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# Improving vision: neural compensation for optical defocus

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Anecdotal reports abound of vision improving in myopia after a period of time without refractive correction. We explored whether this effect is due to an increased tolerance of blur or whether it reflects a genuine improvement in vision. Our results clearly demonstrated a marked improvement in the ability to detect and recognize letters following prolonged exposure to optical defocus. We ensured that ophthalmic change did not occur, and thus the phenomenon must be due to a neural compensation for the defocus condition. A second set of experiments measured contrast sensitivity and found a decrease in sensitivity to mid-range (5–25 cycles deg$^{-1}$) spatial frequencies following exposure to optical defocus. The results of the two experiments may be explained by the unmasking of low contrast, high spatial frequency information via a two-stage process: (1) the pattern of relative channel outputs is maintained during optical defocus by the depression of mid-range spatial frequency channels; (2) channel outputs are pooled prior to the production of the final percept. The second set of experiments also provided some evidence of inter-ocular transfer, indicating that the adaptation process is occurring at binocular sites in the cortex.

**Keywords:** human vision, blur, adaptation, neural compensation

## 1. INTRODUCTION

Spectacle wearers frequently report that their vision improves following a period of time without refractive correction. Such reports may be explained by accommodative effort in hypermetropia (long-sightedness) but are extremely puzzling in cases of myopia (short-sightedness). An increasing tolerance ('habituation') to the continual presence of defocus would hardly be surprising, but spectacle wearers reliably describe an actual improvement in 'seeing'. If seeing is defined as the ability to detect, discriminate, and recognize objects, then a spontaneous improvement in this faculty would constitute a remarkable phenomenon.

Watt (1987) made the following observation with regard to blur: 'The symptoms of [presbyopia] and other defocus conditions are not blurred vision but poor acuity... What has been lost is not the percept of sharpness: as the distribution of "seen" blurs shifts, so too does the distribution of labels' (p. 148). In this passage, Watt draws on the fact that the visual system is never exposed to a 'sharp' edge, as edges are always blurred (to some extent) by both the optics of the eye and the spatial filtering properties of early visual processing. Watt (1987) proposes that the perception of blur is similar to that of size (Blakemore & Sutton 1969) and orientation (Gibson 1933): the edges that are perceived to be sharp are those which are the least blurred in the visual diet. If the eye is defocused then the least blurred edge is, naturally, more blurred than the least blurred edge produced by an optimally focused eye. According to Watt, the visual system responds to defocus, and the resultant change in the diet of blurs, by relabelling measured blurs such that the least blurred edge is labelled as sharp. Such a mechanism of relabelling does not predict an actual improvement of vision following a period of exposure to optical defocus. This then raises the intriguing question of whether myopes actually demonstrate an improvement in vision.

Despite the prevalence of anecdotal reports of improved vision in myopes after a period of time without spectacles, we were only able to find one study of this phenomenon within the research literature. Pesudovs & Brennan (1993) measured vision in a group of ten myopes following two separate 90 min sessions. One session consisted of the myopes wearing their refractive correction during a period of time watching television, whilst in the other session the correction was removed. Although Pesudovs & Brennan reported an improvement in vision when the correction was not worn, the improvement was very small (0.039 logMAR units, see later) and within the measurement error of the charts used to assess eyesight. We therefore sought to discover whether vision (in a letter recognition task) can actually improve following prolonged exposure (30 min) to optical defocus.

