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Retinotopic organisation of cortical mechanisms responsive to colour: Evidence from patient studies

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Abstract

This paper deals with the visual responses of three patients who have impaired colour vision consequent on cortical dysfunction which, in two of them, is associated with demonstrable neuronal damage. The studies to be described are concerned particularly with the spatial attributes of their chromatic response mechanisms. Data are presented which establish that a hemianope GY has coarse chromatic discrimination for large stimuli located within his 'blind' hemifield. GY responds to stimuli containing differently coloured equiluminant components as if the coloured components were averaged over the whole field and it is speculated that such spatial averaging may correspond to the process which, in normal vision, provides compensation for change of illuminant in order to achieve colour constancy. Colour constancy is impaired in a second patient, BL, who has cortical lesions involving the lingual and fusiform gyri, areas which are partially spared in GY. It is shown that movement, but not colour, presented to GY's normal hemifield generates a response localised in his blind hemifield and disinhibitory interaction between movement and colour is illustrated for a patient MW, in whom colour chromatic stimuli generate spreading inhibition of visual responses. This inhibitory interaction is propagated between widely separated stimuli, including those which are located on opposite sides of the vertical meridian. We discuss these experimental results in relation to anatomical and physiological mechanisms of the primate visual cortex. © 1997 Elsevier Science B.V.

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1. Introduction

The eye forms an optical image of the external visual field on the retina, the magnification of which is essentially uniform over whole field. The retinal output is carried by the optic nerve fibres to a number of target sites, the principal of which are the dorsal lateral geniculate nucleus (dLGN) and the superficial layers of the superior colliculus. Following partial decussation at the optic chiasm, nerve fibres in one half of each retina project to the ipsilateral lateral geniculate nucleus, those from the ipsilateral eye innervating layers numbered 2, 3 and 5, and those from the contralateral eye layers 1, 4 and 6. In macaque, the parvocellular layers 1–4 and magnocellular layers 5 and 6 are innervated by two separate fibre groups, the former by axons of P-type ganglion cells and the latter by axons of M-type ganglion cells, the two classes having distinct functional properties (de Monasterio and Gouras, 1975; Leventhal et al., 1981; Perry et al., 1984; Wiesel and Hubel, 1966). The visual field is mapped onto each geniculate layer, the six maps being in register, with the foveal region enlarged relative to the periphery in the non-uniform geniculate representation (Clark and Penman, 1934). Innervation of the striate cortex (visual area V1) from dLGN via the geniculo–calcarine projection is divided, M-type fibres terminating in sub-lamina 4Cα and P-type fibres in striate sub-laminae 4A and 4Cβ. The inputs from the two eyes remain separate in lamina 4, but converge in the other V1 laminae (Wiesel and Hubel, 1974; Levay et al., 1975). Bilateral representation in macaque V1 of a strip of visual field extending some 2° on either side of the vertical meridian has been reported by Stone et al. (1973) and by Bunt and Minckler (1977). The striate cortex projects onwards to the pre-striate cortical areas, the retinotopic representation of which is restricted to the contralateral hemifield (van Essen and Zeki, 1978), although the large receptive fields of single neurones in areas V4 and V5 extend several degrees across the vertical meridian (Zeki, 1978). The half-field representations of the two hemispheres are linked through the calossal fibres (Myers, 1962; Hubel and Wiesel, 1968; van Essen and Zeki, 1978). The callosal innervation at the V1/V2 boundary corresponds to the well-defined representation of the vertical meridian, whereas in pre-striate areas V4 and V5 it is diffuse, corresponding to the complex retinotopic mapping into these areas (Zeki, 1993).

Responses associated with the P-type retino–geniculate projection pathway are colour opponent, with high spatial resolution and sustained (low pass) temporal activity, whereas those of the M-type pathway are non-colour opponent, with lower spatial resolution and transient (band-pass) temporal responses. Evidence of this functional division has been found in the selective responses of single cells in the striate and pre-striate visual areas (Zeki, 1974, 1978; Livingstone and Hubel, 1988; Zeki and Shipp, 1988). The M-pathway input to V1 projects onwards to both pre-striate areas V3 and V5, either directly or via the histologically identified thick stripes of pre-striate area V2. The P-pathway projects via the histologically identified blobs and interblobs of area V1 to area V4, either directly or via the thin stripe and interstripe regions of area V2. The V1 blobs, however, receive input from lamina 4Cα and 4B, both of which are in the M-projection pathway (Lachica et al., 1992), and selec-

tive inactivation of either the magnocellular or parvocellular geniculate layers indicates that although projections from the latter contribute little to V5 responses, those from the former provide significant input to V4, via both blobs and interblobs (Malpeli et al., 1981; Merigan and Maunsell, 1990; Ferrera et al., 1994; Nealey et al., 1994). The multiple reciprocal connections between the different visual cortical areas also provide the basis for interaction between different projection pathways (Zeki and Shipp, 1988).

The retinal projection to the superficial layers of the superior colliculus is formed by axons of M-type and the so-called W-type ganglion cells (Perry and Cowey, 1984; Leventhal et al., 1981). This input is retinotopically organised such that on each superior colliculus, the contralateral hemifield is represented with a magnification factor which varies with eccentricity in a way similar to that of the striate cortical representation (Cynader and Berman, 1972; Goldberg and Wurtz, 1972; Schiller and Koerner, 1971). There are commissural fibres connecting the two superior colliculi. The neurones of these layers are sensitive to transient, particularly fast moving light stimuli presented at high contrast, but although they receive both rod and cone signals (Kadoya et al., 1971; Marrocco and Li, 1977), they do not exhibit colour-opponent responses. The superior colliculus projects to dLGN, making synaptic contact with those geniculate interneurones which project directly to the pre-striate cortex (Kisvárday et al., 1991) and to the pulvinar, which in turn projects onwards to the cortical visual areas. Colour sensitive neuronal responses have been recorded from the pulvinar (Felsten et al., 1983; Benevento and Port, 1995).

