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Research report

There's more to colour than meets the eye

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Abstract

Patients with cerebral achromatopsia, a perceptual disorder caused by ventromedial occipital brain damage, can be completely unable to arrange colours in chromatic sequence and fail most conventional tests of colour blindness. A possible explanation for cerebral achromatopsia is that the colour-opponent parvocellular (P) channel has been selectively and totally destroyed at the level of visual cortex, leaving vision to be mediated by the broad-band magnocellular (M) channel. The persistence of normal occipital visually evoked potentials, and preserved sensitivity to isoluminant chromatic gratings indicates that if this hypothesis is correct the destruction must occur beyond the striate cortex. We have shown that an achromatopsic subject can detect chromatic borders and construct shape from colour, and that he can even perceive the apparent direction of motion of a phase shifted isoluminant chromatic grating where perceived direction depends on knowing the sign of the colour diffence, i.e., which colour is which in the stripes. This and other evidence suggests that perhaps only one part of the cortical P channel has been destroyed. Does the critical area involved in achromatopsia correspond to cortical area V4 of monkeys, often implicated in processing wavelength? When Visual Area 4 is totally ablated in monkeys they have only a mild colour discrimination impairment and easily solve the colour ordering and colour selection tasks that an achromatopsic patient finds impossible. However, monkeys with ventromedial damage rostral to Area V4 *do* perform like achromatopsic patients, suggesting that the role of V4 in the perception of colour is still unclear and that the colour area of the human brain does not correspond to area V4.

Keywords: Achromatopsia; Area V4; Monkeys; Visual area; Colour perception

1. Introduction

One of the most stimulating discoveries about the organisation of the cerebral cortex in macaque monkeys is that roughly half of it consists of about 30 visual areas on each side of the brain [20]. Although the pace at which new visual areas are discovered has slackened in the past few years, suggesting that few if any remain undetected, the total number is impressively high and their existence has greatly influenced views about the functional organisation of the visual cortex. Since visual area 2 (V2) was first demonstrated and mapped in a monkey [10], roughly one further area has been uncovered each year. What are they all for?

Visual areas are defined anatomically in terms of their connections with each other and with the thalamus, by their cyto- and myelo-architecture and, more recently, by their receptor architecture, and most notably by the properties of their visual receptive fields as determined by single cell recording. The fourth visual area (V4) has received as much if not more attention than any other extrastriate visual area, in part because of an initial report that every one of 77 sampled cells showed selective sensitivity to wavelength [68]. Despite subsequent articles demonstrating that many cells in V4 are highly selective to orientation and even indifferent to wavelength (e.g., [18,53]) the idea that V4 plays a special role in colour vision persists. In part this is a sensible response to the evidence that a high proportion of its cells are, indeed, sensitive to the wavelength of visual stimuli (see Fig. 1) and to the report [69] that in V4, possibly for the first time in the visual pathways, cells are sensitive to the colour as opposed to simply the wavelength of a visual stimulus in the receptive field, i.e., that they demonstrate properties consistent with colour constancy. It was probably this combination of evidence that led to V4 being known as 'the colour area'.

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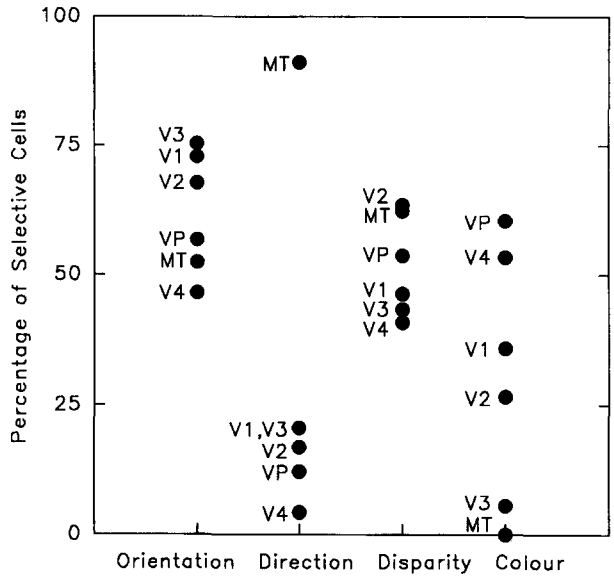

Fig. 1. The percentage of cells in six cortical visual areas that are selectively tuned to orientation, direction of motion, retinal disparity, or colour. Each point is the mean of from 1 to 10 separate investigations. Note that neurons in Area MT (V5) are highly selective for direction of motion and not selective for colour whereas the opposite occurs for neurons in area V4. The points were calculated from the data presented by Felleman and Van Essen [20] and the figure is reproduced from Cowey [11], with permission.

Nevertheless, had it been known then that in parts of the rostral inferior temporal cortex of macaques as many as 66% of neurons respond selectively to particular regions of CIE colour space [34], higher than in recent estimates for V4 [20] this region would have been an even stronger candidate for 'the colour area'. Previous recordings, in which colour selective cells were rare, were confined to the dorsolateral inferotemporal cortex [17].

Human colour vision can be impaired by damage to the cerebral cortex. We shall not discuss here the evidence, still poorly documented neuropathologically, that cortical damage can lead to colour anomia (a disorder of naming colours that is specific to colour words [32,51] or to a disorder of short-term memory that is specific for colours [14] or to impoverished knowledge of the colours of objects as opposed to their many other qualities [39]. The most clear-cut disturbance of the perception of colours that is associated with damage to a particular region of cerebral cortex is known as cerebral achromatopsia (see [43,52,70] for reviews). It is hardly surprising that cerebral achromatopsia has been attributed to the destruction of a region in the human brain purportedly corresponding to area V4 in macaque monkeys and that, *pari passu*, this region of our own brain is increasingly known as 'human V4' (see [74] and many others).

