Clin. Vision Sci. Vol. 6, No. 1, pp. 49–57, 1990
Printed in Great Britain.

0887-6169/90 $3.00 + 0.00

RESEARCH REPORT

ABNORMALITIES IN HUE DISCRIMINATION REVEALED WITH VERY BRIEF STIMULI IN DIABETES MELLITUS AND IN OPTIC NEURITIS

MARK O. SCASE,[1] DAVID H. FOSTER,[1] WILLIAM P. HONAN,[2] JAMES R. HERON,[2]
MARTIN C. GULLIFORD[3] and JOHN H. B. SCARPELLO[3]

[1]Department of Communication and Neuroscience, University of Keele, Keele, Staffordshire ST5 5BG and Departments of [2]Neurology and [3]Endocrinology and Diabetes, North Staffordshire Royal Infirmary, Princes Road, Hartshill, Stoke-on-Trent, Staffordshire ST4 7LN, U.K.

(*Received* 8 *September* 1989; *in revised form* 13 *February* 1990)

Abstract—The effects of reduced stimulus duration on colour vision were assessed in two pathologies differentially affecting the optic nerve and retina. Hue-discrimination thresholds were measured for long and short-duration (500 and 2 ms) stimuli presented on a computer-controlled colour monitor in 10 patients with previous optic neuritis, in 17 diabetic patients, 8 with and 9 without retinopathy, and in 10 normal controls. All patient groups had normal Farnsworth–Munsell 100-Hue scores. Thresholds were obtained by a hybrid-adaptive procedure requiring hue discrimination about a reference white. As flash duration was reduced, normal subjects showed greater threshold elevations along the red-green axis than along the tritanopic axis. For patients with previous optic neuritis, performance was similar to, but uniformly worse than, that of controls. In contrast, for diabetic patients without retinopathy, reduced flash duration had the effect of increasing thresholds more along the tritanopic axis than along the red–green axis. For diabetic patients with retinopathy, this selective temporal effect disappeared. The results suggest that in diabetes significant abnormalities in retinal function may occur early in the disease, and that measures of hue discrimination with short-duration flashes may predict later, more marked, losses in colour vision.

Key words—Diabetes; optic neuritis; multiple sclerosis; colour vision; hue discrimination.

INTRODUCTION

Köllner (1912) was one of the first to classify pathological changes in colour vision. His observations led to Köllner's law which states that diseases of the retina produce blue colour-vision deficiencies whereas diseases of the optic nerve result in red–green deficiencies. This study examines hue-discrimination function in two pathologies: optic neuritis resulting from optic-nerve demyelination, and diabetes which can result in retinal damage.

Colour vision deficiencies often occur in optic neuritis. Types of colour loss include a desaturation in colour appearance (Burde and Gallin, 1975; Glaser, 1976), losses in colour discrimination (Lynn, 1959; Cox, 1960, 1961a, b; Rosen, 1965; Griffin and Wray, 1978; Chisholm, 1979), and raised chromatic thresholds (Fallowfield and Krauskopf, 1984; Mullen and Plant, 1985, 1986, 1987; Foster *et al.*, 1983, 1985). Diabetes is also associated with alterations in colour vision. There may be a reduced sensitivity of the short-wavelength-sensitive mechanism without significant loss of sensitivity of the medium- and long-wavelength-sensitive mechanisms (Adams *et al.*, 1980; Zisman *et al.*, 1981; Zisman and Adams, 1982). Losses have also been reported in colour discrimination (Verriest, 1963; Barca and Vaccari, 1977; Bresnick *et al.*, 1985; Birch and Dain, 1987). Even in patients without retinopathy, abnormalities in colour discrimination have been detected with the Farnsworth–Munsell 100-Hue Test (Kinnear *et al.*, 1972; Lakowski *et al.*, 1972/73; Green *et al.*, 1985).

The effects of reduced stimulus exposure on colour confusion were investigated by Taylor (1984) using the City University Test (Fletcher, 1975) in a small number of diabetic and optic neuritis patients. The City University Test is a plate-matching test in which the subject matches a test colour with one of four surrounding colours. Taylor (1983) had found previously

that when stimulus-exposure time was restricted to 3.75 ms, subjects with normal colour vision gave tritan-like responses (compare Foster *et al.*, 1986; Scase and Foster, 1988). Taylor (1984) tested six diabetic patients, three with retinopathy and found that those patients who were colour normal under conditions of unrestricted viewing time showed the same tritan-type effect observed with normal control subjects when viewing time was reduced. Those subjects without retinopathy gave a similar percentage of tritan errors to those for normal controls. Those subjects with retinopathy were unable to respond to the test at 3.75 ms exposure time. In those cases, however, once a response was obtained by increasing the viewing time, it showed the same tritan-like effect found in normal subjects. The five optic neuritis patients, colour normal in unrestricted viewing, showed a varied response with restricted viewing time in their affected eyes. Both red–green and tritan errors were found.