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## 2. METHODS

### (a) *Experiment 1: blur adaptation*

It is unsatisfactory to explore blur adaptation in natural myopes as degree of refractive error, long-term adaptive effects and subjective expectation are major confounding variables. Vision refers to eyesight without refractive correction, whilst visual acuity (VA) refers to corrected eyesight. We will use the term VA to indicate eyesight through an optical lens even though in this context the lens induces, rather than corrects, myopia. We assessed VA in 15 participants with emmetropia (perfect eyesight) before and after exposure to induced myopia (optical defocus). Letter charts designed to control for crowding, legibility, and measurement linearity (McGraw & Winn 1993) were used in a paradigm that controlled for ophthalmic and psychological variables. Myopia was induced through +1.00 D ophthalmic lenses (it is easiest to express lens power in Dioptres, D: the reciprocal of the lens's focal length in metres), for the 15 emmetropic participants (mean age 17 years; range 16–18) of normal ophthalmic status with no history of spectacle wear. The 15 participants were selected from an initial group of 20. Five of the initial group were found to have small degrees of latent hypermetropia and were therefore not suitable for our study. Measurements were taken of (a) right VA (b) left VA and (c) binocular VA. Visual acuity through the +1.00 D lens was measured before adaptation, following 30 min of induced myopia and after 30 min of normal vision post-adaptation. In a different session run on a different day, the same measures were taken with a 30 min control period during which no lens was worn. These control sessions were randomized across participants with regard to whether they occurred before, or after, the adaptation sessions. These measures controlled for simple learning effects influencing the results. Visual acuity was recorded as the logarithm of the minimal angle of resolution (logMAR). The logMAR is the logarithm to the base ten of the angular subtense of the displayed letter at 6 m:

$$\log\mathrm{MAR} = \log_{10}[\arctan(x/6)], \qquad (1)$$

where $x$ is the size of a letter's stoke width in metres. Each presentation consisted of four letters surrounded by a box whose width equalled the letter stroke, with each presentation decreasing the size of the letters by 0.1 log units (see Bailey & Lovie (1976) or McGraw & Winn (1993) for details). A forced-choice staircase protocol with descending logarithmic steps was employed, with each correctly identified letter scoring 0.025 log units (i.e. if all presented letters were read, the logMAR score would increase by 0.1, whereas the score would increase by 0.05 if only two of the letters were correctly identified). The presentations were altered between conditions to ensure that learning the letters was not possible (measurement time was less than 2 min).

Testing order was randomized within and between participants and measurements were taken over two separate sessions. Pupil size was checked before and after VA measurements (using an ophthalmic ruler) to ensure that no changes occurred (a decrease in pupil size would serve to improve VA). Participants were very carefully examined for the presence of latent hypermetropia using standard clinical ophthalmic measures (e.g. Bennett & Rabbetts 1989) on four different occasions, and refractive state was rechecked after adaptation (but no changes were found). Participants fixated natural objects at optical infinity (they viewed trees through a window in normal daylight) during the adaptation and control periods (importantly, participants did not view text during the adaptation period).

### (b) *Experiment 2: exploring the neural compensation*

In experiment 2, we defocused the right eyes of four participants whilst patching their left eyes. Following the exposure period, we measured the contrast sensitivity function (CSF) and letter acuity in both eyes, to determine whether interocular transfer occurred.

Experiment 2 was similar to the first experiment but had some critical differences: (i) a +2.00 D lens was used; (ii) the ciliary muscle was paralysed; and (iii) participants viewed through an artificial pupil, and only one eye was exposed to the blur whilst the other was covered. In order to ensure that accommodative effort was not a confound, participants were given cycloplegic drops (5 µl of cyclopentolate hydrochloride 0.5%) to temporarily paralyse the ciliary muscle. Cyclopentolate has a secondary mydriatric effect, so that installation of the drops caused an enlarged and unchanging pupil size. The increased pupil size was controllled by the participants viewing through a 5 mm artificial pupil throughout the tests. This measure had the additional advantage of ensuring that changing pupil size was not a factor in the adaptation process. Another control was introduced in experiment 2: refractive error was objectively measured before and after the adaptation period using a modified Canon AutoRef R-1 infrared (IR) objective optometer. The autorefractor determines the position of focus in three meridians, from which the spherocylindrical refractive error can be computed to $\pm 0.12$ D per second.

Prior to the experiments, the emmetropic participants were carefully refracted to the nearest 0.25 D for the 4 m viewing distance using subjective techniques. The refraction occurred approximately 30 min after installation of the cycloplegic agent. VA was measured using two Bailey–Lovie logMAR charts (Bailey & Lovie 1976) viewed through a +2.00 D lens, with the VA taken as the mean of the two measurements. The two charts had a constant illuminance of 600 lux and consisted of different letters to control for learning.