This article evaluates the evidence that area V4 in monkeys is specialised for dealing with wavelength and colour as opposed to other aspects of visual stimuli and that the region of the human cerebral cortex whose destruction leads to cerebral achromatopsia, indeed, corresponds to V4.

2. Where is area V4 in macaque monkeys?

Fig. 2 shows a sketch, like a geographical map, of a side view of the cerebral cortex of a macaque monkey. Some of the fissures have been prised apart in order to reveal their extensive depths. It shows the position of the visual areas that collectively occupy about one half of the cortex. Area V4 occupies much of the prelunate gyrus and extends on to the ventral surface (not shown) where its precise boundaries have not been determined. Note that the dorsal territory of V4 and its slender companion V4t lie next to V5 (also known as area MT). Now area V5 is particularly easily identified by its unmistakeable myeloarchitecture [60], and for the predilection of its cells for moving stimuli [2,47,72] and for the fact that its destruction leads to severe impairments of movement discrimination in the corresponding part of the visual field [12,41,48]. An analogous and probably homologous region has been demonstrated both anatomically and functionally in the human brain [64,75,76] and is known as human V5. It would be odd if human V4 were not adjacent to it, a point taken up below.

3. The characteristics of cerebral achromatopsia

Cerebral achromatopsia is a severe impairment of colour perception caused by damage in the vicinity of the lingual gyrus and the posterior portion of the fusiform gyrus on the ventromedial surface of the occipital and temporal lobes (see Fig. 3). As the name achromatopsia implies all sensation of colour is lost and the world is described as being drab and grey. Patients who are literally achromatopsic provide the clearest indication of the nature of the disorder but patients with less than total loss are much commoner. Strictly speaking they should not be described as having achromatopsia but the alternative term dyschromatopsia is sometimes reserved for a condition where the visual world appears as if it is being viewed through a coloured filter. Some of the disagreements about the nature of cerebral achromatopsia probably stem from not distinguishing clearly between total loss of colour perception and partial loss. For this reason we shall concentrate in this article on the extreme condition.

A patient with frank cerebral achromatopsia cannot arrange a graded series of isoluminent hues in their appropriate chromatic order, i.e., the patient sorts randomly when presented with the Farnsworth-Munsell 100-hue test. On the other hand a graded series of greys is usually sorted nearly normally [30]. The patient is

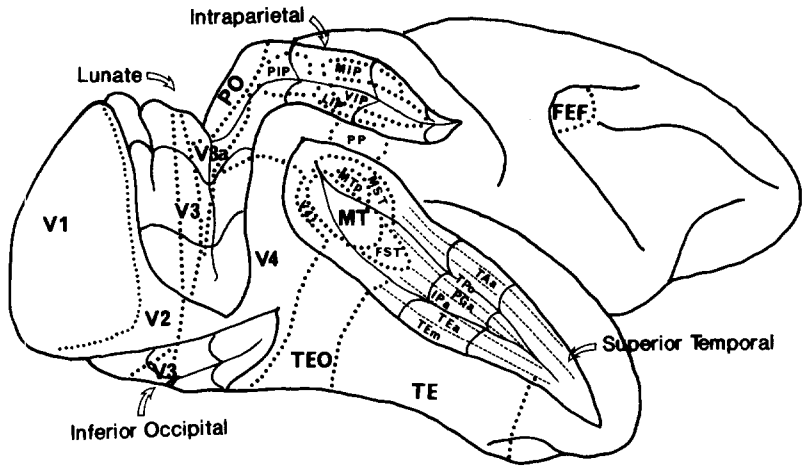

Fig. 2. Sketch of the lateral surface of the right cerebral hemisphere of a macaque monkey, with the caudal pole to the left. The lunate, intraparietal, inferior occipital and superior temporal sulci have been prised apart to expose their depths. Various visual areas are indicated amd the size of the lettering merely reflects their relevance to the present article. Additional areas, not shown, are present on the medial surface. Taken from Cowey [11], with permission.

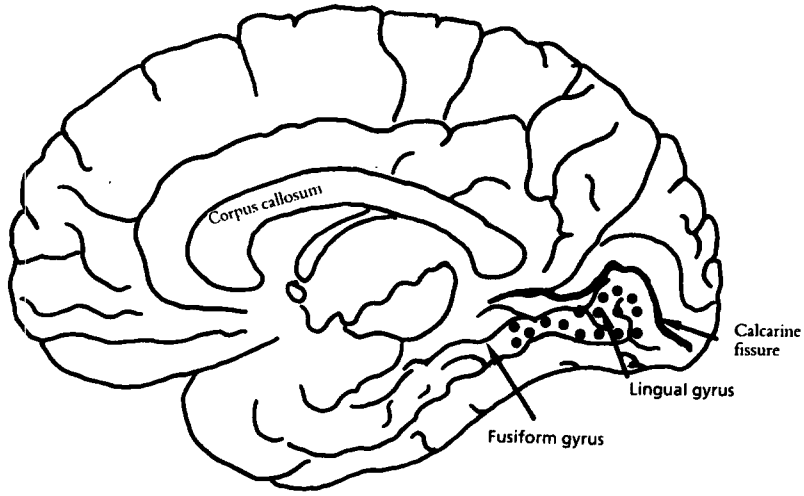

Fig. 3. View of the medial surface of the human brain, with the occipital lobe to the right and showing the region (dotted) whose damage causes cerebral achromatopsia in man. The crucial region was determined by comparing many neuropathological studies of the brains of patients with cerebral achromatopsia and extracting the area common to all of them.

also unable to tell whether two colours matched for brightness are the same or different. Whether the patient fails the other common test of colour blindness (the Ishihara pseudoisochromatic plates) depends, amongst other things, on the distance from which the plates are viewed and the latter has provided an important clue about the nature of the disorder (see below).

