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Cortical Color Blindness is Not ''Blindsight for Color''

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Cortical color blindness, or cerebral achromatopsia, has been likened by some authors to ''blindsight'' for color or an instance of ''covert'' processing of color. Recently, it has been shown that, although such patients are unable to identify or discriminate hue differences, they nevertheless show a striking ability to process wavelength differences, which can result in preserved sensitivity to chromatic contrast and motion in equiluminant displays. Moreover, visually evoked cortical potentials can still be elicited in response to chromatic stimuli. We suggest that these demonstrations reveal intact residual processes rather than the operation of covert processes, where proficient performance is accompanied by a denial of phenomenal awareness. We sought evidence for such covert processes by conducting appropriate tests on achromatopsic subject M.S. An ''indirect'' test entailing measurement of reaction times for letter identification failed to reveal covert color processes. In contrast, in a forced choice oddity task for color, M.S. was unable to verbally indicate the position of the different color, but was surprisingly adept at making an appropriate eye movement to its location. This ''direct'' test thus revealed the possible covert use of chromatic differences.

INTRODUCTION

Covert visual processes are revealed when an observer fails to acknowledge, and indeed denies, awareness of the product of such perceptual processes, which nevertheless yield, or influence, a behavioral response. The phenomenon has been extensively studied in people with brain damage, most notably in cases of blindsight and prosopagnosia. In blindsight, residual discriminative ability can be demonstrated in clinically blind field defects caused by destruction, or deafferentation, of primary visual cortex. Denial of awareness can be accompanied by excellent performance in forced-choice tasks requiring detection, localization, and discrimination of visual stimuli (for reviews, see Weiskrantz, 1986, 1996, 1997). In prosopagnosia, patients are unable to recognize familiar faces yet, when tested appropriately, can ascribe categorical information and show discrimination of familiar from unfamiliar faces, while denying the conscious experience of familiarity (for reviews, see Bruyer, 1991; Young & De Haan, 1992).

Recently, the complete loss of color vision in people following ventromedial occipito-temporal brain damage, called cortical color blindness or *cerebral achromatopsia*, has attracted a degree of attention which contrasts with the comparative rarity of the condition. Doubtlessly, this reflects the potential light it casts on an understanding of the organization of the visual pathways of the brain and, specifically, the role of

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the cluster of visual areas that occupy close to a third of macaque visual cortex (see Zeki, 1990; Cowey, 1994; Cowey & Heywood, 1997, for reviews). But the loss of the phenomenal experience of hue does not, apparently paradoxically, prevent such patients from responding to other visual attributes of the visual scene that are derived from variations in color (more strictly, wavelength). This catalogue of preserved abilities has tempted some authors to refer to them as instances of covert processing or even ''color blindsight.'' Here, we examine what would constitute evidence for such a claim and report the results of testing achromatopsic patient M.S. on a variety of tasks designed to expose the presence of covert processes with respect to color.

OVERT AND COVERT PROCESSES

Covert visual processes require that the presentation of a visual stimulus elicits a response that depends on the very property of the stimulus of which the observer denies conscious, or phenomenal, awareness. The ascription of awareness ordinarily relies on verbal reports, or confidence ratings, in making a perceptual judgment. Low confidence ratings indicate lack of awareness and, normally, there is no correspondence between ratings and performance. Good performance accompanying consistently low confidence ratings in a forced-choice task is the hallmark of covert processing. Such tests are ''direct,'' in that patients are asked to guess about the nature of an unseen visual stimulus, or direct their eyes or hand to its location. In contrast, ''indirect'' tasks do not require patients to respond purposefully on the basis of the properties of the visual stimulus; rather, the stimulus is incidentally introduced into a task which ostensibly probes another ability. For example, when blindsight patients are required to make a manual response to the presentation of a light in an intact hemifield, a second light introduced simultaneously or slightly earlier into the ''blind'' field will affect reaction times (Marzi, Tassinari, Aglioti, & Lutzemberger, 1986). Success in demonstrating covert processes indirectly is not a guarantee of success in direct tests. For example, prosopagnosic patients can fail to discriminate whether a face is familiar, or which of two faces is similar, in forced-choice guessing (a direct test) but be faster at making same/different judgments of pairs of familiar than unfamiliar faces (an indirect test) (Young & De Haan, 1992). Moreover, prosopagnosic patients do not characteristically pass all varieties of either test. There is thus no correspondence between direct and indirect tests, on the one hand, and overt and covert processing, on the other.