In order to measure CSF, participants monocularly viewed sinusoidal gratings from 400 cm, on a standard computer monitor (P4 phosphor) under computer control. Participants had to press a key to indicate whether they did or did not see a grating. Details on the precise technique together with information on the calibration and linearity of the display monitor are provided in Woods *et al.* (1996). An adaptive psychometric procedure (adaptive probit estimation; Watt & Andrews 1981), consisting of approximately 30 trials per spatial frequency, was used to determine the test contrast levels and to estimate contrast sensitivity. The spatial frequency gratings were randomly presented during the procedure. The monitor was masked to give a circular field subtending a visual angle of 2.5°, and the surround luminance was approximately matched to the average monitor luminance of 40 cd m$^{-2}$. The CSFs were measured under different conditions for the participants: (1) the CSF was measured through optical defocus, and spatial frequencies were measured at 2, 4, 6, 9, 12 and 15 cycles per degree (cpd) for participants AR and JR; (2) the CSF was measured without optical defocus at 4, 6, 8, 9, 10, 11, 12, 15, 18 and 21 cpd for participants DS and VG (DS had a small degree of uncorrected hypermetropia (+0.50 D), but this was not in place during testing—this created a 'notch' in her CSF as seen in figure 3); (3) the CSF was measured without optical defocus at 5, 10, 15, 20, 25, 30, 35 and 40 cpd in the final two participants, CM and AR (it should be noted

that AR was unable to detect 40 cpd). The conditions followed a logical progression: the first condition was a direct analogue of experiment 1 (i.e. CSF was measured in the presence of optical defocus), the second condition explored the mid-range spatial frequencies without defocus, whilst the final condition looked at the effect of adaptation across the complete sensitivity range. The total time for all the measurements to be taken was approximately 6 min.

The participants underwent a 30 min adaptation period during which time they watched a 20 inch television through the +2.00 D lens from approximately 400 cm. The participants watched whatever happened to be playing on Australian daytime television in all conditions. After the adaptation period, the initial measures were repeated to determine visual acuity and contrast in each eye. As an additional control measure the participants watched the television without blur for another 30 min period following exposure to optical defocus. Following this additional control period another (identical) set of measures was taken. In the course of running experiment 2, we found an individual (TC) who showed no adaptation to optical defocus. We examined TC using the same procedure as DS and VG to determine what happened to the CSF in this observer.

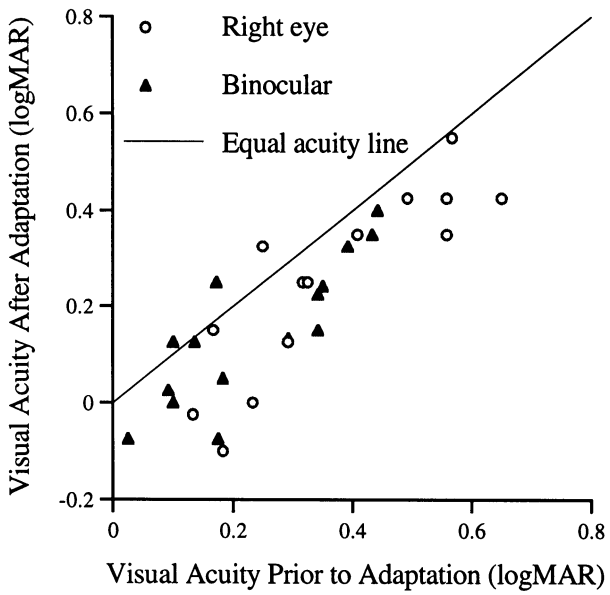



Figure 1. Changes in visual acuity (logMAR) following 30 min exposure to optical defocus. Visual acuity prior to adaptation is plotted against visual acuity (VA) after adaptation. The solid line indicates equal acuity, so points falling below the line show an improvement in VA. The data from the right and left eyes were very similar so, in the interests of clarity, only the monocular data from the right eye, together with the binocular data, are shown. It may be seen that the majority of points fall below the line illustrating the improvement in VA following exposure to optical defocus. The cluster of binocular points appears further to the left than the monocular cluster, owing to the normal advantage of binocular vision over monocular viewing.