In the present investigation, colour-discrimination performance was measured psychophysically by a computer-controlled colour-television technique. Because acquired colour-vision deficiencies may lead to losses in chromatic discrimination for colours of low saturation (Lanthony, 1978) thresholds were determined for the discriminability of a reference white and that white with some colour added (equivalent to a colorimetric purity-discrimination threshold, Wyszecki and Stiles, 1982). The brightness of the stimuli being compared was kept constant so that discrimination was based solely on differences in colour. Traditionally this measurement is repeated for different directions in colour space and a curve plotted in the C.I.E. coordinate system showing the variation in threshold with colour direction, resulting in a MacAdam ellipse (MacAdam, 1942, 1943, 1959; Wright, 1941, 1943). In the present investigation, just four directions were explored, the "cardinal directions of colour space" (Krauskopf *et al.*, 1982), which correspond to independent activity in red–green and blue–yellow opponent-colour pathways.

The hue-discrimination test described here has a number of advantages including the fact that the stimulus is large (2-deg angular subtense), so performance would not be influenced unduly by subjects' visual acuity, as may occur with the Farnsworth–Munsell 100-Hue Test (Brown *et al.*, 1983), and that any direction from any point in colour space (within the phosphor coordinates of the colour monitor) can be explored. Gündüz *et al.* (1988) used a computer-controlled colour television system to detect early losses in colour-contrast sensitivity in patients with ocular hypertension and glaucoma. We wanted here to extend the results of Taylor (1984) to determine how hue discrimination changed as viewing time was decreased in diabetic patients and in optic neuritis patients. In addition to comparing colour function in these two pathologies, we were particular interested in determining whether discrimination might be compromised in patients with diabetes but without overt retinopathy. Two diabetic groups, the one with retinopathy and the other without retinopathy, an optic neuritis group consisting of patients with stable recovered optic neuritis, and a control group were investigated.

A preliminary account of the work described here has been presented elsewhere (Scase *et al.*, 1987).

METHODS

Apparatus and stimuli

A computer-controlled graphics generator (PLUTO, IO Research) driving a high-resolution colour monitor (Mitsubishi 3149E) was used to produce the test stimulus. The graphics generator had its own 16-bit 8088 processor and 512K of memory which enabled it to store and carry out fast graphics changes. The graphics controller provided a display with resolution 640 pixels horizontal by 288 pixels vertical. Input to each phosphor gun on the monitors was controlled by an 8-bit digital-to-analog converter, providing a palette of 16.7 million colours. (As explained later, this system provided sufficient intensity resolution to allow an accurate determination of hue-discrimination threshold.) It was possible to change screen colours within one frame cycle. The colour monitor had a 14-in self-convergence-type dot-phosphor shadow-mask tube screen with an in-line electron gun. The scanning frequency was set at 50 Hz vertical and 15.6 kHz horizontal and the monitor was run in non-interlaced mode. Minimum duration of light output from each phosphor (100% to 10% of maximum luminance) was 2.2 ms. Stimulus presentation and data collection were under the control of a laboratory microcomputer. Photometric and colorimetric calibrations of the display system were performed with a photometer (LMT,

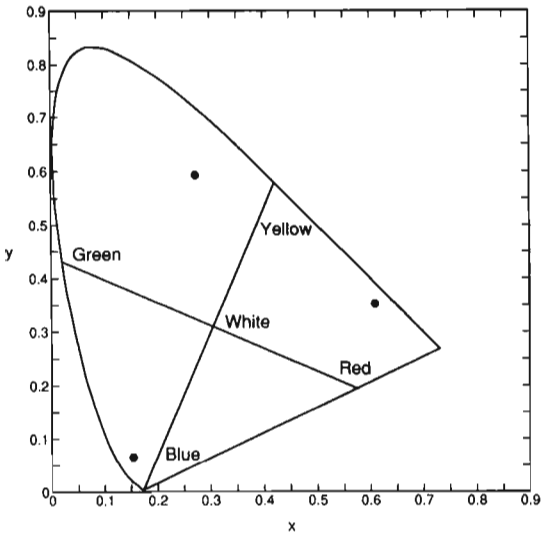

Fig. 1. CIE 1931 x,y-chromaticity diagram showing the chromaticity coordinates of the red, green and blue phosphors of the colour monitor (●). The cardinal directions of colour space are shown with reference to the white point. The line with ends labelled Blue and Yellow corresponds to a tritanopic confusion line. The line with ends labelled Red and Green corresponds to a red–green confusion line.

GmbH) and telespectroradiometer (PR-702A, Photo Research, U.S.A.), each in turn calibrated against standardized reference sources.

The test stimulus was a 2 deg-square horizontally divided bipartite field, of luminance approx. 7 cd m^{-2}. This value was constrained by the requirement that it be no larger than the maximum luminance of the blue stimulus used by patients with diabetes. The chromaticity coordinates of the reference white field produced by the monitor were $x = 0.3049$, $y = 0.3092$ (correlated colour temperature 7150 K). The test stimulus was drawn from one of the four cardinal directions of colour space (Krauskopf *et al.*, 1982, see Fig. 1, labelled conventionally blue, green, yellow and red). Surrounding the stimulus was a neutral field (correlated colour temperature 3100 K), luminance approx. 7 cd m^{-2}, produced by light from two tungsten lamps reflected from white card surrounding the monitor. The background subtended 23 deg by 15 deg with a 4 deg square aperture, luminance approx. 2 cd m^{-2}, in the centre upon which the test stimulus appeared. Stimuli were viewed monocularly at a distance of 2 m where the raster structure of the monitor display was not visible.

Subjects

Colour discrimination was measured in four groups of subjects.

