

SHORT REPORT

A disorder of colour perception associated with abnormal colour after-images: a defect of the primary visual cortex

D Chan, S J Crutch, E K Warrington

Abstract

A 64 year old woman with posterior cortical atrophy secondary to probable Alzheimer's disease is described. Her presenting symptom was of seeing objects as abnormally coloured after prior exposure to a coloured stimulus. Formal testing disclosed that the patient experienced colour after-images of abnormal latency, duration, and amplitude.

The demonstration of prolonged colour after-images in a patient with a cortical disease process provides strong evidence that the generation of colour after-images is mediated at least in part by the visual cortex. A mechanism for the generation of colour after-images is proposed in which abnormal prolongation of the images results from excessive rebound inhibition of previously excited wavelength selective neurons in V1. This may occur as a consequence of the relative sparing of inhibitory interneurons in V1 in the context of the degeneration of excitatory neurons that occurs in Alzheimer's disease.

(*J Neurol Neurosurg Psychiatry* 2001;71:515-517)

Keywords: colour vision; visual cortex; Alzheimer's disease

Dementia Research Group, Department of Clinical Neurology, Institute of Neurology, Queen Square, London WC1N 3BG, UK

D Chan
S J Crutch
E K Warrington

Division of Neurosciences, Imperial College of Science Technology and Medicine, London, UK

D Chan
S J Crutch

Correspondence to:
Dr D Chan
d.chan@dementia.ion.ucl.ac.uk

Received 7 March 2001
Accepted 4 June 2001

Prolonged visual fixation on a coloured stimulus, followed by stimulus removal and fixation on a neutral surface, results in the experience of an after-image of a colour complementary to that of the preceding stimulus. Characteristically these after-images appear after a brief latency and fade away over several seconds. Colour after-images were traditionally thought to be of retinal origin,¹⁻⁴ although other psychophysical experiments indicate that their generation reflects a cortical process.^{5,6}

In this article we document an unusual disorder of colour vision in a patient with posterior cortical atrophy of presumed neurodegenerative aetiology. Her presenting symptoms were suggestive of abnormal colour after-images, and occurred in the context of subjectively preserved colour vision.

Case history

A 64 year old right handed woman began to experience blurring of vision and difficulty in

reading in 1992, with subsequent difficulty in locating objects in visual space. Neuropsychological testing showed visual disorientation, impaired visuospatial skills, and abnormal visual form and object perception. Structural MRI showed atrophy of the posterior cerebral cortex. The clinical picture was consistent with a diagnosis of posterior cortical atrophy, with Alzheimer's disease considered to be the probable underlying cause.

Recently the patient has reported a new problem. After laying out red bed sheets she noticed that her hands were coloured green. This colouration persisted for about 30 to 60 seconds, after which her hands gradually resumed their normal colour.

She has been reviewed regularly and there has been only slight deterioration in her visual functions. Visual acuity has remained normal. Recent visual field testing showed constricted visual fields (about 40° of central vision bilaterally). The visual disorientation has worsened and there has been a further impairment on tests of shape discrimination and visual perception.⁶

Quantitative analysis of two recent MR scans performed 1 year apart disclosed progressive cerebral atrophy which was largely restricted to the occipital pole (fig 1).

INVESTIGATIONS

Occipital pattern visual evoked potentials and pattern electroretinography were normal. Lightness discrimination was normal.

Colour vision was assessed by testing discrimination between pairs of adjacent Farnsworth chips. Ten pair discriminations were made for each major hue at three levels of increasing difficulty, represented by an increase in the difference in hue, value, and chroma between each test pair. A mild but definite impairment of colour vision was noted. Due to her impaired form perception, it was not possible to test her colour vision using Ishihara plates or the Farnsworth-Munsell 100 hue test.

Colour vision was assessed further using a test of chromatic sensitivity. The patient's chromatic sensitivity was reduced by a factor of about three in comparison with normal subjects.

