Colour identification and colour constancy are impaired in a patient with incomplete achromatopsia associated with prestriate cortical lesions

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SUMMARY

We have examined visual functions, including colour vision, in a patient with bilateral cortical lesions involving mainly the fusiform and lingual gyri, areas known to be involved in the central processing of chromatic stimuli. The patient has near normal (6/9) acuity, and his responses to tests of binocular function and spatial vision are normal, as are his discrimination of changes in target speed and surface lightness. He does, however, exhibit minor losses in the upper visual field, mild prosopagnosia and topographical agnosia, all conditions commonly associated with cerebral achromatopsia. Colour matches and spectral response data establish that his cone photoreceptors have normal spectral characteristics and his spectral sensitivity measured against a white background reveals normal postreceptoral chromatic function. The patient's colour discrimination for differences in wavelength, hue or saturation is, however, impaired and his colour naming is significantly disturbed, particularly for blues and greens. We have determined the areas of the chromaticity chart that correspond to his naming categories for surface colours, and show that changes in illuminant cause him to alter the names of surface colours in a manner consistent with the changes in their chromaticities. Other subjects with normal or congenital red–green deficient colour vision make many fewer name changes under changes in illuminant. We conclude that the patient's colour constancy is impaired as a consequence of abnormal central processing of colour vision.

1. INTRODUCTION

Colour vision, the capacity to discriminate between lights that differ in spectral composition, is derived from the responses of different spectral classes of cone photoreceptors in humans. Three spectral classes of cones convert the physical spectral flux distribution into three signals, the magnitudes of which define the physiological stimulus. The cone signals generate wavelength selective responses in postreceptoral and higher visual neurons, and these responses have been studied extensively in non-human primates. There is a well defined region of the macaque prestriate cortex, known as area V4, in which many neurons respond to stimuli of a specific colour, regardless of spectral composition (Zeki 1980). Positron-emission tomography (PET) scanning reveals a colour sensitive cortical area in humans, located in the fusiform and lingual (occipitotemporal) gyri (Lueck *et al.* 1989), and other colour areas are described by Corbetta *et al.* (1991) and Gulyas *et al.* (1994).

Acquired colour vision deficiencies are found in association with various conditions, including neurological diseases that affect the cortical visual areas, particularly the ventromedial occipital cortex (Meadows 1974; Zeki 1990; Plant 1991). Patients with such lesions exhibit a condition known as cerebral achromatopsia, in which colours are variously described as 'shades of grey', 'dirty' or 'pale and washed out'. The loss of colour perception is rarely total, and different patients exhibit different naming errors, although blues and greens are most frequently involved. Correspondingly, some authors have reported that the blue sensitive cone mechanism is selectively impaired in achromatopsia (Pearlman *et al.* 1979; Young & Fishman 1980). Where tested, patients usually perform abnormally on pseudoisochromatic plates (see, for example: Heywood *et al.* 1987; Rizzo *et al.* 1992, 1993) although some respond normally (see, for example, Meadows 1974). Those patients examined with the Farnsworth–Munsell (FM) 100-hue test all give abnormally high error scores, although confusions are sometimes restricted to blue hues (see, for example, Rondot *et al.* 1967).

Achromatopsia is frequently found in association with other neurological conditions, especially prosopagnosia and topographical agnosia, but dissociations between all three conditions are also reported. The extent to which achromatopsia can be isolated from other visual abnormalities has been examined quan-

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titatively in only a few cases. Preservation of spatial vision is indicated by the fact that some patients have normal or near-normal visual acuity, and retention of various degrees of lightness discrimination has been reported by Heywood *et al.* (1987) and Rizzo *et al.* (1992, 1993).

