Suprathreshold Responses of the Visual System in Normals and in Demyelinating Diseases

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It is well-established that diseases affecting the visual pathway can result in the elevation of contrast thresholds. Little is known, however, about how people with decreased sensitivity to contrast perceive targets at suprathreshold levels of contrast. It is known that the normal visual response at suprathreshold levels cannot be linearly derived from the threshold contrast function. It may be expected that threshold abnormalities may not predict the quality of vision for high contrast images which prevail in normal operating conditions. In this investigation, the response to suprathreshold visual stimuli (vertical sine-wave gratings) in multiple sclerosis (MS) and optic neuritis patients with contrast sensitivity deficits was studied. Forty-eight normal eyes served as controls. Suprathreshold response was assessed with a matching procedure. Performance above threshold, for each individual, was estimated by a score which was the ratio of the contrast match to the contrast threshold for a given spatial frequency. The majority of the eyes studied showed that patients performed normally at high contrast levels. Indeed, in a number of cases, the scores implied that patients can perform better than normals. In some eyes, this enhancement of suprathreshold response was correlated with the threshold loss. These results suggest that a "compensation" mechanism helps make the perception of objects independent of the quality of detection at threshold for these patients. In other eyes, an abnormal decrease in the suprathreshold response measure was found, which was not related to the threshold deficit. Thus, suprathreshold responses of MS or optic neuritis patients cannot be predicted from their threshold contrast sensitivity. Invest Ophthalmol Vis Sci 27:1368-1378, 1986

A number of disease processes affecting the visual pathway, such as demyelination, can result in contrast sensitivity (CS) deficits, i.e., alteration of the detection thresholds of visual targets. However, absolute thresholds measure the limits of visibility rather than vision under normal, everyday operating conditions. For instance, it is not clear how patients with threshold sensitivity losses perceive targets whose visual characteristics are well above threshold.

The visual responses at suprathreshold levels in normal observers were reported in earlier studies. The suprathreshold contrast function, i.e., variations in the perceived contrasts as a function spatial frequency (SF), has been described by several investigators. All of these studies have shown that the response function of the visual system at suprathreshold levels cannot be linearly derived from the threshold contrast sensitivity function. In particular, variations in perceived contrast with spatial frequency become much less pronounced as contrast is increased above threshold. This nonlinearity in contrast response has been treated in a recent model. Similar results have been reported by Bowker, who has extended the earlier suprathreshold studies to include the temporal-frequency domain.

These studies confirm previous suggestions that the threshold contrast sensitivity function does not accurately describe the response characteristics of the visual system at suprathreshold levels of luminance and contrast, levels at which the eye, indeed, operates under most normal viewing conditions.

It follows that, in patients with demyelinating diseases, threshold abnormalities, as shown by the CS function, do not provide a complete description of the visual dysfunctions in these patients. For instance, a sensitivity loss will not necessarily be accompanied by an equivalent loss of apparent contrast above threshold, i.e., it may not degrade the patient's ability to perceive high-contrast targets. Hence, the study of suprathreshold responses may be more appropriate for predicting the quality of everyday vision in patients who often complain of unusual visual experiences.
Two studies testing suprathreshold vision in demyelinating diseases have been reported. Regan\textsuperscript{11} studied the relationship between threshold deficits and the discrimination of high-contrast Snellen letters in multiple sclerosis (MS) patients. Tagami and Isayama,\textsuperscript{12} using square-wave patterns as stimuli, reported abnormalities in the Mach-band phenomenon in two optic neuritis patients.

In the present study, we compared contrast detection thresholds and suprathreshold responses (as measured by a contrast-matching procedure) in a group of nine patients with MS or optic neuritis. The study included both stationary and temporally modulated sine-wave gratings. A group of 26 normal subjects was studied as controls. A preliminary report of the results appeared elsewhere.\textsuperscript{13}

### Materials and Methods

#### Apparatus

The stimuli consisted of vertically countermodulated sine-wave gratings. They were generated on a Joyce Company video display unit following the method of Schade\textsuperscript{14} as applied by Campbell and Green,\textsuperscript{15} and were controlled by a microcomputer. A frame rate of 100 Hz was used. Contrast was measured to be linear up to 60%. To account for the slight nonlinearity at higher contrasts, separate calibrations were made for each of the spatial frequencies used. All stimuli were presented at a mean luminance of 60 cd/m\textsuperscript{2}. A white phosphor was used.

