Residual Vision in a Subject with Damaged Visual Cortex

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Abstract

■ It is well known that a lesion in the optic radiation or striate cortex leads to blind visual regions in the retinotopically corresponding portion of the visual field. However, various studies show that some subjects still perceive certain stimuli even when presented in the "blind" visual field. Such subjects either perceive stimuli abnormally or only certain aspects of them (residual vision) or, in some cases, deny perception altogether even though visual performance can be shown to be above chance (blindsight). Research on monkeys has suggested a variety of parallel extrastriate visual pathways that could bypass the striate cortex and mediate residual vision or blindsight. In the present study, we investigated a subject with perimetrically blind visual areas caused by bilateral brain damage. Black and white stimuli were presented at many locations in the intact and affected areas of the visual field. The subject's task was to state, using confidence levels, whether the target stimulus was black or white. The results revealed an area in the "blind" visual field in which the subject perceived a light flash when the experimental black stimulus was presented. We hypothesize that a spared region in the visual cortex most likely accounts for these findings. ■

INTRODUCTION

A postgeniculate lesion of the visual system, in the optic radiation or striate cortex, causes complete blindness in the area corresponding retinotopically to the neural damage (Grüsser & Landis, 1991; Holmes, 1918; Inouye, 1909). However, in a proportion of individuals, residual visual capacity has been shown if testing is rigorous enough. It is usually called "residual vision" if certain visual stimuli or certain aspects of stimuli are consciously perceived. This phenomenon has been observed in subjects over the last 80 years. Most frequently, residual vision has been shown using moving stimuli presented in the affected visual fields of subjects with postgeniculate damage (Barbur, Ruddock, & Waterfield, 1980; Blythe, Kennard, & Ruddock, 1987; Holmes, 1918; Perenin, 1991; Riddoch, 1917). Typically, the subjects reported a sensation of movement (Barbur et al., 1980), a sensation of a dark shadow (Blythe et al., 1987), a vague shadow (Perenin, 1991; Riddoch, 1917), or a light (Perenin, 1991). Residual vision to flicker stimuli (Blythe et al., 1987) or a limited reaction to light (Teuber, Battersby, & Bender, 1960) has also been found.

The properties of residual vision (good detection performance of transient stimuli but poor identification performance) have often led to the conclusion that the superior colliculus (SC) or an extrastriate pathway through the SC was responsible for the residual vision. Studies on destriate monkeys suggest that a secondary, parallel pathway to the occipito-parietal area could receive the input from the retina via the geniculate, colliculo-geniculo, pulvinar, or colliculo-pulvinar pathway. Recent investigations favor the involvement of the occipito-parietal system, V3A, MT, or STP and its associated subcortical regions (Bullier, Girard, & Salin, 1994; Kisvárday, Cowey, Stoerig, & Somogyi, 1991; Milner & Goodale, 1995; Payne, Lomber, Macneil, & Cornwell, 1996).

Residual vision found in 5 out of 25 human subjects with striate cortex lesions may be mediated either by partial destruction of the striate cortex, the LGN-extrastriate pathway, or the colliculo-pulvinar-extrastriate pathway (Blythe et al., 1987). In addition, extrastriate cortical areas such as MT/MST (Perenin, 1991), the magno part of the geniculo-striate pathway, or pathological damage in all geniculo-striate neurons have been suggested as sources of residual vision (Plant & Wilkins, 1988). Reorganization of receptive fields (Celesia, Bushnell, Toleikis, & Brigell, 1991; Schärli, 1997) or artifacts such as scattered light from high-contrast stimuli may have led to the successful performance in some studies that reported residual visual performance (Campion, Latto, & Smith, 1983; Gazzaniga, Fendrich, & Wessinger, 1994; Schärli, Harman, & Hogben, 1999). No study to date has been able to determine conclusively the identity of the neural region responsible for the residual visual sensitivity.

The present study investigated the unusual residual vision of a subject with postgeniculate damage and its possible neural basis. The methods used eliminated or minimized artifacts such as eye movements or stray light.

The project was completed as part of a Ph.D. thesis at the University of Western Australia (Schärli, 1997). Preliminary reports of this work have been published in abstract form (Schärli, Harman, & Hogben, 1995; Schärli, Harman, & Hogben, 1996).

RESULTS

Normal Control Subjects

The results for six normal subjects showed that a stimulus presented in the visual field could be seen, whereas a stimulus presented in the natural blind spot could not (yes/no test). In the forced-choice test, the performance for stimuli presented in the natural blind spot was at chance level, whereas in other parts of the visual field it was almost faultless (for more details, see Schärli et al., 1999).

Lesion Analysis of Subject KF

KF's 1991 stroke was responsible for the lesion that extends from the occipital horn of the left lateral ventricle to the adjacent parieto-temporo-occipital cortex (Figure 1). In addition, some small areas of infarction in the right cerebellar hemisphere were affected. A 1994 CAT scan, taken after a second stroke, showed newly developed, small patchy areas of infarction, situated medially in both occipital lobes. Although the outline of damaged areas in subject KF was clearly visible, the resolution of CAT scans did not permit an analysis of fine detail. Furthermore, brain structure showed considerable individual variation in the occipital lobe (Damasio, 1995). We were able, therefore, to determine that the visual system was damaged in the region of area 17 and area 18 bilaterally and possibly in the upper left optic radiation. However, the true extent of sparing within damaged areas, if any, could not be determined. The scans also revealed some frontal lobe atrophy.