Localization of the underlying lesions in cerebral achromatopsia has been achieved by autopsy and by magnetic resonance imaging (MRI) brain scanning, and in patients with unilateral cortical damage colour vision is disturbed in the contralateral hemifield (see, for example: Verrey 1888; Kölmel 1988). Neurons in area V4 of the monkey prestriate cortex respond selectively to colour appearance, that is, they demonstrate colour constancy under change of illumination. Colour constancy is an attribute of human visual function, and it might be expected that lesions of a cortical area corresponding to V4 would result in failure of this aspect of visual performance. Rizzo *et al.* (1993) comment, however, that the consequences of cerebral achromatopsia are greater than would be expected simply from failure of colour constancy.

In this paper, we present the results of measurements made on a patient who suffers disturbances of colour vision associated with lesions of the prestriate visual areas. We detail a number of his response functions and illustrate the impairment of his colour discriminations. We examine particularly his identification of colours under changes in illumination, for which normal observers demonstrate colour constancy.

2. CASE HISTORY

B.L., a 54 year old male, was admitted to hospital three years ago, after suffering an encephalitic illness believed to be of viral origin. On recovering consciousness, he complained that the world appeared to be in semidarkness, without colour, 'like an old black and white movie'. He experienced difficulty in finding his way around the hospital ward and in recognizing faces. He complained of visual distortion which caused objects to appear narrower or smaller than he knew them to be. Some 20 months after the initial illness, he was examined systematically with standard psychological tests, at which time all his visual problems had partially resolved. He still found it difficult to judge finer aspects of facial appearance, such as the age of the person and was unable to find his way around in environments with which he was not very familiar. His colour vision had recovered partially, but his colour naming was very erratic. He had average reading and verbal skills, but showed some losses of short term memory, and was impaired on tests that required a high degree of concentration or that involved visuospatial conceptualization. He performed a line bisection task normally, and so did not exhibit visual neglect.

An MRI scan of B.L.'s brain reveals bilateral infarctions involving the infracalcarine occipital lobes (figure 1*a*, *b*), with sparing of the calcarine fissure. These bilateral infarcts predominantly involve the fusiform gyri but extend into the lingual gyri, and into the posterio part of the parahippocampal gyri. In addition, there are multiple small juxtacortical lesions in both cerebral hemispheres, considered to be white matter infarcts due to small vessel disease. Extensive ophthalmological examination revealed no retinal abnormality.

B.L. has great difficulty in naming colours perceived visually, although he readily identifies the colour of objects such as banana and grass that are named for him. He reports that common objects, such as grass, do not appear to be correctly coloured and, when a colour is identified for him, he frequently remarks that it 'does not appear as I remember it'. He does, none the less, distinguish between colours, and the almost complete loss of colour discrimination reported clinically immediately after his illness has, therefore, resolved into a milder disturbance, which has remained essentially stable over the period of our study.

3. EXPERIMENTAL OBSERVATIONS

(*a*) *Visual fields*

Visual fields measured with a Goldmann perimeter are normal, except for a scotoma in the upper left quadrants observed with the smallest ($\frac{1}{4}$ mm^2) target under static presentation.

(*b*) *Spatial vision*

His pinhole Snellen acuity is 6/9 in either eye and his ST1 spatial frequency response, which characterizes the early stages of spatial filtering (Barbur & Ruddock 1980), is normal, with a peak at around 4 cycle deg^{-1}. He describes correctly the changes in apparent orientation and in bar width of a set of parallel bars (a grating) induced, respectively, by adaptation to a misaligned grating (Gibson & Radner 1937) or to one of different bar width (Blakemore & Sutton 1969). He also responds appropriately to a number of visual illusions, including those named after Ehrenstein, Kanisza, Müller-Lyer, Necker, Pogendorff and Zöllner (Gregory 1970).

(*c*) *Binocular vision*

His stereoscopic fusion of random dot stereograms (Julesz 1971) is normal, and he experiences binocular rivalry when he views crossed red and green gratings through colour separation spectacles.