The display screen subtended a visual angle of 7.5° vertically and 9.4° horizontally, and was surrounded by a gray field. The surround had a luminance close to the mean luminance of the pattern, and subtended a visual angle of about 27.5° horizontally and vertically.

The viewing distance was set at 150 cm and the subject's position was fixed by means of a chin rest. Subjects were instructed to fixate on the center of the screen. The fixation area was marked by four thin lines positioned orthogonally, which circumscribed a free central area of 2°. This type of fixation was preferred to a fixation point at the center of the screen, which might interfere with pattern detection.

Each eye was tested separately. The non-tested eye was occluded by a translucent eye patch. This arrangement prevented test field "blackening", a sensation due to binocular rivalry when one eye is occluded by an opaque patch. The room lights were baffled but left on during testing. Display luminance and contrast were measured under these ambient light conditions.

#### Test Procedure

**Threshold measurements:** Contrast thresholds were obtained on all subjects with SFs of 0.5, 1, 2, 3, 4, 6, 8, and 12 cycles per degree (cpd), stationary and countermodulating at a rate of 5 Hz. All subjects wore their best correction. The procedure used for the measurement of contrast thresholds was described in a recent study.\textsuperscript{16}

**Suprathreshold contrast measurements:** To obtain a quantitative assessment of the subjective impression of suprathreshold stimuli, a two-alternative temporal forced-choice tracking procedure was employed. The observer had to subjectively match the contrast of a test stimulus to that of a standard whose contrast was kept constant.

The observer was presented with two 3.8-sec intervals demarcated by tones. One contained the test stimulus and the other contained a standard stimulus of fixed contrast. The task was to determine which stimulus was of higher contrast. The pattern was ramped on and off gradually, by multiplying the contrast by half a cosine cycle of 900 msec. Thus, each stimulus pattern was at full contrast for 2 sec. This procedure alleviated the effects of temporal stimulation due to abrupt onset of the high contrast patterns. The standard stimulus, in each case, was a 2 cpd grating, either stationary or countermodulated at 5 Hz. The contrast of this standard stimulus was always set to be 1 log-unit (20 dB) above the observer's threshold.

The test stimuli, presented with the same temporal frequency as the standard, were of eight SFs, 0.5, 1, 2, 3, 4, 6, 8 and 12 cpd. Contrast of the test stimulus varied according to the observers' performance on each trial, as described below.

Gratings presentations were made in pairs; the screen was blank between each pair of presentations. The order of test and standard stimuli was randomly determined on each trial. In each contrast-matching session, the standard (2 cpd) was presented along with one spatial frequency of the test. The test contrast was initially selected at random and was greater or smaller than the standard contrast. The contrast of the test was initially adjusted in 0.06 log-unit steps. For instance, if the observer chose the standard as appearing higher in contrast than the test, the contrast of the test was increased by 0.06 log-unit on the subsequent trial. Conversely, choosing the test as having the higher contrast resulted in its contrast being decreased on the following trial. The observers indicated their choice by pressing a left-hand button if the first interval appeared to be of higher contrast, and a right-hand button if the grating in the second interval was of higher contrast than the first one. The 0.06 log-unit step was used until the observer reversed his decision about which stimulus appeared to be of higher contrast. The step size was then reduced to 0.03 log-units for the remainder of the series. A series was terminated after three reversals were obtained with the smaller step. The mean of the three reversals was...
ter, were retained as controls for this study. Corrected Snellen visual acuities (VA) of 20/25 or better were the same as those in a previous study.\(^{16}\) There were 10 females and 16 males whose ages ranged between 17-51 yr. From these subjects, 48 eyes, with corrected Snellen visual acuities (VA) of 20/25 or better, were retained as controls for this study.

Employing a large number of controls enabled us to make quantitative estimations of the variability of normal data, especially of the suprathreshold responses. In all analyses of the threshold and suprathreshold normal data presented in the results section, “normal limits” refer to the 99% confidence intervals adjusted to preserve SD = 0.01 for each of the eight spatial frequencies.\(^{17}\)

**Subjects**

*Control subjects:* The 26 normal subjects studied here were the same as those in a previous study.\(^{16}\) There were 10 females and 16 males whose ages ranged between 22-60 yr. From these subjects, 48 eyes, with corrected Snellen visual acuities (VA) of 20/25 or better, were retained as controls for this study.