The essence of a ''covert'' process, apart from denial of conscious experience, is that it exerts an influence on behavior and, hence, affords a measurable outcome. In blindsight, this may include a visuomotor act, such as reaching, grasping, or pointing, or a verbal or manual discriminative response in a forced-choice task. It should be noted that there is a further difficulty in assigning the term ''covert'' to a particular ability since the response mode can influence ''perceptual'' judgments (Bisiach, Vallar, & Geminiani, 1989). A further category of responses has fallen under the umbrella of direct tests, namely, autonomic responses and the consequences of central processing, e.g., visual evoked cortical potentials (VECPs). For example, Bauer (1984) paired five names with five photographs of familiar faces, only one of which was correct. Presentation of the photographs to a prosopagnosic patient resulted in

a maximal galvanic skin response (GSR) to the correct pairing on 61% of trials, yet, in a further condition, the patient was unable to select the correct name for the face from among the alternatives (Bauer, 1984). There have been several demonstrations that visually evoked cortical potentials, or VECPs, in response to a purely chromatic pattern (i.e., one devoid of luminance differences) can be recorded from achromatopsic patients (Victor, Maiese, Shapley, Sidtis, & Gazzaniga, 1989; Humphreys, Trosciianko, Riddoch, Boucart, Donnely, & Harding, 1992; Heywood, Nicholas, & Cowey, 1996). However, autonomic or reflexive responses or VECPs may or may not influence behavioral outcome, presumably depending on whether the observer can use them to mediate a discriminative response. For example, the pupillary light reflex can persist in total cortical blindness but with no indication that the patient can ''use'' it to guide other aspects of behavior. Such responses cannot, by themselves, be considered the consequences of covert processing, unless the definition is extended to a point to which it is rendered trivial. For example, the visual reflexes elicited in comatose patients (Keane, 1979) are not helpfully described as covert processing. Thus, while several authors have classified the recording of VECPs and GSRs as direct tests of covert processing, it would perhaps be wise to restrict the definition of such tests to those that require a discriminative response under attentional or voluntary control.

One view of blindsight is that it exposes a modular architecture in which there are multiple routes to visual action. Thus, intact visual pathways can still accomplish visuomotor or other orienting responses, albeit in the absence of conscious awareness of the properties of the visual stimuli that elicit them. This view has been extended to cases of visual form agnosia, notably patient D.F., whose impaired discrimination of orientation and shape coexists with accurate and appropriate visuomotor acts requiring the coding of these stimulus attributes for their successful execution (Milner, Perrett, Johnston, Benson, Jordan, Heeley, Bettucci, Mortara, Mutani, Terazzi, & Davidson, 1991). However, the parallel between cortical blindness and apperceptive agnosia should be applied with caution. That DF ''betrays severely impaired conscious perception. . . . .'' (Milner, 1992, p. 144) conceals the crucial difference that blindsight patients possess no phenomenal vision (with the possible exception of rapidly moving, high contrast stimuli (Barbur, Watson, Frakowiak, & Zeki, 1993)), whereas apperceptive agnosics, including DF, possess qualia, but are grossly impaired at discrimination of shape, i.e., they have phenomenal vision but grossly elevated thresholds for shape and orientation.

While instances of blindsight and apperceptive visual agnosia can highlight the pathways that mediate visual action, cases of cerebral achromatopsia suggest that the loss of phenomenal representation of color does not compromise the use of chromatic cues by alternative pathways in the services of object or pattern vision. Wavelength is phenomenally represented as color. Many cells in visual cortex are sensitive to wavelength variation in their receptive fields, without necessarily being wavelength selective. The contribution of such cells to visual processes need not be restricted to deriving the surface properties (luminance and hue) of objects in the visual scene. It is plausible to suppose that other visual attributes, such as motion, form, and texture, can also be derived from wavelength differences in the absence of phenomenal representation of hue. For example, a ''complex'' cortical cell, unlike its partner, the

''simple'' cell, lacks phase sensitivity and is unable unambiguously to signal the constituent hues of a chromatic edge which falls in its receptive field. Nevertheless, it will indicate the presence of such an edge. Whether such responses are the basis for the ability of achromatopsic patients to detect edges between equiluminant colors, without being able to discriminate isolated patches of its component colors, is unknown. However, it is clear that whatever the neural basis of such an ability, the processing is not covert. Achromatopic patients accurately describe the location and apparent properties of such edges; that is, they are visually aware of contours generated by equiluminant color contrast, despite being unable to tell the colors apart.