One patient who fulfils the above criteria of complete achromatopsia is patient M.S. who has been reported extensively elsewhere [25,26,46]. Following an attack of herpes encephalitis in 1970, M.S. was left with left homonymous hemianopia, with macular sparing, accompanied by achromatopsia in his residual visual hemifield. His Snellen acuity is normal in each eye and he retains three functional cone mechanisms. Examination of M.S. has provided many clues to the nature of, and explanation for, cerebral achromatopsia.

The simplest explanation for cerebral achromatopsia is that the lesion destroys the cortical areas and pathways essential for processing all signals about wavelength and colour. This hypothesis was initially supported by the report that an achromatopsic patient showed normal occipital VEPs to achromatic gratings but none to chromatic gratings [13]. However, other patients have not provided the same result [61] and patient M.S., whose achromatopsia is particularly severe, has apparently normal VEPs elicited by isoluminant sine wave red-green chromatic gratings even though he cannot tell the difference between two patches composed of red and green hues having a luminance equivalent to that of the

grating [50]. Like the patient with incomplete achromatopsia reported by Victor et al. [61], patient M.S. can detect the contours in sinusoidal gratings made up from isoluminant hues and his chromatic contrast sensitivity is normal at low spatial frequencies up to 10 cycles/degree, the highest frequency tested. Furthermore he can detect coloured shapes embedded in static or dynamic grey backgrounds and, perhaps most impressively of all, can identify the direction of movement of an isoluminant chromatic grating which was phase shifted by 90° in either direction, indicating that he has access to the sign of the hues even though he is not phenomenally aware of them [26]. How can this, and similar, paradoxical results be explained?

The syndrome can be interpreted in terms of what is known about the properties of the visual pathways in macaque monkeys [36,37,58,70,71]. Pathways dealing with different attributes of the visual scene such as colour, form and movement, originate in the retina and retain substantial (but far from complete) anatomical and functional segregation up to and including the primary and secondary cortical visual areas [20]. For example, retinal ganglion Pβ cells innervate the parvocellular layers of the dorsal lateral geniculate nucleus (dLGN) which, in turn, project predominantly to layer 4CB of striate cortex and thence to the cytochrome oxidase (CO) rich blobs and the interblobs of cortical area V1. The thin stripes and interstripes of area V2, revealed by cytochrome oxidase staining, receive inputs from the CO-rich blobs and interblobs, respectively, which then project to cortical area V4. This pathway constitutes the colour-opponent P-channel of visual processing. Cells in the CO-rich blobs are largely selective for wavelength but not orientation. Conversely, orientation selectivity is conspicuous in the interblobs of V1, which do not show pronounced wavelength selectivity. The P-channel is thus composed of two branches, even at this early stage of cortical processing, conveying chromatic and orientation information relatively independently to V2, where extensive although not complete functional segregation is maintained in the thin stripes and interstripes, respectively. The second channel, the M-channel, arises from Pα retinal ganglion cells, projects *via* the magnocellular layers of dLGN to area V1 where it terminates chiefly in layers 4B and 4CA. Layer 4B projects both directly, and *via* the cytochrome oxidase thick stripes of V2, to V3 and V5. The orientation and direction selectivity, and absence of wavelength selectivity, of cells in the M system is consistent with a role in processing motion and form. Although the contrasting properties of the P and M channels are broadly accepted the additional idea that they are also segregated into different groups of cortical visual areas now seems untenable. For example, area V4 receives a prominent input from both the colour-opponent P-channel and the broad band M channel [21] and area V5, often described as receiving input from only the M channel, can also be activated via the P channel [42].

A possible explanation for achromatopsia is that the brain damage disrupts the cortical P-channel, although why an encephalitic lesion should have such selective effects is as unclear as the reasons for the occasional selective effect of carbon monoxide poisoning on lightness discrimination [45]. Damage could compromise all or part of the P-pathway, depending on the cortical stage at which the disturbance occurs. However, the locus of the lesion in ventromedial occipital cortex suggests that the functional loss occurs beyond the striate cortex. Nevertheless, the ability of achromatopsic patient M.S. to detect the border between two adjacent, isoluminant hues without being able to detect a difference between the same two hues when they were discontiguous [25] was originally ascribed to cells in the broad-band M-channel, which cannot be rendered unresponsive to chromatic borders or chromatic changes by any adjustment of the relative luminances of the colours [16,31,35]. In addition, M.S. could detect the figures embedded in the Ishihara plates when they were presented at a distance of greater than 2 m, or when they were blurred by optical means. Again, it was suggested that each of these procedures obscured the contours of the small individual dots, of which the plates are composed, enabling the M-channel to detect the now dominant chromatic edge. However, subsequent studies revealed residual colour-opponent processes in the spectral sensitivity curve, suggesting that achromatopsia is not the result of total destruction of the P-channel [25].

The variability of the severity of the colour disturbance in achromatopsia has prompted several explanations for the condition. Preserved ability can extend to colour naming and identification of figures embedded in Ishihara plates [43,61]. The patient described by Victor et al. [61] was poor at colour ordering and identification yet was able to detect colour differences in an oddity task and had normal chromatic colour contrast sensitivity and acuity and a clear visual evoked potential on presentation with an isoluminant chromatic checkerboard. As the figures in the Ishihara plates were also readily identified the authors conclude that the substantial residual abilities were mediated by intact striate cortex. However, an alternative interpretation is that the lesion to the 'colour centre' in the lingual and fusiform gyri was incomplete. In this respect, examination of a patient with complete achromatopsia is informative. M.S. is quite unable to detect chromatic differences in an oddity task or to detect figures in the Ishihara plates at normal reading distance. Nevertheless he, like the patient of Victor et al. [61] has normal sensitivity to sinusoidally modulated isoluminant chromatic gratings which also elicit a conspicuous visually evoked cortical potential [49,50]. This suggests that any residual ability (e.g., detection of colour differences in an oddity task