Humphrey Field Analyzer Test

The extent of the blind visual field as determined using the Humphrey Field Analyzer is shown in Figure 2. The field for the left eye is illustrated; however, the right eye demonstrated a corresponding pattern. The small black dots (and pale gray areas) indicate the regions seen as blind using this technique. There was an extensive loss of vision in the upper left and lower right quadrants of the left eye. The upper right quadrant had less damage, and the lower left quadrant appeared fully intact. In the first Humphrey test, the intact visual field appeared even smaller than shown here by the black dots. Three additional locations, including one in the blind spot region, seemed to be blind in the first test but not in the second (see half-black dots; Figure 2). However, the entire upper left quadrant appeared blind in both tests.

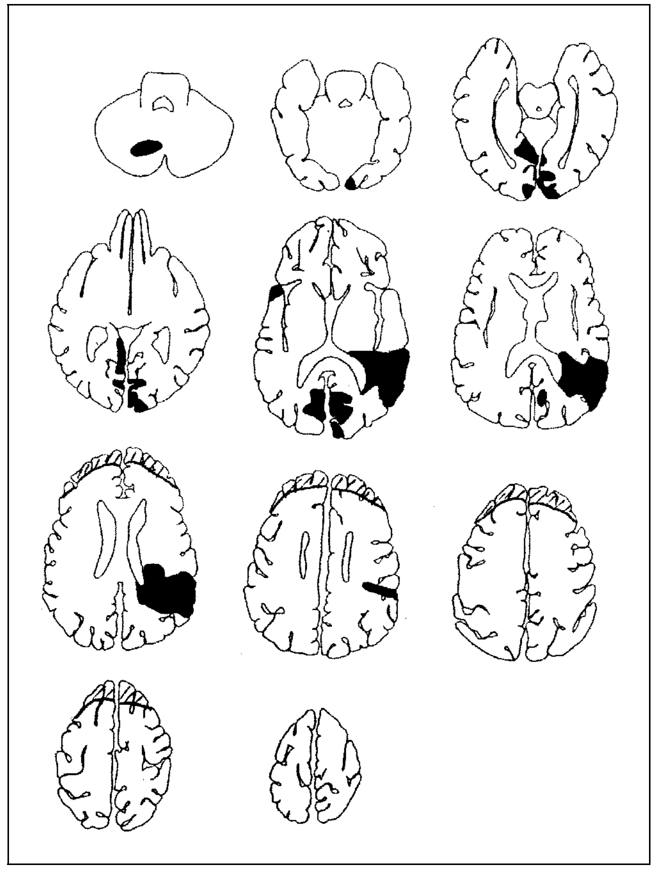

Figure 1. Lesion of subject KF drawn on template. Sections are shown sequentially from lower to higher. Following standard neuroimage conventions, the left hemisphere is on the right and the right hemisphere on the left.

Pretests

During conversations in the pretests, used to delineate the blind regions and test for residual visual performance, it was discovered that KF could, in fact, see many stimuli in his clinically blind visual field. He reported spontaneously that he often perceived "weak white flashes" in the upper left field when the normal black discs were presented. He said that he initially did not report the stimuli because they appeared light and different and because he first believed that the flash was caused by a reflection on the screen from the ceiling lights.

Visual Field Tests

KF's blind visual area appeared to be smaller on the map constructed from the visual field test data when compared to the map that was drawn using the clinical Humphrey data, especially in the upper left area (dark gray versus light gray respectively; Figure 2). In addition, his visual field appears patchy. In about half of the locations, his responses were consistent for each trial (especially in the central and lower left area in which he