CEREBRAL ACHROMATOPSIA

It is commonly reported that cerebral achromatopsia is accompanied by spontaneous complaint that the world is ''drained of color'' and, indeed, more formal testing on tasks requiring color naming or the ordering or matching of equiluminant hues reveals severe deficits consistent with a loss of the phenomenal experience of color. While the results of other conventional tests of color vision can vary among individual cases, it is nevertheless true that the instances and pattern of preserved ability can be as informative as specifying the nature of what is lost.

Perhaps the most striking dissociation is that between essentially normal sensitivity to chromatic contrast in equiluminant, red/green sinusoidally modulated gratings and a complete inability to discriminate between a red and a green patch whose chromatic difference is identical to the peak color contrast in the grating (Heywood et al., 1996). Preserved chromatic contrast sensitivity was also reported by Victor et al. (1989). This ability to derive ''form-from-color'' cannot be the trivial consequence of unintended brightness differences between the component colors since the introduction of random luminance fluctuation fails to mask the detection of a form embedded in a display and only disambiguated by chromatic contour (Barbur, Harlow, & Plant, 1994; Heywood, Cowey, & Newcombe, 1994). However, the use of random luminance masking is precisely the technique adopted in the more conventional Ishihara test for color vision, where each of the exhibits contains a figure camouflaged by additional, static luminance contour. Different achromatopsic subjects read the plates with varying success. When successful, the patient's residual ability has occasionally, but erroneously, been attributed to covert processing. The attribution is erroneous because, in no case, is a patient required to ''guess'' the identity of an invisible figure concealed in the color plates and, indeed, achromatopsics can describe it and trace its outline, revealing that it produces a conscious percept.

The neural basis of responses to chromatic contour has not been determined. There have, however, been attempts to relate it to established properties of the P- and M-channels of primate vision. The P- and M-channels originate from the Pβ and Pα retinal ganglion cells, respectively, which, in turn, innervate the parvocellular (P) and magnocellular (M) cells of the lateral geniculate nucleus (dLGN). The P- and M-cells of dLGN project to different layers of striate cortex (area V1) and the division of labor is essentially maintained in the extrastriate areas beyond. The cluster of visual areas described as the dorsal stream, including area V5, chiefly receive M input while those in the ventral stream, including area V4, receive mixed M and P

inputs. The dorsal and ventral streams have been associated with motion and color processing, respectively. The P-channel conveys color-opponent (responding in an opposite manner to red and green or blue and yellow) information, while the M-channel responds in a broadband manner to a particular wavelength and receives no input from short wavelength cones. The P- and M-channels also differ in their response to luminance contrast and temporal frequency. The M-channel is better able to convey low contrast information and responds to higher temporal frequencies than does the P-channel.

It is now clear that cortical areas assigned to the so-called M-channel of visual processing contain neurons that are color *sensitive,* i.e., broadly tuned to wavelength, without showing color *selectivity*. For example, when a chromatic border is placed in the receptive field of such a cell, its response cannot be silenced by adjustment of the relative luminance of the hues of which the border is composed (Saito, Tanaka, Isono, Yasuda, & Mikami, 1989). However, the relative position of the component colors matters not a jot, and the response of the cell will not distinguish between a red/green and a green/red border. Early demonstrations of the ability of achromatopsic patients to respond to the salience of such borders suggested mediation by the M-channel (Heywood, Cowey, & Newcombe, 1991). However, random fluctuations of the luminance of the pixels or clusters of pixels in a display at a sufficiently high temporal frequency (to which the M-channel is sensitive) will swamp the M-channel with irrelevant signals and render it incapable of distinguishing embedded figures defined solely by chromatic differences. As the detection of chromatic contour survives random luminance masking, and since high temporal frequency luminance flicker would be invisible to its partner, the P-channel, it was proposed that it is P-channel activity that accounts for detection of form-from-color.