*Patients:* The patient group was also the same as that investigated in a recent study.\(^{16}\) However, one patient was rejected because of difficulties in performing the contrast-matching procedure. Thus, nine patients, five males and four females whose ages ranged between 17-51 yr took part in the study. Five patients had multiple sclerosis (MS). Three of them were diagnosed as definite MS, one as probable MS, and one as possible MS, according to the scale defined by Rose et al.\(^{18}\) Among these MS patients, four had a history of unilateral retrolubular neuritis or optic neuritis and one (BB) had no clinical evidence of visual involvement in either eye. The remaining four patients had a previous episode of isolated optic neuritis, unilateral in three cases and bilateral in one. All patients were able to see the fixation target clearly.

Thus, a total of nine affected eyes, i.e., with previous retrolubular neuritis or optic neuritis, and nine clinically unaffected eyes were tested. It should be noted that the selection of patients was such that all patients had normal oculomotor function, and all “affected” eyes had clinically recovered most of their visual function; some patients, however, still complained of some subjective visual disturbance.

All patients had a systematic neuro-ophtalmic investigation before being tested. Details of the clinical data are summarized in Table 1.

Written informed consent was obtained from all control subjects and patients.

### Table 1. Clinical data of patients

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<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnostic</th>
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<th>Pupil defect</th>
<th>Optic discs</th>
<th>Visual fields</th>
<th>VA</th>
<th>VERs</th>
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<td>+</td>
</tr>
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<td></td>
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<td></td>
<td></td>
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<td>pale</td>
<td>scotoma</td>
<td>20/40</td>
<td>abnormal</td>
<td>+</td>
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<td>R = UA</td>
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<td>pale</td>
<td>intact</td>
<td>20/25</td>
<td>N</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>L = RBN</td>
<td>no</td>
<td>pale</td>
<td>scotoma</td>
<td>20/70</td>
<td>abnormal</td>
<td>+</td>
</tr>
<tr>
<td>3 (SO)</td>
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<td>35</td>
<td>MS (definite)</td>
<td>R = UA</td>
<td>no</td>
<td>N</td>
<td>intact</td>
<td>20/15</td>
<td>abnormal</td>
<td>+</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td>L = RBN</td>
<td>no</td>
<td>N</td>
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<td>20/15</td>
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<td>+</td>
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<tr>
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<td>N</td>
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<td>20/15</td>
<td>-</td>
<td>+</td>
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<td></td>
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<td>pale</td>
<td>scotoma</td>
<td>20/20</td>
<td>-</td>
<td>+</td>
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<td>L = UA</td>
<td>no</td>
<td>N</td>
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<td>20/20</td>
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<td>N</td>
<td>intact</td>
<td>20/15</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>L = O.N.</td>
<td>+</td>
<td>N</td>
<td>intact</td>
<td>20/15</td>
<td>-</td>
<td>+</td>
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<td>R = O.N.</td>
<td>+</td>
<td>pale</td>
<td>intact</td>
<td>20/20</td>
<td>abnormal</td>
<td>+</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>L = UA</td>
<td>no</td>
<td>N</td>
<td>intact</td>
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<td>N</td>
<td>no</td>
</tr>
<tr>
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<td>N</td>
<td>intact</td>
<td>20/15</td>
<td>N</td>
<td>no</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>L = O.N.</td>
<td>+</td>
<td>pale</td>
<td>intact</td>
<td>20/20</td>
<td>abnormal</td>
<td>+</td>
</tr>
<tr>
<td>9 (MO)</td>
<td>F</td>
<td>17</td>
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<td>R = O.N.</td>
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<td>pale</td>
<td>intact</td>
<td>20/50</td>
<td>N</td>
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</tbody>
</table>

UA = unaffected eye; RBN = retrobulbar neuritis; O.N. = optic neuritis; VA = visual acuity; VER = visual evoked potential; CT = contrast threshold.