## 3. RESULTS

### (a) *Experiment 1*

These are straightforward: following the exposure to optical defocus, a significant improvement was found in the VA of both eyes when measured individually and together. In condition (a) the right VA changed from logMAR 0.362 to 0.242, in condition (b) the left VA changed from logMAR 0.366 to 0.272, and in condition (c) the binocular VA changed from logMAR 0.239 to 0.15. Figure 1 illustrates the data from before and after adaptation, with the thin line indicating equality between measurements of VA taken with and without exposure to blur. Any points that fall below this line indicate that VA improved following exposure. The data from the control conditions are not illustrated, and only the right eye data are shown as the right and left eye data were very similar. The normal advantage of binocular vision (Campbell & Green 1965) was present for all measurements. Preplanned contrasts between the data from before exposure and the data from after exposure were carried out using Dunn's procedure (Keppel 1982, p. 146). The changes in VA after exposure were found to be statistically reliable (condition (a), $t_{14}=4.758$, $p<0.0003$; condition (b), $t_{14}=3.386$, $p<0.004$; condition (c), $t_{14}=4.156$, $p<0.001$). No significant differences were found in the degree of improvement across the conditions. No significant differences were found between measurements in any of the control tests for conditions (a), (b) or (c).

### (b) *Experiment 2*

The findings of the second experiment were in agreement with the first: VA improves following adaptation to optical defocus. The objective measures of ocular refraction showed no change for any of the participants, and participants showed the same VA following the additional control period as that found in the initial measurements (e.g. the compensation for optical defocus had disappeared). These results confirm those of experiment 1: a genuine improvement in eyesight occurs following exposure to optical defocus, and this improvement cannot be explained by ophthalmic change, psychological strategy, or pattern recognition learning. Comparison of the VA data from the covered and 'adapted' eye indicated that inter-ocular transfer had occurred. An improvement in VA was discovered in both eyes, but the improvement in the covered eye was 34.8% of that found in the exposed eye (0.08 logMAR improvement in the covered eye compared to 0.26 logMAR in the exposed eye).

Figure 3 shows the CSF in the right eye measured before and after exposure to defocus (the measurements taken after the control period are not shown). The results are consistent across participants and measurement conditions: a decrease in sensitivity to mid-range (about 5–25 cpd) spatial frequencies occurs following exposure to optical defocus, with the low and high frequencies remaining unaffected. A change in contrast sensitivity of 0.1 units is considered significant: the CSF is normally stable (the CSFs were similar across control conditions) so that the separation between the functions has genuine clinical significance (Woods *et al.* 1996). Examination of the data from participant TC (who did not adapt to optical defocus) showed that no changes occurred in either the VA or CSF following exposure. These findings suggest that a change in CSF is a necessary component in the neural compensation for blur.