(*d*) *Velocity discrimination*

He was asked to identify the faster of two circular targets, presented sequentially, which moved horizontally, one at 45 deg s^{-1} and the other at a velocity chosen randomly from a series of values in the range 30 deg s^{-1} to 60 deg s^{-1}. He achieved 95% correct identification of the faster target when the velocity difference between the two was ± 10 deg s^{-1}, which is similar to that achieved by normal controls. His incremental threshold for detection of a circular target (3.5° diameter) superimposed on a uniform back-

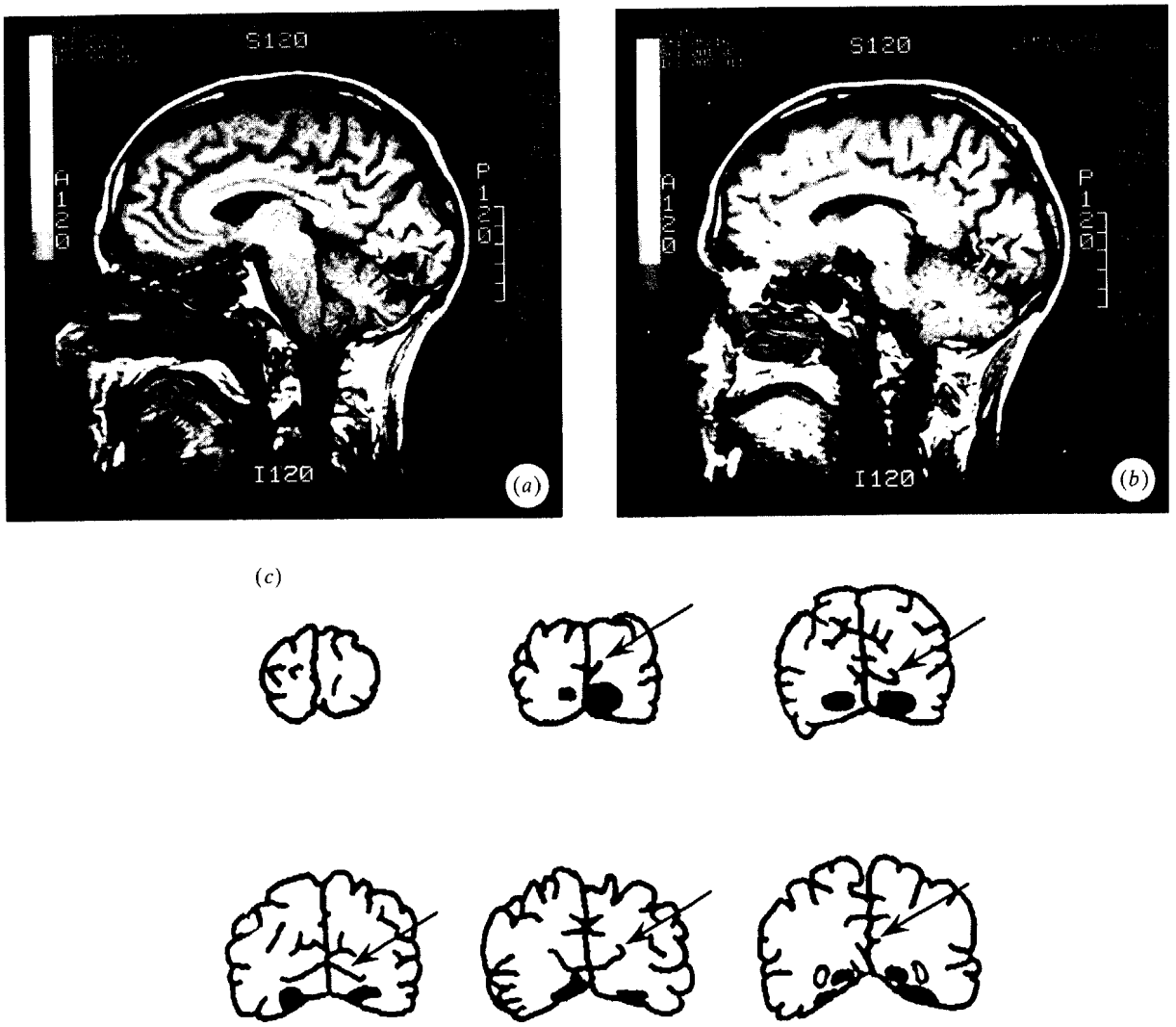

Figure 1. (*a*, *b*) Two T1 weighted parasagittal MRI scans showing cystic degeneration due to infarction in the infracalcarine occipital lobes ((*a*) left, (*b*) right), obtained with a GEC 1.5 T scanner (St Mary's Hospital, London). (*c*) Tracings from the coronal MRI scans, starting posteriorally and passing anteriorally in 9 mm steps. Arrows identify the calcarine fissure and the extent of the infarction is shown in black.

ground was less than 0.5 logarithmic units greater than that set by a normal control, for all photopic background luminance values.

(*e*) *Lightness discrimination*

He ordered correctly in sequence of apparent brightness 46 grey tiles, 5 cm square (Robertson & Wright 1965), making only occasional reversals between adjacent tiles, a performance similar to that achieved by normal controls.

