)	51.4 (2.7)	25.8 (1.7)
Binocular	−0.19 (0.07)	31.1 (1.0)	25.6 (1.1)	56.7 (1.9)	27.6 (1.5)
One-eyed	−0.14 (0.10)	31.0 (1.4)	25.6 (2.1)	56.5 (3.3)	28.8 (2.4)
Recent	−0.16 (0.10)	31.2 (1.1)	26.1 (1.9)	57.3 (2.8)	29.3 (2.6)
Long-past	−0.11 (0.09)	30.6 (1.7)	24.7 (2.2)	55.4 (3.8)	27.9 (1.9)

gpower/),[29] for t-test (2 independent samples), α-error probability of 0.05 and a sample size of 18 (group 1) and 20 (Group 2) rendered a power (1-β error probability) for all indices tested higher than of 0.85. The indices used for statistical analysis were the three AUCSF areas and the cut-off frequency. Any statistical difference between the groups was estimated using independent samples *t*-tests. Differences between the monocular and binocular performance for the normal subjects were estimated using paired t-tests (MedCalc®, version 12.0.0, www.medcalc.org).

Results

Table 2 depicts average (±SD) values of visual acuity scores and contrast sensitivity areas and cut-off frequencies for the control and the one-eyed participants.

All control participants showed an enhanced performance with the two eyes. Average (±SD) best-corrected visual acuity of the control group was better with binocular (−0.19 ± 0.07 logMAR) than with monocular (−0.11 ± 0.09 logMAR) observation (mean difference −0.08, 95% CI from −0.06 to −0.10 $p < 0.001$ - paired *t* test). Similarly, contrast sensitivity was higher with binocular compared to monocular vision at all spatial frequencies (see *Figure 1*). The average binocular summation (binocular minus monocular sensitivity) in contrast sensitivity for all spatial frequencies was 4.2 dB (range 3.8–5.2 dB), which corresponds to about 70% improvement in contrast threshold and was independent of the spatial frequency. Highly significant differences between the two viewing conditions were found for full-, high- and low-AUCSF areas as well as for the spatial frequency cut-off ($p < 0.001$ for all indices; paired t-tests).

Average best-corrected visual acuity in the group with severe visual impairment in one eye (−0.14 ± 0.10 logMAR) was higher compared to the dominant eye of the control group. However, this difference (mean 0.04, 95% CI from −0.03 to 0.10 logMAR) did not reach statistical significance ($p = 0.23$, independent samples *t*-test). On the other hand contrast sensitivity was higher at all spatial

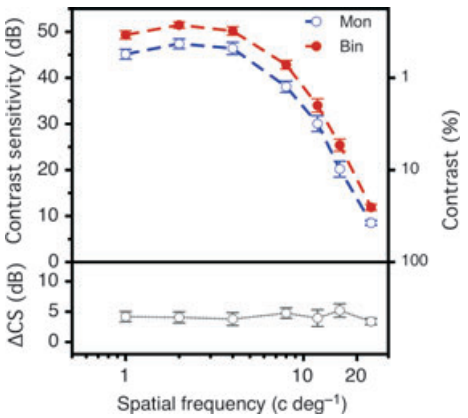

Figure 1. (upper) Average contrast sensitivity functions for the control group (18 participants) under binocular (filled red circles) and monocular (open blue circles) vision; (lower) difference between binocular and monocular contrast sensitivity. The dashed lines form second order regressions. The bars indicate ± 95% confidence intervals.

frequencies in patients with one eye than with the dominant eye of the control group (*Figure 2*). The average difference in contrast sensitivity between the two groups was 5.0 dB, which corresponds to an 83% improvement in contrast threshold. This difference is to some degree more pronounced for high spatial frequencies (approximately 6.5 dB for 16 and 24 c/deg compared to approximately 4.5 for other frequencies). Highly significant differences between the two groups were found for full-, high- and low- AUCSF ($p < 0.001$ for all parameters; independent samples t-tests) as well as for the spatial frequency cut-off (mean 3.0, 95% CI from 1.6 to 4.4 c/deg, $p < 0.001$; independent samples *t*-tests). Note that the contrast sensitivity in patients with one eye was also higher (by 0.84 dB on average) than the binocular values of the control group. These differences were not statistically significant ($p > 0.05$ in all indices).