Functional specialisation in the human visual cortex has been demonstrated by positron emission tomography which has revealed areas activated selectively by movement or colour (Lueck et al., 1989; Zeki et al., 1991; Corbetta et al., 1991; Gulyas et al., 1994) and a colour sensitive region has also been demonstrated by functional magnetic resonance imaging (Sakai et al., 1995). Patients with lesions localised in the fusiform and lingual gyri suffer various deficits of colour vision, including achromatopsia (Meadows, 1974; Zeki, 1990), and in cases of unilateral damage, such losses are restricted to the contralateral hemifield (Verrey, 1888; Albert et al., 1975; Damasio et al., 1980, Kölmel, 1988). Involvement of the corpus callosum in the diffuse spatial interactions involved in colour constancy (Land, 1959; Zeki, 1980) has been demonstrated by experiments on a human patient in whom the callosal projections had been completely resectioned (Land et al., 1983).

In previous studies, we have assessed colour vision in three patients with visual dysfunction of central origin. Two of the patients have well characterized cortical lesions, which in the case of GY give rise to hemianopia (Barbur et al., 1980), and for the patient BL, result in partial cerebral achromatopsia (Kennard et al., 1995). The other patient, MW, has an abnormal response to colour, which is expressed as a steely grey percept which spreads with time beyond the spatial perimeter of the coloured stimulus (Hendricks et al., 1981). In this paper, we present new data for patients GY and MW and examine them in relation to the topographical organisation of cortical mechanisms involved in colour vision.

2. Methods

2.1. Observers

Results are given for two patients, GY and MW, and we outline briefly the relevant background for each of these cases.

Patient GY is a 38 year old male, who at 8 years of age was involved in a traffic accident which caused extensive traumatic damage to his left occipital lobe. Magnetic resonance imaging (MRI) brain scans (Brent et al., 1994) reveal that the extensive lesion involves the left striate and peristriate cortex below the calcarine fissure to within about 1 cm of the occipital pole, and similar areas above the calcarine fissure, not extending to the occipital pole and sparing and fusiform gyrus. The medial aspects of the cuneous to the sulcus parieto–occipital anteriorly, a small strip of the posterior aspect of the pre-cuneous in the parietal lobe and the caudal extremity of the parahippocampal gyrus are also involved. Much of the left geniculo–calcarine tract is damaged. A much more restricted lesion of the right hemisphere involves mainly the inferior parietal lobule, part of the supra-marginal gyrus and juxta–cortical white matter of the adjacent part of the superior parietal lobule, but our studies have not revealed abnormal visual responses to visual stimuli located in the right hemifield. In addition to the MRI study, positron emission tomography (PET) has established activity in visual areas V3 and V5, but not V1, in response to visual stimulation of the right hemifield by movement (Barbur et al., 1993).

Perimetry with stationary stimuli reveals a right homonymous hemianopia sparing the central 3.5°. GY can, however, detect and localise flashed or moving transient lights presented within the 'blind' hemifield, and he reports that such stimuli cause a percept localised within the blind field which he describes as having the appearance of a dark shadow. His residual vision enables him to discriminate between sequentially presented stimuli which differ in flicker frequency or velocity, but his discrimination of spatial parameters such as orientation is severely impaired (Barbur et al., 1980; Blythe et al., 1987; Morland et al., 1996a). We have recently demonstrated that a moving grating presented to his normal left hemifield elicits, in addition to the normal response, activity localised in the blind hemifield (Finlay et al., 1996). Spectral sensitivity functions corresponding to those of the rod and of the Π_4- and Π_5-spectral response mechanisms (Stiles, 1978) have been identified in GY's blind field responses (Barbur et al., 1980; Brent et al., 1994) and the associated colour discrimination responses are described in this paper.

Patient MW is a 46 year old male who, throughout his life has suffered abnormal visual responses to chromatic stimuli. He has been studied at Imperial College since 1970, during which period his visual functions have remained essentially invariant, and anecdotal evidence indicates that the principal symptoms of his abnormal vision have been evident since childhood. MRI brain scans reveal no lesions, although the sulci of the parietal lobe are abnormally wide. Snellen acuity measured with achromatic stimuli is 6/4 in either eye (+1.5 dioptres correction) but with chromatic stimuli it ranges from about 6/12 in yellow light to less than 1/60 in red light (Ruddock and Waterfield, 1978). The underlying reason for these low acuities is an inhibitory re-

sponse elicited by chromatic stimuli, which spreads around the area of the visual field occupied by the chromatic image and is especially severe for reds. MW describes the area within which the inhibition occurs as 'steely-grey' in appearance, without well-defined borders, and within the area occupied by this percept he is unable to detect any other visual stimulus. This phenomenon, first described by Hendricks et al. (1981) is time dependent, as the area occupied by the steely-grey percept and its associated inhibition increases monotonically with the duration of MW's viewing a red stimulus. This inhibitory phenomenon is generated by all saturated chromatic stimuli, except yellow, and its dependence on the colorimetric properties of the stimulus were described by Hendricks et al. MW's highly abnormal threshold responses for spectral stimuli associated with this unique visual dysfunction were described by Bender and Ruddock (1974) prior to the discovery of the spreading inhibition. We have recently demonstrated that moving, but not flickering stimuli elicit an inhibitory effect similar to that found for colour (Morland et al., 1996b) and in this paper, we are concerned with the interactions between movement and colour in the generation of inhibitory visual activity.

