Spectral threshold: measurement and clinical applications

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SUMMARY Photopic spectral sensitivities for a foveal target on a white background are measured for 18 normal eyes, and the results are explained in terms of the function of retinal ganglion cells. Averaged results for diseases such as glaucoma, optic atrophy, tobacco amblyopia, and retinal neuritis are reviewed, and an analysis of the change in shape of the spectral sensitivity curve in these diseases is presented. It is shown how the location of disease sites may be related to characteristic changes in spectral sensitivity.

The assessment of colour abnormalities has long been of interest in the evaluation of the patency of, the visual pathways. Kollner attempted a classification of acquired colour vision defects according to their disease site: a defect in the media or receptors causes a blue/yellow defect (tritan), whereas dysfunction of the visual pathway from the ganglion cell layer to the cortex causes a more pronounced defect in red/green discrimination.

The clinical importance of the assessment of colour vision lies in (a) the susceptibility of the colour detecting system to early disease processes in the eye and its perceptual pathway, and (b) the fact that abnormal colour vision, when the abnormality is due to disease, often precedes and then parallels a decrease in visual acuity. Colour detection and interpretation relies on 3 parameters: saturation, hue, and luminosity. Some common screening devices for colour defects rely on discrimination of a colour from a grey of equal brightness (colourimetric purity or saturation), and their aim is to find congenital red or green colour defects—for example, Ishihara plates. Clinical tests to measure blue/yellow and red/green colour defects often use hue discrimination (e.g., panel D-15 or Farnsworth-Munsell 100-hue), requiring the patient to set in order various wavelength combinations. The spectral sensitivity function described here may be altered by a change in the relative contributions of saturation.

Material and methods

A 1° spot test target of wavelengths 400–650 nm in 25 nm steps is presented monocularly in a Maxwellian view system with a trial lens cell for correcting the patient’s refractive error, and an artificial pupil 2 mm in diameter. Foveal fixation, aided by 4 black fixation spots surrounding the test target, is used against a white or coloured background of intensity 1000 to 10000 trolands. The colour and intensity of the background can be varied and by Marré’s methods; coloured backgrounds can be applied to distinguish cone functions. The patients were adapted to each background: 1 minute for white, 1.5 minutes for blue, 1.75 minutes for magenta, and 2.5 minutes for yellow backgrounds.

For most experiments the test target was presented in 0.5 second flashes from blue (400 nm) through 11 filters to red (650 nm) and in 50 nm steps back to blue. The subject reduced the intensity of the test spot until it was barely visible (threshold). In spectral flicker sensitivity measurement the coloured test spot flickered at 25 Hz on a white background, and the subject set a threshold for flicker. The results are automatically plotted on an X–Y plotter, with mean sensitivity of one or 2 readings (i.e., the reciprocal of threshold) plotted against test wavelength (in nanometers).

Results

Fig. 1 shows the average spectral sensitivity curve for 18 normal subjects (mean age 35 years) determined.
with 0.5-second test flashes on a white background. The 3 peaks near 440, 520, and 600 nm are thought to be derived from the responses of 'tonic' ganglion cells which have an opponent colour organisation.45

Protan and deutan congenital colour defects can be differentially diagnosed, and spectral sensitivities (for 0.5-second flashes on a white background) are given in Figs. 2a and 2b. Although the apparatus is mainly designed to detect subtle acquired colour vision defects, these diagrams are included to show (a) the distinctive 2-peak curve of the dichromat is readily detectable, and (b) even if a patient has a congenital colour defect, loss of colour vision from disease can still be discerned.

In some cases of glaucoma the visual acuity as measured on a Snellen chart may be normal though the peripheral field is restricted. Is it possible to measure any changes of central vision in these cases? An averaged spectral sensitivity for glaucoma patients with visual acuity of 6/9 (or better) is shown in Fig. 2c. Only a small decrease in sensitivity was noted for test spots greater than 500 nm (green to red spectral lights), but the test wavelengths less than 500 nm (blue region of spectrum) showed a sensitivity substantially less than normal.

In toxic amblyopia there is a moderate to severe loss of visual acuity with central scotoma. The visual disturbance caused by overconsumption of tobacco includes the classical loss of central vision for red. A large loss of long wavelength sensitivity along with an even greater loss of narrow-band short-wavelength sensitivity relative to normal was noted in the average spectral sensitivities for 10 eyes with tobacco amblyopia6 (Fig. 2d).