(1) The control group comprised 10 healthy subjects not experienced in psychophysical measurements.

(2) The optic neuritis group comprised 10 patients. Three patients had had isolated optic neuritis and seven had multiple sclerosis and optic neuritis. All patients were in the remission phase of the disease and their vision was stable. The mean duration of optic neuritis ($\pm$SD) was 2.2 $\pm$ 1.9 yr, range 0.3–6 yr. There was no significant difference in age, sex or eye used between the control group and this optic neuritis group ($t = 0.79$, d.f. = 18, $P > 0.2$; $\chi^2_1 = 0.00$, $P > 0.5$; $\chi^2_1 = 0.95$, $P > 0.2$, respectively).

(3) The diabetic group without retinopathy comprised nine patients. There was no evidence for retinopathy on funduscopy or retinal photography. There was no significant difference in age, sex or eye used between the control group and this diabetic group ($t = 1.95$, d.f. = 17, $P > 0.05$; $\chi^2_1 = 0.43$, $P > 0.5$; $\chi^2_1 = 0.09$, $P > 0.5$, respectively).

(4) The diabetic group with retinopathy comprised eight patients. Funduscopic diagnoses of diabetic retinopathy were confirmed by a diabetologist or ophthalmologist. There was no significant difference in age, sex or eye used between the control group and this diabetic group ($t = 1.54$, d.f. = 16, $P > 0.1$; $\chi^2_1 = 3.6$, $P > 0.05$; $\chi^2_1 = 0.18$, $P > 0.5$, respectively).

Clinical details for subjects, including results of the Farnsworth–Munsell 100-Hue Test (Farnsworth, 1943, 1957) and City University Test (Fletcher, 1980), are given in Table 1. All tests were performed under the appropriate illuminant.

Procedure

The subject sat in an ophthalmic chair facing the colour monitor and with the base of the head supported by a headrest. The eye that was not tested was lightly occluded with an eyepatch. Subjects wore spectacles or correcting lenses if appropriate. Heterochromatic flicker photometry was performed at the beginning of the session to ensure that the stimuli used for hue discrimination were matched for luminance. The stimulus was a 2 deg square with the white and test colour interlaced and flickering at 25 Hz. The coloured stimulus corresponded to one of the phosphor guns being driven alone (see Fig. 1 for phosphor coordinates). The luminance of the coloured stimulus was fixed and that of the white was adjusted by a method of limits to determine the range of minimum

Table 1. Clinical details for subjects

	Controls	Diabetic without retinopathy	Diabetic with retinopathy	Optic neuritis
Number[a]	10	9	8	10
Male:female	3:7	4:5	6:2	3:7
Age (yr)[b]	38 (26–60)	49 (22–66)	45 (24–61)	(27–41)
Diabetes duration (yr)[b]	—	3 (0.6–10)	11.5 (2.5–28)	—
Insulin-dependence (proportion)	—	1/9	8/8	—
Blood glucose ($mmol.l^{-1}$)[b]	—	7.7 (7.0–14.6)	10.3 (8.3–18.8)	—
HbA1 (%)[b]	—	9.1 (6.8–12.9)	10.1 (8.9–13.7)	—
Blood pressure (mm Hg)[b]	—	130/85 (115/70–190/100)	120/85 (105/65–150/100)	—
Snellen acuity[b]	6/4 (6/4–6/6)	6/6 (6/5–6/12)	6/7.5 (6/5–6/9)	6/6 (6/4–6/36)
Near-vision acuity	N4.5(N4.5–N5)	N5(N4.5–N9)	N5 (N4.5–N24)	N4.5 (N4.5–N24)
City University Test error score[b]	0 (0–1)	0 (0–0)	0 (0–2)	0 (0–5)
Farnsworth–Munsell 100-Hue total error score[b]	88 (16–160)	104 (72–172)	90 (16–128)	92 (28–716)

[a]Five background retinopathy; 3 proliferative retinopathy with laser therapy/photocoagulation.
[b]Median (range).

flicker. Each match point was based on six measurements for each of the three phosphors.

Hue discrimination was measured using an adaptive two-interval forced-choice procedure (Taylor and Creelman, 1967; Hall, 1981). For each colour direction and stimulus duration, 50 trials were performed in a single experimental run. Each trial comprised two intervals, the one containing a flash with identical, reference white half-fields, the other a flash with different half-fields, one of which was the reference white and the other that white with a small amount of colour added. The temporal ordering of the intervals and the spatial ordering of the different half-fields was chosen pseudorandomly. The subject indicated which of the two intervals contained the different half-fields. No feedback on correctness of response was given. The interval between the two flashes in each trial was 1.5 s and that between each trial approx. 5 s. The amount of colour in the test stimulus was measured in terms of the logarithm of the distance, on the 1931 C.I.E. diagram, between the chromaticity coordinates of the test stimulus and the coordinates of the reference white. This level of the stimulus and the number of trials performed there were under the control of a modified PEST routine (Taylor and Creelman, 1967; Hall, 1981). After each set of 50 trials, which lasted about 5 min, the subject rested and the room lights were turned on for approx. 5 min.

