Blindsight in Subjects with Homonymous Visual Field Defects

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Abstract

■ Brain damage in the visual system can lead to apparently blind visual areas. However, more elaborate testing indicates that some visual ability may still exist for specific stimuli in the otherwise blind regions. This phenomenon is called "blindsight" if subjects report no conscious awareness of visual stimuli but when forced to guess, nevertheless perform better than chance. It has mainly been suggested that secondary visual pathways are responsible for this phenomenon. However, no published study has clearly shown the neural mechanism responsible for blindsight. Furthermore, experimental artifacts may have been responsible for the appearance of the phenomenon in some subjects. In the present study, the visual fields of nine subjects were mapped and residual visual performance was examined in many areas using three different experimental procedures. Artifacts such as stray light or eye movements were well controlled. In addition, confidence ratings were required after each trial in the forced-choice tests. The results show that only one subject with a lesion in the optic radiation had blindsight in two discrete areas of the affected visual field. Spared optic radiation fibers of the main (primary) geniculo-striate visual pathway were most likely to account for this finding. ■

INTRODUCTION

A lesion in the optic tract, lateral geniculate nucleus, optic radiation, or striate cortex (V1) generally leads to blindness in that area of the visual field that corresponds retinotopically to the lesion side (Holmes, 1918; Inouye, 1909). Magnetic resonance imaging (MRI) studies have recently suggested that a lesion in V2/V3 (sparing V1) also produces a homonymous visual field defect (Horton & Hoyt, 1991). The existence of residual (conscious) vision (often the perception of motion) in these affected areas has been claimed for about 80 years (Holmes, 1918; Poppelreuter, 1917; Riddoch, 1917). In the last 25 years, residual performance in visually impaired subjects who suffer from postgeniculate lesions has been confirmed and shown to vary, quantitatively and qualitatively, between subjects. Curiously, some subjects appear to have residual visual functions and yet are not consciously aware of them (Pöppel, Held, & Frost, 1973). This subconscious visual ability was typically inferred from the above-chance performance in a forced-choice task in which the subject claimed to be guessing and has been termed "blindsight" (Weiskrantz, Warrington, Sanders, & Marshall, 1974). Since then many studies have described blindsight, using a number of methods. For reviews on blindsight, see Campion, Latto, and Smith (1983), Weiskrantz (1986), and Weiskrantz (1996).

In monkeys, many parallel, subcortical visual pathways are known that could bypass the damaged visual cortex and be responsible for blindsight (Bullier, Girard, & Salin, 1994; Stoerig & Cowey, 1993; Weiskrantz, 1986). Until recently, the retino-colliculo-pulvinar-extrastriate pathway was favored by many investigators (Perenin & Jeannerod, 1975; Perenin, Ruel, & Hecaen, 1980; Stoerig, 1996). Recent research favors the involvement of the occipito-parietal system, MT, STP, or V3A, and its associated subcortical areas (Bullier et al., 1994; Kisvárday, Cowey, Stoerig, & Somogyi, 1991; Milner & Goodale, 1995; Payne, Lomber, Macneil, & Cornwell, 1996). The occipito-parietal region could receive the input from the retina either via the pulvinar, colliculo-pulvinar, geniculate, or colliculo-geniculo pathway. Besides these systems a number of other theories are possible. Plasticity may have occurred, allowing reorganization of receptive visual fields (Celesia, Bushnell, Toleikis, & Brigell, 1991). In subjects blinded by V2/V3 damage the striate cortex or a striate-extrastriate pathway (e.g., V1-MT pathway) may mediate residual visual performance (Horton & Hoyt, 1991). Recent results with visually impaired subjects indicate that isolated "islands" of the striate cortex may cause patches of degraded vision and be responsible for blindsight (Fendrich, Wessinger, & Gazzaniga, 1992; Wessinger, Fendrich, & Gazzaniga, 1997). No study to date could clearly exclude any of these regions.

The blindsight data is difficult to compare and evaluate because methods of study and residual visual performance have varied between subjects. Moreover, the brain lesion is unique in each subject tested. A great deal

of the published data in blindsight is provided by a handful of selected subjects tested by a few investigators. Artifacts such as light scatter, criterion effects, macular sparing, eccentric fixation, and small occasional eye movements are difficult to control for and may have led to the successful performance reported in some studies (see Campion et al., 1983; Gazzaniga, Fendrich, & Wessinger, 1994, for a discussion on this topic).

Recently, studies on hemispherectomized subjects showed again that light scatter (King, Azzopardi, Cowey, Oxbury, & Oxbury, 1996) or intraocular reflection and diffusion (Stoerig, Faubert, Ptito, Diaconu, & Ptito, 1996) can be a serious problem in visual tests. Stray light has often been claimed to cause blindsight because many authors tested their subjects under conditions that favored scatter effects (e.g., white on dark stimuli presented under mesopic or scotopic conditions, large- or high-contrast stimuli) often without using adequate controls. Another great potential problem is the effect of criterion (Campion et al., 1983; Gazzaniga et al., 1994). Reports on perceptual awareness were usually obtained, after many trials, in a rest break or gathered from a different test (e.g., perimetry) that was carried out previously using different stimuli and procedures (Holtzman, 1984). It is possible that occasional failures to respond "yes" when uncertain may have given a false impression of blindsight when in fact the subject actually did have residual vision (i.e., conscious awareness of the stimulus). Only two recent blindsight studies used confidence ratings after each trial (Fendrich et al., 1992; Weiskrantz, Barbur, & Sahraie, 1995). The effect of criterion has recently been addressed in blindsight subject GY, using signal detection theory (Azzopardi & Cowey, 1997).

