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Short-term memory for colour following posterior hemispheric lesions in man

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SHORT-TERM memory for colour was studied in five patients with circumscribed posterior hemispheric lesions. It was impaired independently of colour discrimination in one and more than colour discrimination in two patients. Two patients were normal in colour short-term memory, one with normal and one with deficient colour discrimination performance. Deficient performance in colour short-term memory was associated with bilateral lesions of the inferior occipito-temporal junction including the lateral part of the fusiform gyrus or with a unilateral lesion of the left parieto-occipital convexity. An additional colour constancy deficit was found in the former but not the latter condition. Thus, colour short-term memory can be affected independently of colour discrimination or colour constancy, and may depend on at least two distinct neural circuits. *NeuroReport* 10:1379–1384 © 1999 Lippincott Williams & Wilkins.

Key words: Adult plasticity; Colour discrimination; Colour memory; Colour naming; Extrastriate visual cortex; Human

Introduction

Several lines of evidence indicate that the lingual and fusiform gyri contribute to colour perception in man. Lesions that include this region were reported to cause achromatopsia and/or colour constancy deficits [1–7], and activation foci to colour tasks were revealed within this region [8–11]. However, it remains unclear how this region contributes to different aspects of colour perception and whether damage restricted to this region and not involving large parts of the surrounding cortex and the white matter is necessary, or sufficient, for colour perception deficits to occur. Other regions such as the occipito-parietal convexity [11–13] may also play an important role. Comparisons of performance in psychophysical tests of achromatopsic patients and macaque monkeys with selective cortical ablations suggest that regions anterior to the human 'colour area' may also have been damaged in achromatopsia [14].

The anatomical substrate of colour memory remains relatively little understood. Roland and Gulyas [15] studied changes in regional cerebral blood flow while subjects encoded, recognized or recalled complex coloured patterns; the lingual and fusiform gyri were activated during encoding and recognition, but not during imagined recall. Davidoff and Ostergaard [16] reported a patient who had impaired short-term memory for colour and colour anomia 2 years after a lesion in the left occipito-temporal cortex, but the lesion extended over large parts of the medial and lateral surfaces.

We report here on colour discrimination, colour short-term memory and colour naming in patients with circumscribed lesions corresponding to some of the areas shown to be involved in colour perception in normal subjects. Preliminary results have been reported in abstract form [17].

Subjects and Methods

Five patients with circumscribed posterior hemispheric infarctions were selected from outpatients of the Neuropsychology Division in Lausanne. The selection criteria were: (1) a completed magnetic resonance imaging (MRI) scan during the present illness showing that the lesion was in the vicinity of regions associated with colour perception; (2) absence of pronounced behavioural or psychiatric problems; (3) absence of retinal chromatic deficits; (4) normal or corrected-to-normal visual acuity; (5) normal auditory–verbal and visual–spatial spans (see Table 1). Lesions were reconstructed from the MRI scans of the patients' brains and are shown in Fig. 1. Peripheral colour perception deficits were excluded in patients and normal subjects. Cataract, glaucoma and macular degeneration were excluded by ophthalmologic examinations. Medication and

Table 1. Patients who participated in this study

	Sex	Age at onset (years)	Lesion	Delay lesion–testing	Visual field	Colour discrimination	Colour memory index	Colour naming	Auditory-verbal span (Hebb)	Visuo-spatial span (Corsi)	Boston naming
GB	F	50	Bilateral inferior temporal hematoma	11 years	Intact[a,b]	N[b]	N[b]	N[b]	6 (C50)[b]	5 (C50)[b]	MD[b]
GP	M	65	Bilateral occipito-temporal infarctions	6 months	L inferior quadrantanopia[a,b]	N[b]	D[b]	D[a], D[b]	5 (C50)[b]	4 (LI–C10)[b]	SD[b]
JD	M	56	Multiple bilateral infarctions	5 months	L superior quadrantanopia[a]	D[b]	N[b]	N[b]	7 (C75)[b]	5 (C50)[b]	C10–C50[a]
JA	M	68	L parieto-occipital hematoma	7 months	Intact[a]	D[b]	D[b]	N[b]	7 (C75)[a]	6 (C75)[a]	C10–C50[a]
MG	F	73	Bilateral inferior occipito-temporal infarctions	13 months	Mosaic quadranopsia[b]	D[b]	D[b]	N[b]	5 (C50)[a]	5 (C50)[a]	C50[a]

All patients were right-handed. Visual fields were defined by dynamic perimetry. Performance is designated as normal when it is $\geq$2.5th centile of the performance of the normal population. [a] In acute stage; [b] in chronic stage; Cxx = centiles of normal performance; D, deficient; F, female; L, left; LI, at lower limit of normal performance; M, male; MD, moderately deficient; N, normal; R, right; SD, severely deficient.