The data derived for each test colour were fitted by a cumulative Gaussian function by a maximum-likelihood procedure (Foster and Bischof, 1987). Threshold was defined as the value of the log of the distance corresponding to a performance level of 75% correct. The standard deviation of this discrimination threshold value was estimated by a bootstrap technique (Foster and Bischof, 1987). Despite the discrete sampling of colour space determined by the intensity resolution of the colour-graphics generator, this procedure enabled accurate threshold estimates to be extracted. (The mean bootstrap standard deviation associated with an individual threshold was 0.079 log unit.)

Discrimination thresholds were measured separately for flash durations of 500 and 2 ms in the four cardinal directions in colour space (see Fig. 1). Total testing time was approx. 1.5 h.

RESULTS AND DISCUSSION

Mean data for the four groups of subjects are shown in Fig. 2. Polar plots of linear discrimination thresholds in the C.I.E. coordinate system are shown in the upper section of the figure (open symbols: thresholds for 500-ms stimuli; solid symbols: thresholds for 2-ms stimuli). The effects of flash duration are shown more clearly in the central section of the figure where log discrimination threshold is plotted against colour direction. Vertical bars show ± 1 SEM. The difference in log thresholds for short and long flashes (i.e. $Red_{diff} = Red_{short} - Red_{long}$, etc.) is plotted in the lower section of the figure (but note the expanded ordinate). A single numerical measure of overall duration-dependent hue discrimination was also calculated as $(Red_{diff} + Green_{diff}) - (Blue_{diff} + Yellow_{diff})$, which quantified the extent of a "short-flash tritanopia". Table 2 shows the loss of sensitivity (in log units) of the patient groups relative to the normal controls, for the different stimulus conditions. The table also shows the standard

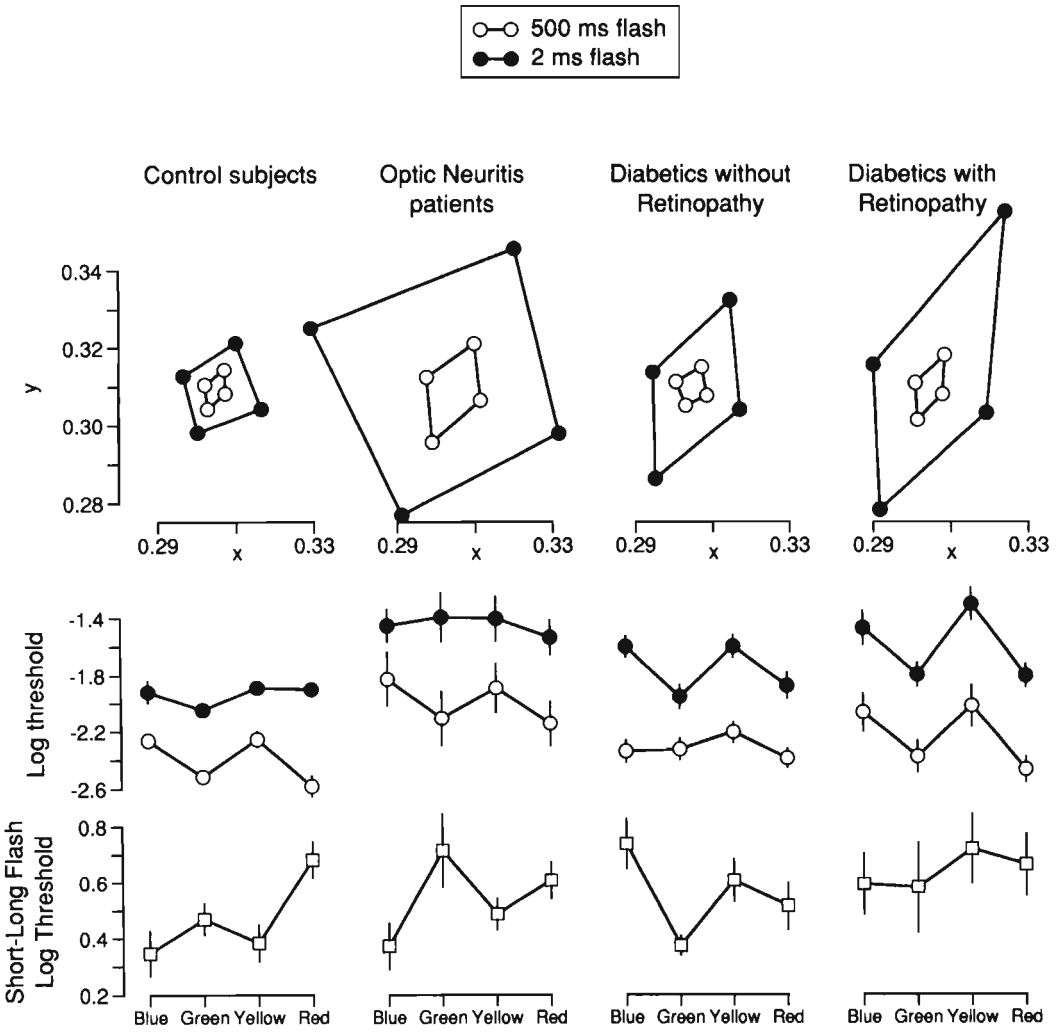

Fig. 2. The top panel shows polar plots of mean linear discrimination thresholds in the CIE coordinate system (○: thresholds for 500-ms stimuli; ●: thresholds for 2-ms stimuli). Thresholds are plotted for control subjects, optic neuritis patients, diabetic patients without retinopathy and diabetic patients with retinopathy. The middle panel shows mean hue discrimination thresholds measured as log distance on the CIE diagram for long (500-ms) flashes (○) and short (2-ms) flashes (●) plotted against test colour. Vertical lines show ±1 SEM where this is sufficiently large. The lower panel shows the numerical difference between the long and short flash threshold plotted against test colour.

deviation (SD) of the difference in threshold (in log units) between groups for each condition and the corresponding P-value (parametric t-test, two-tailed). None of the threshold data were found to be non-normally distributed.