The color-opponent P-channel is certainly indispensable for many of the features of chromatic vision. Selective lesions of the P-channel at the level of the dorsal lateral geniculate nucleus abolish color discrimination within the corresponding part of the visual field in the macaque monkey (Schiller, 1990). However, the M-channel has been implicated in a task assessing color discrimination in achromatopsic patients (Troscianko, Davidoff, Humphreys, Landis, Fahle, Greenlee, Brugger, & Phillips, 1996). In the absence of color qualia, judgments of whether two monochromatic fields differ in luminance should be unaffected by the introduction of a chromatic difference. However, Troscianko et al. (1996) showed that performance was enhanced by introducing color and patients must therefore be processing chroma. Introducing random luminance masking into the display, thereby creating the appearance of checkerboards, affected two patients in a different manner. One patient was affected by static noise, in that judgments were now no better when color differences accompanied overall luminance differences, but was unaffected by rapid flicker. Since the P-channel is sensitive to static noise but blind to rapid flicker, it was concluded that, in this subject, residual color processes were subserved by the P-channel. A second achromatopsic patient showed the opposite pattern of results; rapid flicker, but not static noise, interfered with judgments based on chromatic differences. The improvement present in judgments of luminance differences when color was added to luminance differences in static displays was now abolished. In this subject, it was concluded that the M-channel is implicated in residual color discrimination, with

little or no P-channel contribution. More importantly for the issue discussed here, it was further argued that, in each subject, the successful discrimination was an example of *covert* or *unconscious* color processing, or *color ''blindsight''*. Without casting the slightest doubt on the importance of the authors' results, neither claim is warranted since, as the authors themselves report, the achromatopsic patients commented that introduction of color into the displays resulted in a change of phenomenal vision, i.e., an apparently ''enhanced luminance contrast'' (p. 208). The patients were therefore not guessing about differences they did not consciously perceive. Nor did low confidence ratings accompany poor performance. Instead, residual processing, of whatever origin, resulted in a conscious perceptual change, notwithstanding the absence of color qualia. A more prosaic account is that the processing of wavelength differences contributes more than just the phenomenal representation of hue.

Is there further evidence for P-channel contributions to vision, or to residual processing of wavelength differences, in achromatopsia? The relative luminous efficiency of different wavelengths is usually established by heterochromatic flicker photometry, whereby the luminances of two rapidly alternating colors are adjusted until perceived flicker is minimal. The colors are then said to be equiluminant. The technique relies on the properties of the P- and M-channels, where the rapid alernation of lights is invisible to the P-channel and flicker is detected by the achromatic M-channel. At equiluminance, when the lights differ in chromaticity alone, the M-channel is blind to chromatic differences and perceived flicker is absent or substantially reduced. However, under sustained viewing, or low temporal frequencies which favor the P-channel, and used in heterochromatic direct brightness matching, luminous efficiencies are quite different. In flicker photometry, the addition of two colors results in a hue with a luminance equal to their sum. However, the operation of the color-opponent P-channel results in marked subadditivity, such that when two opponent colors, e.g., red and green, are mixed, hue cancellation occurs and the outcome is a color mixture which is perceptually dimmer than would otherwise be expected (Guth, 1965). Thus, a color-opponent ($red^+/green^-$) receptive field will be maximally excited and inhibited by long and middle wavelength light, respectively. The converse occurs for cells showing opposite opponency ($green^+/red^-$). A yellow light, composed of a mixture of middle and long wavelength light will place excitatory and inhibitory mechanisms of the receptive fields in equilibrium and the nulling of the response results in a perceptually dimmer, subadditive color mixture. Measurements of photopic spectral sensitivity can also uncover opponent cone interactions. Shifts in the peaks of wavelength sensitivity away from the absorbance peaks of the medium and long wavelength cones indicate a contribution from color-opponent, P-channel, mechanisms (Sperling & Harwerth, 1971). Cases of achromatopsia can show both a spectral sensitivity and subadditive brightness perception of color mixture that are inexplicable except by residual P-channel processes (Heywood et al., 1994; Troscianko et al., 1996).

A third example of P-channel processing in the absence of color perception concerns the movement of color displays. There is strong evidence to suggest that the perception of both the color and the motion of a slowly moving, equiluminant chromatic grating is mediated by a color-opponent mechanism (Cropper & Derrington, 1996; Gegenfurtner & Hawken, 1996). But the mechanism does not code velocity

veridically, which presumably accounts for the perceived effect of ''motion slowing'' when the chromatic grating is compared with an achromatic grating drifting at the same speed. The construction of such a grating entails modulating a red and green grating in spatial antiphase. Because of the effects of subadditivity, described above, it is not surprising that such a grating contains apparent brightness variations, particularly where the red and green show maximum overlap and produce yellow midway between the red and green peaks, i.e., at twice the spatial frequency of the R/G grating. Recently, we reported that ''correction'' of the brightness variation, by the addition of frequency doubled luminance, resulted in an even further reduction in the apparent speed of the grating (Heywood, Kentridge, & Cowey, 1998). But this was true for both normal observers and a case of complete achromatopsia, patient M.S., indicating normal processing of slow chromatic motion even when the colors are indistinguishable. The patient showed essentially normal sensitivity to chromatic gratings, along with intact processes for detecting their motion, in the absence of color vision.