Colour constancy was assessed using a test modified to accommodate her visual disorientation. A photograph was projected onto a

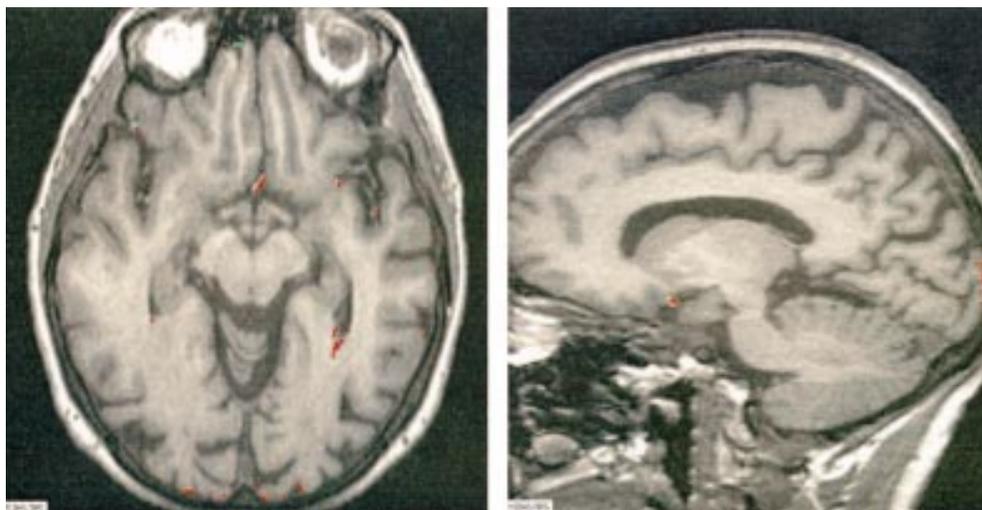


Figure 1 Serial MRI analysis: axial (left) and sagittal (right). Regions of cerebral volume loss are shown in red. Isolated patches of red seen at the posterior edge of the lateral ventricles and at the periphery of the brain (seen on axial section) represent partial volume effects and movement of blood vessels respectively and do not denote true volume loss.

screen using a slide projector modified to project light of different wavelength compositions. She was asked to determine the colour of an object in the photograph (placed next to a Mondrian-type array of coloured blocks) under different lighting conditions. She made no errors in determining the correct colour of the test object in each case, indicating that colour constancy was present.

Under white room lighting conditions, the patient was asked to fixate binocularly on various coloured A5 sized cards of equal luminance placed on a neutral light grey background. Red, blue, green, and yellow cards were presented for a duration of 5, 10, or 15 seconds. After presentation, the test card was removed and the patient was asked to fixate on the grey background and to describe the duration and colour of any after-images. Each colour was presented three times at each presentation duration.

In each case she correctly identified the colour of the test card. The results of after-image testing are summarised in figure 2. For comparison, the averaged results obtained from three age matched controls are provided.

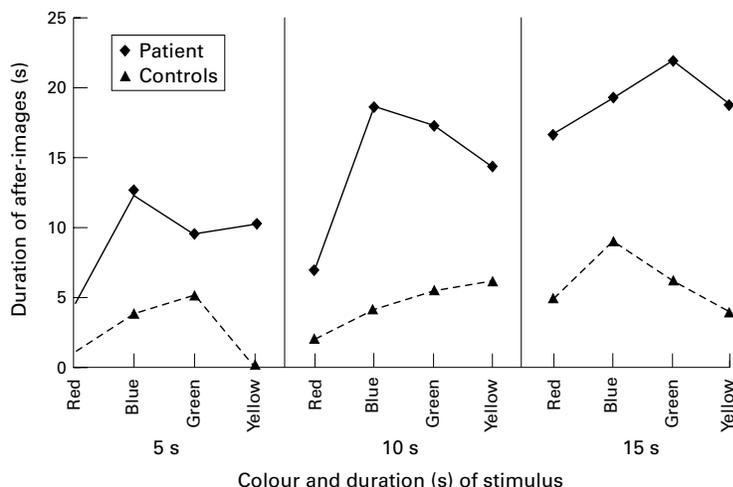


Figure 2 Mean duration of after-images in the patient and controls.