Finally, comparing performance between the two subgroups (recent vs. long-past deprivation) of patients with one eye revealed no statistically significant differences for average (±SD) visual acuity (recent: −0.16 ± 0.10

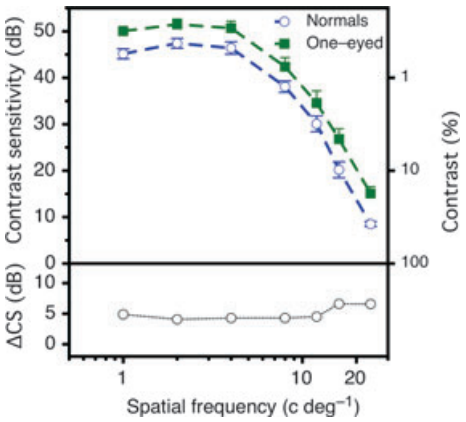

Figure 2. (upper) Average contrast sensitivity functions for the control (18 participants, open blue circles) and the one-eyed group (20 participants, filled green squares) under monocular observation (lower) difference in contrast sensitivity between the two groups (control minus one-eyed). The dashed lines form second order regressions. The bars indicate ± 95% confidence intervals.

logMAR, long-past: -0.11 ± 0.09 logMAR, $p = 0.37$). Contrast sensitivity was found to be higher for the whole range of spatial frequencies in the "recent" subgroup (see table 1), but differences did not reach a statistically significant level ($p > 0.05$ in all indices).

Correlations between visual performance in patients with one eye and the continuous variables were also tested. *Figure 3* depicts plots of linear correlations between logMAR acuity, cut-off frequency of the contrast sensitivity function and the $AUCSF_{high}$ as a function of deprivation time and the age of deprivation onset. Performance tended to reduce with deprivation time and increase with the age of deprivation onset, although none of the individual regressions were significant.

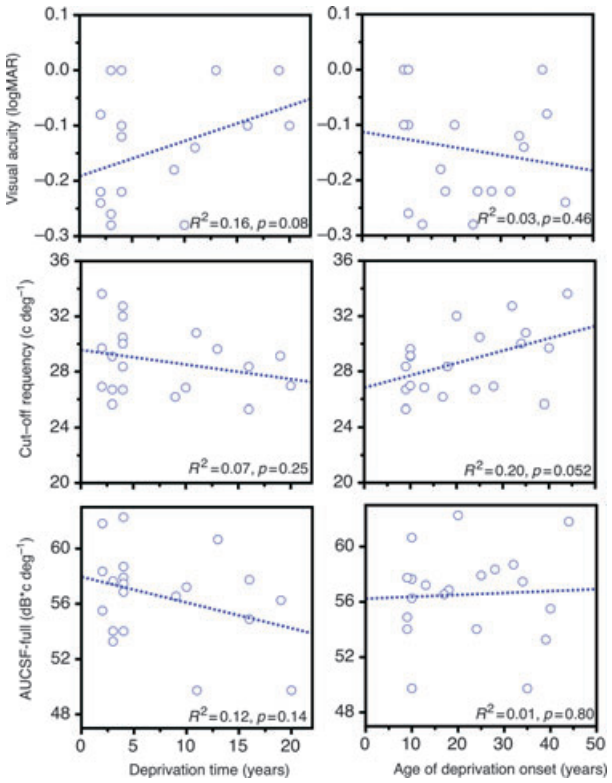

Figure 3. Plots of visual performance (upper: visual acuity; middle: cut-off frequency of contrast sensitivity function; lower: area under the contrast sensitivity function, $AUCSF_{full}$) as a function of deprivation time (left) and onset of deprivation (right). Dotted lines correspond to linear regression fits. Parameters for linear regression fits are also shown.

Discussion

This study shows that monocular deprivation from a variety of causes leads to improved performance of the fellow eye. This was tested in patients who were diagnosed with severe visual impairment in one eye due to ocular pathology or trauma: their contrast sensitivity was found to be significantly better than that of an age-matched control group with normal binocular vision when tested monocularly. The increase in sensitivity was independent of the spatial frequency and similar in magnitude to that found when monocular and binocular sensitivities were compared in normal subjects. On the other hand, visual acuity, measured with high contrast letters, did not reveal any differences between the two groups.