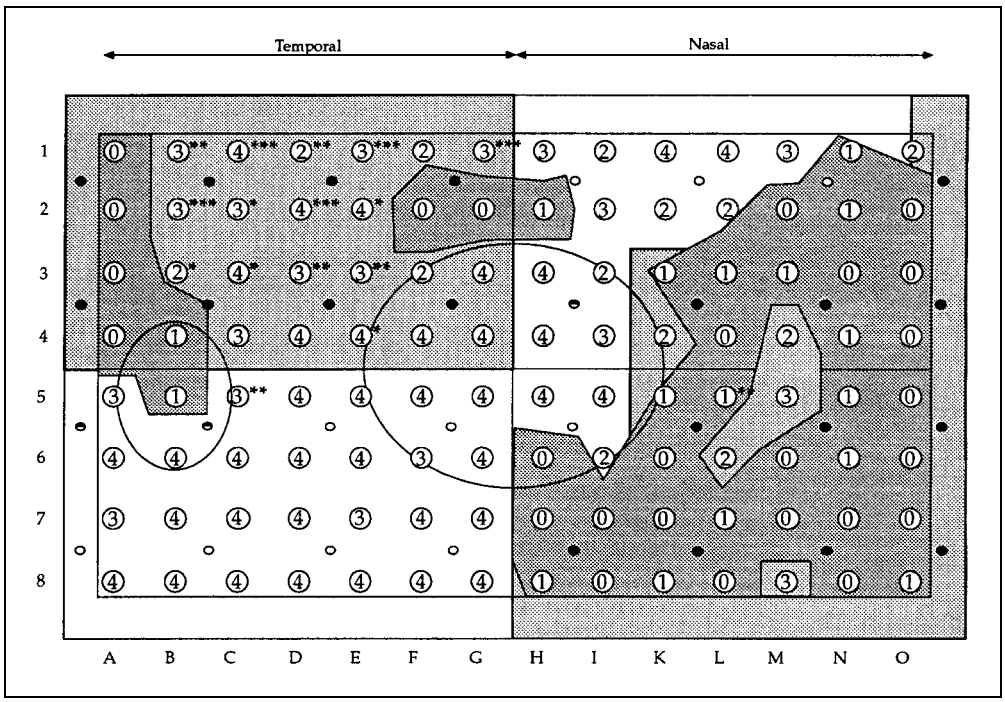

Figure 2. Damaged visual region of subject KF's left eye according to the Humphrey Field Analyzer (light shade) and the test used in our lab (darker shade). The large ellipse indicates the macular region and the small ellipse represents the blind spot (optic nerve head). Four stimuli were presented at every location. The number of Yes responses is shown at each location. In total, four stimuli were presented (two black and two white stimuli) in the first test. Stars indicate locations where black stimuli were seen as white (* = one black was seen as white and one black was seen as black; ** = one black was seen as white and one black was not seen at all; *** = all blacks were seen as white). The small dots indicate the locations tested by the Humphrey Field Analyzer (blank dot, seen; solid black dot, not seen; half black dot, not seen in the first but seen in the second test).

acknowledged seeing the stimuli in most trials). However, at other locations, his responses varied from trial to trial. Subject KF predominantly chose confidence level 1 (very certain) or 5 (very uncertain). Confidence level 3 was used twice only.

Most white stimuli were either perceived as white (a white spot or a white "star") or not seen at all. By contrast, in many locations, black stimuli were seen as white. At 13 neighboring locations (1B, 1C, 1D, 1E, 2B, 2C, 2D, 2E, 3B, 3C, 3D, 3E, and 4E; Figure 2) in the upper left quadrant of the left eye (an area that he never responded to in the Humphrey visual field test), KF reported that he perceived black stimuli mostly as white ("light flashes"). Out of 26 black stimuli that were presented at each of these 13 locations, 17 were perceived as white, 6 were not seen at all, and only 3 were perceived correctly as black (Figure 2). KF described his perception of the stimulus as a "white spot, not clear in shape, like the end of a shooting star." In 10 of the 13 neighboring locations, black stimuli were always seen as white (if seen at all) and in the remaining 3 locations tested, black stimuli were seen as white on one occasion and black on another (Figure 2). There were three further locations in the visual field where black stimuli were seen as white. One of these stimuli was also in the upper left quadrant (location 1G; Figure 2), a second was located just below the vertical meridian on the left side (5C; Figure 2; black was seen as white once), and a third was seen in the lower right blind region (location 5L; Figure 2).

Retests

Table 1 shows the results from all trials (tested and retested locations, left and right eye), listing all upper left quadrant locations and some control locations. The perception of white instead of black was confirmed at five locations that were retested in the upper left area (1C, 2B, 2D, 3B, and 3E; Table 1). Moreover, at three of these five locations, stimuli were nearly always reported as white, if seen, when a black stimulus was presented (1C, 2B, and 2D; Table 1). Subject KF reported that he perceived a "white flash," with no shape, replicating the data from the test above. White stimuli were either perceived as white flashes (similar to the perception of black stimuli) or not seen at all. However, in some regions responses were more mixed. At location 3B (Table 1) stimuli were reported as being seen four times only in the 22 black and 12 white trials. Black stimuli were seen once as white and twice as black, whereas a white stimulus was seen once as white. At location 3E (Table 1) 22 black stimuli were perceived 10 times as black and 7 times as white.

In the intact field, KF perceived the black or white stimulus normally as black or white (see 7D, 8A, Table 1) in all the tests. Stimuli presented in the natural blind spot

Table 1. Number of Times White and Black Stimuli Were Seen in the Left (Right) Eye of Subject KF