Although MW's MRI scans reveal no cortical lesions, his responses to chromatic stimuli are consistent with a visual dysfunction of cortical origin. Thus a red filter placed over one eye suppresses entirely his visual responses through the other, which implies involvement of binocularly controlled response mechanisms. He can, nonethe-less, detect with normal sensitivity two-colour random dot stereograms (anaglyphs; Julesz, 1971), as was reported by Ruddock and Waterfield (1978). Thus those cortical mechanisms responsible for random dot stereopsis are unaffected by the inhibitory effects of chromatic stimuli. Further, evoked potentials recorded from MW's scalp in response to either achromatic or red and black chequer-board patterned stimuli are normal, even though he fails to detect the latter (Ruddock, 1988). Moving but not flickering stimuli elicit an inhibitory response similar to that associated with chromatic stimuli and in macaque, neuronal mechanisms sensitive to movement are a feature of cortical but not of pre-cortical activity (Hubel and Wiesel, 1968).

2.2. Aims and experimental procedures

The experiments for GY were designed to resolve the following questions.

1. Can he utilise residual vision to identify the colour of transient, spatially uniform stimuli presented to the blind hemifield?
2. What is the colour appearance of spatially structured, equiluminant colour mixtures presented transiently to GY's blind hemifield?
3. Is there a colour signal associated with the ipsilateral blind field percept elicited by a moving stimulus presented to GY's normal hemifield?

In these experiments, different chromatic stimuli were presented in a random sequence and after each presentation, GY identified the stimulus colour verbally. Coloured stimuli were generated with a Maxwellian view optical system (see below) and the subject maintained his eye position relative to the instrumental exit pupil by biting on a dental clamp. GY fixated a central target, and he is able to maintain

voluntary fixation to within ±1° over several seconds. He is able to identify immediately any stimuli displayed in the wrong hemifield, as the percepts associated with light falling on the normal hemifield and that falling on the blind hemifield are so markedly different.

The studies on MW examined the following questions.

1. How does the inhibitory area generated by a given stimulus vary with location in the visual field?
2. How do colour and movement interact in the generation of visual inhibition?

In these experiments, MW was presented with matt surface colours, together with achromatic targets against which he could identify the area over which his visual sensitivity was suppressed (see Figs. 4 and 5). He used a pointer to identify the limits of his resolution for the achromatic target and where required, he was provided with a fixation target. Achromatic, spatially sine-waveform moving gratings were generated on a VDU.

2.3. Equipment

The three-channel Maxwellian view optical system used to generate stimuli for the experiments on GY was described in detail by Barbur and Ruddock (1980) and Holliday and Ruddock (1983). Narrow band-width spectral stimuli were produced by placing Balzer B40 interference filters into the channels of the optical system at points where the light beams are parallel, and stimulus luminance was controlled with neutral density filters. The duration of stimulus presentation was controlled by electromagnetic shutters with rapid (<10 ms) opening and closing actions. The observer initiated stimulus display by pressing a push button. Photographic negatives (high contrast Kodalith film) of radial, square waveform gratings were rotated in the back focal plane of the instrumental eye-piece lens to produce 1-D grating images which moved approximately linearly across the visual field. All calibrations were performed in situ, with a PR 650 Spectra Colorimeter (Photo Research Inc.) for chromaticity co-ordinates and a Macam photometer/radiometer for luminance.

3. Results

3.1. Colour vision without striate cortical input

GY's identification of the colour of a uniform field presented transiently to his blind hemifield is illustrated by the data of Fig. 1(a), which show that for large fields, he can discriminate between and categorise spectral colours such as red, green and yellow and long wavelength stimuli of different saturations. He performs less well, however, with spectral stimuli which are not widely separated in wavelength and with short wavelength stimuli. He obtains good discrimination between appropriate spectral stimuli presented at different luminance levels (Fig. 1(b)), so he is not responding simply on the basis of differences in apparent brightness associated with the different spectral stimuli. We measured GY's blind field responses to stimuli

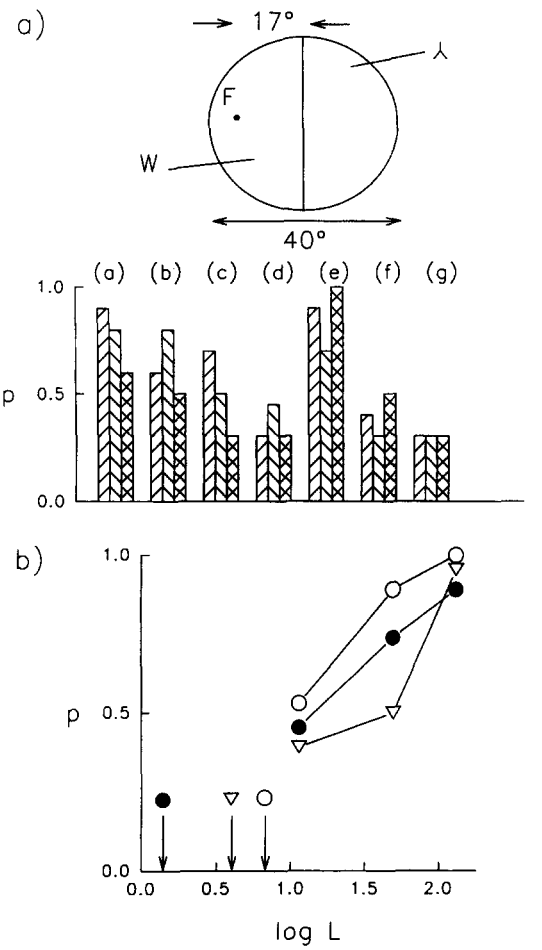
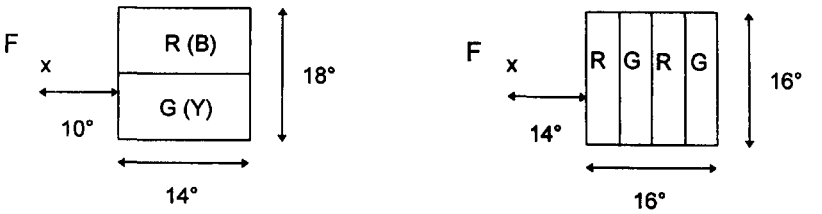