Our findings in control subjects agree with previous studies which reveal that contrast sensitivity, under in-focus conditions, is enhanced by 40–80% with binocular viewing.[4–7,9] Binocular summation decreases with ageing, reflecting deterioration in cortical activity and/or an increasing inter-ocular difference in spatial performance, with the better eye dominating the overall visual performance.[7,10,31] Interestingly, a recent study[32] has shown that binocular vision ameliorates the effect of blur, with the effect being more pronounced the higher the amount of retinal blur. Binocular superiority is significantly reduced for high contrast targets.[5,33–35] Note that the use of non-translucent eye patch during the contrast sensitivity recordings in the control group could have engaged in binocular rivalry with the unpatched eye and possibly influenced light adaptation and monocular threshold. However, it has been reported that this is more common when the eye underneath the patch is open and in cases the dominant eye is patched.[36] Ellingham *et al.*[36] found that only 16% of their patients with the "non-dominant" being patched experience visual disturbances.

Current results on patients with severe monocular vision impairment confirm previous evidence from patients with unilateral deprivation, following surgical removal of one eye, during infancy or childhood.[25] This study reports an

improvement in the non-deprived or 'good' eye of patients with a late onset of deprivation (>9 years of age), after the 'critical period' for normal visual development, during which neuronal activity in the visual cortex can be shaped by natural sensory experience.[13,37,38] This is in agreement with recent studies showing that it is possible to reinstate much greater levels of plasticity in the adult visual system than previously suspected,[16,37,39–43] even in brief periods of monocular deprivation.[41] Perhaps the most telling evidence in humans comes from the treatment of amblyopia. Although, until recently, it was believed that treatment of amblyopia was ineffective for children older than about 8 years (see for review[44]), new clinical and experimental studies provide evidence for neural plasticity beyond the critical period.[45,46] Moreover, there are reports of spontaneous visual acuity improvement in the amblyopic eye of patients following visual loss in the fellow (non-amblyopic) eye due to macular degeneration.[47,48] The "spontaneous" effects of plasticity may also explain the absence of any difference in sensitivity improvement found in the two subgroups (recent incidence of deprivation vs long-past deprivation) tested.

Furthermore, we found that the enhancement in contrast sensitivity in the patients with severe monocular vision impairment is the same for the whole range of spatial frequencies. This pattern is similar to the binocular summation measured in normal subjects, indicating that the remaining eye subserves most of the retino-cortical neuronal circuitry that was previously dominated by the other eye.[39] In contrary, Nicholas *et al.*[25] reported a selective change in the spatial performance of the non-deprived eye in patients with unilateral enucleation during infancy or early in childhood, i.e. the elevation in contrast sensitivity was more pronounced for middle spatial frequencies, They suggested that this was due to a reduction in the number of neurons with large receptive fields in the deprived eye, i.e. the neurons devoted to the Magnocellular (M) pathway, resulting in an expanded M-ganglion cell population in the fellow eye. However, It is now well accepted that the M pathway provides the neural basis of the luminance channel mediating most of the contrast sensitivity function to achromatic patterns.[49–52]

In conclusion, notable improvement in contrast sensitivity is found in the non-pathological or 'good' eye of patients with severe impairment in the other eye due to ocular trauma/pathology at an age after the "critical" period of visual development. The enhancement in sensitivity equals the binocular facilitation observed in normal subjects and is similar for the whole range of spatial frequencies. Current observations are consistent with growing evidence supporting functional changes as a result of altered experience or injury in the adult vision system.