	White Stimulus Presented								*Black Stimulus Presented*							
Location	*White*		*Black*		*Not Seen*		*Total*		*Black*		*White*		*Not Seen*		*Total*	
1A	0	(2)	0	(0)	2	(0)	2	(2)	0	(0)	0	(2)	2	(0)	2	(2)
1B*	2	(3)	0	(0)	0	(0)	2	(3)	0	(0)	1	(3)	1	(0)	2	(3)
1C*R	9	(1)	0	(0)	3	(1)	12	(2)	0	(0)	11	(9)	11	(13)	22	(22)
1D*	1	(2)	0	(0)	1	(0)	2	(2)	0	(1)	1	(1)	1	(0)	2	(2)
1E*	1		0		1		2		0		2		0		2	
1F	2		0		0		2		0		0		2		2	
1G*	1		0		1		2		0		2		0		2	
2A	0	(4)	0	(0)	2	(0)	2	(4)	0	(2)	0	(2)	2	(0)	2	(4)
2B*R	3	(2)	0	(0)	9	(0)	12	(2)	0	(12)	5	(0)	17	(0)	22	(12)
2C*	1	(4)	0	(0)	1	(0)	2	(4)	1	(2)	1	(2)	0	(0)	2	(4)
2D*R	12	(2)	0	(0)	0	(0)	12	(2)	1	(1)	19	(0)	2	(1)	22	(2)
2E*	2		0		0		2		1		1		0		2	
2F	0		0		2		2		0		0		2		2	
2G	0		0		2		2		0		0		2		2	
3A	0		0		2		2		0		0		2		2	
3B*R	1	(2)	0	(0)	11	(0)	12	(2)	2	(2)	1	(0)	19	(0)	22	(2)
3C*	2	(2)	0	(0)	0	(0)	2	(2)	1	(2)	1	(0)	0	(0)	2	(2)
3D*	2	(2)	0	(0)	0	(0)	2	(2)	0	(2)	1	(0)	1	(0)	2	(2)
3E*R	11		0		1		12		10		7		5		22	
3F	1		0		1		2		1		0		1		2	
3G	2		0		0		2		2		0		0		2	
4A	0	(2)	0	(0)	2	(0)	2	(2)	0	(2)	0	(0)	2	(0)	2	(2)
4B	0		0		2		2		1		0		1		2	
4C	1	(2)	0	(0)	1	(0)	2	(2)	2	(2)	0	(0)	0	(0)	2	(2)
4D	2		0		0		2		2		0		0		2	
4E*	2		0		0		2		1		1		0		2	
4F	2		0		0		2		2		0		0		2	
4G	2		0		0		2		2		0		0		2	
5A	2		0		0		2		1		0		1		2	
5BR	0		0		2		2		6		0		6		12	
5C*	1		0		1		2		1		1		0		2	
7DR	12		0		0		12		22		0		0		22	
8AR	2		0		0		2		12		0		0		12	

Note. Data from test and retest trials (R). Locations where black stimuli were perceived as white in the left eye are indicated with an asterisk. Results from the right eye are in parentheses. All locations from the upper left quadrant and some other selected locations are shown.

(5B; Table 1) were seen six times (12 trials were conducted), each time perceived as black.

Right Eye

In the test and retest, 20 locations were chosen for the right eye. Thirteen were located in the upper left quadrant (see Table 1). Nine of these 13 locations were situated in the area where black stimuli were seen as white in the left eye. At four of these locations the results for the right eye were comparable to those for the left eye (1B, 1C, 1D, and 2C; Table 1). However, in contrast to the left eye, black stimuli were also seen as white by the right eye at locations 1A and 2A (Table 1) and a black stimulus was always seen as black at locations 2B, 2D, 3B, 3C, and 3D (Table 1). If seen at all, white stimuli were always perceived as white.

DISCUSSION

Normal Control Subjects

Normal subjects were never able to see the visual stimulus when it was presented in the optic disc, and their guessing performance in the natural blind spot was at chance. These findings therefore indicated that the equipment was not producing artifacts and that there

was no stray light falling on retinal regions outside the optic disc that subjects could have been perceiving.

Residual Vision

According to the Humphrey perimetry charts, obtained from a test before and after the experimental sessions in our lab, KF was totally blind in the upper left visual field to an area that partially included the blind spot. However, together with the results of our experimental test, three qualitatively different visual areas could be distinguished in his clinically blind visual field. At individual test spots, KF was either (1) absolutely blind (confirming the Humphrey test results), (2) could see our experimental stimulus normally, or (3) could see our experimental stimulus abnormally (perceiving black and white stimuli as a light flash). Interestingly, initially KF did not report the upper left stimuli at all but responded as if he were blind. He reported that he had not acknowledged the stimuli because they looked different from the ones "he was supposed to detect." When he reported his visual experience first he believed that the flash was caused by a reflection on the screen from the ceiling lights. This result shows that residual vision can easily be missed. Only suitable stimuli and the constant request for reporting any peculiar perception may lead to its detection. In addition, an informal, friendly atmosphere may also be essential for the reporting of subjective, unusual sensations (Schärli et al., 1999).

Subject KF described the abnormal perception of the stimulus as a light flash that was poorly defined in shape. It is not clear why the stimulus was perceived as light whereas other studies that found residual vision reported the perception of something dark such as a "dark shadow" or a feeling only. Perceiving a black stimulus as white perhaps suggests that, in this subject, the perception of sudden luminance change from light to dark (offset) is defective whereas dark to light (onset) can still be processed. The characteristics of KF's residual vision could be investigated by presenting long-duration black stimuli to see whether he was able to detect the change at the beginning (black) or at the end (white) of the stimulus. An alternative test might be to present four different stimuli with different time-luminance patterns: (1) slow increase and sudden decrease, (2) slow decrease and sudden increase, (3) sudden increase and slow decrease, and (4) sudden decrease and sudden increase. If our hypothesis that the perception of a sudden luminance change from light to dark is defective, whereas dark to light can still be processed, is correct, we would expect the following: KF would be unable to see the sudden decrease in conditions (1) and (4) but would perceive the sudden increase in conditions (2) and (3) as a white flash. By altering the luminance increase/decrease-time relationship, the temporal dependence on perception could also be investigated.