Results

Contrast Threshold Functions

Contrast threshold (CT) functions were obtained by plotting the threshold contrast at each SF. Mean CT functions of the normal subjects with 99% confidence limits are shown in Figure 1, for each of the stationary (solid line) and 5 Hz countermodulated stimuli (dashed line).

The CT function of each of the patient’s eyes was individually compared with the mean control data. In
order to represent the differences between the patients' altered and the normal contrast thresholds clearly, visiongrams were derived by plotting at each spatial frequency, the ratio of the patient's threshold contrast to the mean threshold contrast of the control subjects at the same temporal frequency, steady-0 Hz and 5 Hz (see Figs. 6–10 for examples).

A deficit (i.e., thresholds higher than the 99% confidence limits of normals at more than one SF) was found in 15 out of 18 eyes tested, in all 9 affected eyes, and in 6 out of 9 clinically "unaffected" eyes. Threshold deficits of these patients are described in detail in the previous study.16

Suprathreshold Responses

For each eye tested, contrast threshold and contrast matching functions were obtained by plotting contrast thresholds and contrast matches at each SF. Thus, two sets of curves were obtained for both 0 and 5 Hz stimuli for each eye. Figure 2 shows examples of these functions from two normal subjects. On the left (Figs. 2A, C), threshold (solid symbols) and matching (open symbols) functions obtained with stationary stimuli, and on the right (Figs. 2B, D), the same functions obtained with 5 Hz countermodulated stimuli are shown. As can be seen in these figures, threshold and suprathreshold contrasts vary in different ways with stimulus frequency. That is, contrast needs to be raised by different proportions above threshold for different frequencies in order for those frequencies to be perceived as equal in contrast.

This proportionality was used to describe the suprathreshold response. Following Bowker,10 the ratio of the match contrast to the contrast threshold for each spatial frequency was called "gain" and plotted against spatial frequency.

Gain functions were derived for each of 0 and 5 Hz, and for each eye. For normals, a mean gain function was obtained by averaging the functions from all 48 control eyes. Figure 3 represents these average gain functions, for 0 Hz temporal frequency (solid line) and for 5 Hz (dashed line). In this figure, a gain of 10 represents a condition in which the test and the standard were judged equal when the test was raised 1 log unit above its own threshold. A gain score lower than 10 indicates that the test contrast was raised above its threshold by a smaller proportion (by less than 1 log unit) than the standard in order to be judged equal to it. A gain score higher than 10 represents the conditions where the test contrast had to be raised more than 1 log unit above its threshold in order to appear equal to the standard, which was 1 log unit above its own threshold.

When test and standard were matched in the spatial and temporal frequency domains, e.g., when the test
Fig. 3. The ratio of the match contrast over the threshold contrast plotted against spatial frequency. This function defines the "gain" function. The mean gain functions, averaged from 48 normal eyes, are shown. The solid line depicts the function obtained with the steady stimulus, and the dashed line with the 5 Hz temporally modulated stimulus. The vertical bars, in each curve, represent the 99% confidence limits.

and the standard were both 2 cpd countermodulating at 5 Hz, all observers closely matched the contrast of the test to that of the standard. In this case, the gain score was 10; test contrast needed to be raised by 1 log unit above its own threshold as was the contrast of the standard. As seen in Figure 3, the gain functions for the 0 Hz and the 5 Hz stimuli anchor at 2 cpd, confirming the adequacy of the matching procedure used for the measurement of stimulus apparent contrast.

The situation was different for spatial frequencies that did not match the standard, however. With stationary stimuli, the gain scores were high for frequencies between 2 and 4 cpd; that is, the match contrast was more than 1 log unit above threshold for these frequencies. Interestingly, this is the region of maximum sensitivity, or the lowest contrast thresholds. Gain scores were below 10 for the lower and the higher spatial frequencies; that is, the test stimuli needed to be raised by less than 1 log unit above their own thresholds to match the standard that was 1 log unit above its threshold. These spatial frequencies are the regions in the CT function where the thresholds are high, or fall-offs in sensitivity occur (Fig. 1).

The data with the countermodulated stimuli are similar. Gain scores are low, below 10, at the higher spatial frequencies where thresholds are higher, but gain scores are high, above 10, at the lower spatial frequencies where thresholds are low. In summary, for the control subjects, gain as it is calculated here is related to spatial frequency in a manner that is the opposite of the threshold contrast function. The response at the suprathreshold levels can be said to compensate for the regions of insensitivity at threshold levels.