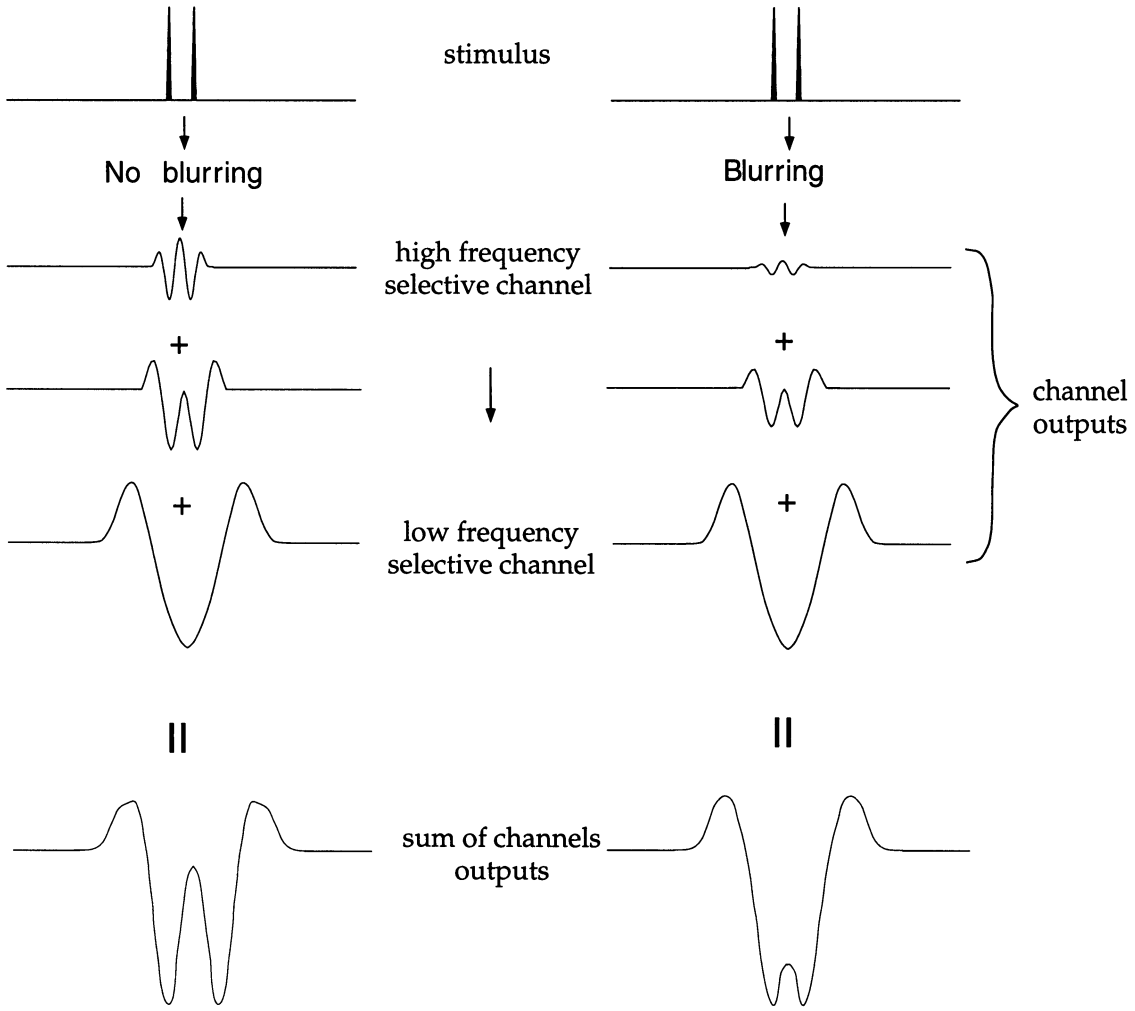


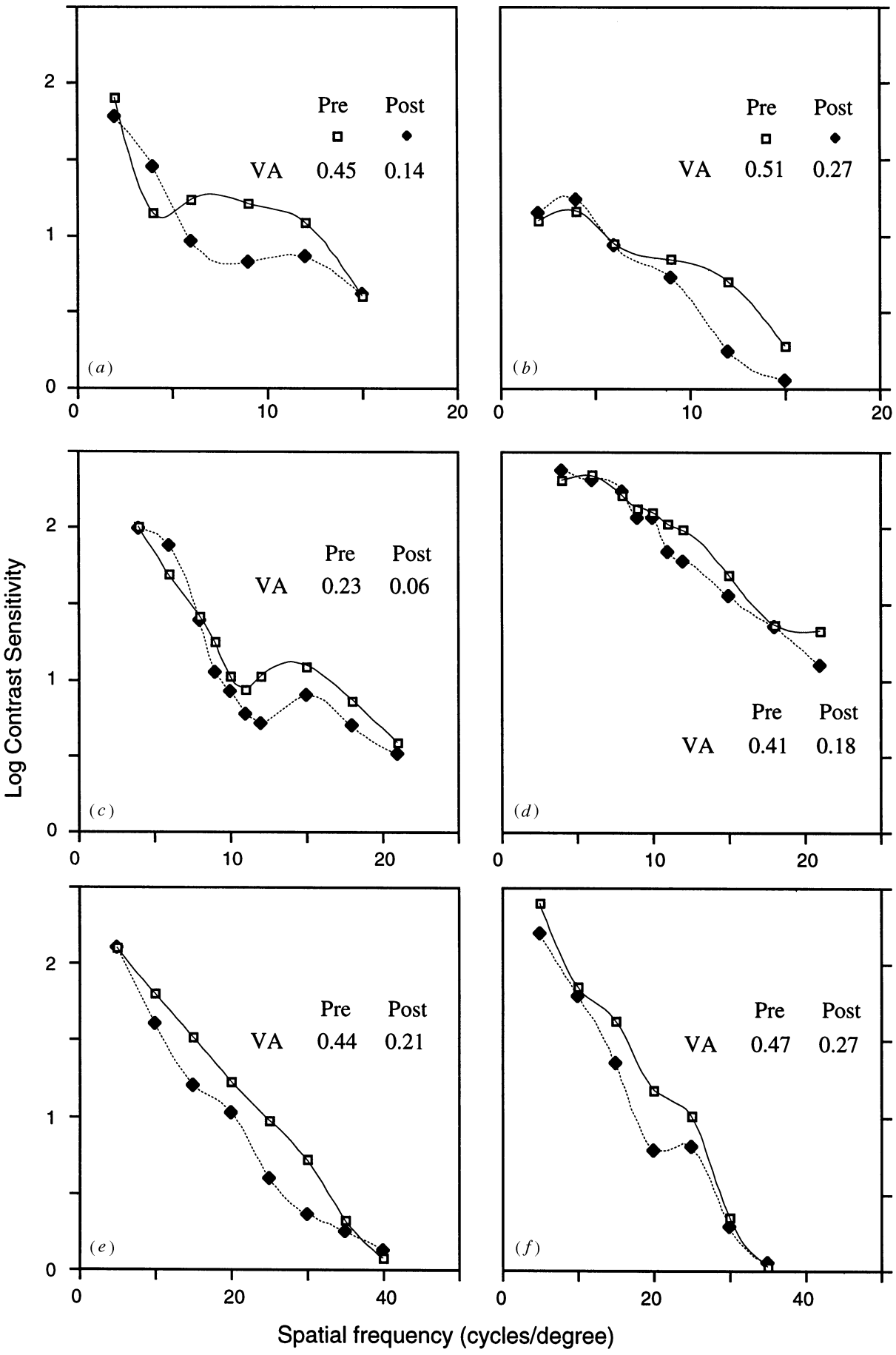

Figure 2. Schematic diagram showing how low-frequency information could mask higher frequency information (adapted from Watt & Morgan, 1985 fig. 7). The stimulus is a one-dimensional two-point luminance profile (top). The panels labelled 'channel outputs' show the responses of spatial filters having a difference of Gaussian-type receptive field organization, which model the response characteristics of the spatial frequency-selective channels in human vision. The bottom panels are the sum of the channel outputs as indicated: overall resolution of the system is based on this sum. The left series of panels shows a hypothetical situation in the absence of additional blur. The series of panels on the right shows that the response of the high-frequency selective filter is greatly attenuated as the result of blurring the stimulus. It is easier to resolve the two stimulus points in the situation on the left since there are pronounced peaks in the summed response. The peaks are much reduced on the right and the summed response is dominated by the low-frequency filter output.

## 4. DISCUSSION

### (a) *Experiment 1*

Following exposure to blur, participants were able to obtain information from the retinal image which they were unable to access prior to exposure. The blurring lens is a low pass spatial filter, and so attenuates high spatial frequency components whilst leaving lower frequency components relatively unchanged. A possible strategy for improving visual resolution might be to increase the sensitivity of high spatial frequency selective channels. If this were possible, then frequency components that were previously sub-threshold could be rendered supra-threshold. There is no evidence, however, that the human visual system is able to increase its sensitivity to high spatial frequencies. The best the visual system can hope to do in this respect is to change the gains of the spatial frequency selective channels. Such an operation cannot, in general, improve sensitivity since