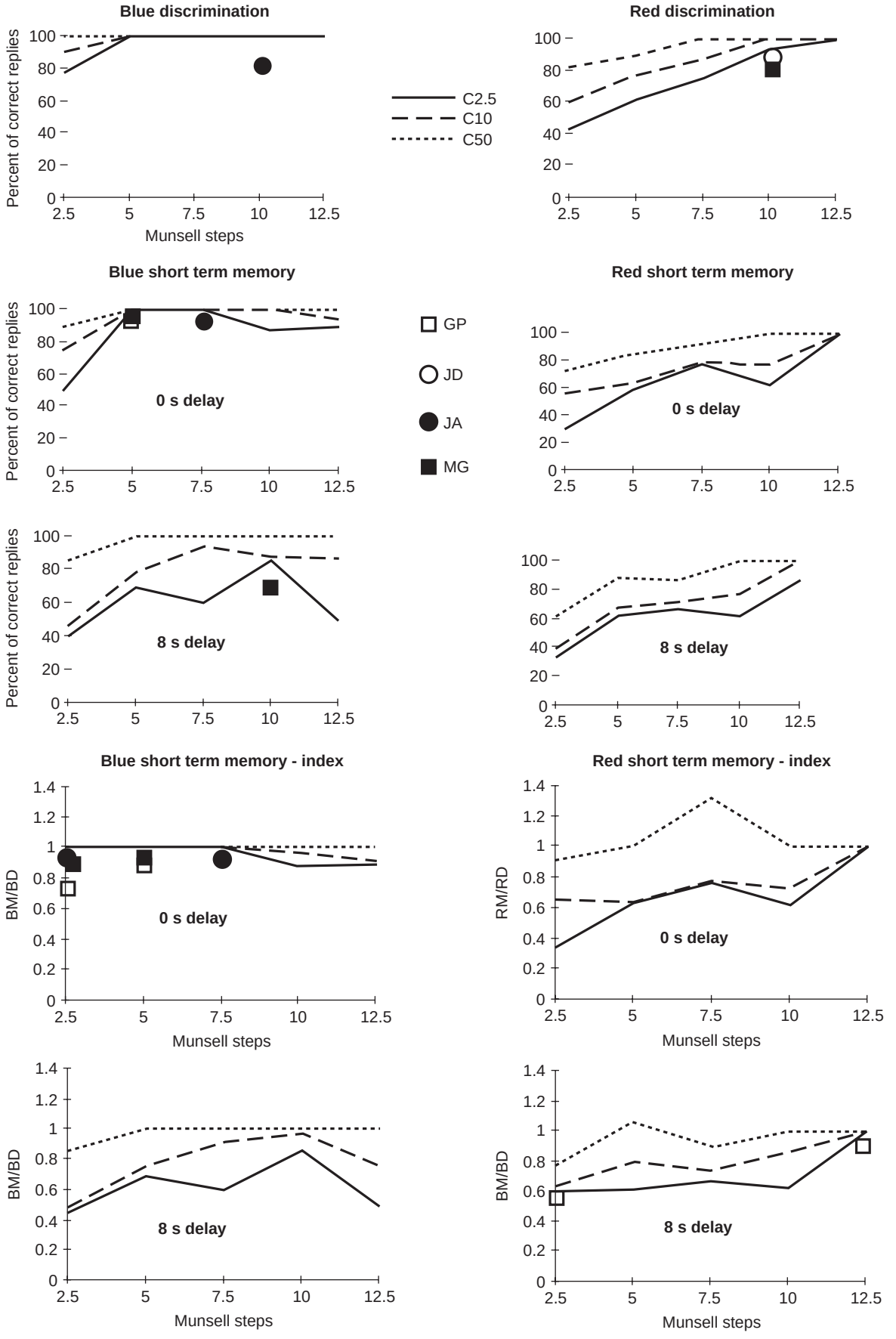
FIG. 1. Sites of lesions in as visible in MRI. The normalized coordinate system introduced by Talairach and Tournoux [23] was used to compare lesions in different patients. This system uses a proportional grid and a coordinate system anchored in the forebrain commissures. Three planes are placed within the brain, corresponding to a horizontal plane through the anterior and posterior commissures and two coronal planes through the anterior and posterior commissures respectively. The hemispheres are subdivided into 12 horizontal (four below and eight above the bicommissural plane), 11 coronal (four anterior to the anterior commissure, three between the anterior and posterior commissures, four posterior to the posterior commissure), and eight sagittal slices (four for each hemisphere). Centres of foci selectively activated by colour tasks are indicated.

chronic alcohol intake were assessed anamnestically (control subjects) or by consulting the medical record (patients).

All patients had a clinically oriented neuropsychological assessment during the acute and chronic stages and were examined with psychophysical tests in the chronic stage (defined $>$2 months since the occurrence of the lesion; see Table 1).