The search for the responsible neural substrate in blindsight has been based mainly on monkey studies. The results from monkeys with striate cortex lesions show that training generally leads to good recovery from the damage (Dineen & Keating, 1981; Pasik & Pasik, 1982). Monkeys can consistently detect, localize, and discriminate visual stimuli, indicating that extrastriate pathways are able to mediate complex visual abilities. By contrast, residual performance to visual stimuli in perimetrically blind visual areas of humans with postgeniculate lesions appears to be rare and weak. It has been suggested that additional extrastriate damage may be responsible for the rarity of blindsight in humans (Weiskrantz, 1996). However, extended striate damage including visual areas 18 and 19 does not lead to total blindness in monkeys (Denny-Brown & Chambers, 1976; Pasik & Pasik, 1971). This indicates that one should be cautious in simply extrapolating from monkey data to humans (Fendrich, Wessinger, & Gazzaniga, 1993).

This study investigated the neural basis for blindsight by examining nine subjects who had a variety of lesions in the visual system and comparing their residual visual performance with lesion site located by MRI or computerized axial tomography (CAT) scan. We used procedures designed to eliminate or minimize artifacts such as eye movements, stray light, or altered response criterion.

This project has been 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

In the yes/no test, results for all six normal subjects showed that a stimulus presented in the natural blind spot could not be seen (6 to 63 presentations for each subject; 99% correct "no" responses; mean confidence level = 4.9). When the stimulus was presented in the visual field, subjects responded "yes" (12 to 134 presentations for each subject; 99% correct "yes" responses; mean confidence level = 5.0). For the temporal forced-choice test the performance for stimuli presented in the natural blind spot of all six subjects was at chance level (6 to 70 presentations for each subject; 50% correct responses; mean confidence level = 1.1). However, when the stimulus was presented in other parts of the visual field, their performance was almost faultless (18 to 146 presentations for each subject; 97% correct responses; mean confidence level = 4.9). In the spatial summation test all of the 22 tested subjects showed a faster median reaction time for double stimuli than for single ones. In 17 subjects, the reaction time was significantly faster for doubles than for singles (Mann-Whitney-tests; $p < 0.05$). When averaged over all 22 subjects, the reaction time for double stimuli was significantly faster than for single ones (double, 291 msec; single, 306 msec; $t = 10.14$; $p < 0.0001$).

Visually Impaired Subjects

Lesion Analysis

One subject was found to have a lesion in the LGN-optic tract area (subject AN) and two, in the optic radiation (subjects BG and PM). The remaining six subjects had damaged visual cortex (see Table 1). CAT scans do not allow a high-resolution analysis of damage in the occipital lobe because of limited resolution and because the brain structure varies from person to person (Damasio, 1995). For subject RB an MRI scan was available, allowing a more precise evaluation. Figure 1 shows the estimated site and size of the lesion of each subject tested.

In general, the yes/no visual field test provided a similar but more accurate map of vision than the Humphrey test map (Figures 1 and 2). All subjects were mostly very certain about their "yes" or "no" response

Table 1. Description of the Visually Impaired Subjects

Subject	*Age*	*TSA*[a]	*Sex*[a]	*Lesion and Aetiology*[a]	*Visual Lesion*	*Affected Visual Field*[b]
RB	44	6–15 m	M	Left, small occipital infarction (L-PCA) (calcarine artery)	Left upper area 17 (V2?)	Right lower quadrantanopia
HC	62	20–28 m	F	Right occipital infarction (R-PCA)	Right area 17, 18, 19	Left homonymous hemianopia
KF	69	15–22 m	M	Bilateral infarctions		
				Left temporo-parietal and left/right occipital	Left/right area 17, 18 (patchy)	Left upper & right lower quadrantanopia
				Right cerebellar hemisphere (small) Bilateral frontal lobe atrophy		Partial damage in the two other quadrants
AH	74	5 y	M	Bilateral infarctions Left/right occipital and right parietal	Left/right area 17, 18, 19 (patchy)	Mostly left lower quadrantanopia
PC	51	5 y	M	Left occipital infarction (L-PCA)	Left area 17, 18, 19	Right homonymous hemianopia
AS	77	5y	F	Right occipital-parietal infarction (R-PCA) (possibly with a haemorrhagic component)	Left area 17, 18	Left homonymous hemianopia
PM	47	32 y	M	Bilateral infarction (accident) Right anterior temporal and inferior frontal Left posterior occipital Bilateral cerebellar hemispheres (small)	Left optic radiation	Right homonymous hemianopia
BG	35	5 y	F	Left, small infarction (accident) (postgeniculate)	Left optic radiation (anteriorly)	Right homonymous hemianopia
AN	24	9 y	M	Right infarction, ischaemic, avascular necrosis (accident) Right thalamus and adjacent internal capsule Right frontal, parietal and temporal Right optic nerve	Right optic tract Possibly right LGN	Left hemifield defect in left eye (right eye is blind)

[a] L (R)-MCA/PCA, left (right) middle/posterior cerebral artery infarction; y, year(s); m, month(s); M, male; l F, female; TSA, time passed since the accident.
[b] All field defects are homonymous.

(confidence level = 5). Only occasionally, confidence levels 1 to 4 were chosen. There was no apparent difference between the detection performance of black and white stimuli in the visual field test of all subjects. The responses close to the vertical meridian were mostly very consistent, indicating a steep border between intact and affected visual fields. In addition, this result showed that the subjects fixated well. Curiously, one subject (KF) with visual damage in areas 17 and 18 showed evidence of residual (conscious) vision. He could still see white flashes in one area of the upper left quadrant when a black or white stimulus was presented using our experimental procedure; however, he appeared blind for the stimuli used in the two conducted Humphrey tests (Schärli, Harman, & Hogben, in press). Apart from subject PM (see below), nobody showed a consistent, above chance, performance for the blind visual field in the temporal forced-choice tests (FC-A and FC-B tests). Confidence level 5 (very certain) was mostly used for stimuli that were presented in the intact field and level

