

## Hemianopic colour blindness

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**SYNOPSIS** A man developed cortical blindness after cerebral infarction in the distribution of both posterior cerebral arteries. When he recovered from this condition, he was found to be colour blind in the left visual field, but not in the right. This unusual situation resulted in apparently contradictory performances on hemifield and free-field tasks of colour discrimination, naming, and recognition. The contradictions may be explained by interhemispheric competition between a hemisphere which could discriminate colours and a hemisphere which was colour blind.

In 1965 Critchley stated, 'No case has been described where a patient with intact visual fields has shown a colour defect of hemianopic distribution'. In the course of evaluating a patient with a cerebral infarction in the distribution of the posterior cerebral arteries, we discovered that he had such a colour defect, and we believed that this unusual observation warranted being placed on record.

### CASE REPORT

This man had an unusual cluster of neuropsychological symptoms including associative visual agnosia without alexia, and he has been described in detail elsewhere (Albert *et al.*, 1975). The current report will concentrate on the disorder of colour vision, which has not previously been described.

The patient was a 59 year old, right-handed, university professor who was seen by us for evaluation of a brief episode of loss of consciousness followed by visual and memory defects. Twenty-five years previously, the patient had been wounded in his right eye, which was subsequently enucleated. Four years before the current admission he suddenly developed a mild left hemiparesis which improved within a few days. During the three to four years before the present admission he suffered from attacks of confusion and disorientation, each lasting a few hours. He was always able to return to his normal activities shortly after an attack.

General physical examination on the present admission was within normal limits except for a prosthesis in his right eye. Neurological examination revealed gross visual defects (see below), mild paresis of the lower half of the face on the left, very mild paresis of the left leg, and hyperreflexia in the left limbs with a Babinski sign present on the left. Several hours after he had regained consciousness, he was awake, alert, attentive, and socially appropriate. His immediate recall was normal (digit span seven forward); he had severe impairment of recent memory; remote memory was mildly impaired. He had no aphasia. He could carry out complex arithmetical calculation mentally; and he had no difficulty in manipulating abstract mental concepts.

His visual disorders passed rapidly through several stages to a final point of stability. The early evaluation of his visual defects showed the following progression. Immediately after his period of unconsciousness he provided no evidence of being able to see anything, although he denied being blind. Two days after the onset of his illness he was able to distinguish light from dark and he was able to follow a moving object with his eye. He was unable to perform any other visual discriminations. Seven days after the event he was able to read letters, numbers, and some words. He could not name colours; however, he could distinguish lighter from darker shades of the same hue. He could neither name nor recognize objects presented visually, although he could describe or draw the features of the objects. On the eighth day his reading ability was normal; his visual acuity with his usual correction was 6/7.5 (Jaeger); and Goldmann perimetry

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revealed an upper quadrant defect in the right visual field of his left eye. From this point in time his visual defects stabilized.

Radioactive gamma encephalography performed 15 days after the onset of the illness revealed a mildly-to-moderately increased uptake of radioactive material in both occipital regions, posteriorly and inferiorly. A second brain scan nine days later showed complete clearing of the abnormality on the left, with fading of the abnormality on the right. (These results should be compared with the results of visual evoked potentials; see Table.) Standard electroencephalography, repeated several times during his hospital stay, showed persistent slowing with occasional sharp activity in both occipital regions.

**VISUAL EVOKED POTENTIALS** Visually evoked potentials, obtained from occipital leads, using standardized procedures of the Vision Research Laboratory, were recorded from each hemisphere on the 19th and 21st days after the onset of the illness. Results are shown in the Table.

