Balance between pattern and flicker sensitivities in
the visual fields of ophthalmological patients

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SUMMARY We measured the balance between visual sensitivities to pattern and to flicker rather
than measuring absolute sensitivities to pattern or flicker. The test target was a 2-cycle deg⁻¹
sinewave grating that was counterphase modulated at 8 Hz. Seventeen points in the visual field
were tested out to eccentricities of 24°. We examined 10 control subjects, 6 patients with glaucoma,
10 with ocular hypertension, and 10 with multiple sclerosis. For controls pattern sensitivity was
lower than flicker sensitivity in central vision. The converse held in peripheral vision. The balance
between pattern sensitivity and flicker sensitivity was markedly abnormal in part or all of the visual
field for many patients. There were examples in all patient groups. In some patients flicker
sensitivity was depressed relative to pattern. In others the converse was true. Of 10 patients with
ocular hypertension and no perimetric field loss 8 had a significantly abnormal ratio between
pattern sensitivity and flicker sensitivity at some point in the visual field. The balance between
pattern and flicker sensitivity was more sensitive to visual pathology than absolute sensitivity to
either pattern or flicker. We conclude that the relationship between pattern and flicker sensitivity
may be more sensitive to visual field damage than is conventional perimetry or visual acuity
perimetry.

A moving or flickering pattern may look quite
different at threshold and at suprathreshold contrast
levels. A near threshold pattern does not necessarily
look like a fainter version of its suprathreshold
counterpart. As stimulus contrast is slowly raised
from zero the first impression can be one of a motion-
less spatial pattern, and not until the stimulus has an
appreciably higher contrast is flicker or motion visible
as well as pattern.¹⁻³ In other cases it has been
reported that there is no impression of pattern until
stimulus contrast is somewhat higher than flicker
threshold, though this second observation was not
confirmed when a forced choice method was used⁴
rather than the method of adjustment.

It has been proposed that the existence of different
contrast thresholds for pattern and for flicker percep-
tions reflect the operation of 2 neural mechanisms,
one signalling the presence of a stationary pattern
and the other signalling motion or flicker.⁵⁻⁷

In both kinetic and static clinical perimetry either
of these mechanisms might detect the target. There-
fore, if some visual disorder affected one mechanism
while sparing the other, clinical perimetry might fail
to detect the visual loss. A further point is that the
balance between visual thresholds for pattern
perception and flicker perception might be a more
sensitive indicator of pathophysiology than is visual
acuity or pattern vision alone. Exploring these ideas
we measured the ratio of thresholds for pattern per-
ception and for motion perception at 17 sites in the
visual field using a single 2-cycle deg⁻¹ target that
was counterphase modulated at 8 Hz (16 reversals
s⁻¹). It is known that before spatial vision is affected
visual disorders can affect the temporal properties of
the visual system⁸ and upset the dynamic response
coefficient (DRC) measure (defined as the mean of
visual sensitivities to a grating and to a flickering
homogeneous field⁹). Previous studies, however,
used different stimuli to test spatial and temporal
properties, and few explored the visual field outside
the macular region. Here we compare 2 different
percepts produced by a single stimulus, so that our
findings can be directly compared with the relevant
body of basic research.²⁻⁴⁻⁶⁻⁷

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Material and methods

A sinewave grating stimulus of spatial frequency 2 cycles deg⁻¹ and mean luminance 20 cd m⁻² was generated on a cathode ray tube by special purpose electronics. The grating was circular, subtended 3.5° diameter, and was viewed from 145 cm. It was surrounded by a green adapting field subtending 40x40°. Contrast thresholds were measured by a tracking technique described previously. Subjects were instructed to fixate either in the centre of the square or at a fixation mark located at eccentricities of 4°, 8°, 16°, or 24° along the 4 oblique half meridia. The experimenter had the subject's eye in view throughout each measurement. The unused eye was occluded. Subjects were instructed to set separately for: (a) pattern just visible, (b) flicker or contrast just visible, and (c) screen just different from blank. Otherwise the procedure was as described previously. Each point was measured at least twice during each session. If the measurements did not agree to within 3 dB further repeats were recorded.

We calculated the ratio between pattern and flicker thresholds at 17 sites in the visual field for each individual studied. The statistical treatment of these data was as follows. First, we calculated the ratio of flicker threshold to pattern threshold at each of the 17 sites for each control eye. Then we averaged over all control eyes to obtain the mean ratio and its standard deviation at each of the 17 sites. An abnormal balance between pattern and flicker thresholds at any of the 17 sites was defined as a ratio that fell more than 2.5 standard deviations beyond the control mean. Only 1 in 100 control subjects would fall outside this limit (99% confidence).