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References

1. Campbell FW & Green DG. Monocular versus binocular visual acuity. *Nature* 1965; 208: 191–192.
2. Pirenne MH. Binocular and uniocular threshold of vision. *Nature* 1943; 152: 698–699.
3. Hubel DH & Wiesel TN. Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. *J Physiol* 1962; 160: 106–154.
4. Arditi AR, Anderson PA & Movshon JA. Monocular and binocular detection of moving sinusoidal gratings. *Vision Res* 1981; 21: 329–336.
5. Home R. Binocular summation: a study of contrast sensitivity, visual acuity and recognition. *Vision Res* 1978; 18: 579–585.
6. Legge GE. Binocular contrast summation–I. Detection and discrimination. *Vision Res* 1984; 24: 373–383.
7. Pardhan S. A comparison of binocular summation in young and older patients. *Curr Eye Res* 1996; 15: 315–319.
8. Legge GE & Rubin GS. Binocular interactions in suprathreshold contrast perception. *Percept Psychophys* 1981; 30: 49–61.
9. Meese TS, Georgeson MA & Baker DH. Binocular contrast vision at and above threshold. *J Vis* 2006; 6: 1224–1243.
10. Ross JE, Clarke DD & Bron AJ. Effect of age on contrast sensitivity function: uniocular and binocular findings. *Br J Ophthalmol* 1985; 69: 51–56.
11. Wiesel TN & Hubel DH. Effects of Visual Deprivation on Morphology and Physiology of Cells in the Cats Lateral Geniculate Body. *J Neurophysiol* 1963; 26: 978–993.
12. Wiesel TN & Hubel DH. Comparison of the effects of unilateral and bilateral eye closure on cortical unit responses in kittens. *J Neurophysiol* 1965; 28: 1029–1040.
13. Hubel DH & Wiesel TN. The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *J Physiol* 1970; 206: 419–436.
14. Hubel DH, Wiesel TN & LeVay S. Plasticity of ocular dominance columns in monkey striate cortex. *Philos Trans R Soc Lond B Biol Sci* 1977; 278: 377–409.
15. Barlow HB. Visual experience and cortical development. *Nature* 1975; 258: 199–204.
16. Hooks BM & Chen C. Critical periods in the visual system: changing views for a model of experience-dependent plasticity. *Neuron* 2007; 56: 312–326.