Twenty-one normal subjects between the ages of 50 and 75 years and 31 subjects between the ages of 20 and 35 years served as controls. We used the SAS System as adapted in JMP 2.0.5 to analyse the scores of the normal population and to investigate the effect of age on normal performance. The Shapiro-Wilk W test was used to evaluate the normality of the distribution. None of the relevant distributions

FIG. 2. Deficient performance of patients with chronic circumscribed posterior lesions in colour discrimination (top), colour short-term memory (middle) and colour short-term memory relative to colour discrimination (memory index; bottom) compared with the performance of the normal population (expressed in centiles). Testing in the blue region of colour space is summarized on the left, that in the red region on the right. All patients listed in Table 1 were tested for blue and red discrimination and for blue and red short-term memory with 8 s delays. All but patient 2 were tested for blue and red short-term memory with 0 s delays. Note that none of the patients performed at chance level in colour discrimination or short-term memory.


Blue discrimination
Red discrimination
Percent of correct replies
Munsell steps
C2.5
C10
C50
Blue short term memory
Red short term memory
0 s delay
GP
JD
JA
MG
8 s delay
Blue short term memory - index
Red short term memory - index
BM/BD
RM/RD

were normal, so we used the non-parametric Wilcoxon test. No statistically significant differences were observed between the performance of the younger and older groups. Centile reports for individual scores (Fig. 2) served for assessing the performance of brain-damaged patients. The lower limit of normal performance was fixed at centile 2.5. The consent of all subjects, brain-damaged patients and healthy controls, was obtained according to the declaration of Helsinki and the project was approved by the local ethical committee.

The colour stimuli used to test colour discrimination and memory were simulated Munsell chips from two regions of colour space. One set of stimuli were 2.5R, 5R, 7.5R, 10R, 2.5YR, 5YR and 7.5YR; colours in this set would be perceived as red, orange or yellow. The second set of stimuli were 2.5PB, 5PB, 7.5PB, 10PB, 2.5P, 5P, 7.5P and 10P; these would be perceived as a range of blue, violet and pink. All stimuli were simulated with a Munsell Value of 5 and Chroma of 10 and were presented at 6 cd m^{-2}. This particular set of stimuli was chosen because they are evenly spaced within the colour domains explored by our tasks and were within the gamut of the monitor phosphors. The monitor was calibrated with a spot photometer (PR 1500). Munsell stimuli were simulated using the method described by Lucassen and Walraven [18] (their appendix B). Knowing the CIE (x, y) chromaticity co-ordinates [19] and the chosen luminances (Y) of the stimuli, tristimulus values are calculated as $X=(x/y)Y$, $Y=Y$, $Z=(z/y)Y$ where $z=1-x-y$. From these values the luminance value required for each phosphor (Y_r, Y_g, Y_b) is calculated using

$$\begin{pmatrix} x \\ y \\ z \end{pmatrix} = \begin{pmatrix} x_r/y_r & x_g/y_g & x_b/y_b \\ 1 & 1 & 1 \\ z_r/y_r & z_g/y_g & z_b/y_b \end{pmatrix} \begin{pmatrix} y_r \\ y_g \\ y_b \end{pmatrix}$$

Testing was carried out on a PC-controlled touchscreen monitor at which the patients sat within comfortable arm's reach (approximately 57 cm). The subjects and patients were mesopically adapted while doing the discrimination and memory test. Unlimited viewing time was allowed and no fixation point was required. The whole discrimination test took typically 10 min, and the memory test 20–25 min. A trial began with the presentation of a coloured square (the sample), subtending ~4° of visual angle, with its lower edge two degrees above the centre of the screen. In the discrimination task, touching the sample was followed by immediate presentation of two squares while the sample remained on the screen. In the memory task the sample disappeared when touched and the two match stimuli appeared after a delay of either 0 or 8 s. The match stimuli always differed from each other but one of them was always identical with the sample. In all conditions the task of the subject was to touch the stimulus which was the same hue as the sample. Matches differed by a maximum of 12.5 and a minimum of 2.5 hue steps. Subjects received an average of ten trials on each hue step in each condition. Feedback on performance was not given during the test session.

In a pilot study (not described here) we conducted the memory tests with delays of 0, 4, 8, and 12 s in normal subjects. No decrease in performance was observed between 0 and 4 s, with a decrease for small hue differences with 8 s. The decrease was more pronounced for 12 s and made the memory task for red stimuli too poor to be a measure of deficient performance. The use of a 12 s delay made the test too long (35–45 min) for many of the brain-damaged subjects.

Results

The performance of each patient in the colour discrimination task was compared with the performance of the control population. One patient (JA) was deficient in discrimination even at 10 hue steps in the blue region of colour space (Fig. 2 top). Two patients (JD, MG) also showed discrimination deficits in the red region of space at 10 hue steps.