(*f*) *Higher visual functions*

He continues to display mild prosopagnosia and topographical agnosia, but neither has been assessed formally.

(*g*) *Colour vision*

B.L.'s colour vision is the principal topic of this investigation, and a number of relevant functions have been measured.

(i) *Colour matching*. Both 1° 20′ and 10° colour matches were examined with a tristimulus colorimeter (Wright 1946). B.L. made a normal Rayleigh match for a yellow (590 nm) test against a red (650 nm) and green (530 nm) mixture, although his accuracy in matching was subnormal. He failed to make consistent trichromatic matches for a blue–green (494 nm, plus 650 nm desaturation) test.

(ii) *Spectral sensitivity functions*. These were measured with a three beam Maxwellian view system (Barbur & Ruddock 1980), with use of Balzer B40 interference filters to control the spectral composition of the stimuli. Two-colour increment threshold measurements (Stiles 1978) isolated the Π_3 (blue-sensitive) spectral response mechanism. B.L.'s spectral sensitivity function for detection of a target (diameter 4.5°) against a white background (colour temperature 3200 K, luminance 3.0 log troland, diameter 17 deg) was normal, with prominent minima at around 580 nm and 490 nm. Such responses are attributed to detection by post-receptoral, colour opponent response mechanisms (Sperling & Harwerth 1971; King-Smith & Carden 1976).