The question is, does the suprathreshold response in optic neuritis and MS patients also show effective compensation for relative insensitivity observed at detection levels?

Suprathreshold Responses in Patients

Threshold and matching functions obtained from eight patients with the steady grating are given in Figure 4. Figure 5 shows the results from the remaining patient obtained by the 5 Hz stimuli as well.

The relation of the two functions is represented more economically by gain functions. Accordingly, gain functions were derived for the 18 eyes of patients, 15 of which showed a contrast threshold deficit, and 3 of
which had normal threshold responses (These three eyes from patients 2, 5, and 8 exhibited normal gain functions as well, and will not be discussed further). There were two major types of suprathreshold responses for the 15 eyes with a threshold deficit.

First, with a steady stimulus, 12 eyes of eight patients (both eyes for patients 1, 3, 4, and 7, and one eye of patients 2, 5, 8, and 9) showed compensation; that is, the gain scores of these patients were either within normal limits or they were lower. Second, for two patients (6, 9) and three eyes, again with a steady stimulus, there was undercompensation; that is, the gain scores were higher than normal for at least two spatial frequencies.

Individual cases representing variations within the first group of 12 eyes which showed compensation are discussed below, and represented in Figures 6–8. In these figures, the effect of 5 Hz countermodulation is also shown.

In a group of seven eyes within the 12 (from patients 1, 3, 5, 7, and 9), gain scores were significantly lower than the normal gain limits obtained from the control group. That is, these patients “overcompensated”; they needed a lower contrast, in relation to their threshold, than did the normals to match the test to the standard. Most importantly, for five eyes (patient 3 RE, LE; patient 7 RE, LE; patient 5 RE), overcompensation oc-

Fig. 5. Contrast threshold (solid symbols) and match functions (open symbols) from patient 4 obtained with the steady (solid lines) and 5 Hz stimulus (dashed lines). Panels A and B show the right and panels C and D the left eye.

Fig. 6. Threshold elevation (visuogram) on the left and gain functions on the right panels for patients 3 (A, B), and patient 4 (C–F). The average normal visuogram is depicted by the heavy horizontal line with ratio of 1. It is derived by averaging the ratio of the contrast threshold of each control subject to the mean threshold of all controls. Vertical bars are the 99% confidence limits of the mean ratio. Ratios higher than these limits represent a CT elevation. The average gain functions, shown in the right panels, are depicted by the heavy lines. Gain scores lower than 10 indicate a suprathreshold match that was less than 1 log unit above threshold for that spatial frequency; gain scores higher than 10 indicate a match contrast higher than 1 log unit above threshold for that particular SF. Panels A and B represent the visuogram and the gain function of patient 3, LE obtained with the steady stimulus. Panels C and D represent the visuograms and gain functions of patient 4, RE (circles), LE (triangles), obtained with the 5 Hz stimulus. Note that, in these examples, there is a correlation between the SF range where a threshold deficit occurs and where the gain scores are lower than 10, indicating frequency selective overcompensation. Panel E shows the threshold deficit revealed with the steady stimulus for the same patient. Panel F depicts the corresponding gain function. In this case, the gain scores are within normal limits, with the exception of 12 cpd, indicating normal compensation. Panels C–F together show the effect of 5 Hz modulation in increasing compensation.

curred selectively at the SF region where a deficit was found at threshold. This finding is depicted in Figure 6A–D.

In order to represent the relationship between threshold elevation and gain, threshold responses in the form of the visuogram are given on the left panel, and the gain functions on the right panel. The visuogram shows the degree of the threshold deficit loss in
Fig. 7. Visuograms (A, C) and gain functions (B, D) for patient 7, RE (circles), LE (triangles), obtained with the steady (solid lines) and 5 Hz (dashed lines). This patient had a unilateral threshold deficit, and yet presented a bilateral "compensation" for frequencies above 3 cpd. Heavy lines represent the normal functions with 99% confidence limits shown.