and correct identification of the Ishihara plates) in cases of incomplete achromatopsia is unlikely to depend on the integrity of striate cortex and is better accounted for by a partial lesion to those areas that are indispensable for the conscious representation of hue. Such areas are assumed to be integral to the P-channel of visual processing. If, however, cerebral achromatopsia is the result of destruction of the P-channel, then the weight of evidence favours the view that such damage must occur beyond striate cortex. The characteristic shape of the spectral sensitivity function in M.S., which shows diminished sensitivity for wavelengths displaced from peak cone sensitivities, is strong evidence for colour-opponent processing, an exclusive property of the P-channel. This, along with the demonstration that M.S. can detect form that is concealed by random luminance masking (which ensures that chromatic contour cannot be detected by the broad-band neurones of the M-channel) leads to an alternative interpretation. If damage is confined to the cytochrome oxidase 'blobs' of the P-channel while the 'interblob' pathway remains largely intact, then the coding of orientation in the latter pathway, in the absence of substantial chromatic selectivity, may well account for the surprising ability of M.S. to detect form defined by chromatic contour without any accompanying awareness of hue.

Cerebral achromatopsia is often described as a deficit restricted to the perception of colour even though it usually occurs alongside a severe disturbance in the visual recognition of objects. The customary and reasonable explanation for the association of disorders is that the cortical lesion is prone to involve adjacent areas that are separately concerned with colour and form. However, the question of whether cerebral achromatopsia can occur in isolation has become more important now that the ventro medial damage that gives rise to it is increasingly referred to as damage to human V4, the reason being that damage to V4 in macaque monkeys always impairs shape discrimination learning and retention (e.g., [24]). In the handful of instances where it is reported that the visual impairment in patients is restricted to colour vision [13,33,40,71] it is far from clear that the disorder was genuine achromatopsia and/or that the tests for form vision were sufficient. For example, although the patient referred to by Zeki ([71], p. 45) could draw and copy skilfully, this is common in patients with dense associative object agnosia. Achromatopsic patient M.S. also suffers from severe visual object agnosia but he can readily draw and copy what he cannot identify. This particular issue is at present unresolved.

The recent evidence of Allison et al. [3], in which brain potentials evoked by chromatic stimuli were recorded via penetrating electrodes that reached the lingual and fusiform gyri, indicates that wavelength-selective neuronal activity is prominent in this region and that when these areas are stimulated through the same electrodes the patient experiences colour sensations. However, the same report reveals that wavelength selective activity is also present in the inferior temporal gyrus and in a region of dorsal lateral cortex, where electrical stimulation also elicited sensations of colour. Stimulation of this lateral region also evoked sensations of colour. Unfortunately, there were no tests for responsiveness to shape or movement of visual stimuli, nor exploration with chromatic stimuli of other cortical regions implicated in colour processing by functional imaging. However, most of human area V4 was also placed in lateral occipital cortex by Drasdo et al. [19] on the basis of VEPs elicited by isoluminant coloured or achromatic checkerboards (Fig. 4c).

4. Is human V4 on the lingual gyrus and caudal fusiform gyri?

The answer to this question is usually based on reports that an unusually high proportion of cells in V4 in macaque monkeys respond selectively to some aspect of the wavelength of a visual stimulus and that only a lesion involving the ventromedial region of the human brain leads to cerebral achromatopsia. Therefore, the argument runs, as V4 must be of great importance in colour vision it must lie ventromedially in our own brain. But there are other, neuroanatomical, ways in which human V4 might be located on the map. Cyto- and myeloarchitecture, cytochrome oxidase immunocytochemistry, and the patterns of terminal degeneration of callosal fibres in the normal hemisphere of patients who died following damage to the occipital and temporal lobes of the other hemisphere, have been used to localise areas V1, V2, V3, V4 and V5 in the human brain [5–7,77]. Fig. 4a,b shows the outcome. None of these studies centres or even places V4 in the lingual gyrus although this alone might not be seen as a serious problem, given that the damage responsible for cerebral achromatopsia probably always involves the caudal fusiform gyrus as well. However, although the medial edge of 'human V4' is shown as occupying part of the fusiform gyrus it is principally more ventrolateral, continuing up to the heavily myelinated lateral region thought to be V5, i.e., roughly where one where one would expect V4 to be on the basis of its position relative to other visual areas in macque monkeys. It does not correspond to the 'hot spot' first demonstrated by positron emission tomography and called the 'colour area' [74].

5. Localization by functional neuroimaging

Functional imaging by positron emission tomography (PET) and magnetic resonance (MR) seems ideal for revealing the location of the colour area in the human brain, if there is, indeed, only one area. In the first such