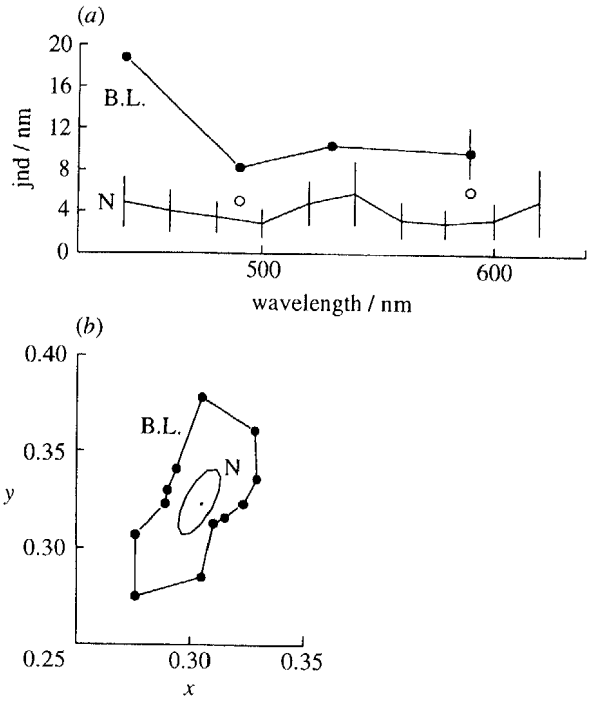

Figure 2. (*a*) Wavelength discrimination steps measurements for two contiguous rectangular fields (40′ × 80′) arranged as shown in the figure. The wavelength of the variable, lower hemifield was altered until it appeared just different in colour from the upper field. Data are given for the patient B.L. (full circles), with a typical error bar, and for eight naive normal observers, with the standard deviations in their data indicated by the error bars. Also shown are two values measured for B.L. with a bipartite 10° circular field (open circles). (*b*) Chromaticity discrimination around a reference white colour, the CIE 2 deg coordinates of which are denoted by the central spot. The full circles denote the chromaticities at which the grating target can just be distinguished from the white background by the patient B.L. The ellipse marked N indicates the area enclosed by the equivalent data for normal controls.

(iii) *Colour discrimination.* B.L. made between zero and three errors in reading the Ishihara pseudo-isochromatic plates and had error scores of between 400 and 600 on the Farnsworth–Munsell 100-hue test, with no well defined confusion axis. B.L.'s wavelength discrimination steps, measured at selected points in the spectrum with the Wright colorimeter, were three to five times those measured for a naive, normal trichromat (figure 2*a*). An increase in field size from 1° 20′ to 10° reduced the size of his discrimination steps, but they remained larger than those recorded for the normal control. B.L. could perform the wavelength discrimination task with sequential presentation of the two stimuli, although these discrimination steps were slightly larger than those for simultaneous, side-by-side presentation. His discrimination for detection of a colour difference between a white field and an equiluminant coloured grating (Barbur *et al.* 1992) followed the same pattern as that for a normal observer, but the steps were about three times larger (figure 2*b*).

(iv) *Colour naming.* This was examined with 57 reference colours, generated on a visual display unit screen and located at the centre of a Mondrian display (figure 3*a*). The Mondrian was constructed from