17. Harwerth RS, Smith EL 3rd, Duncan GC, Crawford ML & Von Noorden GK. Multiple sensitive periods in the development of the primate visual system. *Science* 1986; 232: 235–238.
18. Hoffmann KP & Sherman SM. Effects of early monocular deprivation on visual input to cat superior colliculus. *J Neurophysiol* 1974; 37: 1276–1286.
19. Cynader M, Timney BN & Mitchell DE. Period of susceptibility of kitten visual cortex to the effects of monocular deprivation extends beyond six months of age. *Brain Res* 1980; 191: 545–550.
20. Blakemore C & Vital-Durand F. Effects of visual deprivation on the development of the monkey's lateral geniculate nucleus. *J Physiol* 1986; 380: 493–511.
21. Shatz CJ & Stryker MP. Ocular dominance in layer IV of the cat's visual cortex and the effects of monocular deprivation. *J Physiol* 1978; 281: 267–283.
22. Casagrande VA, Guillery RW & Harting JK. Differential effects of monocular deprivation seen in different layers of the lateral geniculate nucleus. *J Comp Neurol* 1978; 179: 469–485.
23. Freeman RD & Bradley A. Monocularly deprived humans: nondeprived eye has supernormal vernier acuity. *J Neurophysiol* 1980; 43: 1645–1653.
24. Osuobeni EP. Monocular vernier acuity in normally binocular, monocular, and amblyopic subjects. *Optom Vis Sci* 1992; 69: 550–555.
25. Nicholas JJ, Heywood CA & Cowey A. Contrast sensitivity in one-eyed subjects. *Vision Res* 1996; 36: 175–180.
26. Colenbrander A. Measuring Vision and Vision Loss. Duane's Clinical Ophthalmology. Lippincott, Williams and Wilkins: Philadelphia, PA, 2010.
27. Plainis S, Tzatzala P, Orphanos Y & Tsilimbaris MK. A modified ETDRS visual acuity chart for European-wide use. *Optom Vis Sci* 2007; 84: 647–653.
28. Plainis S, Moschandreas J, Giannakopoulou T *et al.* Validation of a modified ETDRS chart for European-wide use in populations that use the Cyrillic, Latin or Greek alphabet. *J Optom*. 2013. doi:10.1016/j.optom.2012.06.008
29. Applegate RA, Howland HC, Sharp RP, Cottingham AJ & Yee RW. Corneal aberrations and visual performance after radial keratotomy. *J Refract Surg* 1998; 14: 397–407.
30. Jiang BC, Scialfa CT, Tyrrell RA, Garvey PM & Leibowitz HW. Bandwidth of the contrast sensitivity function as an index of spatial vision with application to refraction. *Optom Vis Sci* 1990; 67: 260–267.
31. Gagnon RW & Kline DW. Senescent effects on binocular summation for contrast sensitivity and spatial interval acuity. *Curr Eye Res* 2003; 27: 315–321.
32. Plainis S, Petratou D, Giannakopoulou T, Atchison DA & Tsilimbaris MK. Binocular summation improves performance to defocus-induced blur. *Invest Ophthalmol Vis Sci* 2011; 52: 2784–2789.
33. Heravian JS, Jenkins TC & Douthwaite WA. Binocular summation in visually evoked responses and visual acuity. *Ophthalmic Physiol Opt* 1990; 10: 257–261.
34. Horowitz MW. An analysis of the superiority of binocular over monocular visual acuity. *J Exp Psychol* 1949; 39: 581–596.
35. Pointer JS. Influence of selected variables on monocular, interocular, and binocular visual acuity. *Optom Vis Sci* 2008; 85: 135–142.
36. Ellingham RB, Waldock A & Harrad RA. Visual disturbance of the uncovered eye in patients wearing an eye patch. *Eye (Lond)* 1993; 7: 775–778.
37. Morishita H & Hensch TK. Critical period revisited: impact on vision. *Curr Opin Neurobiol* 2008; 18: 101–107.
38. Mitchell DE. The long-term effectiveness of different regimens of occlusion on recovery from early monocular deprivation in kittens. *Philos Trans R Soc Lond B Biol Sci* 1991; 333: 51–79.
39. Fischer QS, Aleem S, Zhou H & Pham TA. Adult visual experience promotes recovery of primary visual cortex from long-term monocular deprivation. *Learn Mem* 2007; 14: 573–580.
40. Hofer SB, Mrsic-Flogel TD, Bonhoeffer T & Hubener M. Lifelong learning: ocular dominance plasticity in mouse visual cortex. *Curr Opin Neurobiol* 2006; 16: 451–459.
41. Lunghi C, Burr DC & Morrone C. Brief periods of monocular deprivation disrupt ocular balance in human adult visual cortex. *Curr Biol* 2011; 21: R538–R539.
42. Shibata K, Kawato M, Watanabe T & Sasaki Y. Monocular deprivation boosts long-term visual plasticity. *Curr Biol* 2012; 22: R291–R292.
43. Spolidoro M, Sale A, Berardi N & Maffei L. Plasticity in the adult brain: lessons from the visual system. *Exp Brain Res* 2009; 192: 335–341.
44. Wu C & Hunter DG. Amblyopia: diagnostic and therapeutic options. *Am J Ophthalmol* 2006; 141: 175–184.
45. Levi DM. Prentice award lecture 2011: removing the brakes on plasticity in the amblyopic brain. *Optom Vis Sci* 2012; 89: 827–838.
46. Lewis TL & Maurer D. Multiple sensitive periods in human visual development: evidence from visually deprived children. *Dev Psychobiol* 2005; 46: 163–183.
47. El Mallah MK, Chakravarthy U & Hart PM. Amblyopia: is visual loss permanent? *Br J Ophthalmol* 2000; 84: 952–956.
48. Vereecken EP & Brabant P. Prognosis for vision in amblyopia after the loss of the good eye. *Arch Ophthalmol* 1984; 102: 220–224.
49. Kulikowski J. The role of P and M systems: (c) psychophysical aspects. In: Seeing contour and colour. (Kulikowski J, Dickinson C & Murray I editors).Pergamon Press: Oxford, 1989; pp. 232–237.
50. Lee BB, Martin PR & Valberg A. Sensitivity of macaque retinal ganglion cells to chromatic and luminance flicker. *J Physiol* 1989; 414: 223–243.
51. Plainis S & Murray IJ. Magnocellular channel subserves the human contrast-sensitivity function. *Perception* 2005; 34: 933–940.
52. Murray IJ & Plainis S. Contrast coding and magno/parvo segregation revealed in reaction time studies. *Vision Res* 2003; 43: 2707–2719.