Fig. 1. (a) Colour naming data for GY, obtained with a semi-circular field (diameter 40°; duration of presentation 500 ms, wavelength λ), located 17° off-axis in the blind hemifield, as shown in the upper diagram. F denotes the fixation point, and the white light semicircular field, W (luminance 2.1 log trolands) was continuously visible. In each set of measurements, three stimuli were each presented 10 times, in random sequence, at luminance 2.1 log trolands, and the histogram bars give the probability, p, of GY naming correctly the stimulus. The stimulus combinations used in each experiment were, in the sequence open bar; hatched bar; double hatched bar (a) 665 nm, red; 535 nm green; 440 nm, blue (b) 665 nm, red; 615 nm orange; 585 nm, yellow (c) 615 nm orange; 585 nm yellow; 535 nm, green (d) 535 nm green; 490 nm, blue; 455 nm, purple. (e) 665 nm, red; white, W, and 665 nm added in equal photometric proportions to give pink; W, white (f) 535 nm green; white, W, and 535 nm added in equal photometric propotions to give pale green; W, white (g) 440 nm blue; white W , and 440 nm added in equal photometric proportions to give pale blue; W, white. A probability of 0.75 is statistically significant at 0.01 level. 1(b) Stimulus as in Fig. 1(a), but with F located 10° from the vertical diameter of the field. Three stimuli, 665 nm red , 550 nm green and 440 nm blue were each presented 20 times at one of three luminance levels, L, in random sequence, to GY's blind hemifield. The probability, p, of GY's correctly naming the stimulus colour is given by open circles (red), full circles (green) and triangles (blue). The arrows denote GY's thresholds for detection of the different stimuli.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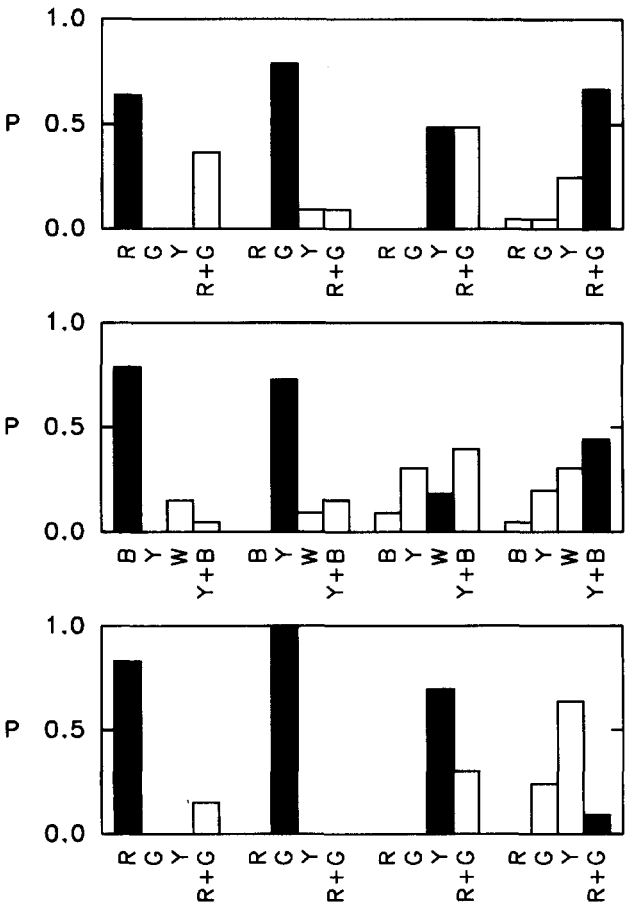
16°

a)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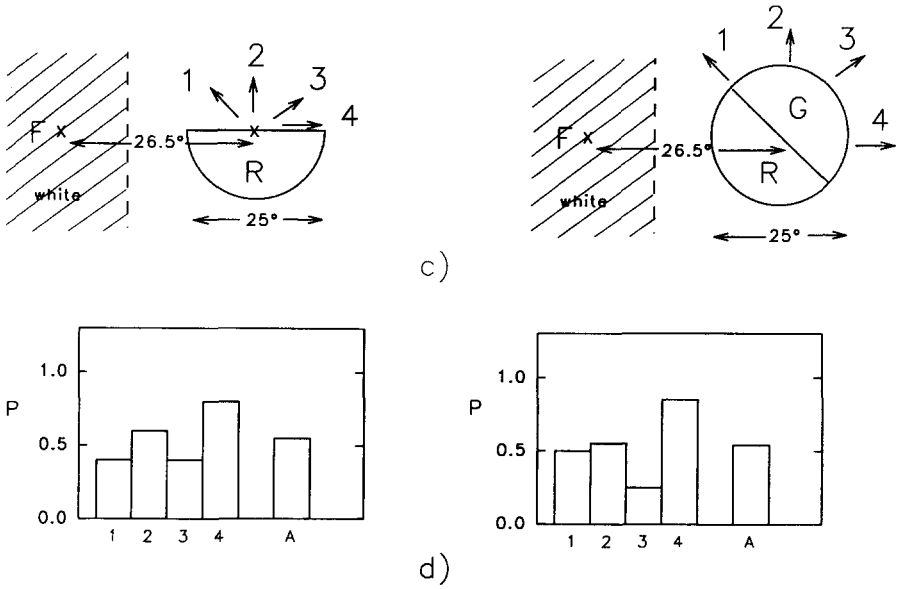