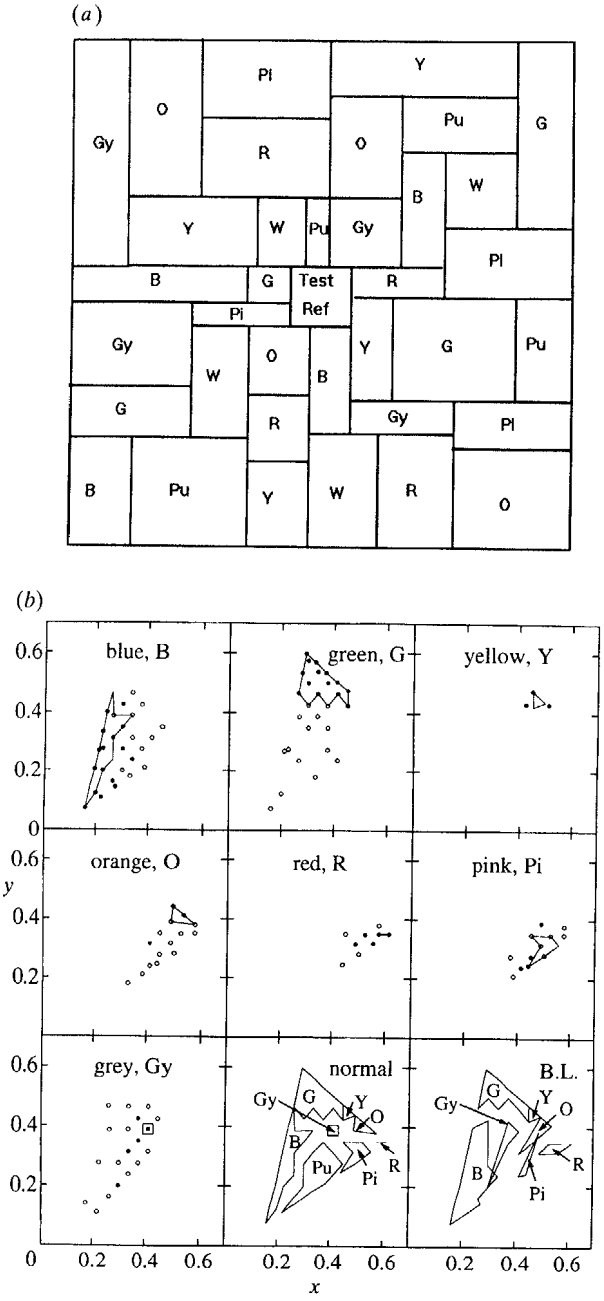

Figure 3. (*a*) The Mondrian display used to investigate B.L.'s colour naming. The test and reference samples were placed in the central square, of side 1.2°, and the whole display was 10° square. The Mondrian colours, and their CIE 2° chromaticity coordinates, measured for the white illuminant, were grey ($x = 0.412$; $y = 0.425$; $L = 12.0$ cd m^{-2}), orange (0.525, 0.428, 12.0), red (0.577, 0.360, 8.5), pink (0.505; 0.375, 12.5), yellow (0.464, 0.483, 32.5), green (0.336, 0.508, 9.0), blue (0.315, 0.377, 6.0), purple (0.381, 0.357, 7.0) and white (0.412, 0.426, 36.5). (*b*) The CIE chromaticity coordinates of reference samples to which B.L. gave the different colour names denoted at the top of the panels. Full circles denote identification on three or more of five presentations and open circles identification on one or two of five presentations. The full lines connect the values associated with the different colour names made by a normal subject, for whom all samples corresponded unambiguously to one or other of the colour names. In the 'grey' panel, the square surrounds the single chromaticity which appeared grey to the normal. The last two panels summarize the different areas for the normal subject and the patient B.L. Note that the latter did not use the description 'purple' (Pu on the normal panel).

diffusely scattering papers cut into square or rectangular elements and arranged into a $10° \times 10°$ square. The display was illuminated with a white light projector beam, modified as required by inclusion of a broad-band red, green or blue filter, and all colours were calibrated *in situ* with the PR 650 Spectra Colorimeter (Photo Research Inc.).

The average luminance of the Mondrian was 15 cd m^{-2}, and the 57 reference colours were generated at two luminances, three measurements being made at 5 cd m^{-2} and two at 12 cd m^{-2}, but as the two sets of data were indistinguishable they have been pooled. The five different sets of measurements were made on different days, at roughly two week intervals. Although B.L. was allowed to choose colour names freely, he selected seven of the names identified as those categories required by subjects with normal colour vision (Walsh *et al.* 1992), plus white or grey but without purple. He used brown and orange interchangeably; so we have represented both under 'orange'. The colours associated with each name are plotted in the CIE chromaticity diagram of figure 3*b*, together with data for a normal subject with trichromatic colour vision. For the normal, all samples were identified unambiguously by one or other of the colour names specified in the figure. We also asked the patient to identify the colour of isolated patches, the luminances of which were varied over a 100:1 range, and found that his naming of a given patch remained independent of luminance.