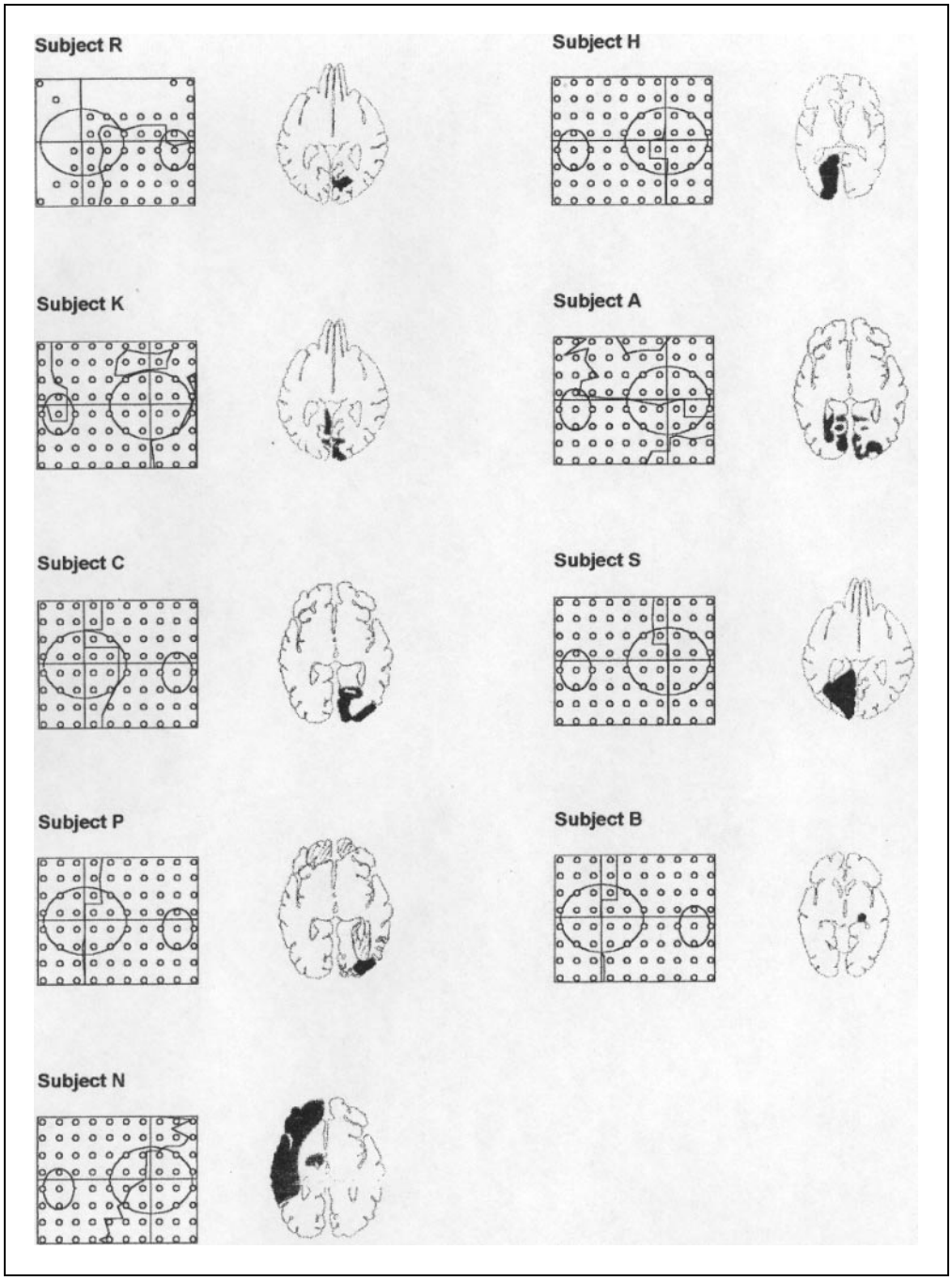

Figure 1. Sections through area 17 of the brain and visual field maps (yes/no test) for each subject.

1 (very uncertain) was used for stimuli that were presented in the affected field. Levels 2, 3 and 4 were used only occasionally. In the spatial summation test, the median reaction time to double stimuli was not faster than the median reaction time to single stimuli for any subject tested (at any individual location or summed over all locations). Many of the visually impaired subjects often missed stimuli (single and double ones). In most subjects a few single stimuli were also presented in the blind field. However, none of these subjects ever responded to a single presentation in the blind field.

Subject PM

In the yes/no test, subject PM occasionally reported that he was uncertain in some areas of the visual field and reported he "sensed" something. However, his performance was at chance at the locations that were initially tested in the temporal forced-choice test (FC-A test). Eighteen additional locations were chosen in regions of PM's hemianopic field where he occasionally reported to feel something in the yes/no test. They were tested extensively in 68 blocks of trials using a slightly different temporal forced-choice procedure (FC-B test). At seven

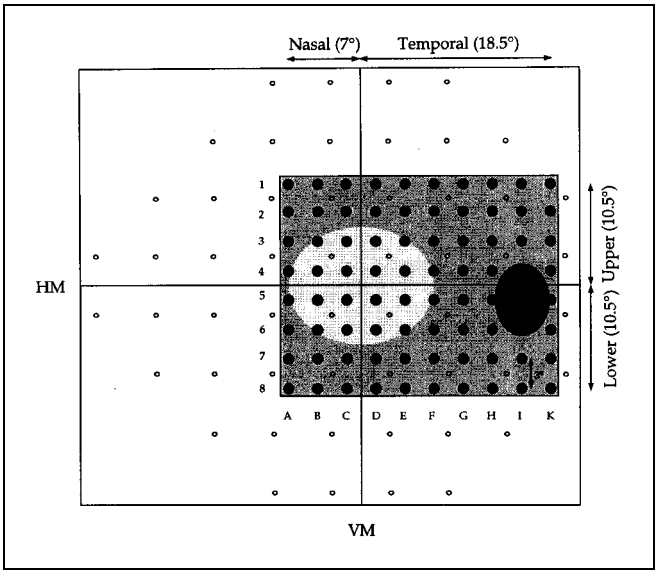

Figure 2. Dimensions and locations of the visual stimuli of the Humphrey visual field test (outer square, small circles) and the experimental computer screen (inner square, bigger black discs) drawn for the right eye. Horizontal meridian (HM) and vertical meridian (VM) meet in the fixation cross. The natural blind spot (dark oval in the temporal field) and macula (light region around the fixation cross) are also drawn.

of these locations, subject PM's performance was consistently and repeatedly above chance (locations 3F, 3G, 8D, 8E, 8F, 8G, and 8H, see Table 2). These above-chance locations are in two different areas in the upper and lower right visual field (Figure 3). The upper area appeared very small and was surrounded by locations with chance performance. The lower area was larger, borders the intact visual field, and may have extended further inferiorly.