Fig. 2. GY's spatial discrimination for coloured stimuli. (a) The stimulus configurations for colour naming; the component colours were red (R, 665 nm), yellow (Y, 585 nm), green (G, 535 nm) and blue (B 440 nm). The observer fixated at F and stimuli were presented for 100 ms. (b) The probability, p, of GY identifying correctly the colour and spatial structure of the stimulus. Each block of histograms refers to one of the four stimuli presented, and the histogram corresponding to the (correct) identification of that stimulus is blocked, the other three histograms identifying the different erroneous names given to it. The two upper sets refer to the two component grating and the lowest to the four component grating shown in part (a). Each stimulus was presented 20 times in random sequence, at a luminance of 3 log troland, and p equal to 0.5 is statistically significant at 0.01 level. (c) The stimuli used to examine GY's orientation discrimination. The diameter of the red (R) semicircle and of that seperating the red (R) and green (G) semicircles was set at one of four orientations denoted by the arrows. The red and green were spectrally broadband corresponding to Wratten filters Nos. 25 and 47b respectively. Each stimulus orientation was presented 20 times, in random sequence, for 3 s. (d) The probability, p, of GY's identifying correctly the orientation of the diameter of the semicircular field (left hand plot) and that of the red–green border (right hand plot). Values are given for each orientation, as noted below the histogram bars, and the overall average value is given by A. For the individual orientations, p equal to 0.5 is statistically significant at 0.01 level.

which contained equiluminant coloured components (Fig. 2(a)). He was able to distinguish e.g. a red/green compound stimulus from a uniform red or a uniform green of the same luminance, but confused it with an equiluminant uniform yellow (Fig. 2(b)). He identified the orientation of the diameter of a semicircular red stimulus at greater than chance level, but his performance was not improved by providing red/green contrast across the diameter (Fig. 2(c)), even though he could discriminate accurately between the two component colours when they were presented separately. We examined the effect of colour on the interhemispheric transfer of movement which we have observed in GY's blind field responses (Finlay et al., 1996). GY reports that he detects no colour associated with his blind field response

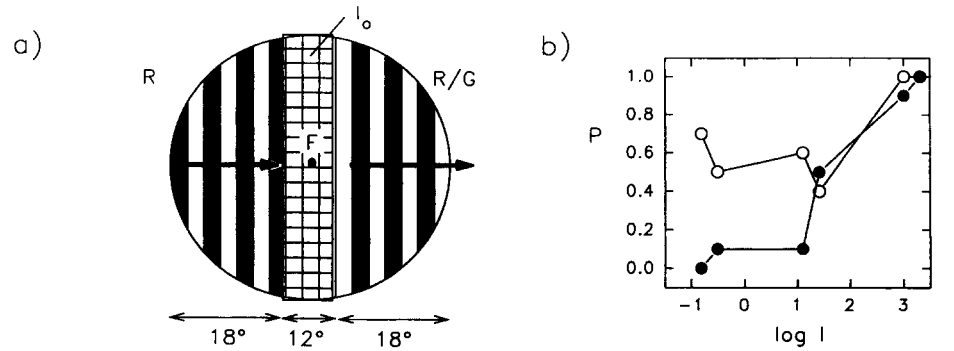

Fig. 3. Characteristics of the percept induced in GY's blind hemifield by movement localised in his normal hemifield. (a) The stimulus used to explore responses induced in GY's blind hemifield by moving stimuli presented to his normal hemifield. Both gratings were square-waveform (periodicity 0.5 cycle deg^{-1}) and moved left to right at 15 deg s^{-1}. That presented to the normal hemifield was red (655 nm) with fixed luminance 3 log trolands and that to the blind right hemifield was either red (655 nm) or green (540 nm). Both the coloured gratings were set at one of three luminance levels I, and the different luminance and colour combinations were each presented six times, in random sequence. The two gratings were presented simultaneously for 1 s and the central white strip (luminance I_0 equal to 3 log troland) was continuously visible. The observer fixated on the central fixation point F. GY was asked to identify both direction of movement and the colour of the percept in this blind hemifield. (b) The probability, p, with which GY reported the movement percept in the blind hemifield as moving left to right (full circles) and as appearing to be the same colour as the right hand grating (open circles). Note that as I falls, the direction of apparent motion reverses, whereas the identification of colour remains essentially random.

to moving, spectral stimuli presented in the normal hemifield, and to investigate this systematically we used the stimulus configuration shown in Fig. 3(a). The left field grating was red (655 nm) of fixed luminance (2 log trolands), whereas that in the right, blind field grating was either red (655 nm) or green (542 nm), each set at one of three possible luminance levels. The six different colour/luminance combinations were each presented 10 times, in random sequence, and GY was asked to name the colour and direction of motion of the percept he perceived in the right blind hemifield. He identifies both the colour and direction of motion (left to right) as being those of the right field grating when this is at high luminance, but as its luminance decreases, his identification of colour becomes essentially random, whereas the identified direction of motion reverses to right to left (Fig. 3(b)). We conclude that the blind field percept, which on every presentation was induced by the stimulus in the normal hemifield, is reversed in direction of apparent motion relative to the direction of stimulus motion, whereas it possesses no identifiable colour.