Interestingly, KF's perception of white flashes appears to be similar to the way in which direct electrical stimulation of the striate and extrastriate cortex has been perceived by subjects. Direct stimulation apparently leads to the perception of small circumscribed phosphenes, described as being like white sparks or flashes (Penfield & Rasmussen, 1952). One hypothesis might be that KF's similar sensations in a small area of the visual field are the result of visual stimulation activating small numbers of spared cells in the striate (or extrastriate) cortex.

KF appeared to be able to perceive a single feature of the stimulus only. In the clinic it is well known that a progressive visual defect first impairs shape and color perception before it affects size, movement, or brightness perception. The reverse takes place during recovery (Grüsser & Landis, 1991). In a recovering visual field, the perception of movement has been reported to reappear first (Riddoch, 1917). Similarly, the detection of moving stimuli and bright flashes (but not color and stationary objects) was observed in a recovering field 7 months after the accident (Perenin, Ruel, & Hecaen, 1980). The subject "could probably see weakly;" however, he behaved as if he were completely blind in everyday life. Therefore, possibly the most robust feature of a stimulus is movement or simply the perception of light. In comparison to the Humphrey stimulus, our stimulus type was a flicker, was larger, and was presented for a shorter duration. Presumably, then, some feature present in our stimulus but not in the Humphrey stimulus, such as larger size or several onsets and/or offsets (flicker), were the features still able to be perceived in some regions of the visual field.

Alternatively, the acoustic warning sound and the presentation tone, together with verbal reports and individually controlled presentations, that were used in this study may have facilitated the focusing of attention and consequently may have led to a better performance. Therefore, the difference in experimental procedure in the two tests, not the stimulus type, may have caused the different performance in the upper left area. It has been shown previously, in discrimination tasks, that the threshold is lower if the location in visual space or the temporal interval of stimulus presentation is known (Westheimer & Ley, 1996). Moreover, it has recently been found that in some locations, subject GY was only above chance when he was informed about the stimulus location (Kentridge, Heywood, & Weiskrantz, 1997).

Inconsistent Responses

Interestingly, subject KF occasionally reported that he saw the stimulus when it was presented in the natural blind spot region. Furthermore, we did observe, on one examination with the Humphrey test, that KF responded to a stimulus presented in the blind spot. It is possible that KF used an eccentric retinal point for fixation (Verhoeff, 1943). The eye monitoring technique employed

here could detect eye movements of as little as 1° but could not detect extracentral fixation. We are confident that there were no eye movements during trials. Eccentric fixation is common but usually less than 1.5° in hemianopic or commissurotomized subjects, and miniature eye movements occur in about 4 to 10% of trials (Sugishita, Hamilton, Sakuma, & Hemmi, 1994; Sugishita, Hemmi, Sakuma, Beppu, & Shiokawa, 1993). Therefore, a slightly abnormal position of KF's natural blind spot relative to the expected position, caused by eccentric fixation or an anatomical abnormality, could well have caused the occasional perception of the stimulus in the blind spot. We would probably not have been able to detect eccentric fixation as an offset position of the blind spot because the blind spot was already located right at the edge of our experimental field and almost within a blind region of the visual field.

Subject KF's performance was inconsistent at several locations, and it is well known that performance fluctuates when a task requires concentration. The inconsistency suggests that KF may have perceived stimuli only very weakly at these locations. Although it must be remembered that KF was 70 years old, it is also possible that he had attentional problems with a consequent fluctuation in concentration. Parietal lesions have been generally linked with a pathological disorder of attention (Petersen, Robinson, & Currie, 1989). KF's brain damage involved, in part, the temporo-parietal area (in the left hemisphere), an area that is known for its importance in spatial attention. In any case, his most valuable data, seeing black stimuli as white flashes in the upper-left "blind" visual field, is not related to these problems. His reports of white flashes were fairly consistent and restricted to one area.

Responsible Neural Area

The results from the left and right eye show that the phenomenon of seeing black stimuli as white flashes roughly corresponded in the two eyes and was therefore not a retinal effect. It strongly indicates that some part of KF's postgeniculate lesion in the visual cortex is responsible for the phenomenon. Subject KF's residual vision of seeing light flashes was restricted to a small area in the upper left quadrant. The lower right occipital lobe that is responsible for the upper left visual field showed patchy damage in areas 17 and 18. This finding suggests that his residual vision in the upper left visual field is mediated by a small area in the visual cortex that may be only partly damaged and corresponds to the site of abnormal perception. In line with this finding, some studies on residual vision (perception of motion) found that it was frequently restricted to one region of the affected visual field (Plant & Wilkins, 1988; Riddoch, 1917). Interestingly, residual vision without conscious awareness (blindsight) has also been found to be restricted to isolated areas of the visual field. The authors of these studies suggested that isolated spared visual cortex or optic radiation may be responsible for blindsight (Fendrich, Wessinger, & Gazzaniga, 1992; Wessinger, Fendrich, & Gazzaniga, 1997; Schärli et al., 1999).