Of particular interest to our Manchester group measuring acquired colour deficiencies with the spectral threshold technique are diseases which affect the optic nerve. The results of our measurements on 3 eyes with DIDMOAD (diabetes insipidus, diabetes mellitus, optic atrophy, and nerve deafness) syndrome and 10 eyes in a family of late-onset, dominant, hereditary optic atrophy are averaged in Figs. 2e and
These patients had little or no colour vision with the Farnsworth-Munsell 100-hue test, and their visual acuity ranged from 6/12 to 6/36. These results have previously been interpreted as a loss of tonic ganglion cells, and the observed spectral sensitivity for 0.5-second flashes—i.e., a smooth single curve peaking at 555 nm instead of the normal 3-peak composite opponent curve—is thought to be due to a sparing of the other major class of retinal ganglion cells, namely, phasic cells.

Electrophysiological research on the phasic ganglion has shown a primarily noncolour opponent system that responds well to fast flicker and has a single peak of spectral sensitivity at 555 nm. Flickering the test spot at 25 Hz (the flicker sensitivity test), we can predict the detection of the flicker that would be accomplished by the phasic ganglion cells in the normal eye. The spectral flicker sensitivity in our procedure yielded a single peak sensitivity at 550 nm in the normal eye (dashed curve, Fig. 3a). An average of 10 eyes with optic atrophy (Fig. 3b) showed their spectral flicker sensitivity to be very similar to normal in both amplitude and shape.

In some cases of retrobulbar neuritis (RBN) a decrease in the critical fusion frequency (CFF) has been observed. The spectral threshold (1 Hz) for eyes recovering from RBN showed a decrease in sensitivity, especially in the blue range (<500 nm), in our test on a white background. The spectral sensitivity for 25 Hz flicker of the RBN patients is an example of severe loss of flicker sensitivity (Fig. 3c), while the detection of 0.5-second flashes (e.g., at about 550 nm) is nearly normal.

Discussion

The results of the diseased eyes for 0.5-second flashes may be divided into 2 categories: the region of the spectral sensitivity curve less than 500 nm (referred to as ‘blue’), and the curve in the region greater than 500 nm (‘green/red’). This division is convenient for relating the results to the subclassification of tonic opponent-colour ganglion cells categorised as red/green and blue/yellow. In all the above diseases a loss in the blue sensitivity is noted. This correlates with the findings of other workers that the blue mechanism seems vulnerable early in the course of many eye disorders.

In the red/green region 2 systems are contributing to the spectral threshold—the colour opponent tonic ganglion cells and the phasic ganglion cells. In cases of generalised sensitivity loss—that is, when the curve for detection of 0.5-second flashes retains a normal shape—the loss may be due to nonspecific damage of all types of optic nerve fibres. After a specific loss of the red/green tonic cells we might predict a change in the shape of the spectral threshold, so that the 2 peaks near 520 nm and 600 nm would be replaced by a single peak at 555 nm (phasic cell peak sensitivity). King-Smith and others have interpreted the spectral sensitivities of patients with optic atrophy in terms of this selective damage to tonic cells. The loss of red/green sensitivity for the 0.5-second flashes seems to correspond well with acuity loss, while loss of only blue sensitivity seems to precede a measurable loss in central visual acuity.

Damage to the phasic cell system can be seen in the reduced sensitivity to the spectral flicker sensitivity at 25 Hz. In cases of retrobulbar neuritis (Fig. 3c) reduction in sensitivity of both types of optic nerve fibre occurs even after substantial recovery of visual acuity.

Conclusion

Many sensitive and different tests are available for the investigation of visual function of acuity, field, and colour. Some are purely objective and some have a psychophysical approach. Arden extensively reviewed spatial contrast sensitivity and discussed its application in clinical ophthalmology. Arundale studied contrast sensitivity function in ocular pathology, and Minassian et al. found a grating test simple and valuable in screening a large population for ocular disorders. The present study of spectral threshold is another sensitive measure of changes in macular function.
Location of damage in certain diseases may be indicated by selective reduction in sensitivity to colour or duration of the test light—for example, the selective loss of tonic ganglion cell function in the cases of optic atrophy discussed above. Spectral sensitivity curves may provide supportive evidence in diagnosis by comparison with average responses in known cases of disease. Depression in the spectral sensitivity curve relative to the normal may indicate early changes in central vision in patients who are otherwise asymptomatic.

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References