Table 2. Differences in threshold between patient groups and normal controls

		Optic neuritis patients vs normal controls			Diabetics without retinopathy vs normal controls			Diabetics with retinopathy vs normal controls		
Duration	Colour	Log threshold	SD	P	Log threshold	SD	P	Log threshold	SD	P
Long	Blue	0.437	0.606	0.035	−0.076	0.255	0.365	0.198	0.407	0.145
	Green	0.409	0.611	0.049	0.190	0.232	0.019	0.138	0.321	0.192
	Yellow	0.365	0.574	0.060	0.048	0.277	0.598	0.235	0.452	0.124
	Red	0.437	0.548	0.021	0.191	0.302	0.069	0.112	0.337	0.329
Short	Blue	0.463	0.444	0.004	0.316	0.333	0.010	0.446	0.426	0.006
	Green	0.654	0.554	0.002	0.094	0.286	0.315	0.251	0.269	0.011
	Yellow	0.483	0.516	0.008	0.286	0.273	0.004	0.585	0.354	0.000
	Red	0.363	0.405	0.011	0.024	0.304	0.813	0.094	0.260	0.285

As flash time decreased, control subjects showed a non-uniform increase in discrimination threshold. The thresholds for red and green flashes were raised more than those for blue and yellow (Fig. 2, lower section), suggesting a greater loss in discrimination along an axis corresponding to the red–green confusion line than along the orthogonal axis. Taylor (1983) reported a time-induced tritan error when measuring colour confusion using the City University Test. That result appears to conflict with the present measurements, but may be a consequence of the distribution of colour differences in the City University Test.

Differences between control subjects and patients with optic neuritis

The optic neuritis group had significantly poorer Snellen acuity than the normal controls (Mann–Whitney $U = 17$, d.f. $= 1$, $P < 0.01$). There was no significant difference between optic neuritis and control subjects as assessed with the Farnsworth–Munsell Test ($t = 1.25$, d.f. $= 18$, $P > 0.2$) or City University Test ($U = 39$, d.f. $= 1$, $P > 0.2$). For long-duration flashes, optic neuritis patients showed a mild loss in discrimination in all colour directions apart from yellow (blue: $t = 2.28$, d.f. $= 18$, $P < 0.05$; green: $t = 2.11$, d.f. $= 18$, $P < 0.05$; red: $t = 2.53$, d.f. $= 18$, $P < 0.05$). For short-duration flashes, the optic neuritis group was much worse than the control group with all elevations in threshold being more highly significant (blue: $t = 3.30$, d.f. $= 18$, $P < 0.01$; green: $t = 3.74$, d.f. $= 18$, $P < 0.01$; yellow: $t = 2.96$, d.f. $= 18$, $P < 0.01$; red: $t = 2.83$, d.f. $= 18$, $P < 0.05$). There was no significant difference between the groups in the effects of flash duration, that is short-minus-long flash thresholds (blue: $t = 0.22$, d.f. $= 18$, $P > 0.5$; green: $t = 1.73$, d.f. $= 18$, $P > 0.05$; yellow: $t = 1.19$, d.f. $= 18$, $P > 0.2$; red: $t = 0.81$, d.f. $= 18$, $P > 0.2$), or in the short-flash tritan index defined earlier ($t = 0.32$, d.f. $= 18$, $P > 0.5$).

These results suggest a general rather than specific colour discrimination loss in optic neuritis. Relative to the control group, thresholds were elevated approximately equally in all colour directions with long and short flashes. There was, however, a greater increase in thresholds using the short flash than the long flash suggesting that the short-flash paradigm is more sensitive in detecting colour loss. The mean difference in threshold between the controls and optic neuritis patients with the long flash was 0.41 log unit and 0.49 log unit with the short flash. These results of a generalized colour loss with no selective loss of the red–green or blue mechanism are consistent with previous data on colour discrimination in optic neuritis (Fallowfield and Krauskopf, 1984; Mullen and Plant, 1985, 1986) obtained with longer-duration displays.

Differences between control subjects and diabetic patients without retinopathy

Snellen acuity was worse in the diabetic group without retinopathy than in the normal control group ($U = 14$, d.f. $= 1$, $P < 0.01$). There was no significant difference between diabetic patients without retinopathy and control subjects as assessed by the Farnsworth–Munsell 100-Hue Test ($t = 1.37$, d.f. $= 17$, $P > 0.1$) or City University Test ($U = 50$, d.f. $= 1$, $P > 0.2$). For long-duration flashes, diabetic patients without retinopathy showed a mild loss in discrimination relative to the control subjects in the green direction only ($t = 2.58$, d.f. $= 17$, $P < 0.05$). In contrast, for short-duration flashes, a major loss in discrimination occurred both in the blue direction ($t = 2.91$, d.f. $= 17$, $P < 0.01$) and in the yellow direction ($t = 3.28$, d.f. $= 17$, $P < 0.01$). This loss was reflected in a difference between the groups in the values of short-minus-long flash threshold in the blue direction ($t = 3.28$, d.f. $= 17$, $P < 0.01$) and in the yellow direction ($t = 2.23$, d.f. $= 17$, $P < 0.05$). Comparison of the overall performance of control subjects and the diabetic subjects over the range of colours and stimulus durations with the short-flash tritan index showed a highly significant difference ($t = 9.10$, d.f. $= 17$, $P < 0.001$) for the diabetic subjects.