Detection of a drifting grating does not, however, require the subject to code the sign of the color, i.e., which portion is *red* and which *green*. If a grating is phase shifted by 180°, where the red bars become green, and vice versa, then its apparent direction of motion is ambiguous. Such ambiguity is removed, for a normal observer, by a phase shift of 90°, but not, presumably, for an achromatopsic patient, who is unable to distinguish red from green. Remarkably, patient M.S. instantly and flawlessly reported the correct direction of apparent movement, indicating sensitivity to the sign of the colors that were moving (Heywood et al., 1994).

Again, such demonstrations are consistent with the processing of wavelength differences to derive motion, without recourse to notions of ''covert'' processing of color. In judgments of form or motion from equiluminant chromatic cues, achromatopsic patients readily provide a commentary on what they *see*, rather than having to be cajoled to guess about the nature of the displays. Their percepts may be unusual but they are indubitably percepts.

Complete and Incomplete achromatopsia

The extent and character of residual ability among achromatopsic patients show a variability which presumably reflects the nature of the brain damage. For M.S., a profound achromatopsic, residual color processes are not mediated solely by the M-channel. Furthermore, the location of the lesion in the ventromedial occipital cortex suggests that any P-channel involvement must occur beyond striate cortex. One explanation as to why some patients have greater access to chromatic signals is that their achromatopsia is ''incomplete,'' possibly because of subtotal damage to regions in extrastriate cortex, i.e., the ''color center'' (Zeki, 1990), which process such information. Alternatively, it has been proposed that it is not the remnants of a color center which mediate performance, but intact striate cortex. Hence, in those patients where the striate cortex has been additionally damaged, impairments are more severe. Several authors have distinguished between the greater complexity of processes required for higher-order tasks like color identification, classification, and ordering, compared with the processes that underlie lower-order tasks like color discrimination, chromatic

contrast sensitivity, and the preservation of color elicited VECPs (Victor et al., 1989). The authors plausibly attributed a normal VECP to chromatic checkerboards, normal acuity, and color contrast sensitivity to the absence of damage to striate cortex in their patient. They similarly suggested that intact color-opponent mechanisms in striate cortex could mediate the correct identification of 8 of 9 Ishihara pseudoisochromatic plates and enable the patient to select a colored square embedded in 39 irrelevant squares of the complementary opponent color. In contrast, deficits in the identification and sorting of colors were proposed as being the results of damage to extrastriate ventromedial regions. A similar pattern of performance has been described in a another patient (Humphreys et al., 1992). However, these are instances of *incomplete* achromatopsia, where it is difficult to conclude whether intact striate cortex or remaining tissue in extrastriate regions mediates spared function. Certainly, striate cortex is minimally, if at all, involved in patient M.S., whose chromatic contrast sensitivity and VECPs are normal. Yet, he is severely impaired at color oddity and identification of the Ishihara plates at normal reading distance. This suggests that these tasks do not depend on striate cortex but are more likely to depend on the intact regions that survive a partial lesion to the color center.

Whatever cortical visual areas mediate an achromatopsic subject's performance on color oddity, matching color samples, or reading of the Ishihara plates, the performance need not and should not be described as covert. In cases of incomplete achromatopsia, sensitivity to chromatic variation is sufficient to perform some tasks (color oddity, Ishihara test and rudimentary color matching). Whether mediated by the remnants of a partially damaged color center or other intact neural pathways, as is presumed in complete achromatopsia, the subjects make clear that they are performing a conscious discrimination of a depleted percept. What, then, would constitute covert color vision in achromatopsia?