Figure 3. Contrast sensitivity functions for the 'exposed' right eyes of six observers before 30 min exposure to defocus (pre) and after this period (post). The curves are simple cubic spline interpolations between the data points. Spatial frequency is shown on the abscissa, the logarithm of sensitivity to contrast is shown on the ordinate. A change in contrast sensitivity of 0.1 log units is considered to be a real difference: the CSF is normally stable and the CSFs were similar across control conditions (the measurements taken after the control period are not shown), so that the separation between the functions has genuine clinical significance (Woods *et al*. 1996). The CSFs of (*a*) JR and (*b*) AR (top) were measured under conditions of optical defocus both pre and post exposure. Those of (*c*) DS, (*d*) VG, (*e*) CM and (*f*) AR were measured without defocus. Also shown are the VA measurements in logMAR units for the viewing distance of 4 m (see text for details). Participants JR and DS both demonstrate 'notches' in their CSF (Woods *et al*. 1996) due to optical defocus.


Pre Post
VA 0.45 0.14
(a)
Pre Post
VA 0.51 0.27
(b)
Pre Post
VA 0.23 0.06
(c)
Pre Post
VA 0.41 0.18
(d)
Pre Post
VA 0.44 0.21
(e)
Pre Post
VA 0.47 0.27
(f)
Log Contrast Sensitivity
Spatial frequency (cycles/degree)

both signal and noise are amplified equally. It might be possible, however, to use this method if the gains of the channels were initially relatively low. In these conditions near-threshold high-frequency components would give rise to small amplitude channel outputs, which could be swamped in noise at later stages in the system. The small amplitude output would prevent these signals from making a contribution to the visual percept, and increasing the channel gains could improve the system's resolution in such circumstances.

A second possible mechanism for the improvement of resolution exists if low spatial frequency information can 'mask' the higher frequency components in an image (Watt & Morgan 1984). A simple scheme in which such masking would occur is shown in figure 2. It may be seen that as the amplitude of the signal from higher spatial frequency-selective channels gets smaller, so the masking of high-frequency components by low-frequency components increases. Conversely, if the amplitude of the lower spatial frequency-selective channels decreases, their masking effect is reduced. It follows that resolution in a system of this kind could be improved by reducing the amplitude of the low-frequency-selective channels. The human visual system could achieve such amplitude reduction by decreasing the gains of the low-frequency channels.

These two mechanisms can both, at least in principle, account for improved visual resolution following exposure to blurred images. They make different predictions, however, about the changes that would occur in the visual system's sensitivity to individual spatial frequency components. The first mechanism predicts that sensitivity to lower frequency components should be unchanged, whereas sensitivity to higher frequency components should increase. The second mechanism predicts that sensitivity to high frequencies should be unchanged, whereas sensitivity to lower frequencies should decrease. A combination of both mechanisms is also possible. In order to determine whether there was any evidence for the operation of either type of mechanism, we measured the contrast sensitivity function of several observers prior to, and following, half an hour's exposure to a blurred visual diet.

### (b) *General discussion*

The results demonstrate a consistent reduction of sensitivity to spatial frequencies between about 5 and 25 cpd, but little or no change at frequencies above 25 cpd or below 5 cpd following exposure to optical defocus. The finding of reduced sensitivity to mid-range spatial frequency, combined with the clear improvement in letter acuity is consistent with a masking type of explanation, as proposed above: prolonged exposure to blur leads to an unmasking of low contrast, high frequency luminance changes.

The finding of a reduced sensitivity to mid-range spatial frequency components cannot be the result of the fatigue or habituation type mechanism often proposed as an explanation for selective adaptation effects (Georgeson & Harris 1984). Following a short period of exposure to a high contrast sinusoidal luminance grating, the human visual system subsequently displays elevated thresholds at the frequency of the adapting grating. Optical defocus does not increase the contrast of any frequency components, indeed, its effect can only be to decrease contrast or leave it unchanged. Thus, contrast at frequencies showing decreased sensitivity following exposure to blur will at best have been the same during exposure as prior to exposure. It may be concluded that fatigue or adaptation of associated spatial frequency channels is an implausible explanation for the observed reduction in sensitivity.