The same measurements were carried out on a total of 10 patients with ocular hypertension (age range 45 to 69, mean 59 years), 6 patients with glaucoma (age range 40 to 65, mean 58 years), and 10 patients with definite multiple sclerosis (age range 21 to 52, mean 36 years). Ocular hypertensives all showed a pressure of 22 mmHg or greater measured by applanation tonometry on at least 2 occasions over a one-year period. All had normal fields on Goldmann perimetry and were normal on fundus examination. Normal limits for the ratio of flicker to pattern thresholds were established with 10 control subjects. Findings on patients were reconfirmed at subsequent visits.

Conventional visual fields were obtained on the Goldmann perimeter and/or the Octopus automatic perimeter, and were assessed 'blind' by an ophthalmologist. Snellen acuities for control subjects ranged from 6/4-5 to 6/6.

Results

Anecdotally it has long been known that pattern thresholds are higher than flicker thresholds in central vision for most control subjects while the reverse obtains in peripheral vision, but this observation had not previously been quantified. Fig. 1A plots mean pattern thresholds (continuous line) and flicker thresholds (broken line) versus eccentricity in the visual field for 10 control subjects. For 9 out of 10 of the control subjects pattern thresholds were higher than flicker thresholds from 0° eccentricity to about 15° after which the reverse applied.

In view of the strikingly clear difference between the 2 thresholds that is evident subjectively it may seem surprising that the difference in mean flicker
pattern threshold and mean flicker thresholds is not larger in Fig. 1A. That is because the treatment of the data in Fig. 1A brings out the mean thresholds but is a clumsy way to reveal the balance between pattern and flicker motion thresholds. It is statistically preferable to calculate the ratio of pattern to flicker motion thresholds in each individual control subject before averaging. This is the reason why we calculated ratios separately for each subject before averaging and then plotted the mean ratios versus eccentricity in Fig. 1B. Numerical control data with upper normal limits are set out in Table 1.

In several patients sensitivity to flicker was reduced while sensitivity to pattern was comparatively unaffected. This could occur in part or in the whole of the visual field. Fig. 2 gives one example for a male patient aged 65 with ocular hypertension. His Snellen acuity was 6/6. Vertical bars indicate where the

### Table 1

<table>
<thead>
<tr>
<th>Position in visual field (degrees)</th>
<th>Fovea 0</th>
<th>Q 4</th>
<th>Q 8</th>
<th>Q 16</th>
<th>Q 24</th>
<th>C 4</th>
<th>C 8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pattern to flicker sensitivity range</td>
<td>CT, mean</td>
<td>-4.6</td>
<td>-4</td>
<td>-2.1</td>
<td>0.3</td>
<td>2.6</td>
<td>-3.5</td>
</tr>
<tr>
<td></td>
<td>CT, upper normal limit</td>
<td>2.4</td>
<td>2.2</td>
<td>3.1</td>
<td>7.5</td>
<td>10</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>CT, lower normal limit</td>
<td>-11.6</td>
<td>-10.2</td>
<td>-7.3</td>
<td>-7.2</td>
<td>-4.9</td>
<td>-8.5</td>
</tr>
</tbody>
</table>

CT = contrast threshold ratio.
Balance between pattern and flicker sensitivities

![Graph showing balance between pattern and flicker sensitivities](image)

Fig. 3 Vertical black bars indicate an abnormal balance between pattern threshold and flicker threshold. Stars indicate where absolute sensitivity to pattern was depressed. The same experimental procedure was used as for the control subjects of Fig. 1A, but the data are quite different. Normal limits were set at the 99% significance level. Snellen acuity was 6/7.5 in this patient with glaucoma.

balance between pattern and flicker thresholds was abnormal (99% significance level). Stars mark where absolute flicker threshold was abnormally depressed (99% significance level), showing that the balance of thresholds was a more sensitive indicator of pathology than absolute thresholds to either pattern or flicker. This patient's field by Goldmann perimetry was normal. The Octopus field was normal in all the areas marked as abnormal in Fig. 2 except for the top right field, where there was an equivocally small (4–8 dB) depression of sensitivity.