This exaggerated compensation, as demonstrated by reference to the horizontal solid line which represents the average visuogram of normal controls. Results from the LE patient 3 (Fig. 6A, B) under stationary conditions of stimulus showed a deficit (higher thresholds than normal), affecting selectively the high SFs above 2 cpd (Fig. 6A). The gain function under the same condition of stationary stimuli (Fig. 6B) showed an overcompensation at SFs higher than 2 cpd, and at 0.5 cpd as well. Thus, gain varied with SF in a proportionately inverse fashion as the threshold elevation. The same suprathreshold overcompensation phenomenon was observed in both eyes of patient 4 (Fig. 6C, D) with the 5 Hz countermodulated stimuli, but not with the steady stimulus (Fig. 6E, F). In both eyes, threshold responses to 5 Hz stimulus showed a CT elevation affecting mainly medium and high spatial frequencies above 2 cpd (Fig. 6C). The 5 Hz gain function (Fig. 6D) showed lower scores (overcompensation) for SFs higher than 2 cpd. Moreover, above 2 cpd, the magnitude of the loss (Fig. 6C) increased regularly with SF in both eyes, in much the same way as the gain changed. With the steady stimulus, this patient showed a threshold elevation affecting all spatial frequencies almost equally (Fig. 6E). Contrary to the effect found with the 5 Hz stimulus, the suprathreshold gain scores are within normal limits with the exception of 12 cpd.

This facilitory effect of 5 Hz countermodulation was common, with the exception of one case where gain scores with 5 Hz stimulus indicate undercompensation (Fig. 8F).

Figures 7 A–D represent enhancement of compensation in another subject, patient 7. This patient is particularly interesting, because overcompensation occurred in the eye (LE) which was clinically unaffected as well. She had recovered from an acute episode of optic neuritis in her right eye. Figure 7A and C show her visuograms for steady and 5 Hz stimuli, respectively; and Figure 7B and D show the gain functions with steady and 5 Hz stimuli, respectively. With steady stimuli, the right eye showed a threshold elevation across all SFs; the left eye showed no deficit (Fig. 7A). Similarly, with 5 Hz stimuli, there was a threshold deficit in the right eye, but no corresponding deficit in the left eye (Fig. 7C). In the gain functions, overcompensation was observed, for most high and medium SFs, for both left and right eyes (Fig. 7B). With 5 Hz stimuli, undercompensation was also observed in both the normal and deficient eyes. Moreover, the gain tended to vary with increases in SF (Fig. 7B, D), as did the magnitude of the threshold loss (Fig. 7A, C).

This exaggerated compensation, as demonstrated by
the suprathreshold responses in patients with threshold deficits, which occur at SFs of poor detection, is similar to the compensation effect observed in normals for the less sensitive (higher thresholds) regions of the CT function. This effectively serves to enhance the suprathreshold discrimination of poorly detected SF.

The exaggerated elevation of compensation could also be uncorrelated with the threshold deficit. Two examples are given in Figure 8. In the case of subject 1 (RE) with steady stimuli (Fig. 8A, B), a non-selective deficit was found at threshold (Fig. 8A). Above threshold, overcompensation occurred (Fig. 8B) at many (0.5, 1 cpd and 3, 4, 6 cpd) but not all SFs. In the case of subject 3 (LE) with 5 Hz stimuli (Fig. 8E, F), a threshold deficit occurred at most intermediate SFs (Fig. 8E), but overcompensation was observed at all SFs above 1 cpd (Fig. 8F), and undercompensation occurred at frequencies lower than 1 Hz. For still another example of overcompensation not correlated with the threshold deficit, see Figure 6E and F.

In Figure 8, we also present a case where the threshold deficit (LE) is not reflected in the suprathreshold response. However, while the gain scores for both eyes were within normal limits, they did not vary with spatial frequency the way the normal gain function does.