The performance of each patient in colour short-term memory was compared with that of controls (Fig. 2 middle). In the blue region of colour space, three patients (GP, JA, MG) were deficient at 5 or 7.5 hue steps with 0 s delay, and one patient (MG) at 10 hue steps with 8 s delay. None of the patients was deficient in the red region of colour space. To obtain a measure of colour short-term memory performance relative to colour discrimination, a short-term memory index was calculated by dividing the percentage of correct replies for a given hue step in the memory task by the percentage of correct replies for the same hue step in the corresponding discrimination task. Several of the patients produced memory index values below that of normal subjects (Fig. 2 bottom). At 0 s delay, in the blue region of colour space, two patients (GP, MG), whose lesions included bilaterally the 'classical' colour area [1,3,8], were deficient at 2.5 and 5 hue steps. Patient JA, with a lesion restricted to the left occipito-parietal convexity, including regions activated by colour attention and discrimination tasks in normal subjects [11,12], was impaired at 2.5 and 7.5 hue steps. In the red region of colour space, only one patient (GP) showed a deficit in memory at 2.5 and 12.5 hue steps with 8 s delay. This relative lack of deficits in the red region of space could be an example of floor effects (the control population performed less well

with the red stimuli than with the blue stimuli) or to the not uncommon asymmetries of performance in different regions of colour space following brain lesions [5].

Colour naming, was tested by asking the patient to name 12 clearly defined colours ('typical' red, blue, green yellow, brown, orange, yellow, violet, pink, black, grey, turquoise) and was deficient in only one patient (GP). He was able to name only six colours (against 10–12 in normal subjects), despite having premorbid experience with colours (he was a house painter). He was equally deficient in object naming (assessed with shortened French version of the Boston naming test), and his colour naming deficit was thus part of a more general naming deficit. The reverse pattern, namely impaired object naming with selectively preserved colour naming was observed in patient GB.

Discussion

The results demonstrate three features of cortical colour processing. First, short-term memory deficits for colour can occur in the absence of deficits in colour discrimination (GP) or impairments in verbal or visuo-spatial memory (GP, JA, MG). Second, colour short-term memory deficits can occur in the absence of colour naming deficits (JA, MG). The converse, however, appears not to be true and our patients who had naming deficits also had memory impairments, similar to that reported in a previous single case study [16]. Third, there are at least three regions of the human extrastriate visual cortex that contribute to colour short-term memory.

In activation studies, three regions of the cortex have been identified as important for colour processing: (i) left inferior occipito-temporal junction [8,9,11–13,20]; (ii) right inferior occipito-temporal junction [8,9,11–13,20]; and (iii) left occipito-parietal convexity [11–13,20]. The left hemisphere seems to be more active irrespective of whether the viewing is passive or active. As is clear from Fig. 1, four of our patients had lesions coincident with regions activated in studies of colour perception. The lesions in patients GP, JD and MG involved bilateral damage to the classical colour area and two of these patients (GP, MG) were achromatopsic in the first month following the infarction, but both of them recovered much of their colour vision. Patient JD never experienced achromatopsia and the only evidence of colour-related deficits was in this patient's colour discrimination abilities. Thus, bilateral lesions of a region selectively activated by viewing coloured displays [8,9] produced a temporary achromatopsia followed by long-lasting discrete colour discrimination deficits. Unilateral left sided damage to the dorsolateral occipito-parietal focus of activation found in imaging studies of colour perception in normal subjects [11,12] did not produce achromatopsia, but the patient was worse than controls in colour discrimination. When lesions were outside the inferior occipito-temporal and occipito-parietal foci (case GB) or touched them only marginally (JD), colour short-term memory was within normal limits.

Colour short-term memory was impaired, albeit only slightly, following damage to either the lingual-fusiform areas or the occipito-parietal convexity. And in normal subjects, the lingual-fusiform region was previously shown to contain activation foci in a delayed match-to-sample colour task [21]. In a preceeding activation study of memory for complex colour patterns [15] both foci were activated during recognition but not recall.

We showed in a previous study [7] that colour constancy was impaired following bilateral lesions of the inferior occipito-temporal junction (patients GP, MG), but not after a lesion of left occipito-parietal convexity (patient JA). Thus, a close link between colour constancy and colour memory, recently demonstrated psychophysically [22], appears to be true for some, but not all colour processing areas.

Conclusion

Small lesions of the fusiform and lingual gyri cause temporary achromatopsia followed by long-lasting discrete colour discrimination deficits and more pronounced colour short-term memory deficits. Such long-lasting deficits are, however, not only caused by lesions of the fusiform and lingual gyri but also by damage to the left occipital convexity.

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