Confidence

In general, subject PM found it hard to choose a confidence level. At 11 locations subject PM never reported that he saw or sensed the presented visual stimulus at any time (mean confidence level = 1), and his performance was at chance. By contrast, at the seven "above-chance" locations he occasionally sensed something and reported levels 2 to 4 sometimes (mean confidence > 1 at each of these seven locations; see Table 2).

Reports

Apart from the confidence level, it is important also to consider subject PM's verbal reports that were given after each block of trials (perception, see Table 2), although they allow a qualitative assessment only. After most blocks he reported that he did not *see* anything at all and that he was purely guessing on each trial. However, he admitted he *sensed* something sometimes (mostly very weakly). Subject PM had difficulties putting his experience into words. He was generally very uncertain about his performance and apparently needed a lot of concentration to pick the correct interval. He described his occasional perception as his sixth sense or "a feeling, which was neither visual nor auditory," "a feeling that you can have when something makes you turn around." Subject PM never perceived any color or shape in his affected visual field. His feelings were not the same in "strength" at all of the above-chance locations. Moreover, his feelings fluctuated at one stimulus location. Its strength was generally not correlated with overall performance.

On at least three blocks of trials, at locations 8G and 8H, subject PM never reported that he sensed anything at all, but his performance was clearly above chance. In four locations (3F, 8D, 8E, and 8F) he consistently reported after each block that he "had a feeling" on some trials as to whether the stimulus was presented in the first or second interval. At location 8H his reports varied a lot from block to block.

At two locations (3F and 8G), in the final block of trials, he occasionally reported he could "see something very weakly." At location 8G, in the first block he reported that he did not see anything at all and that he was only guessing. In the next two blocks he noted he sensed something sometimes and in the last three blocks (carried out 3 months later) he reported he saw something, very weakly sometimes. At location 3F, his perception was fairly stable until the last block of trials when he reported that he saw something very weakly sometimes. Interestingly, at the locations where his performance was at chance, he mostly had no "feelings" at all.

Training

In general, PM's performance did not improve with time. In addition, good performance at one location did not improve performance at another, different location. However, at two locations (3F and 8G) his perception changed with time (see above).

Feedback

In general, feedback did not appear to improve subject PM's performance; however, he felt more confident and motivated in blocks with feedback. At one location (8H) his performance was at chance in the first two blocks but above chance for the first time in the third block when he received feedback as to the correctness of his response. However, in the following blocks he no longer improved. At two other locations (3G and 8G), although his performance was at chance, he reported that he sensed something for the first time in the second block, which were trials with feedback.

Table 2. Subject PM's Above-Chance Locations

Location	*Feedback*	*Mean Confidence*	*Correct*	*%*	χ^2	*Significance*[a]	*Perception*[b]
3F	√	1.0	21	75	7.5	**	+
	–	1.0	18	64	1.9	*ns*	(+)
	√	1.0	26	93	20.5	***	(+)
	√	1.0	22	79	8.2	**	+
	–	1.7	24	86	14.3	***	+
	√	1.6	25	89	17.3	***	+++
			136	81	**64.4**	***	
3G	–	1.0	15	54	0.31	*ns*	–
	√	1.0	16	57	0.58	*ns*	+
	√	2.4	23	82	13.6	***	+
			54	**64**	**6.8**	**	
8D	–	1.1	21	75	7.5	**	+
	–	1.4	21	75	6.6	*	++
	√	1.0	24	86	14.6	***	+
			66	**79**	**27.2**	***	
8E	–	1.0	26	93	20.7	***	+
	√	1.0	28	100	28	***	++
	–	2.8	21	75	7	**	+
	√	1.9	27	96	24.2	***	++
			102	**91**	**76**	***	
8F	–	1.0	28	100	28	***	+
	√	1.0	27	96	24.3	***	++
	√	1.0	28	100	28	***	+
			83	**99**	**80.1**	***	
8G	–	1.0	20	71	5.2	*	–
	√	1.0	16	57	0.44	*ns*	+
	√	1.0	21	75	7	**	+
	–	2.6	27	96	24.2	***	+++
	√	2.4	24	86	13.68	***	+++
	√	3.0	25	89	17.31	***	+++
			133	**79**	**59.345**	***	
8H	–	1.1	15	54	0.08	*ns*	+
	–	1.1	15	54	0.53	*ns*	+
	√	1.0	23	82	8.4	**	+
	–	1.0	21	75	8.4	**	+
	√	1.0	16	57	0.22	*ns*	–
	√	1.0	22	79	8.8	**	–
	–	1.1	21	75	4.2	*	–
	√	1.0	16	57	0.74	*ns*	–
	√	1.0	16	57	0.07	*ns*	–
	–	1.6	22	79	8.8	**	(+)
	√	2.2	20	71	5.3	*	+
	√	1.3	18	64	4.4	*	+
			225	**67**	**35.8**	***	

[a] **Significance:** *, $0.5 > p > 0.01$; **, $0.01 > p > 0.001$; ***, $p > 0.001$.
[b] **Perception** is a qualitative measure of PM's perception over a block of trials (–, never seen, sensed, or felt; +, very weak feeling in some trials; (+), very weak feeling in some trials but very unsure; ++, feeling stronger or in more trials; +++, strong feeling in most trials or very weak seeing in some trials).