It is possible that residual vision and blindsight originate from the same neural substrate. Both blindsight and residual vision have been shown to exist in the blind visual field of subject GY, who has been investigated on a number of occasions by several authors. This subject showed many residual visual abilities, with or without conscious awareness, depending on the stimulus employed (Barbur, Forsyth, & Findlay, 1988; Barbur, Harlow, & Weiskrantz, 1994; Barbur et al., 1980; Blythe, Branley, Kennard, & Ruddock, 1986; Blythe et al., 1987; Weiskrantz, Barbur, & Sahraie, 1995; Weiskrantz, Harlow, & Barbur, 1991). The level of impairment may be related to the amount of spared tissue in one small area or the severity of destruction over a larger area in the striate cortex or optic radiation. Strong, salient stimuli may still produce degraded vision and low-level performance in certain subjects (Schärli et al., 1999; Wessinger et al., 1997). Depending on the size and site of the spared area (and the spared connections to extrastriate cortex), visual performance may be normal or abnormal, with or without conscious awareness for certain stimuli. These observations suggest that the spared striate cortex may be responsible for the residual vision described in subject KF. It has been shown that the "residual visual performance" phenomenon (denying any or most visual sensitivity but performing above chance) also exists in normal subjects or neurological patients with other (nonvisual) impairments (Schärli et al., 1999). This is in line with the view that the phenomenon may generally be caused by degraded sensitivity.

It is unlikely that the midbrain, striate, or extrastriate cortex as a whole was responsible for KF's residual vision because it did not occur at all the locations tested. Moreover, in a recent study, the majority of subjects with postgeniculate lesions, tested extensively under the same conditions, did not show any ability to perceive visual stimuli in their blind visual fields. This was the case even in two subjects with lesions that were most likely restricted either to the striate cortex or optic radiation (Schärli et al., 1999). In general, it is rare to find residual sensitivity to visual stimuli in perimetrically blind visual areas of humans. In addition, no residual visual performance was found in a number of subjects whose lesion appeared to be restricted to V1 (Gazzaniga et al., 1994; Schärli et al., 1999).

An alternative explanation for the residual vision is plasticity. *Plasticity* means the taking over of or growth into a damaged or denervated brain region by neighboring neurons and appears to take place in a limited way in the cortex, particularly in younger individuals and over a long period of time (Blythe et al., 1987; Gazzaniga et al., 1994; Payne et al., 1996). However, it seems unlikely that plasticity is responsible for the residual vision

we saw in this subject. KF was completely blind in most regions of the visual field surrounding the residual vision. Therefore, we assume that the surrounding cortical regions were mostly completely damaged and unlikely to regrow into neighboring regions. Furthermore, plasticity seems to be less likely in an older subject, KF was over 60 when he had his strokes.

The possibility cannot be excluded that, in subject KF, a small number of spared isolated fibers from the dLGN or pulvinar to extrastriate cortex were responsible for the residual vision. Recently, tests on subject GY using different stimulus parameters, revealed that, at 2 out of 15 locations, performance was always at chance (Kentridge et al., 1997). The authors suggested a parallel extrastriate pathway responsible for the phenomenon. Thorough testing at many locations in the impaired visual field of more subjects with well-described postgeniculate lesions is needed to resolve the present issues in residual vision and blindsight.

METHODS

The subjects' consent was obtained and the research was approved by the Committee for Human Rights of the University of Western Australia and complies with NH & MRC guidelines.

Normal Control Subjects

A yes/no and temporal forced-choice test were performed on six control subjects to test the equipment and obtain control data (Schärli et al., 1999). The natural blind spot was used to simulate the "affected" region.

Visually Impaired Subject

Subject KF (69-year-old male) was born in 1926 and suffered two strokes, causing bilateral occipital and left parieto-temporal brain damage. He had his first stroke in May 1991, which temporarily affected his motor functions. After his second stroke, in June 1994, he was blind in both hemifields for more than a month. He then bumped into a wall and claims he could suddenly see again in parts of his visual field (first "misty" but then more clearly within a few days). He was tested in our lab in September and October (1995) and in April (1996). KF was well motivated, patient, and reliable. His visual orientation was remarkably good, and he walked with confidence. Apart from his visual impairment, his left hearing was also slightly impaired. KF participated voluntarily and apart from travel expenses, no money was paid. Informed consent was obtained before testing.

Lesion Analysis

Brain damage was assessed as carefully as possible using the available CAT scans. The extent was estimated and outlined on templates (Damasio & Damasio, 1989). See Figure 1.

Clinical Perimetry

Each eye was tested on a standard Humphrey Field Analyzer (stimulus size, 25.9 min; presentation time, 200 msec; intensity, 0.25 to 3183 cd/m^2; background luminance, 10 cd/m^2) before and after experimental examination in our lab. The central visual field tested was 45 × 50° in size.