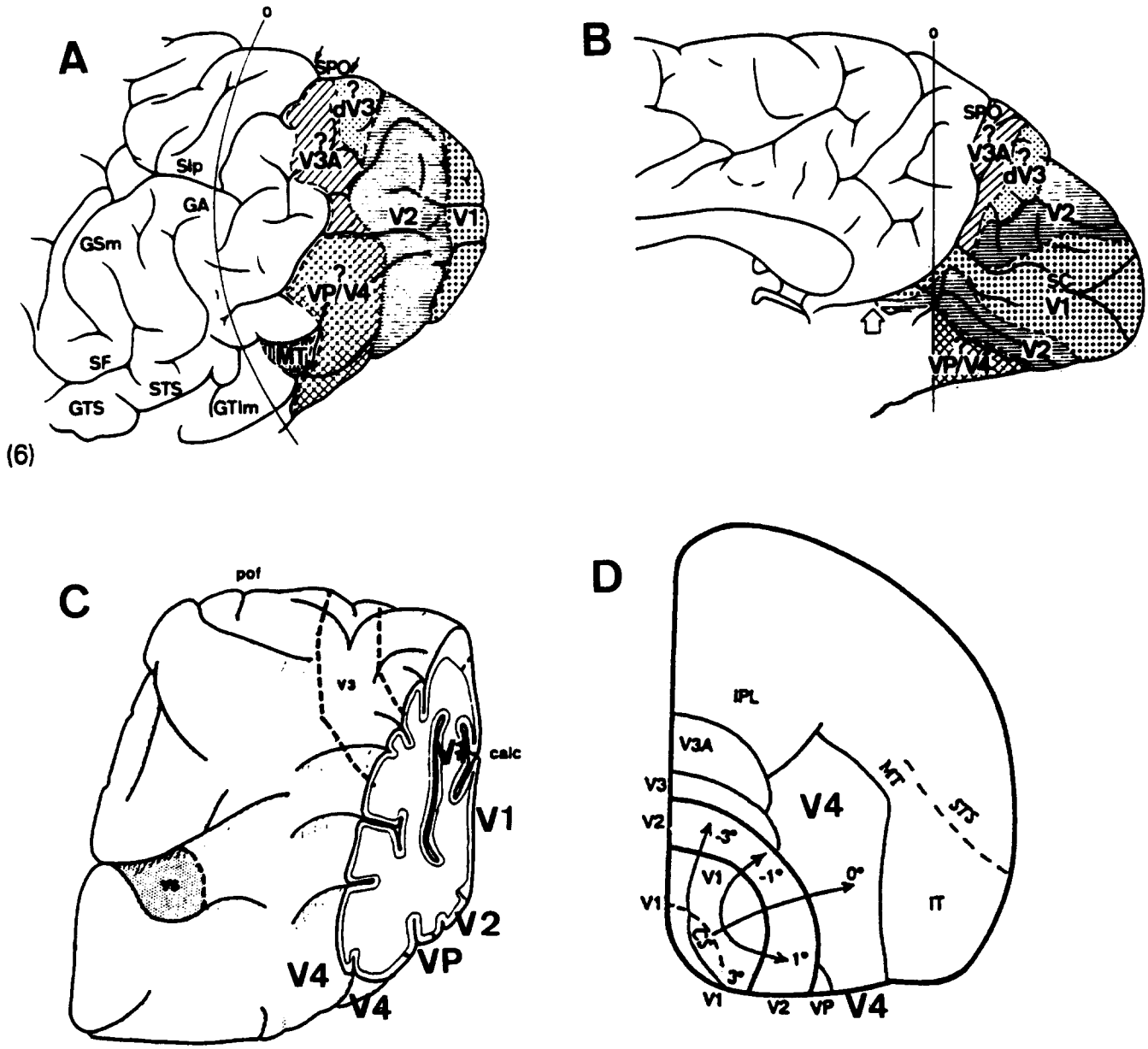

Fig. 4. Maps of the human visual cortical areas in lateral (A) and medial (B) views of the left occipital lobe. The areas were defined by cyto- and myeloarchitectonic criteria. Note that area V4 is predominantly lateral and abuts area V5 (MT). Modified from Zilles and Schleicher [77]. (C) Lateral view of the left human occipital lobe showing the location of human visual areas based on the distribution of callosal afferents and corresponding roughly to the view shown in (A) above. Note that area V4 occupies a latero-ventral position and does not extend on to the lingual gyrus. Area VP is the ventral component of V3. Modified from Clarke [5]. (D) Outline diagram of a caudal view of the right human occipital lobe showing visual areas determined from evoked responses to different visual stimuli and taken from Drasdo et al. [19]. Note that area V4 occupies much of the lateral surface, that its centre, i.e., 0°, is also lateral, and that it abuts area V5. A–D are taken from [78], with kind permission from Elsevier Science Ltd., Kidlington, UK.

experiment [38,74] subjects inspected a pattern of isoluminant coloured patches or an identical pattern of greys. When the levels of cerebral blood flow in relation to the latter were subtracted from those related to the former it was revealed that the cortex in the vicinity of the lingual gyrus showed a strong elevation in blood flow that was related to inspecting the coloured display. This very clear result, more than any other, probably led to the now common description of this region as 'the colour area' and in addition as 'human V4'. However, it was not clear even in the first reports that elevated blood flow was absent from other regions and different experimenters using active viewing (where the viewer had to make perceptual discriminations and attend to some particular aspect of the display such as the colour, orientation or shape of its components) rather than passive viewing, demonstrated additional regions, notably on the lateral surface of the occipital lobe, adjacent

and caudal to the region known as V [9,23]. These results indicate that chromatic differences are processed in several segregated cortical areas but that it is the region in or close to the lingual gyrus that is indispensable for the phenomenal awareness of colour. In this narrow sense it is therefore appropriate to refer to it as the colour area as long as it is remembered that wavelength is analysed in other regions for other purposes, such as determining form, motion and texture from chromatic cues. It is probably the activity of these other areas that allows a patient with dense achromatopsia to use hues he cannot appreciate to generate shape in an apparently paradoxical manner, and which is detectable in the VEPs generated by isoluminant chromatic displays.

6. Perceptual effects of removing V4 in monkeys

If V4 in macaque monkeys really is functionally equivalent to the 'colour area' in the lingual and fusiform gyri of the human brain, its total removal should lead to achromatopsia. It does not. But it does impair other aspects of visual perception! Even before the boundaries of area V4 were well established Dean [15] examined the effects of removing extrastriate cortex that included the present V4 but was somewhat larger than V4. Macaque monkeys were barely impaired at hue discriminations using Munsell coloured chips. However, there was no unequivocal evidence that the hues were isoluminant for the animals, who might conceivably have relearned the discriminations on the basis of lightness. This problem was also present in evaluating the lack of effects on colour discrimination of similar lesions in other experiments [22]. When luminance cues were eliminated or made irrelevant and hue discrimination thresholds were measured there was a small threshold elevation following complete removal of area V4 in macaque monkeys but the impaired performance was still within the normal range of other monkeys and trichromatic human observers [24,27] (see Fig. 5). The latter study also demonstrated that in a task of colour ordering, like a simplified version of the FM 100-hue test, monkeys with removal of V4 are not impaired – unlike a subject with cerebral achromatopsia who was presented with the same stimuli.