This discrimination loss in diabetic patients without retinopathy was not revealed by the Farnsworth–Munsell 100-Hue Test though this test has been used to detect changes in colour vision in large diabetic groups including patients without retinopathy (500 patients tested in the same study by Kinnear *et al.*, 1972 and by Lakowski *et al.*, 1972/73) where tritan-like losses have been reported. The results from the short-flash threshold measurements show that even when the 100-Hue error score is normal and there is no evidence of retinopathy, there may be significant subclinical colour-vision deficiencies signifying some abnormal changes in retinal function.

Table 3. Differences between diabetic patients with and without retinopathy

Duration	Colour	Log threshold	SD	P
Long	Blue	0.274	0.450	0.092
	Green	−0.052	0.381	0.696
	Yellow	0.187	0.471	0.257
	Red	−0.079	0.322	0.487
Short	Blue	0.130	0.409	0.363
	Green	0.157	0.353	0.218
	Yellow	0.299	0.405	0.047
	Red	0.070	0.359	0.580

Differences between control subjects and diabetic patients with retinopathy

As expected, Snellen acuity was worse in the diabetic group with retinopathy than in the normal control group ($U = 12$, d.f. $= 1$, $P < 0.05$). There was no significant difference between this diabetic group and the control group as assessed with the Farnsworth–Munsell 100-Hue Test ($t = 0.18$, d.f. $= 16$, $P > 0.5$) or City University Test ($U = 29$, d.f. $= 1$, $P > 0.1$). For long-duration flashes, there was no significant difference between the two groups in any colour direction (see Fig. 2). For short-duration flashes, however, a major loss in discrimination occurred both in the blue direction ($t = 3.18$, d.f. $= 16$, $P < 0.01$) and in the yellow direction ($t = 5.17$, d.f. $= 16$, $P < 0.001$) and there was a mild loss in the green direction ($t = 2.88$, d.f. $= 16$, $P = 0.05$). These diabetic patients were also significantly worse in the short-minus-long flash discrimination threshold in the yellow direction ($t = 2.55$, d.f. $= 16$, $P < 0.05$).

In contrast to the result for diabetic patients without retinopathy, there was no significant difference between the short-flash tritan index for the diabetic patients with retinopathy and the normal controls ($t = 1.68$, d.f. $= 16$, $P > 0.1$), a consequence of the fact that, for this diabetic group, losses in sensitivity along the tritanopic axis were as severe for the long-duration flash as for the short-duration flash.

Differences between diabetic patients with retinopathy and diabetic patients without retinopathy

Table 3 shows the loss in sensitivity (in log units) of the diabetic patients with retinopathy relative to the diabetic patients without retinopathy, for the different stimulus conditions. The SDs of the thresholds are shown with corresponding P values (parametric t-test, two-tailed). Apart from their differences in relation to the normal control group, the two diabetic groups were functionally closely similar: there were no significant differences between the two groups according to the Farnsworth–Munsell 100-Hue Test, the City University Test, and the visual-acuity measures. There were also no significant differences between the two diabetic groups in blood-glucose levels ($t = 0.55$, d.f. $= 11$, $P > 0.5$) or in the longer-term measure of blood-glucose control, the HbAl percentage ($t = 1.55$, d.f. $= 14$, $P > 0.1$). It should be noted that the proportion of diabetics in the two groups on insulin therapy was different. The extent of the hue-discrimination loss (the short-flash tritan index) in the diabetic patients grouped together was not significantly correlated with blood-glucose level (Pearson correlation coefficient $r = -0.15$, $n = 13$, $P > 0.2$) or HbAl percentage ($r = -0.02$, $n = 16$, $P > 0.2$).

CONCLUSIONS

In subjects with normal colour vision, a reduction in stimulus-flash duration to 2 ms produced a greater increase in hue-discrimination threshold along the red–green axis than along the tritanopic axis. Optic neuritis patients showed a similar pattern of results to the control subjects but were uniformly worse. In diabetic patients, a reverse pattern of loss occurred in which the blue and yellow colour directions were most affected. This loss was not revealed by the total error score of the Farnsworth–Munsell 100-Hue Test. Because it depended on flash duration, the hue-discrimination loss could not be attributed to passive optical effects, and presumably arose from subclinical changes in function at receptor or post-receptoral level.