COVERT PROCESSING IN ACHROMATOPSIA

One demonstration of covert processing of color would require that a subject deny any phenomenal experience of hue differences, but nevertheless perform at better than expected by random responding during forced-choice testing. This would constitute a ''direct'' test. A test of this sort was conducted by Brent, Kennard, and Ruddock (1994) on a blindsight patient G.Y. The authors first demonstrated normal spectral responses in his blind hemifield, where the subject reported that, during threshold detection measurements, his responses were based on either the presence or absence of a percept. However, when required to name the color of large, 40° diameter, chromatic stimulus briefly presented in his field defect, and where the luminance of the stimulus provided no clue to its color, correct performance was unaccompanied by a conscious percept of the stimulus. This demonstation of ''color blindsight'' is quite different from a claim of covert color processing in achromatopsia. A brief report of a study performed on achromatopsic patient H.J.A. (Humphreys et al., 1992) describes the patient as being 40–50% correct at both color naming and being asked to point to a named color in a collection of colored patches. Performance deteriorated to 19 and 31%, respectively, when static luminance noise was added to the patches. The extent to which these abilities reflected incomplete achromatopsia is hard to

establish. However, in a task requiring matching a sample to 1 of 20 colored patches which were widely separated in color space, H.J.A. performed at 60–65% correct (random responding would yield 5% correct) with and without static luminance noise. When the chromatic range of the samples was reduced, performance fell to 40% correct. A feature of covert processing is that low confidence in making a discriminative response can nevertheless accompany proficient performance. For H.J.A., better performance was evident for widely spaced, compared with narrowly spaced, colors. However, confidence ratings remained unaltered at 50% of correct judgments rated as ''unsure'' or ''very unsure.'' As mentioned above, introducing static noise into widely spaced colors left performance essentially unaffected. But it reduced confidence such that 77% of correct judgments were now rated as unsure or very unsure. This pattern of confidence ratings and performance scores, especially in view of the small number of trials presented, could be, but need not be, interpreted as evidence of covert processing. Humphreys et al. (1992) demonstrated that a change in a subject's confidence in a judgment need not be accompanied by a change in performance. This is more akin to the transition between the ''unaware'' and ''aware'' modes of perception found in the blindsight subject G.Y. by Weiskrantz, Barbur, & Saharie (1995) than to the commonly accepted criterion for blindsight in which change in performance occurs, although confidence remains very low. It is, in fact, debatable whether a subject who is confident about 50, or even 23%, of their judgments is really demonstrating blindsight—they are showing a normal co-occurrence of confidence and discrimination on half or a quarter of all trials.

Sporadic descriptions of achromatopsia as color blindsight, and the infrequency with which covert processing of color has been appropriately assessed, prompted us to test patient M.S., a complete achromatopsic, on tasks designed to reveal such processes, should they exist. M.S. is a 49-year-old man who suffered an attack of herpes simplex encephalitis in 1970. He has been repeatedly tested since 1972 and his condition is stable, consisting of severe achromatopsia along with a left homonymous hemianopia with macular sparing. Extensive studies have been published elsewhere (Heywood et al., 1991, 1994, 1996) and the nature of his residual vision has been briefly commented on above. We recently tested M.S. on direct tests of color oddity that required a verbal report of, or an eye movement to, the target location. In addition, we administered a single indirect test of letter identification.

Stimului were generated using a Cambridge Research Systems VSG2/3 visual stimulus generator driving an Eizo T784 color monitor. Red and green and neutral grey stimuli had CIE coordinates of $x,y = (0.64, 0.33)$, $(0.29, 0.60)$, and $(0.30, 0.30)$, respectively, and were displayed against a black background (0.1 $cd.m^{-2}$). A white central fixation cross (52.5 $cd.m^{-2}$) was available.

Direct Tests

The display for the verbal report of color oddity was composed of a vertical line of four 1.5° diameter disks with a center-to-center separation of 1.625° and displaced 4.5° to the right of a 0.5° fixation cross. In 2 separate blocks of trials, M.S. was instructed to report verbally which of the four disks in each trial looked different

from the others. Each trial started with a fixation followed by a 2 s stimulus presentation, during which he was free to move his eyes. Responses were followed by a 1.5 s intertrial interval. The first block consisted of 80 trials of 4 grey discs, 3 dim distractors (5.5 $cd.m^{-2}$) and a brighter target (20.0 $cd.m^{-2}$). In the second block of 40 trials, the 4 discs had the same luminance (5.5 $cd.m^{-2}$) but the target was red and the distractors were green. In each block, the spatial position of the target was randomly assigned from trial to trial, ensuring equal numbers of trials in which the target appeared in each of the four locations.