A major abnormality in suprathreshold responses was observed in a group of three eyes. Here, gain scores were higher than normal limits; that is, the patients used higher contrast than normal controls to achieve a 1 log unit suprathreshold match over a variable number of SFs. Examples are shown in Figure 9. Again, visuograms on the left show threshold deficits, and gain functions on the right show suprathreshold response. Patient 6 (Fig. 9A-D) presented threshold deficits in both eyes with steady (Fig. 9A) and 5 Hz stimuli (Fig. 9C). With steady stimuli, gain scores were higher than normal (Fig. 9B) at all SFs above 2 cpd for the right eye, and SFs above 3 cpd for the left eye. It should be noted that the decrease in compensation in the right eye occurred at SFs of normal threshold sensitivity (Fig. 9A), and was more pronounced than that of the left eye, which had the larger threshold loss. Moreover, in both eyes, undercompensation tended to become more pronounced as SF increased above 2 cpd. With 5 Hz stimuli (Fig. 9C, D), a comparable reduction in compensation was observed in both eyes (Fig. 9D). However, in the right eye, undercompensation shifted to higher SFs and was less pronounced (i.e., gain scores were closer to normal limits) with 5 Hz than with steady stimuli. As can be seen from these figures, the decrease in gain was not correlated with the threshold deficit. The missing gain score for 12 cpd reflects the subject's inability to make a match for this spatial frequency. Finally, a question could be asked whether the suprathreshold response types are correlated with the clinical assessment of the eye. A chi-square test for the independence of the state of the eye (affected and unaffected) and compensation (classified on three levels: overcompensation, compensation to normal level, and undercompensation) revealed no association between clinical history and suprathreshold response pattern. Neither was there a correlation between the response types and optic neuritis and MS.

**Discussion**

It has been demonstrated\(^8,10\) previously that contrast-matching to stationary high contrast stimuli show some characteristic features. In general, perceived contrast, as a function of spatial frequency, does not reflect the regions of attenuation of sensitivity observed at threshold levels, and apparent contrast becomes progressively more independent of frequency as the standard contrast increases.

It has also been demonstrated\(^10\) that contrast-matching with counter-modulated stimuli at 8 Hz show an enhancement in the suprathreshold response at high SFs (above 2 cpd). This is highly correlated with the 8 Hz contrast sensitivity function, in which attenuation occurs only at high SFs.

The present results on normal observers, which represent averaged data across 48 eyes, agree well with those reported on data from two individual subjects by Georgeson et al\(^8\) and two by Bowker\(^10\) for comparable contrast levels. Our data gathered at standard
contrast levels 1 log unit above threshold show some attenuation of apparent contrast at the low and high spatial frequencies, but not to the same degree as at threshold levels. The data gathered with 5 Hz temporal modulation show similar features. It may be presumed that a process exists which leads to compensation for physiological threshold attenuation due to neural blur at low spatial frequencies, or optical blur at high spatial frequencies. The main question posed in this study concerned the existence of a similar compensatory process in pathological conditions, such as demyelination of the visual pathway, which lead to high contrast thresholds.

The results obtained with 18 eyes of 9 patients with MS and optic neuritis showed that seven observers (12 eyes) exhibited varying degrees of improvement in the matching task over the threshold deficit. Moreover, for the majority, effective compensation above threshold was significantly increased as compared to that of normals. In some of these cases, such enhancement occurred at the SF region that was affected by the threshold loss. An exaggeration of the compensation process described in normal subjects could operate in these patients to help make the perception of objects independent of the quality of their detection at threshold. This might explain the absence of visual complaints in all of our MS patients with large threshold deficits.

As a general conclusion, it can be stated that the compensation process does not only operate when visual information is degraded by optical and neural limitations of the eye; it can also be observed under conditions of neural deficits due to pathological processes. A similar effect of compensation has been reported in astigmats by Georgeson et al. who tested 13 subjects with a residual elevation of CT, and by Hess and Bradley in amblyopic patients. Georgeson reported that "... some observers showed varying degrees of improvement in the matching task over the threshold... indicating at least partial correction." However, no correlation was found between the compensation scores and the amount of threshold deficit for any of their astigmatic subjects. Similarly, some patients in the population that we studied showed little correlation between the degree of compensation and the extent and type of threshold deficit.

The compensation effect rather resembles loudness recruitment, observed in patients with lesions in the peripheral auditory pathway (for review, see Moore). In loudness recruitment, the patient may not be able to hear very faint sounds, but sounds of high intensity are just as loud to him as to a normal listener. However, it has been pointed out that sounds which are highly audible to a patient with recruitment may not be readily intelligible. Analogously, in patients with threshold visual deficits and compensation effects above threshold, the overall quality of vision may not necessarily be rendered better.