The results reported here for optic neuritis patients broadly agree with a series of studies by Mullen and Plant (1985, 1986, 1987). Mullen and Plant (1985, 1986), also using a colour monitor display, measured threshold contrast sensitivity to sinusoidal chromatic red–green and blue–yellow gratings all with a spatial frequency of 1 c/deg. They tested 10 patients with unilateral optic neuritis comparing the vision in the good eye with that in the bad eye and found an equal loss in sensitivity using red–green and blue–yellow gratings. Recently, Mullen and Plant (1987) quantified the changes in the appearance of colours in optic neuritis by measuring the perceived changes in hue, chroma (saturation) and value (lightness) in the affected eye compared to the unaffected eye. An interocular matching technique was used to measure

colour appearance of Munsell chips. Hue discrimination was assessed by the Farnsworth–Munsell 100-Hue Test. There were seven patients tested though complete results were obtained from only four. No control subjects were tested. Mullen and Plant found that the predominant deficit in the ON patients was a loss in saturation which could occur for any hue. The saturation changes occurred such that the hues tended to shift in appearance towards one of the four unique hues.

These previous studies and the present investigation are clearly inconsistent with the aspect of Köllner's law concerning optic-nerve disease. Demyelination appears to act nonselectively with regard to chromatic function. The data from patients with diabetes were more consistent with Köllner's law concerning diseases affecting the retina. Diabetic patients, even with no evidence of retinopathy, had significant blue colour-discrimination losses as assessed by the short-flash hue-discrimination technique.

The extent of colour-discrimination loss found in the diabetic patients was not significantly correlated with disease duration or blood-glucose level. Aspinall *et al.* (1983), in a much larger sample (295 patients), found that disease duration and blood-glucose control were good predictors of the onset of retinopathy and hence, presumably, visual loss. Losses in colour discrimination in diabetic patients with and without retinopathy have been reported by other workers using the Farnsworth–Munsell 100-Hue Test (compare Kinnear *et al.*, 1972; Lakowski *et al.*, 1972/73; Green *et al.*, 1985) though the diabetic groups there were much larger than those investigated here. The fact that colour-vision losses dependent on flash duration were found in patients without retinopathy and who had normal Farnsworth–Munsell 100-Hue Test scores suggests that significant abnormalities in visual function may occur relatively early in the course of the disease. It is possible that measures of hue-discrimination performance with short-duration flashes may predict subsequent, more profound, losses in colour-vision function (Gündüz *et al.*, 1988).

Acknowledgements—We thank G. B. Arden, S. J. Dain, K. J. Hardy and P. R. Kinnear for review of an earlier version of this paper.

REFERENCES

Adams A. J., Zisman F. and Cavender J. C. (1980) Sensitivity loss in chromatic processing channels of diabetics. *Invest. Ophthal. visual. Sci.* **19** (Suppl.), 169.

Aspinall P. A., Kinnear P. R., Duncan L. J. P. and Clarke B. F. (1983) Prediction of diabetic retinopathy from clinical variables and color vision data. *Diabetes Care* **6,** 144–148.

Barca L. and Vaccari G. (1977) On the impairment of color discrimination in diabetic retinopathy. A report of 24 cases. *Atti Fond. G. Ronchi* **32,** 635–640.

Birch J. and Dain S. J. (1987) An averaging method for the interpretation of the Farnsworth–Munsell 100-Hue Test—II. Colour vision defects acquired in diabetic retinopathy. *Ophthal. physiol. Opt.* **7,** 281–291.

Bresnick G. H., Condit R. S., Palta M., Korth K., Groo A. and Syrjala S. (1985) Association of hue discrimination loss and diabetic retinopathy. *Archs Ophthal.* **103,** 1317–1324.

Brown L., Govan E. and Block M. T. (1983) The effect of reduced visual acuity upon Farnsworth 100-Hue Test performance. *Ophthal. physiol. Opt.* **3,** 7–11.

Burde R. M. and Gallin P. F. (1975) Visual parameters associated with recovered retrobulbar neuritis. *Am. J. Ophthal.* **79,** 1034–1037.

Chisholm I. A. (1979) Acute optic neuritis. In *Congenital and Acquired Color Vision Defects*, edited by Pokorny J., Smith V. C., Verriest G. and Pinckers, A. J. L. G., pp. 308–309. Grune & Stratton, New York.

Cox J. (1960) Colour vision defects acquired in diseases of the eye. *Br. J. physiol. Opt.* **17,** 195–216.

Cox J. (1961a) Colour vision defects acquired in diseases of the eye. *Br. J. physiol. Opt.* **18,** 3–32.

Cox J. (1961b) Colour vision defects acquired in diseases of the eye. *Br. J. physiol. Opt.* **18,** 67–84.

Fallowfield L. and Krauskopf J. (1984) Selective loss of chromatic sensitivity in demyelinating disease. *Invest. Ophthal. visual. Sci.* **25,** 771–773.

Farnsworth D. (1943) The Farnsworth–Munsell 100-Hue and dichotomous tests for color vision. *J. opt. Soc. Am.* **33,** 568–578.

Farnsworth D. (1957) *The Farnsworth–Munsell 100-Hue Test for the examination of color discrimination: manual.* Munsell Color Company Inc., Baltimore.

Fletcher R. J. (1975) *The City University Colour Vision Test.* Keeler Instruments Ltd, London.

Fletcher R. J. (1980) *The City University Colour Vision Test.* (Second edn). Keeler Instruments Ltd, London.

Foster D. H. and Bischof W. F. (1987) Bootstrap variance estimators for the parameters of small-sample sensory-performance functions. *Biol. Cybernet.* **57,** 341–347.

Foster D. H., Snelgar R. S. and Heron J. R. (1983) Abnormal chromatic and luminance sensitivity in multiple sclerosis. *EEG clin. Neurophysiol.* **56,** 16P.