Less common was the converse pattern of loss, an elevation of pattern threshold relative to flicker threshold. Fig. 3 illustrates this pattern of loss with data for a male patient aged 59 with glaucoma. His Snellen acuity was 6/7.5. Vertical bars indicate where the balance between pattern and flicker-motion thresholds was abnormal (99% significance level). Stars indicate visual field locations where absolute pattern threshold was abnormally depressed (99% significance level), showing that absolute pattern threshold was abnormal at only one point, while the balance between pattern and flicker thresholds was abnormal at 7 points. The Octopus field was normal in all the areas marked as abnormal in Fig. 3 except for the top right field, where there was a peripheral field defect of 11–14 dB. Other patients gave examples of completely normal Octopus and Goldmann fields associated with depressed flicker sensitivity or depressed pattern sensitivity in part or of all of the visual field tested.

A third pattern of loss was shown by glaucoma patients with absolute scotomata. Both pattern and flicker thresholds were similarly raised in the vicinity of absolute scotomata.

In total, all 6 glaucoma patients, 6 out of 10 patients with multiple sclerosis, and 8 out of 10 patients with ocular hypertension had an abnormal balance between pattern and flicker thresholds at one or more points in the visual field. Fifteen of the eyes with these occult field defects, were normal to conventional perimetry.

Discussion

As mentioned above it has been proposed that the human visual pathway has somewhat separate mechanisms for pattern and flicker. Evidence for this idea has so far been gathered from normally sighted individuals. Our finding that the balance of visual sensitivities to pattern and to flicker can be tilted in either way in retinal or visual pathway disease adds pathophysiological support to the idea of somewhat
separate mechanisms. Further to this point, no patient showed a relative depression of pattern sensitivity in one part of the field and a relative depression of flicker sensitivity in another part. If one sensitivity was significantly depressed at one location it was depressed to some extent through the field. This finding is consistent with the existence of sufficiently separate pattern and flicker mechanisms that pathophysiology is much more likely to affect one or other mechanism over the whole of the visual field than to involve both mechanisms simultaneously. Of the 8 patients with ocular hypertension who had an abnormal sensitivity balance 7 (88%) had a relative depression of flicker sensitivity. Results were somewhat more mixed in multiple sclerosis. Of 6 patients with an abnormal balance 4 (67%) had a relative depression of pattern sensitivity. This preliminary finding is consistent with different sites of pathology, presumably the retina in ocular hypertension, while multiple sclerosis can involve axons at levels ranging from optic nerve to cerebral white matter.

As to the clinical implications of our findings, the main conclusion is that in ocular hypertension the balance between pattern and flicker thresholds can be upset in the parafoveal (4° eccentricity) or peripheral field without significant change in absolute thresholds either for pattern or for flicker-motion, and before any field defect is evident to conventional perimetry. That the loss was usually of flicker rather than pattern perception may be related to previous findings that double flash resolution and critical fusion frequency are affected in some patients with glaucoma, though our main point is that the balance of sensitivities is more sensitive than sensitivity to either flicker or pattern alone.

In none of the patients with glaucoma or ocular hypertension did the central visual field show abnormal visual responses. One implication of this apparent rarity of central abnormality is that it may limit the effectiveness of Arden plates in glaucoma screening.

We propose the following pathophysiological basis for our finding that visual responses to a sinewave grating of low spatial frequency can be abnormal in a part of the visual field where acuity is spared. It is known on morphological grounds that ganglion cells' dendritic trees have a wide range of sizes even within a single class of ganglion cells. Suppose that the dendritic sites most distant from the cell body are more vulnerable to functional loss in ocular hypertension and glaucoma than are dendritic sites less distant from the cell body, perhaps because of the greater distance that a signal must travel from the outer boundary of the tree before reaching the cell body. Then, for any given dendritic tree, the most distant dendritic processes would be the first to lose their ability to transmit neural signals. The biggest dendritic trees would be most vulnerable, and these presumably belong to the ganglion cells with large receptive fields that govern visual sensitivity to our low spatial frequency test grating. Less vulnerable would be the ganglion cells with smaller dendritic trees that presumably govern visual acuity.

Finally, we note that this hypothesis is consistent with retinal ischaemia as a possible causative factor in visual field loss (additional and distinct from the widely discussed causative factor of ischaemia in the disc region13-16).

Although the number of patients in this preliminary study was small our finding of 80% of patients with ocular hypertension with unbalanced pattern versus flicker sensitivities far exceeds the highest (16%–20%) percentage of patients with OHT who subsequently develop glaucomatous field defects. This can be understood in terms of our hypothesis of dendritic pathophysiology. The ganglion cells whose dendritic dysfunction causes reduced contrast sensitivity are not necessarily the cells whose death due to axonal pathophysiology causes glaucomatous field defects.

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References
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