The role of area V4 can also be examined by removing only part of it and comparing visual performance when stimuli are presented in and confined to the affected or unaffected regions of the visual field, allowing each animal to be used as its own control. Using this technique similar slight changes in colour discrimination were found [54–57]. But there was a prominent disruption of the animals' ability to find and respond to stimuli, whether characterized by luminance or colour or motion or depth, that are less visually salient than others around them [54]. In striking contrast to the small effects on hue discrimination, the total or partial ablation of V4

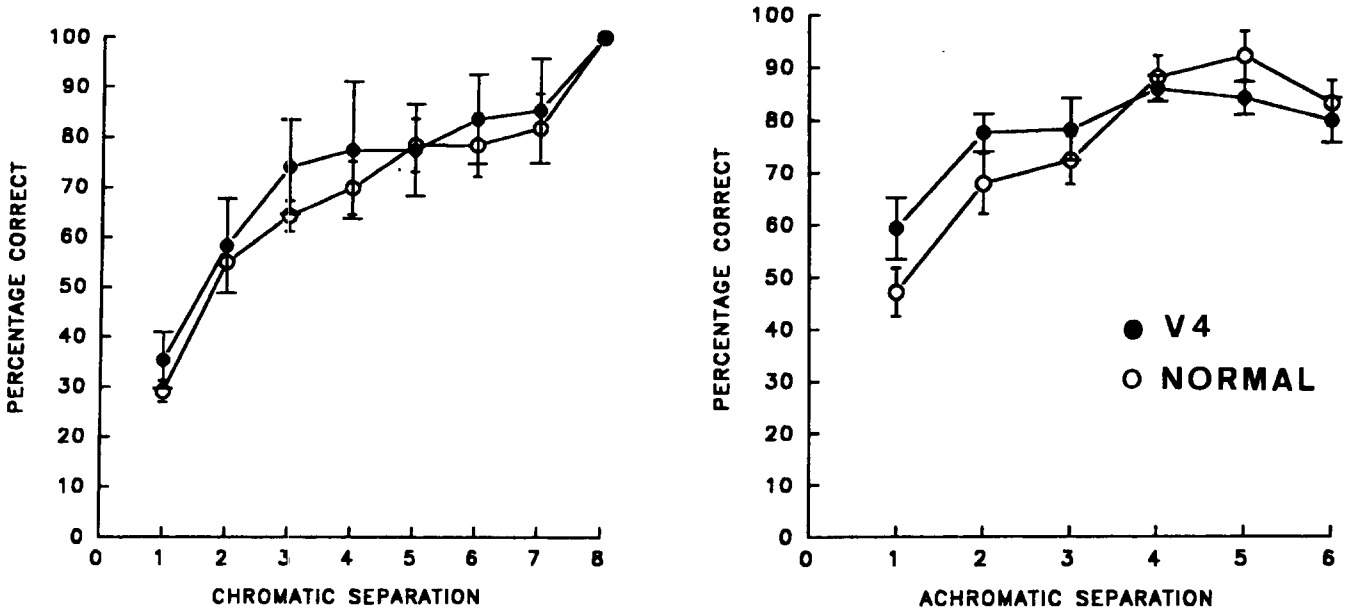

Fig. 5. (A) Percentage correct performance of six macaque monkeys on a task where the monkey had to touch the odd one out in a display of nine coloured targets, one of which was a different hue from the other eight. Error bars represent standard errors for the performance of the unoperated control group (open symbols) and monkeys with total removal of area V4 (solid symbols). Chromatic separation refers to the chromaticity difference between the target and the distractors, all of which were in the red-green range. The monkeys without area V4 are not impaired. (B) The results with the same monkeys when the target was a grey that differed in luminance from the other eight greys. Achromatic separation refers to the luminance difference between the target and distractors. The groups were not significantly different. Taken from [24] by permission of Oxford University Press.

in several of these experiments produced a clear impairment of form discrimination. Commenting on experiments like these [73], the author remarks "I do not refer here to the carefully controlled experimental lesions in monkeys, which have been the single worst guide to the organisation of the visual cortex imaginable,.....' (p. 157). There is a danger here of reviving the phrenologists" objections to lesion experiments that failed to confirm their views (e.g., Coombe [8]). It will be recalled that the lesion experiments carried out on the cerebellum by Flourens, which indicated that 'the cerebellum serves for the regulation of muscular motion' (Coombe [8], p. 155), were dismissed by contemporary phrenologists because of phrenological demonstrations that the cerebellum was the organ of amativeness. The effects of removing V4 in monkeys provide the strongest possible indication that the area is involved in selective attention to a rather broad range of visual stimuli that include colour but go far beyond colour.

If cortical area V4 in monkeys is not essential for hue discrimination and ordering of hues, what is its contribution to colour vision? One possibility is that its role is subsidiary in the sense, mentioned above, that it is concerned with the analysis of colour and wavelength in relation to the perception of shape. Other possibilities are that it is concerned with colour constancy [62,65] or the categorisation of colours [63,64] or with memory for recently perceived colours.

A corollary to the question raised above is: can cerebral achromatopsia be caused in monkeys by any other cortical lesion? The answer is yes. For many years it has been known that inferotemporal ablation in macaque monkeys *does* impair colour discrimination and to a much greater extent than ablation of V4 [1,22,29]. More recently we have specifically examined the role of area V4, the occipital cortex immediately adjacent to it ventromedially, and the rostral inferior temporal cortex and its medial extension to the parahippocampal gyrus [28]. Fig. 6 shows the performance of a macaque monkeys with the rostral inferotemporal lesion, which includes the region shown by Komatsu et al. [34] to contain a large percentage of colour selective cells, on an oddity task. The task required monkeys to select the odd-one-out when presented with a target and eight identical distracters and the chromatic or luminance difference between target and distracters was systematically varied. The figure shows that the monkey, like three others, was achromatopsic and its performance was almost indistinguishable from that of achromatopsic patient M.S. who was tested with an almost identical display. Furthermore, when required to detect the coloured target from eight grey targets varying in lightness neither M.S. nor monkeys with inferotemporal lesions performed any better than would be expected on the basis of random responding [25,28]. It may be worth noting that three of the four monkeys had some indirect retrograde damage to the lateral geniculate