3.2. Abnormal inhibitory effects of chromatic stimuli

The size of the inhibitory region generated by a red stimulus was measured as a function of the time for which MW viewed the stimulus and of the location in the visual field. The size increases monotonically over a 20 min viewing period (Fig. 4(a)) and subsequent to cessation of viewing, decreases at a similar rate. A similar inhib-

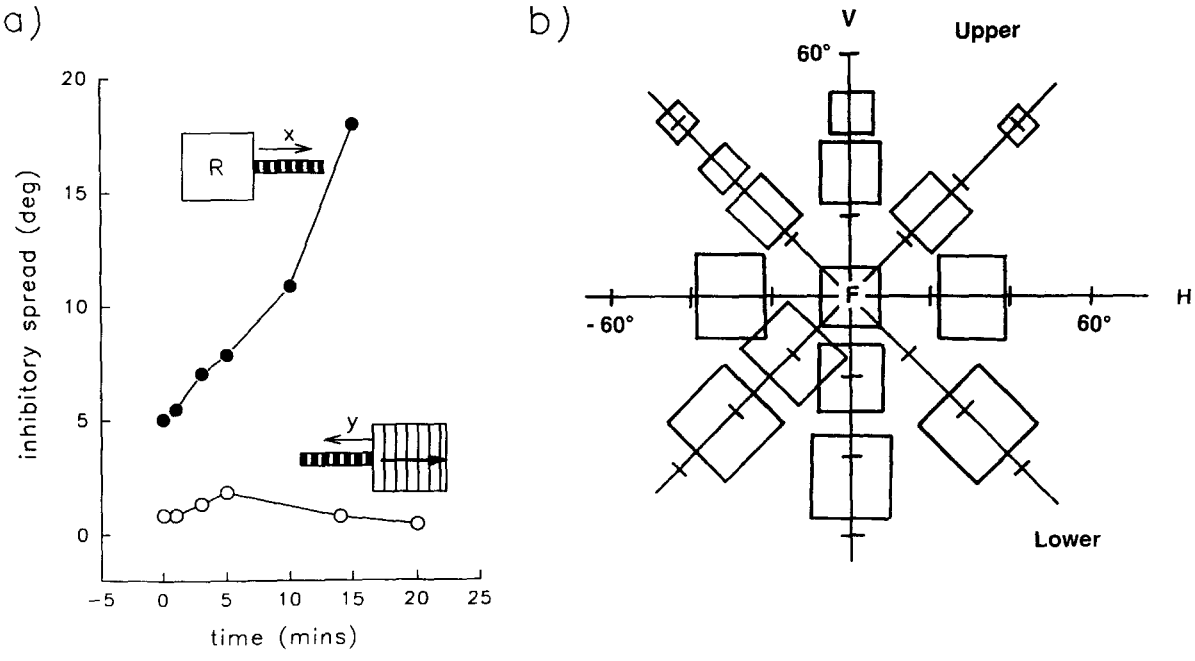

Fig. 4. Spreading inhibition for observer MW. (a) The extent of inhibitory spread, measured as the distance from the stimulus at which the high contrast black and white grating (period 0.25°) could just no longer be detected, plotted against the duration of viewing the stimulus. The red (R) stimulus was a 4° square, with C.I.E. 2° chromaticity co-ordinates $x = 0.577$; $y = 0.360$ luminance; 20 cd m^{-2}, for which the distance of inhibitory spread, x, is given by full-circles. The 4° square achromatic grating (periodicity 1.36 cycle deg^{-1}) moved horizontally at 6 deg s^{-1}, for which the distance of inhibitory spread, y, is given by open circles. (b) The size of the inhibitory region around a red 4° square with C.I.E. 2° chromaticity co-ordinates $x = 0.577$; $y = 0.360$, luminance, 12 cd m^{-2}, presented briefly at different points in the visual field. H denotes the horizontal and V the vertical meridian.

itory effect generated by a moving grating does not, however, increase over time. For a fixed 30 s presentation of the red stimulus, the inhibitory area varies in size over the visual field, and is much smaller in the upper than in the lower field (Fig. 4(b)).

A moving pattern placed adjacent to a red stimulus suppresses the spread of inhibition around the stimulus during continuous viewing, but when the movement stops, the inhibition spreads rapidly to cover the same area as it would had the moving stimulus not been present (Fig. 5(a)). This disinhibitory action of movement is observed when the red patch and the moving grating are well separated in the visual field, even in the case when the grating is presented to one hemifield and the red patch to the other (Fig. 5(b)). Despite the long range nature of this disinhibitory action, it is closely tuned to the direction of grating movement relative to the line joining the location of the coloured area to that of the moving stimulus (Fig. 5(c)).