For each card the patient reported that the after-image hue was complementary to that of the preceding card (for example, green after red, yellow after blue), as did the controls.

The patient was then placed in a room illuminated by a light source adapted to emit light of different wavelength compositions (red, blue, green, yellow). After 60 seconds of exposure, the light source was switched off and normal (white) room light was resumed. In each test condition, she noted that the entire room was abnormally coloured, with the perceived hue of the room colour complementary to that of the preceding light. On each occasion, she stated that the room was uniformly coloured, irrespective of any colour contrasts in the room (for example, floor/wall, table/people).

This phenomenon was then tested under monocular viewing conditions: she was asked to close one eye while the room was illuminated. When the coloured light source was switched off and the room lights were reactivated, she was asked to cover up the exposed eye and to open the closed eye. In most instances she reported that the room was coloured cream or an indeterminate dark colour but on one occasion, after exposure to orange light, she reported that the room was coloured blue when viewed through the previously unexposed eye.

Discussion

We have described a patient with posterior cortical atrophy who presented with abnormal prolongation of colour after-effects. Prior exposure to light of a particular wavelength results in the perception that her visual environment is coloured in a hue complementary to that of the preceding light. This phenomenon has certain invariant characteristics: it is experienced immediately after a change in the wavelength compositions of illuminating light; the entire visual environment is affected; the environment is uniformly coloured, regardless of the different colour of the constituents or of any colour boundaries; the

experience is transitory; normal colour vision is restored thereafter.

The patient had abnormal colour after-images after prolonged fixation on coloured stimuli. Several aspects of her after-images were unusual. Firstly, there was no latency period between removal of a coloured stimulus and the appearance of the after-image: she invariably experienced after-images immediately after removal of the stimulus, whereas normal observers experience a latency of 1–2 seconds. Secondly, the after-images were always abnormally prolonged, with durations up to 400% of normal. Thirdly, the after-images were unusually powerful, and in fact were powerful enough to colour non-neutral objects upon which her gaze might be fixed (as exemplified by her observation that her hands looked green after laying out red bed sheets).

These findings indicate that colour after-image generation is at least partly mediated by the cerebral cortex. She has no history of retinal or ophthalmological disorders and her underlying disease is cortical (probable Alzheimer's disease). Furthermore, the demonstration of interocular transfer on one occasion is difficult to explain in terms of a mechanism at the level of the retina or lateral geniculate nucleus and instead is indicative of a cortical process. Finally, it is interesting to note that this patient's symptoms bear some similarity to those reported in other patients with cortical disorders.^{7,8}

Electrophysiological studies have detailed the properties of V1 neurons in the macaque monkey.^{9–12} Of particular interest in the present context are the wavelength selective neurons in V1, which respond only to light of a certain spectral composition. Many of these cells display chromatic opponency characteristics, in that they are excited by light of a certain wavelength and inhibited by light of complementary spectral composition (red/green, blue/yellow).

It is proposed that the properties of these wavelength selective cells in V1 may mediate the generation of colour after-images, according to the following mechanism. Presentation of a coloured stimulus illuminated by achromatic light results in excitation of the appropriate wavelength selective cells in V1. These neurons remain active for the duration of the coloured stimulus, with response adaptation if there is prolonged stimulus presentation. After stimulus removal, the adapted neurons become inactive and undergo postexcitation rebound inhibition. Upon fixation on a neutral surface, illuminated as before by achromatic light, there is activation of the previously inactive (and therefore unadapted) wavelength selective cells in V1. This results in the perception of an after-image of a colour complementary to that of the previously presented stimulus. The generation of colour after-images is thus considered to be modulated by the rebound inhibition of adapted wavelength selective cells in V1.