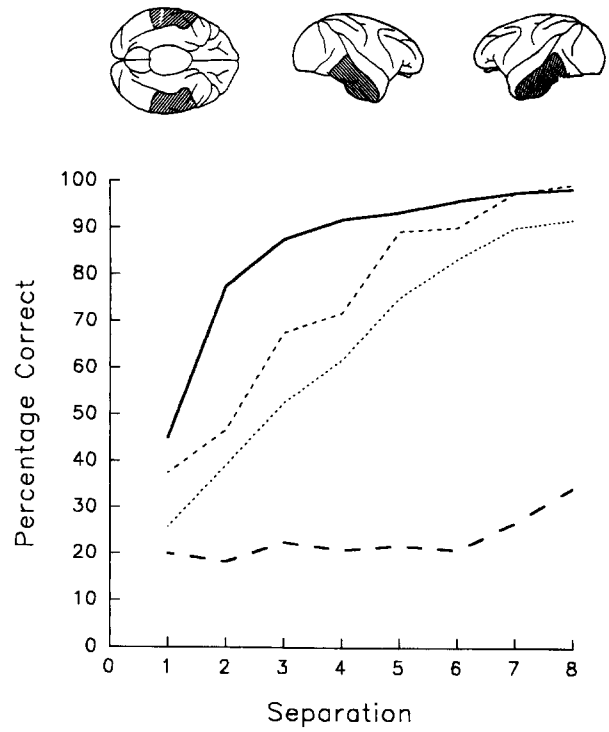

Fig. 6. (Top) Ventral and lateral views of the cerebral hemispheres of a macaque monkey with ablation of area TE, entirely sparing area V4. (Bottom) The performance of the monkey on a task in which he had to touch the odd one out in a display of nine targets, either one coloured among eight other different colours or one grey that was brighter or dimmer than the other eight identical greys. Separation, on the abscissa, refers to the chromatic or achromatic difference between the target and the distractors. Solid and long dashed lines respectively illustrate pre- and postoperative performance for hues. Short dashed and dotted lines respectively illustrate pre- and postoperative performance for greys. Performance with colours was severely disrupted whereas performance with greys was only mildly affected, as in patients with cerebral achromatopsia. Taken from Heywood et al. [28], by permission of Oxford University Press.

nuclei that would cause a superior altitudinal field defect and that the latter is a common although not invariant feature of cerebral achromatopsia in human patients [43]. However, it important to stress that, as in human achromatopsia, the field defect is not the cause of the impaired colour vision. One achromatopsic monkey had no geniculate damage. Finally, the small decrement in discrimination of grey is consistent with only *relative* preservation of achromatic discrimination in achromatopsic patients.

7. Pupillometry

The pupil of the eye responds to both achromatic and chromatic changes even when there is no change in overall space-averaged luminance. The response to purely chromatic changes is thought to indicate some cortical influence on the pupil, given the sparse evidence for chromatic processing in the mid-brain and brainstem.

Would the putative cortically mediated control over the chromatically triggered pupillary response be abolished in cerebral achromatopsia, while sparing the achromatic response. The answer is yes, at least in the only two patients yet studied [4]. But what about monkeys? Our first, still unpublished results [44], on one adult male macaque with unilateral removal of the rostral region of inferotemporal cortex (the same lesion that causes the achromatopsia shown in Fig. 6) show that the response to chromatic, but not achromatic, gratings was abolished when they were presented in the hemifield contralateral to the lesion, further indicating that if macaque monkeys have a 'colour area' functionally equivalent to the ventromedial area of the human brain it is not area V4. We are now extending these experiments to include ablation of V4.

8. V4 in the scheme of things

Brain maps like that shown in Fig. 2 do not and can not show how the many visual areas function in a hierarchy or in parallel or in some computational network that combines hierarchical and parallel processing. This is one reason why box and arrow flow diagrams are often preferred. However, a third approach is to use all the available quantitative information about the connections between different visual areas (number, reciprocity, length) and, by optimization analysis, derive a connection matrix that best fits these properties and provides a topological model of the visual areas [66,67]. The result of doing this with the visual areas of the macque monkey is shown in Fig. 7, which reveals that the extra-striate visual areas are, indeed, divided into two major pathways (as first suggested on the basis of studies of brain lesions [59], and that particularly area V4 appears to be a staging post for many aspects of visual information, e.g., orientatation, wavelength, disparity, that are processed in the ventral pathway to the temporal lobe. Little surprise that its removal leads primarily to complex disorders of object perception and visual attention rather than to simple unitary deletions of sensory qualities such as hue.

9. Conclusions

The existence of achromatopsia along with its partner, akinetopsia, has fuelled the belief that each of the