4. Discussion

GY, like other patients with striate lesions who exhibit residual vision and/or 'blindsight', is selectively sensitive to transient light stimuli presented within the blind hemifield (Barbur et al., 1980; Ruddock, 1991). The ability to discriminate colours has been reported in only a few patients (Blythe et al., 1987; Stoerig, 1987; Stoerig and Cowey, 1992), and GY's colour discrimination, described originally by Brent et al. (1994), is illustrated in Fig. 1. Failure to perform spatial discriminations is also a common attribute of blind field responses of such patients, although some exceptions have been noted (e.g. Weiskrantz, 1986, 1990). The data of Fig. 2 show that GY is grossly impaired in the identification of spatial structure, including orientation, represented in the chromatic variation of images presented to his blind hemifield. It should be noted that when they are presented separately, GY is able to identify correctly the component colours of the compound images, and he also distinguishes between these and the compound stimulus. Thus GY distinguishes the red/green compound stimuli from either the red or the green components presented in isolation, but confuses them with an equiluminant yellow (Fig. 2). He is unaware of the spatial structure in the compound images, and responds to them as if the spatially distributed chromatic components mix together to give a percept corresponding to the space averaged colour. We conclude that the mechanisms which mediate chromatic discrimination in the absence of striate input are unable to partition the visual field spatially or to signal effectively edge orientation.

The striate cortex is the first stage of the macaque visual system at which single neuronal responses are sensitive to spatial parameters such as edges or bars (Hubel and Wiesel, 1968). Neurones in visual area V4 are selectively sensitive to chromatic stimuli (Zeki, 1978, 1980) but orientation sensitivity is also a feature of their responses (de Yoe and van Essen, 1988). The retinotopic representation in V4 is complex, single V4 neurones having large receptive fields which for neighbouring cells can be centred at widely different points in the visual field (Zeki, 1980, 1993). In contrast to V1 and V2 neurones, those in area V4 respond selectively to stimuli of a fixed colour appearance rather that to those of a specific spectral composition, thereby exhib-

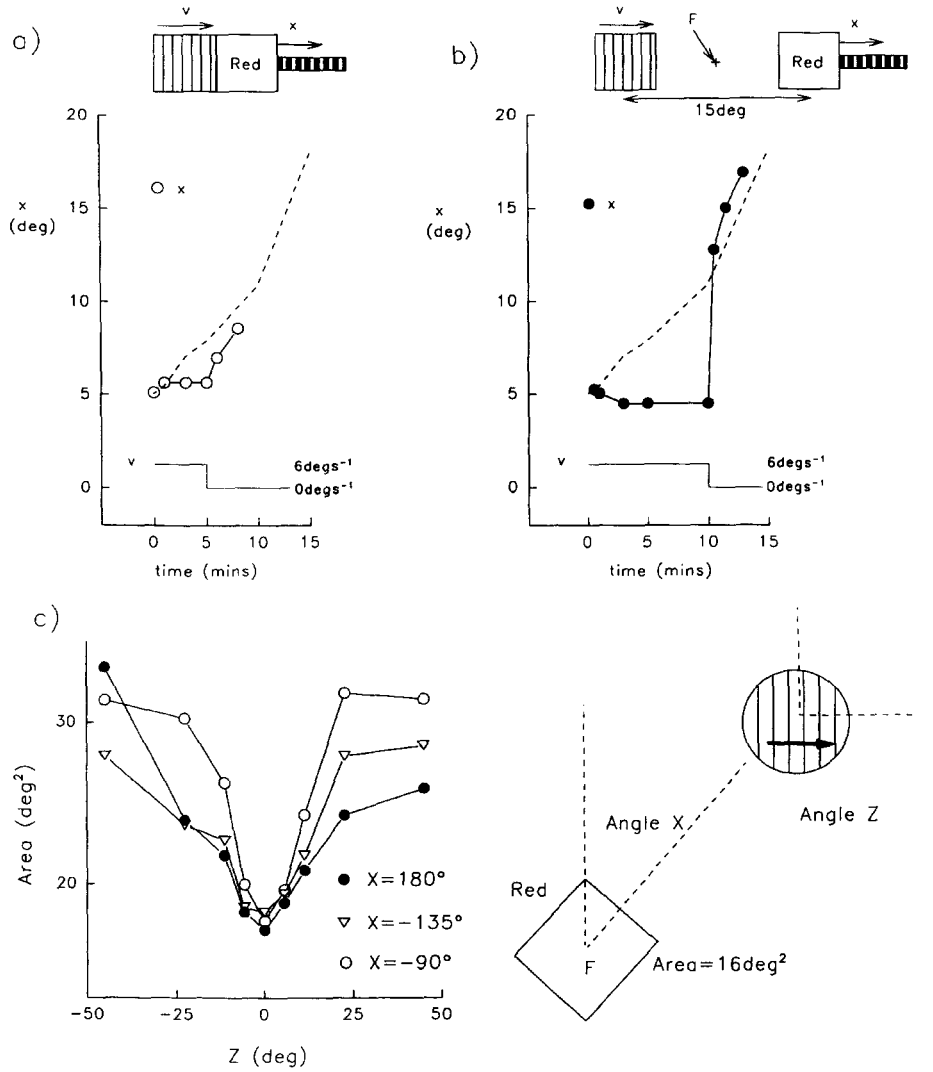

Fig. 5. Disinhibitory effect of motion on the inhibitory spread around a red stimulus. Observer MW. (a) The red square and the moving grating to which the data of Fig. 4(a) refer were placed adjacent to each other and the inhibitory spread from the red square in the direction x was measured as a function of time (open circles). The grating movement was stopped after 5 min viewing. The dotted line shows equivalent measurements in the absence of the moving grating (taken from Fig. 4(a)). (b) As for (a), but with the moving grating and the red square separated horizontally by 15°, on opposite sides of the central fixation spot F. The grating movement was stopped after 10 min viewing. (c) The size of the inhibitory area around the red 4° square, plotted as a function of the angle Z between the direction of motion and that joining the centre of the square to the centre of the circular patch. Z equal to 0° corresponds to movement directed at the centre of the red square, which was always aligned along the Z equal 0° direction. The circular patch (diameter 6°) contained an achromatic 1-dimensional grating (periodicity 1.36 cycles deg^{-1}; velocity 6° s^{-1}) and the centre to centre separation of the red square and the circular patch was 20°. MW fixated at the edge of the inhibitory area when determining its limit.