TABLE  
VISUAL EVOKED RESPONSES

Days after ictus	Wave	Left hemisphere		Right hemisphere	
		Amplitude ( $\mu V$ )	Latency (ms)	Amplitude ( $\mu V$ )	Latency (ms)
19	Initial positivity	< 1	60	1	20
	Primary	< 1	80	4	45
	Secondary	1	150	5	80
	Tertiary	3.5	240	10	245
21	Initial positivity	< 1	25	< 1	30
	Primary	1	50	4	50
	Secondary	5	90	5.5	85
	Tertiary	7.5	260	10	250

Analysis of the evoked potentials demonstrates a clear improvement of the recordings from his left hemisphere. The impulses reached the visual cortex earlier by far on the second set of recordings than they did on the first. Little change was noted in the records measured above the right hemisphere.

**TESTS OF COLOUR DISCRIMINATION, NAMING, AND RECOGNITION** 1. *Ishihara test of colour discrimination* On the ninth day after the event, he was unable to read any of the numbers or trace any of the lines, a performance consistent with trichromatic

colour blindness. Eight days later he continued with total failure.

2. *Farnsworth—15 hue test* On this test on the ninth day after the event, his performance was totally impaired, the colour discs being placed randomly. Eight days later his performance had markedly improved; he made a single error only.

3. *Weigl-Goldstein-Scherer colour-form sorting test* On this test, administered 17 days after the event, the patient's performance was normal. There was no difficulty in making the transition from forms to colour; no pattern formation was observed; and definitions were given on the abstract level.

4. *Evocation of colour names from memory* On the ninth day after the onset of his illness he was 80% accurate in providing colour names from memory in response to such questions as, 'What is the colour of a tomato?' His correct responses were not precise—for example, he would say 'reddish' instead of 'red'. On the 15th day his performance was normal.

5. *Colour naming to confrontation* When stimuli were presented free-field with no time limit, he was correct 80% of the time. His errors were on pastel shades rather than on primary colours.

6. *Colour naming to confrontation—hemifield presentation* Following the technique for hemifield presentation described previously (Albert *et al.*, 1975), we presented coloured slips of paper individually to each hemifield separately. He was totally unable to name colours presented to his left visual field; he was 90% correct in naming colours presented to his right visual field.

7. *Colour matching* Coloured slips of paper were placed before him, free-field. His task was to select all the slips of one colour from a mixed group of coloured slips. He succeeded in this task. However, he took 26 to 48 seconds to take out five slips of the same colour. Normal subjects could perform the same task in three to five seconds.

8. *Colour matching—hemifield presentation* The contradictions noted in the responses on the several tests above prompted us to devise a colour matching test for hemifield presentation. A single coloured slip of paper was presented to one hemifield as the target item. This was followed immediately by the serial hemifield presentation of five coloured slips of paper, including one of the same colour as the target. The task was to indicate, without

naming, which slip matched the target. Ten trials were presented to each hemifield. When stimuli were presented to the left visual field, the patient failed completely. When stimuli were presented to the right visual field, the patient always succeeded.

*9. Object matching—hemifield presentation* This test served as a control for the hemifield colour matching test described above. Single objects were presented to one hemifield as target items and not verbally identified. The target stimuli were immediately followed by four additional objects, including the target. The task was to match the target with itself. With stimuli presented to either field, responses were 100% correct. However, reaction times were greater for stimuli presented to the left field.

#### DISCUSSION

This patient was unable to match colours presented to his left hemifield, but was able to match colours presented to his right hemifield. By contrast, he was equally capable of matching objects presented separately to either visual field. He was totally colour blind, but only in his left visual field. Colour vision in his right visual field was normal. This disorder of colour vision seems all the more striking when we consider that he had no visual field defect on the left; but he did have a partial, superior quadrantanopia on the right.

Colour blindness is demonstrated by impaired performance on tests of colour discrimination and colour matching. Our patient failed the Ishihara test on two occasions and failed, but then succeeded, with the Farnsworth test. With free-field presentation of a colour matching test he was successful, but only with an abnormally long reaction time. His performance on the colour-form sorting test was normal. We believe that interhemispheric competition between a hemisphere which could discriminate colours and a hemisphere which was colour blind resulted in these apparently contradictory findings.