A second set of eight test colours, consisting of diffusely scattering papers, were presented at the centre of the Mondrian, which was illuminated by one of the four different illuminants, and B.L. was asked to name them. The full set of test colours were presented once, in random order, for a given illuminant, and successively for each of the different illuminants. The procedure was repeated four times, on four different dates at approximately fortnightly intervals. The chromaticities of the eight test colours under the four illuminants are plotted in figure 4*a*, and responses for two test colours are illustrated in figure 4*b*. It should be noted that the chromaticity shifts induced by the changes in illumination are very much greater in magnitude than B.L.'s discrimination steps (figures 2, 4*a*). The four chromaticities associated with each test colour, one for each of the different illuminants, corresponds to a colour name or, in intermediate cases, two or more names, specified in figure 3*b*. These predicted colour names were compared with those chosen by B.L., and correspondence between the two would imply that B.L. perceives colour in terms of the spectral composition of the samples, without adjustment for the illuminant. Of the 24 changes in chromaticity coordinates, corresponding to the change from white to red, to green or to blue illuminant for each of the eight test samples, 18 result in a shift in the colour category associated with the chromaticity coordinates (figure 4*b*). B.L. made 16 changes in colour names, eight of which corresponded to those predicted on the basis of the chromaticity coordinates and seven more were in the appropriate direction but corresponded to a chromaticity change of intermediate magnitude (see the caption to figure 4*b* for an example). Only one name change made by B.L. was not predicted from consideration of the change in chromaticity coordinates, and he failed to make three of the predicted changes. Two subjects with normal colour vision made no name changes in this test.

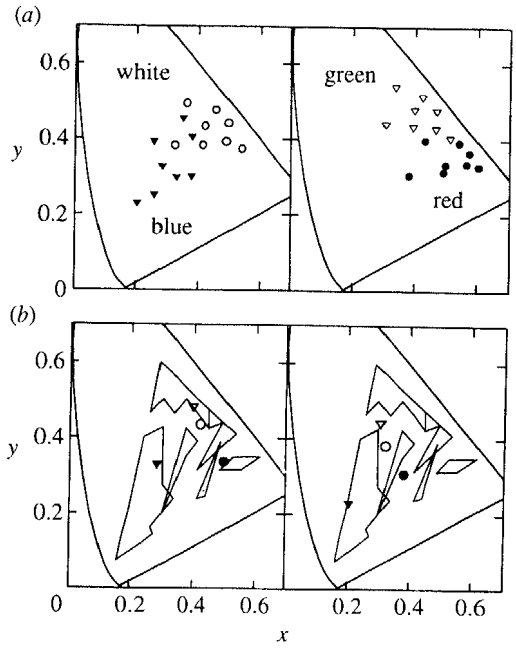

Figure 4. (*a*) The chromaticity coordinates of the eight test samples under each of the four illuminants: white (open circles), red (filled circles), green (open triangles) and blue (filled triangles). (*b*) The chromaticity coordinates of two samples, under the white (open circles), red (filled circles), green (open triangles) and blue (filled triangles) illuminants. Also shown are the colour categories, taken from the bottom right panel of figure 3*b*. For the left hand panel, B.L. named the samples white, yellow or green (white illuminant), pink (red illuminant), yellow (green illuminant) and blue (blue illuminant). The corresponding names for the right hand panel were green or blue (white illuminant), grey (red illuminant), green, grey or blue (green illuminant) and blue (blue illuminant).

DISCUSSION

B.L. suffers lesions of the prestriate visual cortical areas similar to those that, in other patients, are associated with disturbances of colour vision. Certain features of B.L.'s response functions, including the mild impairment restricted to the upper visual fields, his mild prosopagnosia and topographical agnosia, are frequently observed in cases of cerebral achromatopsia (Meadows 1974). Except for his reduced (6/9) Snellen visual acuity, B.L.'s spatial vision is essentially normal, as is his binocular function, including stereoscopy. Like the two achromatopsic patients of Rizzo *et al.* (1992), his responses to moving stimuli, in this case assessed by measurement of detection sensitivity and velocity discrimination, are also normal. Our observations therefore support the view that localized lesions of the fusiform and lingual gyri have little effect on visual responses to spatial pattern or to movement. His discrimination of lightness is normal; thus he has no overall impairment of fine visual discriminations. We did not examine constancy in B.L.'s identification of

spatial pattern but we note that in contrast to his problems with colour he has no apparent difficulty in identifying shapes and objects viewed under different conditions.