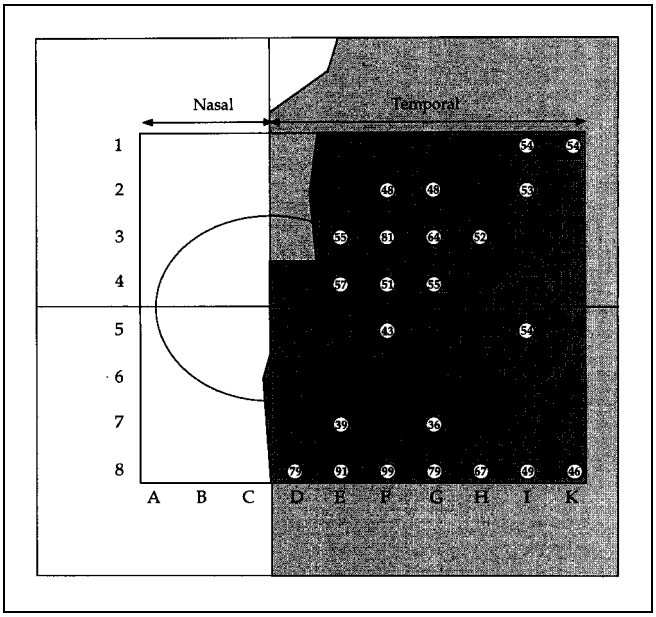

Figure 3. Temporal forced-choice tests (FC-A and FC-B tests) on subject PM's right eye. The percentage of correct responses of the selected locations are shown. The affected right temporal hemifield is drawn in shades according to the Humphrey test (light gray) and the experimental yes/no test (dark gray). Stars (*) indicate locations with overall above-chance performance (**, $0.01 > p > 0.001$; ***, $p > 0.001$).

Subject PM claimed that knowing the location of presentation and getting feedback made the task a little easier. After a few trials of stimuli presented at the same location, he could usually guess the location of the stimulus correctly (especially in feedback blocks). In three blocks, subject PM was informed about where the stimulus was presented. Interestingly, this did not lead to a better performance.

DISCUSSION

Experimental Controls

The results from normal subjects show that the experimental setup and design were a rigorous test for the subjects with homonymous visual field defects. Normal subjects could never 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 using. These findings also show that the subjects' fixation was in fact steady. In the spatial summation test, normal subjects consistently showed a faster reaction time for double stimuli than for single ones, confirming the results of a former study (Tassinari, Morelli, & Berlucchi, 1983). The map created by the yes/no test revealed a clear border between the intact and blind areas of all impaired subjects. This finding generally shows that fixation was maintained well. Even greater accuracy (<1°) could, in future, be achieved using an image stabilizer (Fendrich et al., 1992). Therefore, the size of a spared area could be determined even more accurately.

Blindsight

Evidence of blindsight was only found in subject PM using the temporal forced-choice test. This is in line with another study that also found residual visual performance in only a minority of the subjects tested (Blythe, Kennard, & Ruddock, 1987). Interestingly, in the present study, subject PM was above chance at seven locations that were clustered in two different areas in his affected visual field. At all other locations tested his performance was at chance. He reported that he saw nothing at any time but noted he had sometimes non-visual feelings and that he occasionally felt where the stimulus was presented. This is in line with other blindsight studies (Fendrich et al., 1992; Perenin, 1991; Shefrin, Goodin, & Aminoff, 1988; Weiskrantz et al., 1974; Zihl, 1980).

The stimulus used in this study was chosen to minimize artifacts and maximize detectability. Interestingly, the stimulus used by the Humphrey Field Analyzer was not seen, although it was more intense. However, in contrast to our stimulus, the Humphrey stimulus was not a flicker; it was smaller and of longer duration. It seems probable therefore that the nature of the stimulus is relevant when detecting regions of residual visual performance. Thus, it is quite possible that yet another type of stimulus may have led to the discovery of even more spared areas. The number of locations in the affected visual field that could be tested was limited. Therefore, it is possible that even more extensive forced-choice testing may have revealed additional isolated regions of residual performance.

Although, in the present study, the forced-choice test provided some evidence for blindsight, the spatial summation test did not. In subjects with homonymous visual field defects, no reaction time differences between single stimuli (presented in the intact field) and double stimuli (one presented in the blind field, one in the intact field) could be found. This is in contrast to two former studies that showed some evidence of spatial summation in several but not all sessions with four subjects (Corbetta, Marzi, Tassinari, & Aglioti, 1990; Marzi, Tassinari, Aglioti, & Lutzemberger, 1986) and in a recent study that tested hemispherectomized subjects (Tomaiuolo, Ptito, Marzi, Paus, & Ptito, 1997). Moreover, in the present study the impaired subjects showed some difficulties with the spatial summation test. In contrast to the normal subjects, many of the clinical subjects missed single and double stimuli in many trials. The reaction time was substantially slower in clinical subjects (median, 507 msec; range, 371 to 606 msec) in comparison to normal ones (median, 306 msec; range, 254 to 358 msec), al-

though normal and clinical subjects were not matched for age. Nevertheless, it is possible that there was no spatial summation in clinical subjects because their vigilance was generally impaired (Tomaiuolo et al., 1997). Future studies could also address the possible effect of altering stimulus onset asynchronies. A significant difference in reaction time between single and double stimuli was found when doubles were presented asynchronically in monkeys (Stoerig & Cowey, 1997) and humans (Corbetta et al., 1990).