What has occurred during the exposure period is that the amount of image contrast at lower frequencies has increased *relative* to that at higher frequencies. We propose that it is this relative change in contrast levels that causes the observed type of adaptation effects. Georgeson & Sullivan (1976) suggested a mechanism for contrast constancy which could show these adaptation effects. This mechanism is basically an adjustment rule applied to the amplification factors (gains) of the spatial frequency-selective channels of early vision. The gain adjustment rule works as follows: the visual system learns from its diet of images over an extended period of time that visual stimulation has a particular pattern of spatial frequency content (amplitude spectrum). In response to this pattern, the system develops a rule by which the gains of spatial frequency channels are adjusted so as to preserve the learned amplitude spectrum in the processed image. Georgeson & Sullivan suggested, for illustrative purposes, that this spectrum is flat: it is now known that the amplitude spectrum of natural images falls off with spatial frequency ($f$) in an approximately $1/f$ fashion (Burton & Moorhead 1987; Field 1987; Tolhurst *et al*. 1992).

According to Georgeson & Sullivan's scheme, the gains of the channels are in a constant state of change, and are adjusted by the system so that the average responses of the channels are equal over a relatively extended time-scale. This mechanism compensates for systematic biases in the relative responses of the channels as long as the biases exist over a time-scale which exceeds the time-scale of gain adjustment. The adjustment of gain could be implemented by modulation of the inhibitory interactions between channels (Dealy & Tolhurst 1974; Greenlee & Magnussen 1988). The mechanism cannot improve the signal-to-noise ratio in any spatial frequency-selective channel (both signal and noise are amplified by the same gain factor), and Georgeson & Sullivan state that gain adjustment 'would not affect the threshold function' (p. 651). It is evident, however, that changing channel gains could affect the CSF; for example, if the gains were zero, then all sensitivity would be lost so that decreasing the channel gain can, in principle, elevate thresholds. It does not follow, however, that increasing the channel gain will lower thresholds. This may be the reason why no increases in sensitivity were observed at higher spatial frequencies. Since the contrast at these frequencies is normally rather low (Burton & Moorhead 1987; Field 1987; Tolhurst *et al*. 1992) the gains of the high spatial frequency-selective channels are likely to be relatively high, and so these channels may be operating close to their maximum sensitivity. This would mean that high-frequency channel sensitivities cannot be significantly increased.

A two-stage mechanism involving an adaptation process of the kind discussed above, followed by a combination of the outputs of the spatial frequency-selective channels, is sufficient to explain both the increased letter acuity and the decreased mid-range spatial frequency sensitivity following exposure to blur. The channel output combination must occur at a stage in the visual pathway prior to

object recognition and scene analysis. The available psychophysical evidence certainly suggests that the channel outputs are combined at a relatively early stage in visual analysis (Watt 1987; Watt & Morgan 1983, 1985). Furthermore, it appears that the outputs of low-frequency selective channels cannot be accessed independently of high-frequency channels (Harmon & Julesz 1973; Jamar & Koenderink 1985; Morgan & Watt 1984) and that high-frequency channels cannot be accessed independently of low-frequency selective channels (Watt & Morgan 1984). On the other hand, the simple illustrative version of the mechanism shown in figure 2 is unlikely to provide a reasonable model of human vision. Watt has argued convincingly that the problems of locating luminance boundaries in a process that simply adds or averages the outputs of individual spatial frequency-selective channels make the schema illustrated in figure 2 an implausible model for human vision (Watt 1987; Watt & Morgan 1985).

In conclusion, neural adaptation to blur may occur by the unmasking of high spatial frequencies via a two-stage process: (1) the pattern of relative channel outputs is maintained during optical defocus by the depression of mid-range spatial frequency channels; (2) channel outputs are pooled prior to the production of the final percept. Such a mechanism ensures the optimal percept of retinal images and would allow neonates to maximize vision during development. Neural compensation would also optimize vision during age-related ophthalmic changes, and may explain why the onset of myopia and cataract are so well tolerated. Anecdotal reports abound of spectacles worsening vision and controversial therapies for improving myopia often emphasize the removal of spectacles. These reports and therapies are generally dismissed as there is no evidence of objective refractive change. It appears, however, that the myopic population may be justified in claiming an improvement in vision without spectacles.

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