Contrary to the effect described above, a deficit in compensation was found in some other patients. This attenuation in suprathreshold response was not correlated with threshold loss; in one observer, the suprathreshold response was consistently worse than normal at SFs where threshold sensitivity remained normal. Similarly, in his study of MS patients for discrimination of high-contrast Snellen letters, Regan reported opposite findings in two observers. In one patient with threshold loss, a deficit in discrimination was found, whereas, in another observer, despite the threshold loss, discrimination was not degraded. The latter might be interpreted as a result of a compensation effect.

The use of the term gain and compensation, although intended here as descriptive device, necessarily implies the existence of a neural mechanism which amplifies or attenuates the signals generated by high contrast signals. Georgeson and, later, Bowker suggested the presence of a mechanism in the visual system which effectively corrects for attenuation in sensitivity at threshold levels by adjusting the response of the detectors (or channels) to suprathreshold stimuli. Further evidence for an effective compensation mechanism has been previously provided by other authors. Indeed, if apparent contrast were coded by the magnitude of the signal, then threshold and match curves should have been parallel, and the "gain", as we have calculated it, should have been 10 for each frequency. This is not the case. It may be argued, however, that it is not necessary or even proper to invoke a compensatory neural circuitry as a basis for the results. First of all, one could argue, gain of spatial mechanisms as such (response-contrast curves for each spatial frequency) were not studied, and the way of expressing gain by the difference between match and threshold contrast obscures the possibility that the dynamic range as well as sensitivity in some patients may be reduced. In addition, the suprathreshold performance may well be unassociated with threshold performance. For example, an increase in threshold may be the result of increased neural noise. A high contrast signal may not be affected by the increased variability, and suprathreshold contrast matching may be normal. Another reason for threshold increase may be a decrease in spatial summation as a result of a decrease in the number of functioning cells. Suprathreshold response, it is known, is not influenced by spatial summation. In other words, the increase in gain, as we calculate it, may well reflect a threshold deficit accompanied by normal suprathreshold match, rather than a true adjustment of the high contrast signal by some neural mechanism. There is indeed evidence from animal and human electrophysiology, as well as psychophysics, which suggests that contrast perception depends on a high and
a low contrast mechanism and that, under some pathological conditions, they can be dissociated. These arguments would lead one to expect that, in the case of increased or "normal" compensation, the match curve would be normal while the threshold curve would show the deficiencies. To examine this possibility, a "normal" match curve was constructed. Figure 10A shows the results from the only eye for which the suprathreshold response was within normal limits while the threshold response showed frequency specific deficits. In all other cases of increased or normal gain, the threshold and match curves were both displaced from normal; it was the ratio between the curves that was either smaller or the same as the normals. An example of what can be interpreted as true increased gain is shown in Figure 10B. Here, there is interocular transfer of gain; in the left eye, the threshold curve is near normal, while the match curve is significantly better than the normal, yielding smaller ratios than normal. The right eye shows a threshold and match deficit, but the ratio by which a stimulus had to be raised above threshold in order to be judged equal to a standard is simply not as high as the normal population, especially at the regions where the threshold deficit occurred. In Figure 10C, a case of undercompensation is presented. Here, the threshold curve is normal for the right eye for the higher frequencies, but the match curve is significantly less sensitive than normal, yielding higher than normal ratios between these curves. This may be a case of true attenuation. For the left eye, both curves are displaced in such a way that the ratio between the curves is larger than the normal.

It could be argued that the match curves for the RE (open circles) in Figure 10B and the LE (open triangles) in Figure 10C may be "normal" for the standard contrast used. In this experiment, there is no way of judging this possibility. The data state, however, that patients with demyelinating diseases while showing compensation for threshold deficits, as does the control population, do not always perceive stimuli 1 log unit above their own thresholds as the control population does.

The data, in general, do not supply unequivocal evidence for dissociated performance of low and high contrast systems. Nor can all cases be interpreted as giving evidence for a gain mechanism that amplifies or attenuates the incoming high contrast signal according to threshold performance.

In summary, these findings indicate that threshold is not the only aspect of perception that can be impaired by demyelination of the visual pathway. Perception of stimuli at suprathreshold levels of contrast can either be attenuated or enhanced. This attenuation or enhancement may or may not depend on threshold losses. It is not clear why some observers exhibit suprathreshold attenuation, while the majority show compensa-

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