Experimental Apparatus and Stimuli

A personal computer generated the stimuli and presented them on a 70-Hz refresh rate display. The stimulus was a dark or light disc (1° in diameter) that appeared three times for 14.3 msec in 100 msec on a light gray background (flicker stimulus). The two intervals in between lasted for 28.6 msec each and were blank (background luminance). The stimulus appeared either almost black or almost white and will therefore be referred to as black or white from now on. The stimulus contrast chosen was relatively low. The luminance for white was 82 cd/m^2, and the luminance for black was 12 cd/m^2. The background luminance was equidistant from the white and black stimulus shades (47 cd/m^2). The tests were conducted under constant photopic light conditions. The experimental setting minimized screen reflections, after-images, eye movement during stimulus presentation, and the possibility of scattered light falling into intact regions.

The subject sat 0.5 m from a PC screen, with one eye covered with a patch (monocular stimulus presentation). A warning sound before the stimulus presentation indicated to the subject when to look at the fixation cross. The subject was instructed to look at the fixation cross and not to move the head, which was stabilized by a chin and forehead rest. On every trial, the investigator could observe the subject's fixating eye on a TV screen, which was monitored and magnified by a video camera. Fixation control was assisted by the light reflection on the pupil and the help of a reference grid drawn on the screen. Eye movements of a magnitude of about 1° could be detected with this equipment. The trial was discarded whenever eye or head movement between warning sound and stimulus presentation was detected. The rejected stimulus was presented again in a later trial.

Stimuli and procedures used have been described in more detail in an earlier study (Schärli et al., 1999).

Pretests

Subject KF was first tested using a yes/no and temporal forced choice task to determine the limits of the intact visual field and to test for residual visual performance in

the blind visual area. The two tests were part of an earlier study (for more details, see Schärli et al., 1999).

Tests

Many locations in both the blind and intact regions of the visual field were tested to find out whether performance differed at a variety of locations. The subject's left eye was tested thoroughly at 112 locations in the central 21 × 37° of the visual field (see Figure 2), presenting black and white stimuli. In one block, 12 locations (distributed over the screen) were chosen and tested on four trials at each location (two black and two white stimuli). To test for correspondence in the two eyes, 20 locations were also tested in the right eye (1A, 1B, 1C, 1D, 1H, 1K, 2A, 2B, 2C, 2D, 3B, 3C, 3D, 4A, 4C, 5I, 7I, 8B, 8D, and 8K; see Figure 2). The subject was instructed to respond orally whether (1) he saw the stimulus or not, using confidence levels 1 to 5 (1 = seen, very certain; 2 = seen, certain; 3 = cannot decide; 4 = not seen, uncertain; 5 = not seen, very certain) and (2) what color the stimulus appeared to be. KF was encouraged to use all confidence levels.

Retests

A few locations were tested more thoroughly in blocks of 10 trials. Twenty black and 10 white stimuli were presented at each of five locations in the upper left impaired visual field (1C, 2B, 2D, 3B, and 3E; Figure 2). At locations 5B and 8A (Figure 2) only 10 black stimuli were presented. The natural blind spot (5B; Figure 2) and two locations in the intact field were tested as controls (7D and 8A; Figure 2).

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REFERENCES

Barbur, J. L., Forsyth, P. M., & Findlay, J. M. (1988). Human saccadic eye movements in the absence of the geniculocalcarine projection. *Brain, 111,* 63–82.

Barbur, J. L., Harlow, A. J., & Weiskrantz, L. (1994). Spatial and temporal response properties of residual vision in a case of hemianopia. *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences, 343,* 157–166.

Barbur, J. L., Ruddock, K. H., & Waterfield, V. H. (1980). Human visual responses in the absence of the geniculocalcarine projection. *Brain, 103,* 905–928.

Blythe, I. M., Branley, J. M., Kennard, C., & Ruddock, K. H. (1986). Visual discrimination of target displacement remains after damage to the striate cortex in humans. *Nature, 320,* 619–621.

Blythe, I. M., Kennard, C., & Ruddock, K. H. (1987). Residual vision in patients with retrogeniculate lesions of the visual pathways. *Brain, 110,* 887–905.

Bullier, J., Girard, P., & Salin, P.-A. (1994). The role of area 17 in the transfer of information to extrastriate visual cortex. In A. Peters & K. S. Rockland (Eds.), *Primary visual cortex in primates* (vol. 10, pp. 301–330). New York, London: Plenum Press.

Campion, J., Latto, R., & Smith, Y. M. (1983). Is blindsight an effect of scattered light, spared cortex and near-threshold vision? *Behavioral and Brain Sciences, 3,* 423–486.

Celesia, G. G., Bushnell, D., Toleikis, S. C., & Brigell, M. G. (1991). Cortical blindness and residual vision: Is the "second" visual system in humans capable of more than rudimentary visual perception? *Neurology, 41,* 862–869.

Damasio, H. (1995). *Human brain anatomy in computerized images.* New York: Oxford University Press.

Damasio, H., & Damasio, A. R. (1989). *Lesion analysis in neuropsychology.* New York: Oxford University Press.