The defect reported here was not colour agnosia, which is the inability to name a colour in the presence of normal colour discrimination. Our patient had normal colour discrimination only in the right visual field, but not in the left. The defect was not amnesic colour anomia (Goldstein, 1948), which is the inability to remember colour names. Our patient could

name colours when they were presented free-field or to his right visual field. The defect was not a visual-verbal disconnection syndrome for colours (Geschwind, 1965), in which colours presented to the right hemisphere cannot be named because stimuli reaching the right hemisphere are anatomically separated from the language zone of the left hemisphere by a lesion in the corpus callosum. It is correct that our patient could not name colours presented to his left visual field (right hemisphere); however, he could not match them either. Although he could not name objects presented to his left visual field, he could match them.

Immediately after recovery of consciousness our patient had cortical blindness. Ordinarily, in recovering from cortical blindness, a patient passes through several stages; first is the discrimination of light from dark; then comes perception of movement; then there is the slow return of form discrimination, separation of figure from background. Accompanying the return of form discrimination is the slow return of colour discrimination. From greyness, first reds appear; then greens, then blues. Our patient followed this pattern, with the exception that, although form discrimination returned to both hemispheres, colour discrimination returned only to a single hemisphere.

Reinhard (1887) reported a case which bears some resemblance to ours. The patient had a visual field defect in the right visual field, but not in the left. In the left visual field, objects and colours were poorly discriminated, colours appearing 'greyish'. We cannot be certain, however, that colour vision was preserved in the right visual field, since this detail is lacking in the original report and since Reinhard's patient subsequently developed a complete right homonymous hemianopia.

Neurophysiological studies with evoked potentials and single cell analyses have demonstrated evidence of colour responsive cells beyond the retina. A complete colour vision system may be represented in the two dorsal layers of the lateral geniculate nucleus of the rhesus monkey (De Valois *et al.*, 1958); and the lateral geniculate nucleus of the macaque has a mechanism for the discrimination of colour purity (De Valois and Marrocco, 1973). Wiesel and Hubel (1966) demonstrated that more than 75% of cells in the

lateral geniculate body of the rhesus monkey exhibit colour and spatial specificity. Even beyond the lateral geniculate body, colour specific cells have been found. Gouras (1970) described trichromatic mechanisms in single neurones of the foveal projection region of rhesus monkey striate cortex. Dow and Gouras (1973) found a large number (20% of the units recorded) of colour opponent cells in the foveal striate cortex of the rhesus monkey. In humans the possibility that different colour stimuli may excite different cortical regions has been raised (Regan, 1973). The current study extends the results of these previous studies to man. Additionally in man, however, the factor of cerebral dominance also seems to play a role.

Certain speculations regarding cerebral dominance may be supported by the observations in this case. De Renzi *et al.* (1969) have suggested a differential specialization of the two hemispheres for visual recognition—the right hemisphere being more involved in the process of discrimination and comparison of sensory data; the left hemisphere being more involved in associative and cognitive functions. In our patient, the contradictory findings could, perhaps, be explained on the basis of this speculative hypothesis. When colour stimuli were presented to the right visual field, he could both name the colours and match them without naming. With stimuli presented to his left visual field, he could neither name the colours nor match them, although he could match objects. With stimuli presented free-field, he failed the tests requiring colour discrimination as the main component—for example, Ishihara test—but was successful for colour tests involving a certain degree of abstract reasoning ability—for example,

the colour-form sorting test. His right hemisphere may be dominant for the task of colour discrimination and, being colour blind, could not perform normally. His left hemisphere may be dominant for the colour task involving abstract reasoning and, since his left hemisphere was not colour blind and his verbal reasoning capacities were intact, he was successful.

Electrophysiological studies were conducted in the Vision Research Laboratory of Hadassah University Hospital (Professor E. Auerbach, Director).

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