Given that the patient could name correctly the colours of presented stimuli, and has partial preservation of colour vision, it is likely that the

excitation responses of wavelength selective cells are at least partially intact. In addition, the fact that she perceives the appropriate colour of after-images indicates that the basic mechanism for generation of the complementary after-image colour is intact. However, the other characteristics of the abnormal colour after-images suggest that there is a defect in the nature of the post-excitation response. In view of the fact that Alzheimer's disease affecting the visual cortex results in degeneration of projection neurons with relative sparing of interneurons,¹³ it is tempting to speculate that this disorder reflects the relative overactivity of preserved local circuit inhibitory interneurons. Excessive post-excitation inhibition, causing a greater degree and duration of response inhibition, would then result in colour after-images of abnormal amplitude and duration.

Posterior cortical atrophy represents an atypical variant of Alzheimer's disease which usually presents in the form of parietal lobe syndromes and disorders of higher visual processing.¹⁴ Involvement of the primary visual cortex is rare. The patient described here presents with atrophy of V1 which is confirmed on MRI and dysfunction of the visual cortex which manifests as a disorder of colour after-image generation. This disorder is considered to reflect excessive inhibition of the wavelength selective neurons, and these data provide evidence for a cortical role in the generation of colour after-images.

We are particularly grateful to Professor John Barbur for his assistance with the tests of chromatic sensitivity and colour constancy. We also thank Drs Neil Burgess, Jules Davidoff, and Peter Rudge for their opinions, and Professor Martin Rössor for his support.

- 1 Craik F. Origin of visual after images. *Nature* 1940;145:512.
- 2 Brindley GS. Two new properties of foveal after images and a photochemical hypothesis to explain them. *J Physiol* 1962;164:168–79.
- 3 Poppel E. Long-range colour-generating interactions across the retina. *Nature* 1986;320:523–5.
- 4 Barbur JL, Weiskrantz L, Harlow JA. The unseen color after effect of an unseen stimulus: insight from blindsight into mechanisms of color afterimages. *Proc Natl Acad Sci USA* 1999;96:11637–41.
- 5 Loomis JM. The photopigment bleaching hypothesis of complementary after images: a psychophysical test. *Vision Res* 1972;12:1587–94.
- 6 Zeki S. Colour coding in the cerebral cortex: the reaction of cells in monkey visual cortex to wavelengths and colours. *Neuroscience* 1983;9:741–65.
- 7 Kinsbourne M, Warrington EK. A study of visual perseveration. *J Neurol Neurosurg Psychiatry* 1963;26:468–75.
- 8 Blythe IM, Bromley JM, Ruddock KH, et al. A study of systematic visual perseveration involving central mechanisms. *Brain* 1986;109:661–75.
- 9 Zeki S. Colour coding in the cerebral cortex: the responses of wavelength selective and colour coded cells in monkey visual cortex to changes in wavelength composition. *Neuroscience* 1983;9:767–81.
- 10 Livingstone MS, Hubel DH. Anatomy and physiology of a color system in the primate visual cortex. *J Neurosci* 1984;4:309–56.
- 11 Michael CR. Color vision mechanisms in monkey striate cortex: simple cells with dual opponent color receptive fields. *J Neurophysiol* 1978;41:1233–49.
- 12 Ts'o DY, Gilbert CD. The organization of chromatic and spatial interactions in the primate striate cortex. *J Neurosci* 1988;8:1712–27.
- 13 Hof PR, Morrison JH. Quantitative analysis of a vulnerable subset of pyramidal neurons in Alzheimer's disease: II. Primary and secondary visual cortex. *J Comp Neurol* 1990;301:55–64.
- 14 Berthier ML, Leiguarda R, Starkstein SE, et al. Alzheimer's disease in a patient with posterior cortical atrophy. *J Neurol Neurosurg Psychiatry* 1991;54:1110–11.