Responsible Neural Area for Blindsight

Why did subject PM show blindsight when the other eight subjects did not? The absence of any residual visual performance was expected in subject AN, whose lesion involved the right optic tract and possibly the dorsal lateral geniculate nucleus (dLGN), consequently cutting the input to the dLGN and midbrain. However, the other remaining seven subjects had variable postgeniculo-striate lesions but none had blindsight. The visual system of subject PM was damaged in the optic radiation but areas 17 and 18 were apparently spared. The lesion in the optic radiation would affect input to V1. However, the restriction to the optic radiation or striate cortex is not likely to be critical, first, because blindsight was only found in two areas of subject PM's blind visual field and, second, because subject BG, who had a small optic radiation lesion, and subject RB, who had a small lesion that was likely to be restricted to striate cortex, did not show any ability to perceive visual stimuli in their blind visual fields.

The results from the nine subjects tested in this study do not support the hypothesis that the midbrain (e.g., superior colliculus) or an undamaged pathway that bypasses V1 is responsible for the residual visual performance because PM was the only subject who showed blindsight and he clearly showed residual performance in some areas but not in others. The results from hemispherectomized subjects are contradictary. The latest research on hemispherectomized subjects generally supports the view that the midbrain cannot mediate any residual visual performance in humans (King et al., 1996; King, Frey, Villemure, Ptito, & Azzopardi, 1996; Perenin, 1991; Stoerig et al., 1996). However, older studies have claimed residual visual performance (Perenin & Jeannerod, 1978; Ptito, Lassonde, Lepore, & Ptito, 1987; Ptito, Lepore, Ptito, & Lassonde, 1991), and one recent study does appear to show spatial summation in certain hemispherectomized subjects (Tomaiuolo et al., 1997). In any case, a positive result must be interpreted cautiously. The intelligence of hemispherectomized people is known to be below average, and more importantly, the brain damage in all the studied subjects occurred in early childhood, usually prenatally, at birth, or shortly thereafter.

Plasticity is also unlikely to be responsible because the blindsight location in the upper visual field was surrounded by locations with chance performance. Instead, the occurrence of blindsight in one small isolated area in the upper visual field and in some but not all locations in the lower visual field of subject PM suggests that spared optic radiation geniculate fibres that were not damaged by the trauma (and could not be seen on the neuroimages) were responsible for the residual visual sensitivities. Moreover, subject RB, who had a lesion that was probably restricted to V1, did not show any blindsight. If fibers from the dLGN or pulvinar to the extrastriate cortex were responsible, subject RB should have shown blindsight in all or at least part (if V2 is critical and partly damaged) of his affected visual field. In addition, other subjects with a lesion that was restricted to V1 and did not have blindsight have been reported (Gazzaniga et al., 1994). This interpretation is in line with an earlier study that claimed spared V1 in a subject with an occipital lesion who showed blindsight in a small isolated area only (Fendrich et al., 1992). Recently, these investigators confirmed their findings and found "islands of sparing" with variable residual visual performance in three more subjects (Wessinger et al., 1997).

It cannot be excluded that, in subject PM, although the optic radiation may be extensively damaged, a small number of spared isolated fibers from the dLGN or pulvinar to the extrastriate cortex were responsible for the blindsight. Recently, Kentridge, Heywood, and Weiskrantz (1997) tested subject GY at many locations and found that his performance depended on luminance of the stimulus, temporal discriminability in the temporal forced-choice test, and whether he was informed of the stimulus location. His performance remained always at chance at 2 out of 15 locations tested. However, the authors believe that an extrastriate pathway, not the isolated spared striate cortex, was responsible in their case because of the relatively large blindsight area.

The absence of residual visual performance in seven subjects with postgeniculate lesions suggests that spared fibers within a lesion are not common. However, a long-standing lesion, especially when incurred early in life, may be critical for residual visual performance (Blythe et al., 1987; Gazzaniga et al., 1994; Payne et al., 1996). Subject PM, who showed blindsight, had a long-standing lesion (32 years), and the trauma that led to the brain damage occurred at a relatively early age (15 years). By contrast, subjects RB (V1) and BG (optic radiation) had their accident at an older age than subject PM and their lesion was not as old. The notion that the age when the lesion occurred may be important is also supported by several other studies. Wessinger et al., (1997) found blindsight in two isolated areas in a subject who appeared to have had no functional brain tissue left in the right occipital lobe since childhood. In another study, 3 of 25 tested subjects with residual visual sensitivity had long-standing lesions (> 12 years) that were incurred before the age of 11, another one had the lesion when he was 23 years old, 4 years prior to testing, and a fifth

subject's date of accident was unknown (Blythe et al., 1987). Interestingly, one of their subjects was subject GY, who has shown blindsight and residual conscious vision in many other studies. Subject DB, another well-documented subject, may also have had brain damage early in life (Weiskrantz, 1986).

Blindsight and Conscious Awareness

Weiskrantz (1986) defined blindsight as "residual visual capacity in a field defect in the absence of acknowledged awareness." Using this definition, subject PM must be considered to have blindsight. However, it is possible that blindsight subjects do not realize that a very weak or differently perceived stimulus is the target stimulus and therefore do not report it. The fidelity of a subjective report cannot be verified and therefore the crucial dependence on subjective reports in blindsight has been criticized (Campion et al., 1983).

Subject PM's occasional nonvisual feelings at most locations at which he performed above chance (but rarely at the ones he did not) indicate that his guessing was often based on something he felt he was vaguely aware of, even though it may not have felt visual. A feeling that occurs with the presentation of a stimulus can be regarded as one attribute of perception and is therefore not strictly subconscious. Weiskrantz's (1986) definition allows an objective investigation of blindsight. However, it cannot be determined whether the observed above-chance performance was visually subconscious or just visually degraded.