M.S. had no difficulty in correctly identifying the target when it differed in luminance from the distractors. However, consistent with previous reports of oddity discrimination (Heywood et al., 1991), he was no better than would be expected by random responding for the 40 color trials, correctly identifying the target on only 9 occasions

We then went on to test whether M.S. covertly processes hue when required to make a different behavioral reponse, namely saccadic eye movements. Eye movements were recorded using a Fourward Technologies Dual Purkinje Image Eyetracker and collected on an Apple Macintosh Quadra 650 computer fitted with a National Instruments LabNB data acquisition board. Again we used an oddity task, but now M.S. was instructed to move his eyes to the ''odd one out.'' To facilitate measurements, we used horizontal bars of the same height as the discs (1.5°) and with the same gap between them (0.125°) but extending over the whole 40° width of the screen. We used only three bars, colored either red or green. The center of the middle bar coincided with the fixation cross. On each trial, two of the bars were the same color and one was odd. The odd color bar was always in the top or bottom location to allow it to elicit an eye movement away, up or down, from the initial fixation position. Each trial started with the display of a 0.5° white fixation cross against a black background. After approximately 2 s (under the control of the experimenter), the stimulus appeared and remained visible for 2 s, after which the subject had to refixate the central cross for the next trial. There were three blocks of 25 trials with approximately equal number of odd targets in the upper and lower locations and an approximately equal number of each color of odd target. Presentation order was randomized within blocks. Eye movement records were initially analyzed to allow rejection of data from trials in which no saccade could be detected or in which the tracker failed to remain locked onto the subject's pupil. The aim of our analysis was to determine whether the odd location influenced the saccadic eye-movements of M.S. Unfortunately, on a number of trials, M.S. hardly moved his eyes from fixation. Although small saccades could be detected on these trials they left M.S.'s gaze within the central bar. All trials in which the vertical amplitude was less than 0.75° (i.e., within the central target) were therefore discarded from the analysis. Of the remaining 39 trials, M.S. made eye movements in the direction of the odd target on 26 occasions as shown in Fig. 1. The probability of this occurring by chance is 0.012 (one-tailed binomial), suggesting that M.S. may indeed show covert processing of hue in his eye-movement system but that these responses cannot be used to mediate performance in verbal tasks. When asked about the task, he reported that it was difficult because the bars looked the same. We are encouraged to pursue this possible dissociation between eye movements and phenomenal experience.

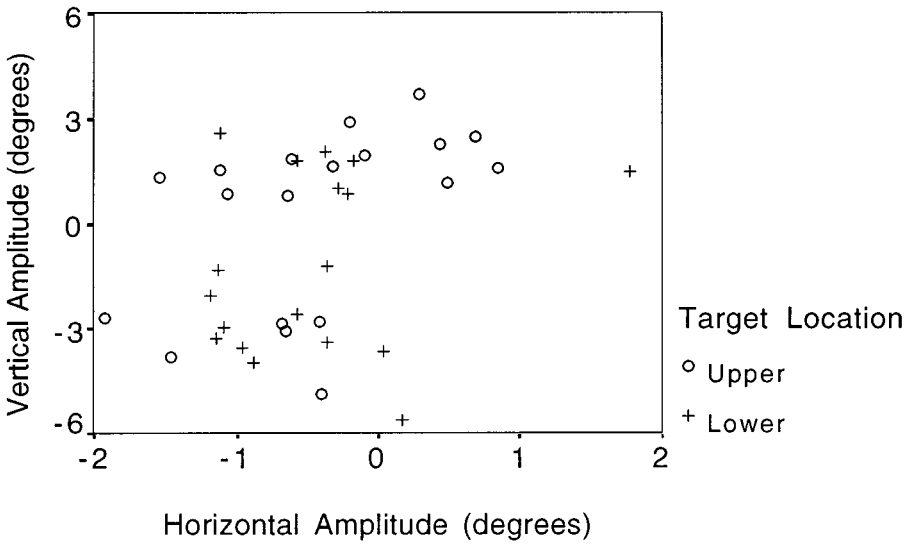

FIG. 1. The figure shows the amplitude of the first saccade, made by M.S., from the center bar at fixation (0,0) to the bar of a different color that lay above (O) or below (+) it.

Indirect Test

If M.S. can distinguish hues covertly, he should be able to use this information to segregate nonadjacent elements of a stimulus on the basis of hue alone. In normal subjects, such grouping by color has been shown to affect performance in tasks unrelated to color. For example, the extent to which peripheral elements of a stimulus display interfere with identification of an element at the fixation point in the middle of the display is impaired if the element at fixation and those in the periphery are differently colored (Baylis & Driver, 1992). We used a task of this type to assess the extent to which M.S. might have covert access to hue.