iting colour constancy in their responses (Zeki, 1980; Walsh et al., 1992, 1993). This response feature of V4 neurones requires that single neuronal responses are normalised with reference to the colour of the illuminant. We have reported that colour constancy fails in a patient BL (Kennard et al., 1995), who has lesions localised in the fusiform and lingual gyri, and this observation is consistent with functional homology between V4 and the damaged areas of BL's pre-striate cortex. One interpretation of the space-averaged responses to spatially structured chromatic stimuli found in GY's residual vision is that they correspond to the reference signals through which, in normal vision, colour constancy is achieved. We were, however, unable to induce changes in the apparent colour of a grey test patch presented foveally by exposing GY's blind hemifield to a Mondrian pattern under different illuminants, whereas with the Mondrian located in the normal hemifield, GY reported changes in the apparent colour of the grey patch.

The existence of multiple interhemispheric connections established by callosal fibres at the boundaries between the different striate and pre-striate visual areas of the macaque cortex has been adduced as evidence of parallel rather than serial organisation (Zeki, 1993). GY's data demonstrate induced responses to movement but not to colour (Fig. 3(b)) and further experiments have established that this effect involves input from the normal hemifield which terminates at the vertical meridian in the blind hemifield, a localisation pattern consistent with interhemispheric transfer via callosal fibres. The fact that a moving stimulus in the normal hemifield induces a percept of movement but not of colour in the blind hemifield suggests that there are selective callosal connections in human vision and that in GY's case, those involved with movement, perhaps connecting the human V5 pre-striate areas, generate the observed responses. Land et al. (1983) established that interhemispheric transfer via callosal fibres contributes to colour constancy, but we were unable to demonstrate such activity in GY's responses.

Although brain scans reveal no lesions, MW's visual responses are consistent with dysfunction at cortical level, as was discussed in our review of his case history (see Methods). MW's experimental data provide strong evidence of activity which arises in several distinct visual response mechanisms. Those sensitive to chromatic stimuli generate abnormal inhibitory effects which extend beyond the boundary of the visual image itself and the affected area increases during continuous viewing of the image (Fig. 4(a)). Mechanisms sensitive to moving achromatic stimuli give rise to a localised inhibitory effect which does not, however, increase during prolonged viewing of the stimulus (Fig. 4(a)). Moving stimuli also produce disinhibitory suppression of the spreading inhibitory effects around a coloured stimulus (Fig. 5). Mechanisms sensitive to stationary achromatic stimuli are, in contrast, entirely normal. Such specificity in MW's responses to different attributes implies a high degree of functional independence in the organisation of the (cortical) mechanisms which process colour, spatial pattern and movement.

The size of the inhibitory area generated in MW by a given chromatic stimulus changes significantly with location in the visual field (Fig. 4(b)). It is probable that this dependence on retinal location, which differs markedly from the M-scaling used to describe many visual phenomena (Drasdo, 1977; Rovamo et al., 1978), reflects

the visual field representation of those chromatic mechanisms which generate the inhibitory response. As was discussed in Section 1, the retinotopic organisation in monkey V4 is complex and there is separation of the representations of the upper and lower quadrants (Gattas et al., 1988) which, if also present in the homologous area of the human cortex, could give rise to the differences between the upper and lower field responses shown in Fig. 4(b). In contrast, the size of the inhibitory area generated by movement is essentially independent of location in the visual field, again different from M-scaling, and apparently representative of retinotopic organisation of the underlying movement-sensitive mechanisms. Thus MW's responses provide evidence of retinotopic organisation of (cortical) mechanisms sensitive to colour and movement which differs markedly from that usually described by M-scaling.

The disinhibitory action of movement in suppressing the inhibitory spread around chromatic stimuli is markedly non-retinotopic, as it involves interactions between visual field areas which are widely separated from each other, including those on opposite sides of the vertical meridian (Fig. 5). Cessation of stimulus movement causes the inhibitory effects of a chromatic stimulus to expand rapidly to cover the area which would have been affected had the moving stimulus not been presented (Fig. 5). It appears therefore, that movement suppresses the spreading inhibitory effects of chromatic stimuli by acting at the point at which the inhibition is expressed rather than on the chromatic mechanisms which generate the inhibition during continuous viewing. The transmission of disinhibitory effects over large distances across the vertical meridian is inconsistent with the topographical organisation of the striate and pre-striate visual areas, in which only the contralateral hemifield is represented. Such diffuse interactions may involve trans-callosal connections such as those observed in monkey V4 and V5, or may involve higher level interactions, for example in the parietal lobe or the inferior temporal lobe, where visually driven neurones receive inputs from both hemifields (Andersen, 1987; Bruce et al., 1986). The strong directional tuning evident in the disinhibitory action of the moving grating (Fig. 5(c)) demonstrates that some spatial ordering is retained in the convergent pathway which links MW's responses to movement with those to colour.

In summary, experiments on patients with visual deficiencies of cortical origin reveal spatially distributed responses of a kind not readily demonstrated in patients with normal vision. We have presented experimental data which describe such responses and have examined them in relation to cortical visual organisation of non-human primates.

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