It was generally observed that subject PM's criterion became more lax with time and that he reported higher confidence levels more frequently in the final sessions, without however, improving his performance. At one location subject PM's perception changed from "purely guessing," to a "feeling" and then to a "seeing very weakly." Subject KF who had bilateral damage including the occipital lobe, could still perceive weak light flashes in one small restricted area (residual vision with conscious awareness). Interestingly, subject KF did not at first report the stimuli that were presented in the perimetrically blind upper left area. However, later his potential "blindsight" turned into residual vision he was consciously aware of, indicating that his primary failure to report the visual stimuli was based on conservatism. He said that he initially did not report them because they looked different and were not the same as the ones he used to see in the intact field (Schärli et al., in press, for a full discussion on this case). This is in contrast to subject PM, who consistently and repeatedly reported that he did not see anything throughout all the testing. KF's findings suggest that his perceptual improvement was likely to be based on learning the association between sensation and stimuli (i.e., learning how to respond to a weak or unfamiliar degraded signal). Reported residual visual performance that is based on degraded vision (or artifacts like stray light, eccentric fixation, or eye movement search strategies) is likely to depend on practice (Bach-y-Rita, 1983; Balliet, Blood, & Bach-y-Rita, 1985). Similarly, in normal subjects, it is well known that the boundaries of the natural blind spot depend not only on the method used and on the intensity and kind of stimulus but also on "training" (Armaly, 1969). Moreover, in clinical perimetry it is known that the size of a scotoma varies in every test and that they appear larger when subjects are tired and smaller when they are well trained (Frisen, 1990).

The blindsight phenomenon (reported unawareness or low confidence but above-chance performance) can also be shown in experiments with normal subjects who do not have any brain damage (Kolb & Braun, 1995; Meeres & Graves, 1990; Schärli, 1991; Schärli, Landis, & Regard, 1992) or in subjects with damage in a nonvisual sensory system (Gabrieli, Milberg, Keane, & Corkin, 1990; Goodale, Milner, Jakobson, & Caray, 1991; Marshall & Halligan, 1988). Many studies suggest that a variety of neurological syndromes could exhibit nonconscious or implicit knowledge of stimuli that cannot be recollected consciously or perceived explicitly (Farah, 1994; Schacter, 1992). These findings suggest that blindsight may not be a phenomenon that is directly related to the lesion. Haber (1983) suggested that performance in a detection task may be superior to an identification task because it requires less salient stimuli. Similarly, the performance in a forced-choice task may be superior to a yes/no task. Sufficiently degraded stimuli may generally result in a dissociation of behavior and awareness (Schärli et al., in press; Wessinger et al., 1997). In visually impaired subjects with postgeniculate damage, spared small regions of V1 may not allow normal (conscious) visual perception, but subconscious or degraded processing may still be possible using certain simuli.

CONCLUSION

In the present study, blindsight was found in two areas of the perimetrically blind hemifield of one subject. It cannot be excluded that blindsight over the whole affected field exists under different circumstances. However, the results here and those from Fendrich et al., (1992) and Wessinger (1997) strongly indicate that isolated regions of the major geniculo-striate pathway led to degraded vision in corresponding visual areas and may explain blindsight at least in some cases. The results also indicate that residual visual performance is more likely in subjects with a long-standing lesion that was incurred early in life.

More systematic research is necessary to solve these issues. A study on subjects who suffered circumscribed lesions in the optic radiation, V1 and V2/V3, that originated from strokes and excised tissue at various ages in their childhood and adulthood (tested shortly or long after the accident) would help to find the critical

parameters for visual awareness and residual visual performance in homonymous visual field defects. Technological advances in brain scanning will assist and lead to higher-resolution images, allowing better analysis and identification of lesions.

METHODS

Normal Control Subjects

The three main tests, described below, were performed on a total of 28 normal subjects (university staff and students) in order to test the equipment and obtain control data. Six subjects participated in the yes/no and temporal forced-choice test, and 22 subjects did the spatial summation test. The natural blind spot was used as the "affected" region.

Visually Impaired Subjects

Nine subjects were tested who had a homonymous visual field defect due to a lesion in the visual system (Table 1). Eight subjects had an optic radiation or visual cortex lesion that was caused by a cardiovascular accident (CVA) or accident. One subject had damage in the LGN/optic tract area. The mean age was 54 (range 24 to 77). The majority had their accident 5 years or more prior to testing. The mean time that had passed since the accident was about 7 years (range, 6 months to 32 years). Only reliable and motivated volunteers with homonymous visual defects who had no other impairments that may have influenced their visual performance were selected. All had normal or corrected to normal vision in their intact field. Minor nonvisual neurological problems associated with the brain damage were found in three subjects. Subjects AH and AN showed weak left hemiparesis and subject PM had light (long-term) memory problems. All subjects lived independently at home. Four were employed full-time (subjects RB, HC, PM, and AN). All the subjects tested had pleasant personalities and were attentive, patient, and reliable. Informed consent was obtained from all subjects. Apart from travel expenses, no money was paid.

Lesion Analysis

The lesion from the CAT or MRI scan was assessed as carefully as possible, outlined on templates, and the extent was estimated (Damasio & Damasio, 1989, Figures A.12, A.13, A.14).

Clinical Perimetry

All the visually impaired subjects had each eye tested on a standard Humphrey Field Analyzer (stimulus size, 25.9′; presentation time, 200 msec; intensity, 0.25 to 3183 cd/m^2; background luminance, 10 cd/m^2). The visual field tested was 45 × 50° in size. Three subjects were tested twice (subjects RB, HC, and KF).

Experimental Apparatus and Visual Stimuli

Using small stimuli, a variety of locations across the visual field were tested. The region of examination extended over 21 × 25.5° (7° nasally and 18.5° temporally; see Figure 2). The experimental setup was specifically chosen to minimize screen reflections, after-images, eye movement during stimulus presentation, and the possibility of perceiving scattered light. To ensure that a performance difference was not simply based on the stimulus type used, the same stimuli were used to map the blind visual field (yes/no test) and to test for residual visual performance (forced-choice and spatial summation tests). In addition, the normal visual field and the natural blind spot in normal and visually impaired subjects was used as a control for artifacts. Special care was taken to control for extraneous stimuli.