Foster D. H., Snelgar R. S. and Heron J. R. (1985) Nonselective losses in foveal chromatic and luminance sensitivity in multiple sclerosis. *Invest. Ophthal. visual Sci.* **26,** 1431–1441.

Foster D. H., Scase M. O. and Taylor S. P. (1986) Anomalous loss in blue–green hue discrimination in very brief monochromatic stimuli presented to the normal human eye. *J. Physiol., Lond.* **381,** 64P.

Glaser J. S. (1976) Clinical evaluation of optic nerve function. *Trans. Ophthal. Soc., U.K.* **96,** 359–362.

Green F. D., Ghafour I. M., Allan D., Barrie T., McClure E. and Foulds W. S. (1985) Colour vision of diabetics. *Br. J. Ophthal.* **69,** 533–536.

Griffin J. F. and Wray S. H. (1978) Acquired color vision defects in retrobulbar neuritis. *Am. J. Ophthal.* **86,** 193–201.

Gündüz K., Arden G. B., Perry S., Weinstein G. W. and Hitchings R. A. (1988) Color vision defects in ocular hypertension and glaucoma. Quantification with a computer-driven color television system. *Archs Ophthal.* **106,** 929–935.

Hall J. L. (1981) Hybrid adaptive procedure for estimation of psychometric functions. *J. Acoust. Soc. Am.* **69,** 1763–1769.

Kinnear P. R., Aspinall P. A. and Lakowski R. (1972) The diabetic eye and colour vision. *Trans. Ophthal. Soc., U.K.* **92,** 69–78.

Köllner H. (1912) *Die Störungen des Farbensinnes, ihre klinische Bedeutung und ihre Diagnose.* Karger, Berlin.

Krauskopf J., Williams D. R. and Heeley D. W. (1982) Cardinal directions of color space. *Vision Res.* **22,** 1123–1131.

Lakowski R., Aspinall P. A. and Kinnear P. R. (1972/73) Association between colour vision losses and diabetes mellitus. *Ophthal. Res.* **4,** 145–159.

Lanthony P. (1978) The new color test. *Documenta ophth.* **46,** 191–199.

Lynn B. H. (1959) Retrobulbar neuritis. A survey of the present condition of cases occurring over the last fifty-six years. *Trans. Ophthal. Soc., U.K.* **79,** 701–716.

MacAdam D. L. (1942) Visual sensitivities to color differences in daylight. *J. opt. Soc. Am.* **32,** 247–274.

MacAdam D. L. (1943) Specification of small chromaticity differences. *J. opt. Soc. Am.* **33,** 18–26.

MacAdam D. L. (1959) Small-field chromaticity discrimination. *J. opt. Soc. Am.* **49,** 1143–1146.

Mullen K. T. and Plant G. T. (1985) Colour and luminance sensitivity in human optic neuritis. *J. Physiol., Lond.* **360,** 23P.

Mullen K. T. and Plant G. T. (1986) Colour and luminance vision in human optic neuritis. *Brain* **109,** 1–13.

Mullen K. T. and Plant G. T. (1987) Anomalies in the appearance of colours and of hue discrimination in optic neuritis. *Clin. Vision Sci.* **1,** 303–316.

Rosen J. A. (1965) Pseudoisochromatic visual testing in the diagnosis of disseminated sclerosis. *Trans. Am. Neurol. Assoc.* **90,** 283–284.

Scase M. O. and Foster D. H. (1988) Anomalous loss in blue–green wavelength discrimination with very brief monochromatic stimuli presented to the normal human eye. *Ophthal. physiol. Opt.* **8,** 193–197.

Scase M. O., Honan W. P., Gulliford M. C., Scarpello J. H. B., Heron J. R. and Foster D. H. (1987) Colour vision loss in diabetic subjects. *Diabet. Med.* **4,** 581A.

Taylor M. M. and Creelman C. D. (1967) PEST: efficient estimates on probability functions. *J. acoust. Soc. Am.* **41,** 782–787.

Taylor S. P. (1983) A time induced tritan effect. *Vision Res.* **23,** 745–748.

Taylor S. P. (1984) Acquired colour defects under restricted viewing time: a new diagnostic technique? *Br. J. Ophthal.* **68,** 139–143.

Verriest G. (1963) Further studies on acquired deficiency of colour discrimination. *J. opt. Soc. Am.* **53,** 185–195.

Wright W. D. (1941) The sensitivity of the eye to small colour differences. *Proc. phys. Soc.* **53,** 93–112.

Wright W. D. (1943) The graphical representation of small color differences. *J. opt. Soc. Am.* **33,** 632–636.

Wyszecki G. and Stiles W. S. (1982) *Color Science: Concepts and Methods, Quantitative Data and Formulae* (Second edn), pp. 571–574. Wiley, New York.

Zisman F. and Adams A. J. (1982) Spectral sensitivity of cone mechanisms in juvenile diabetics. In *Colour Vision Deficiencies VI*, edited by Verriest G., pp. 127–131. Junk, The Hague.

Zisman F., Adams A. J. and Cavender J. C. (1981) Chromatic, luminosity, and contrast sensitivy changes in diabetes. *Invest. Ophthal. visual. Sci.* **20** (Suppl.), 92.