B.L.'s colour matching and two-colour increment threshold data establish that he has normal trichromatic colour vision and isolation of his Π_3 spectral response demonstrates that in contrast to the achromatopsic patients of Pearlman *et al.* (1979) and Young & Fishman (1980), but like that of Mollon *et al.* (1980), B.L.'s blue-sensitive cone mechanism is functional. His spectral sensitivity function measured against a white background is, like that of the patient of Heywood *et al.* (1991), normal and exhibits clearly the characteristics attributed to activity of postreceptoral colour opponent response mechanisms. B.L.'s impaired colour discriminations for saturation and wavelength (figure 2), and particularly the large errors he makes in arranging the samples of the Farnsworth–Munsell 100-hue test, are typical of responses described for patients with incomplete cerebral achromatopsia. Although chromatic sensitivity is usually attributed to the parvocellular projection pathway, the magnocellular pathway can also respond to equiluminance borders (Saito *et al.* 1989; Hubel & Livingstone 1990), and can contribute to colour discrimination in achromatopsia (Heywood *et al.* 1991, 1994). B.L., however, was able to perform wavelength discrimination when pairs of spectral stimuli were presented sequentially and our saturation discrimination test is designed so that border contrast disappears at threshold (Barbur *et al.* 1992).

The areas of the chromaticity chart occupied by B.L.'s colour naming categories correspond closely to those associated with normal colour naming, but are much less tightly defined (figure 3*b*), as would be expected from consideration of his difficulties with colour identification. B.L.'s naming of the 57 reference samples was unaffected by reducing the luminance of the reference stimuli by a factor of 2.4.

Our major new findings are that B.L.'s identification of surface colours changes with change of illumination, and that these changes can be predicted on the basis of the spectral composition of the light reflected by the coloured samples (figure 4). As well as modifying their chromaticity coordinates, the illumination changes also reduced the luminance (Y) factors of the samples, the luminance of the test varying by up to 4:1 for the different illuminants, albeit with similar, but smaller, changes in the average luminance of the surround Mondrian. As stated previously, we found that the names given to the reference samples remained unchanged for a 2.4 change in relative luminance, which was the maximum range over which we could generate a constant luminance set of reference colours. With isolated patches, however, his naming remained invariant over 100:1 change in luminance and these observations show that luminance changes do not influence his colour naming. Constancy in colour naming is reported to be robust in those with normal colour vision (Troost & de Weert 1991); so its impairment in our patient is particularly noteworthy. In experiments similar to those reported here, B.L. made 16 name changes in identifying the colours of the Mondrian display, whereas a deuteranomalous subject, whose overall wavelength discrimination was very similar to B.L.'s, made only three changes and a group of deuteranopic dichromats made, on average, only 5.5 changes (Morland *et al.* 1995). The central origins of B.L.'s discrimination losses are different from those that give rise to congenital abnormalities of red–green colour vision, and these findings imply that impairment of B.L.'s colour constancy is much more severe than that in subjects with retinal abnormalities who have comparable or more severe wavelength discrimination losses.

In summary, our experiments on a patient with bilateral lesions of the fusiform and lingual gyri and associated small juxtacortical lesions reveal selective disturbances of colour vision, manifest as impaired discriminations and failure of colour constancy. Many of the response features found in this patient are consistent with those observed in others with incomplete achromatopsia, but colour constancy has not previously been studied systematically. Ablation of area V4 in the macaque leads to mild losses of hue discrimination accompanied by more severe impairment of colour constancy (Walsh *et al.* 1993) and by disruption of pattern discrimination (Heywood & Cowey 1987; Heywood *et al.* 1992) although the basic colour categories appear to be unaffected in such monkeys (Walsh *et al.* 1992). Several aspects of our patient's responses are, therefore, similar to those produced by V4 ablations, but he has essentially normal pattern discrimination, and in this respect his responses diverge from those of monkeys that lack V4 (Heywood *et al.* 1992).

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