Each stimulus was composed of a single alphanumeric character or a triplet of characters (each 1.5° wide × 2.4° high), separated by 0.3°. The characters were either red or green, with a luminance of 5.5 cd.m^{-2}. The subject's task was to identify whether the character presented at fixation was a ''C'' or an ''N'' and respond as quickly as possible by pressing one of two buttons to produce a reaction time. The different character strings provided different types of interference for this task. Strings ''NCN'' and ''CNC'' provide maximum conflict between the responses elicited by the flanking and central letters. A number of control conditions provide lesser degrees of conflict. The triplets ''BCB'' and ''BNB'' have a flanking character which is equally confusable with C or N (Townsend, 1971); hence some interference would still be expected, although less than that between C and N. The triplets ''*C*'' and ''*N*'' have a neutral flanking character which is not readily confusable with C or N. Finally, the single characters C and N have no flanking character to elicit conflicting responses. The triplets could be presented with the central character either in red or

green, and both flanking characters of either the same or a different color. On each trial, a white (52.5 cd.m^{-2}) fixation cross of 0.25° was presented against a black (0.1 cd.m^{-2}) background for a random interval of between 1.0 and 2.0 s. The cross was then replaced by a stimulus. As soon as the subject had made a response, the next trial began. Each test block consisted of 60 trials, approximately equally distributed between these conditions. There were 6 blocks of testing.

All trials with outlier reaction times (more than 2 standard deviations from the original mean) were discarded. An analysis of all possible conditions in a two-way ANOVA, with Letter-Combination and Color as Factors, yielded no overall significant differences for either factor or their interaction (all F values < 1). However, planned comparisons reveal a significantly faster reaction time to identify the central letter when it is adjacent to neutral flankers (*C* and *N*; mean 595.3 ms, *S.E.*= 7.6), compared with those reported to result in maximal interference, NCN and CNC; mean 616.9 ms, *S.E.* = 7.7; $F(1, 455) = 4.0$, $p < 0.05$). There was, however, no effect of introducing a color difference between the target and flankers ($F(1, 455) = 0.37$, ns). Any effect of covert hue processing should have revealed itself in the interaction, which does not reach statistical signficance ($F(1, 455) = 0.01$, ns). When we conducted an identical experiment with a normal observer, the same analysis showed a significant effect of interfering flanking letters (*C* and *N*; mean 420.6 ms, *S.E.* = 3.4; NCN and CNC, mean 459.1 ms, *S.E.* = 4.1; $F(1, 429) = 53.94$, $p < .001$) and no overall effect of a color difference between the central letter and its flankers ($F < 1$), just as we found for M.S. In the case of the normal observer, however, there was a significant interaction between the color-difference and flanking-interference factors ($F(1, 429) = 4.46$, $p < .05$) indicating that the normal observer utilizes color to isolate the central letter from the flankers (*C* and *N*, same color mean 414.4 ms, *S.E.* = 4.2; different color mean 427.0 ms, *S.E.* = 5.4; NCN and CNC, same color mean 464.2 ms, *S.E.* = 5.4; different color mean 454.6 ms, *S.E.* = 5.9). The absence of such an interaction for achromatopsic patient M.S. shows that an indirect test, of the sort described here, fails to reveal covert processing of color.

In conclusion, M.S. shows no covert processing of color in a forced-choice oddity task with a verbal response. His performance is consistent with his spontaneous comment that his responses are ‘‘guesswork.’’ Neither is such processing revealed by a single indirect test of letter identification. However, the experiments conducted are far from exhaustive and the surprising ability of M.S. to make appropriate first saccades to chromatically differing targets warrants further investigation. It remains to be seen whether this ability is accompanied by low confidence judgments when such judgments are gathered more formally on a trial-by-trial basis. Certainly, M.S. expressed no overall confidence in his ability to move his eyes to the target, implying that information about eye position cannot cue the correct verbal response. It is, nevertheless, possible that this may be rendered possible if M.S. were provided with sufficient practice along with trial-by-trial feedback of his performance. What has been described is an instance where chromatic differences exert a measurable effect on behavior in the absence of any phenomenal representation of hue. Nevertheless, many of the abilities displayed by M.S., such as extracting form and motion from

wavelength variation across the visual scene, are better characterized as properties of residual vision, i.e., the survival and perceived awareness of some aspects of the normal processing of wavelength.

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