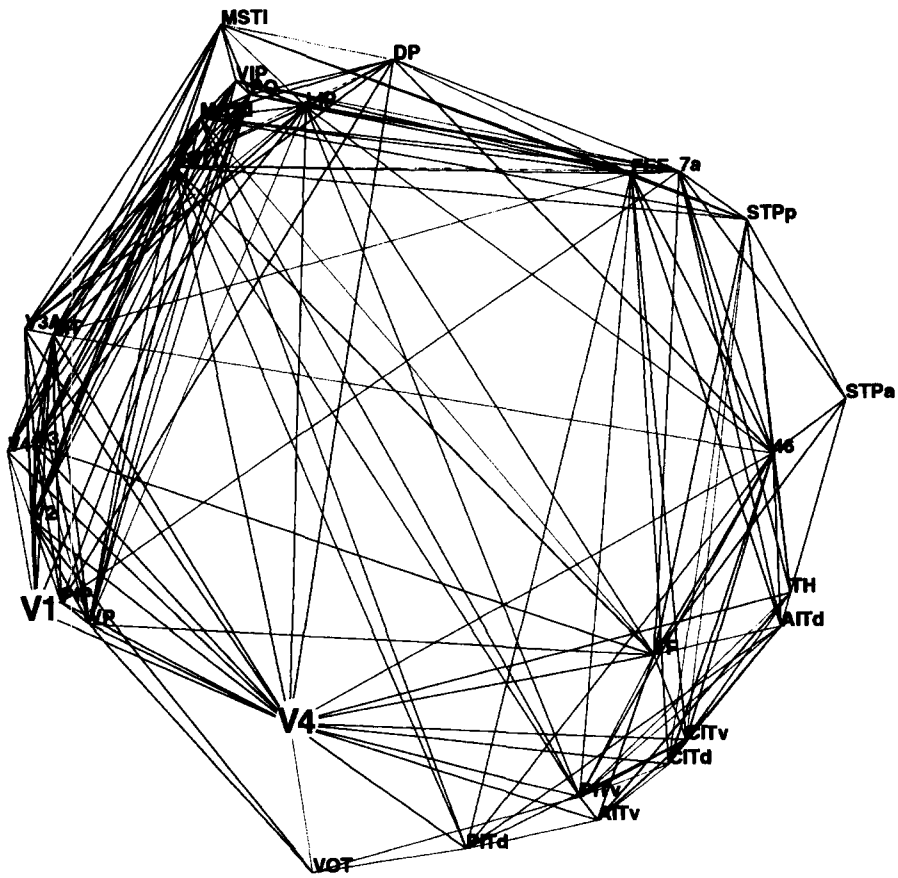

Fig. 7. The topological arrangement of the cortical visual areas of the macaque monkey as determined by multidimensional scaling. Note the position of area V4 in the so-called ventral pathway. Adapted from Young [66], and represented with permission from MacMillan Magazines Ltd. (*Nature*, 358 (1992) 152–154).

patchwork of visual cortical areas is concerned with the processing of a single psychophysical dimension or a single visual experience, such as colour or motion. Quite apart from the fact that it is difficult to think of as many psychophysical dimensions or visual qualities as there are visual areas, there are other major problems with this view. First, in even the 'purest' example of akinetopsia yet reported [75,76] the lesion includes far more than cortical area V5 and the patient's performance on an adequate range of static perceptual tasks has not been reported. Second, the results of experimental lesion studies on monkeys have yet to lend any convincing support to this belief. Although there is a consistent resemblance between the short-term effects of lesions confined to area MT in the monkey and the characteristics of human cerebral akinetopsia, and several lines of evidence point to a convincing homology between human V5 and cortical area MT, the only cortical lesion that severely and permanently disturbs (but does not abolish) the discrimination of movement in macaques extends beyond area MT [12,41], suggesting that several cortical visual areas, all concerned with the perception of movement, have to be involved. We have argued in this article that the parallel often drawn between the human 'colour centre' and cortical area V4 is even less convincing.

Since colour vision, but not just colour vision, is essentially abolished following damage to cortex in the monkey that is rostral and ventral to V4 [28], whereas large bilateral lesions to V4 have little or no effect on colour discrimination as assessed by tasks that an achromatopsic patient cannot perform, it is reasonable to suppose that the lingual and caudal fusiform regions in the human brain do *not* correspond to area V4 in macaque monkeys (see also Merigan [44]). This conclusion is supported by the results of neuroanatomical studies. The analogous consequences of ventromedial occipital damage in people and inferotemporal cortical ablation in monkeys suggests a very different homology. The proposal that the second temporal convolution in the human brain is the homologue of the superior temporal sulcus in the macaque implies a migration of tissue corresponding to inferotemporal cortex to a more medial location. This is consistent with the lingual and caudal fusiform gyri being functionally equivalent to anterior temporal cortex in monkeys. An alternative and rarely discussed possibility is that achromatopsia is a result of white matter damage, especially beneath the collateral sulcus, which disconnects striate and extrastriate areas from more rostral and inferior temporal cortex.

Achromatopsia is perhaps the result of destroying, or disconnecting, an area which is indispensable for the cortical registration and phenomenal awareness of hue. It may also be interpreted as the loss of one arm of the P-channel, the 'blob' pathway, leaving intact the ability to extract form from chromatic contour, a role for which the 'interblob' pathway is well suited. While form discrimination is also compromised by temporal lobe lesions in the monkey it is as yet unknown whether anterior inferotemporal lesions spare the ability to *detect* isoluminant chromatic form.

A very different interpretation of achromatopsia is that it results from a breakdown of colour constancy, the ability to perceive unvarying hue despite substantial changes in the spectral composition of the illuminant. This view derives in part from the observed responses of cells in V4 of monkeys which demonstrate such a property, in addition to their large receptive fields which are ideally suited to the colour interactions across large areas of the visual field predicted by many computational accounts of constancy mechanisms. However no one has yet explained, or even attempted to explain, why impoverished colour constancy should lead to an inability to sort a range of hues into their 'correct' chromatic sequence under constant illumination or why the world appears grey whatever the illumination. Furthermore, deficits in colour constancy in monkeys following ablation of V4 [65] have not yet been shown to be selective for colour constancy rather than an example of a general deficit on all difficult visual perceptual tasks. Even if the severe loss of colour vision following rostral inferotemporal cortical ablation is consistent with the view that it is constancy that is at fault, it merely locates the 'colour centre' elsewhere in the primate brain than in area V4.

The suggestion that cerebral achromatopsia is a failure to 'construct' colour raises the possibility that patient M.S. retains a 'local' process, enabling him to detect colour discontinuities, but has lost the 'global' filling-in mechanism which gives a colour to any region of space. This could readily be tested by examining lightness constancy in patients like M.S. and, just as informatively, in monkeys with inferotemporal cortical ablation.

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