Fendrich, R., Wessinger, C. M., & Gazzaniga, M. S. (1992). Residual vision in a scotoma: Implications for blindsight. *Science, 258,* 1489–1491.

Gazzaniga, M. S., Fendrich, R., & Wessinger, C. M. (1994). Blindsight reconsidered. *Current Directions in Psychological Science, 3,* 93–96.

Grüsser, O. J., & Landis, T. (1991). *Visual agnosias and other disturbances of visual perception and cognition* (vol. 12). London: Macmillan Press.

Holmes, G. (1918). Disturbances of vision by cerebral lesions. *British Journal of Ophthalmology, 2,* 353–384.

Inouye, T. (1909). *Die sehstöhrungen bei schussverletzungen der kortikalen sehsphähre. nach beobachtungen an verwundeten der letzten japanischen kriege.* (2nd ed.). Leipzig: Wilhelm Engelmann.

Kentridge, R. W., Heywood, C. A., & Weiskrantz, L. (1997). Residual vision in multiple retinal locations within a scotoma: Implications for blindsight. *Journal of Cognitive Neuroscience, 9,* 191–202.

Kisvárday, Z. F., Cowey, A., Stoerig, P., & Somogyi, P. (1991). Direct and indirect retinal input into degenerated dorsal lateral geniculate nucleus after striate cortical removal in monkey: Implications for residual vision. *Experimental Brain Research, 86,* 271–292.

Milner, A. D., & Goodale, M. A. (1995). *The visual brain in action.* Oxford: Oxford University Press.

Payne, B. R., Lomber, S. G., Macneil, A., & Cornwell, P. (1996). Perspective evidence for greater sight in blindsight following damage of primary visual cortex early in life. *Neuropsychologia, 34,* 741–774.

Penfield, W., & Rasmussen, T. (1952). *The cerebral cortex of man*. New York: Macmillan.

Perenin, M. T. (1991). Discrimination of motion direction in perimetrically blind fields. *NeuroReport, 2,* 397–400.

Perenin, M. T., Ruel, J., & Hecaen, H. (1980). Residual visual capacities in a case of cortical blindness. *Cortex, 16,* 605–612.

Petersen, S. E., Robinson, D. L., & Currie, J. N. (1989). Influences of lesions of parietal cortex on visual spatial at-

tention in humans. *Experimental Brain Research, 76,* 267–280.
Plant, G. T., & Wilkins, A. J. (1988). Preserved movement sensitivity following occipital lobe damage: a case report of spatio-temporal contrast sensitivity in the Riddoch phenomenon. *Clinical Vision Science, 2,* 321–329.
Riddoch, G. (1917). Dissociation of visual perceptions due to occipital injuries, with especial reference to appreciation of movement. *Brain, 40,* 15–57.
Schärli, H. (1997). *Blindsight in subjects with homonymous visual field defects.* Unpublished dissertation, University of Western Australia, Perth.
Schärli, H., Harman, A. M., & Hogben, J. H. (1995), Testing residual vision in patients with striate cortex lesions (abstract). *Proceedings of the Australian Neuroscience Society,* February 1–3, Perth (Australia), *6,* 192.
Schärli, H., Harman, A. M., & Hogben, J. H. (1996). Residual vision in patients with damaged optic radiation or striate cortex (abstract). *First Kongress der Neurowissenschaftlichen Gesellschaft,* February 24–27, Berlin.
Schärli, H., Harman, A. M., & Hogben, J. H. (1999). Blindsight in subjects with homonymous visual field defects. *Journal of Cognitive Neuroscience, 11,* 52–66.
Sugishita, M., Hamilton, C. R., Sakuma, I., & Hemmi, I. (1994). Hemispheric representation of the central retina of commissurotomised subjects. *Neuropsychologia, 32,* 399–415.
Sugishita, M., Hemmi, I., Sakuma, I., Beppu, H., & Shiokawa, Y. (1993). The problem of macular sparing after unilateral occipital lesions. *Journal of Neurology, 241,* 1–9.
Teuber, H.-L., Battersby, W. S., & Bender, M. B. (1960). *Visual field defects after penetrating missile wounds of the brain.* Cambridge, MA: Harvard University Press.
Verhoeff, F. H. (1943). A new answer to the question of macular sparing. *Archives of Ophthalmology, 30,* 421–425.
Weiskrantz, L., Barbur, J. L., & Sahraie, A. (1995). Parameters affecting conscious versus unconscious visual discrimination with damage to the visual cortex (V1). *Proceedings of the National Academy of Sciences of the United States of America, 92,* 6122–6126.
Weiskrantz, L., Harlow, L., & Barbur, J. L. (1991). Factors affecting visual sensitivity in a hemianopic subject. *Brain, 114,* 2269–2282.
Wessinger, C. M., Fendrich, R., & Gazzaniga, M. S. (1997). Islands of residual vision in hemianopic patients. *Journal of Cognitive Neuroscience, 9,* 203–221.
Westheimer, G., & Ley, E. (1996). Temporal uncertainty effects on orientation discrimination and stereoscopic thresholds. *Optical Society of America, 13,* 884–886.