A personal computer generated stimuli and presented them at one of the set of possible locations on a display (70-Hz refresh rate). See Figure 2. A small, relatively low-contrast stimulus was chosen that was still saliently perceived by the visual system and was suitable for all experiments with both visually impaired and normal subjects. The stimulus was a flickering dark disc (filled; 1° in diameter; 12 cd/m^2) that appeared three times for 14.3 msec in 100 msec on a light gray background (47 cd/m2) under constant photopic light conditions. The two intervals in between were blank and lasted for 28.6 msec each. The stimulus appeared almost black and will therefore be referred to as black from now on.

The subject sat 0.5 m from a PC screen and was instructed to look at the fixation cross and not to move his or her head, which was stabilized by a chin and forehead rest. The stimulus was always presented monocularly in all tests (one eye was covered with a patch) in the affected temporal field and intact (or partly intact) nasal field of each subject. Fixation was monitored by a video camera on every trial of all experiments. The image of one eye was magnified and presented on a monitor where it could be observed by the investigator. The subject's eyes could be monitored accurately by a light reflection on the pupil or the border of the pupil with the help of a reference grid on the screen. A warning sound was heard before stimulus presentation, indicating that the subject should look at the fixation cross. The cross remained on the screen as long as required. Pretests with normal subjects showed that eye movements of a magnitude of about 1° could be detected with this equipment. If eye or head movement between warning sound and stimulus presentation was detected, the trial was discarded. The discarded stimulus was presented again in a later trial. Subjects either reported orally or through buttons on a hardware box that also measured reaction time in milliseconds.

Experimental Tests

The same basic set of tests and procedures were completed by all subjects. Some subjects were then tested in more detail. The additional locations tested and number of trials completed depended on the subjects' results from the basic tests, their visual pattern of damage, their ability to persevere, and availability. First, the yes/no test was chosen to map the visual field and determine whether subjects were able to "see" the target stimulus used in all the tests in our lab. Two different paradigms were used to estimate the potential residual performance in the blind visual field. First, a two-alternative temporal forced-choice test was used to find out whether subjects were able to guess whether or not a stimulus was presented that they did not actually "see" in the yes/no test. Second, an implicit spatial summation test was used. This method is not only criterion-free but is also nonguessing. Consequently, subjects did not have to make an uncomfortable "blind decision" but could respond automatically to stimuli they always saw.

To evaluate the state of awareness in the yes/no and forced-choice tests, subjects were repeatedly encouraged to adopt a lax criterion and report any visual or nonvisual sensation. Confidence ratings (range, from very uncertain = 1 to very certain = 5) were employed to measure subjective awareness at any time and account for fluctuations or change in perception and attention or the possible influence of practice. All normal subjects responded by keypress in all tests. Initially, clinical subjects also responded by keypress. However, this was changed to an oral response procedure (in the yes/no and forced-choice tests) because subjects tended not to report small changes in perception by keypress. Presumably, pressing buttons encouraged automatic responses that were not based on reflection. The clinical subjects were also asked to comment on their perceptual experience after each block of trials.

Yes/No Test

Procedure

First, a cross appeared on the screen, followed by a warning sound (signal to fixate on the cross). Then, together with another sound, a target stimulus was presented either in the blind or intact field. A location was randomly chosen by the computer before each trial. After each trial the subjects' task was to decide whether they had seen the stimulus ("yes" response) or not ("no" response) and how certain they were of their decision (confidence rating). See Figure 4a.

Impaired Subjects

For each of the subjects the entire test region of the visual field was systematically sampled with an evenly spaced grid of sample points (Figure 1). The order of stimulus presentation was, however, random. Fifty-six locations in the affected temporal field and 24 locations in the (partly) intact nasal field (exception: subject RB, 45 temporal locations, 8 nasal locations) were tested on four trials at each location. The other eye with the major defect in the nasal hemifield was tested in all but two subjects (AN and AS, for methodological reasons). Many locations (18 to 30 in the affected field and 6 to 10 in the intact field) were tested more thoroughly in 5 to 20 more trials in all but one subject (BG, unable to attend further sessions due to limited availability).

Normal Subjects

The target stimulus, which was shown at one of four possible locations, was either a stimulus or a blank (50% blanks, 50% stimuli). One location was the natural blind spot (location 5I for the right eye; see Figure 2). The three other locations were 2 to 3° up, down, and left from the natural blind spot (locations 3I, 7I, and 5G for the right eye; see Figure 2).

Temporal Forced-Choice Test

Procedure

First, the fixation cross, immediately followed by a warning sound, was presented. Then, the target stimulus was shown in *one* of two intervals at one of the possible locations (interval and location were randomly chosen by the computer before each trial). The intervals were announced acoustically. After the presentation, subjects had to decide whether they had seen the stimulus in the first or the second interval and how certain they were of their decision (confidence rating). If they had seen nothing at all, they had to guess. See Figure 4b.

Impaired Subjects

FC-A Test. In total, 5 locations (exceptions: subject RB, 12 locations; subject AS, 2 locations) in the affected field and 2 locations (exception: subject RB, 3 locations) in the intact field were chosen. One block consisted of 50 trials (exceptions: subject RB, 24 trials; subject AS, 26 trials) at each location. For all nine subjects, the initial locations chosen were close to the four corners of the screen (locations 1A, 1K, 8A, and 8K; see Figure 2) and one was the natural blind spot (location 5I for the right eye; see Figure 2). The remaining locations were chosen individually for each subject based on the results from the yes/no test.

FC-B Test. After the preliminary testing, four chosen subjects were tested or retested extensively at 5 to 18 individually selected locations (subject PC, 5 locations; subject PM, 18 locations; subject RB, 7 locations; subject HC, 7 locations). The chosen locations were distributed over the testing area and based on the results from the

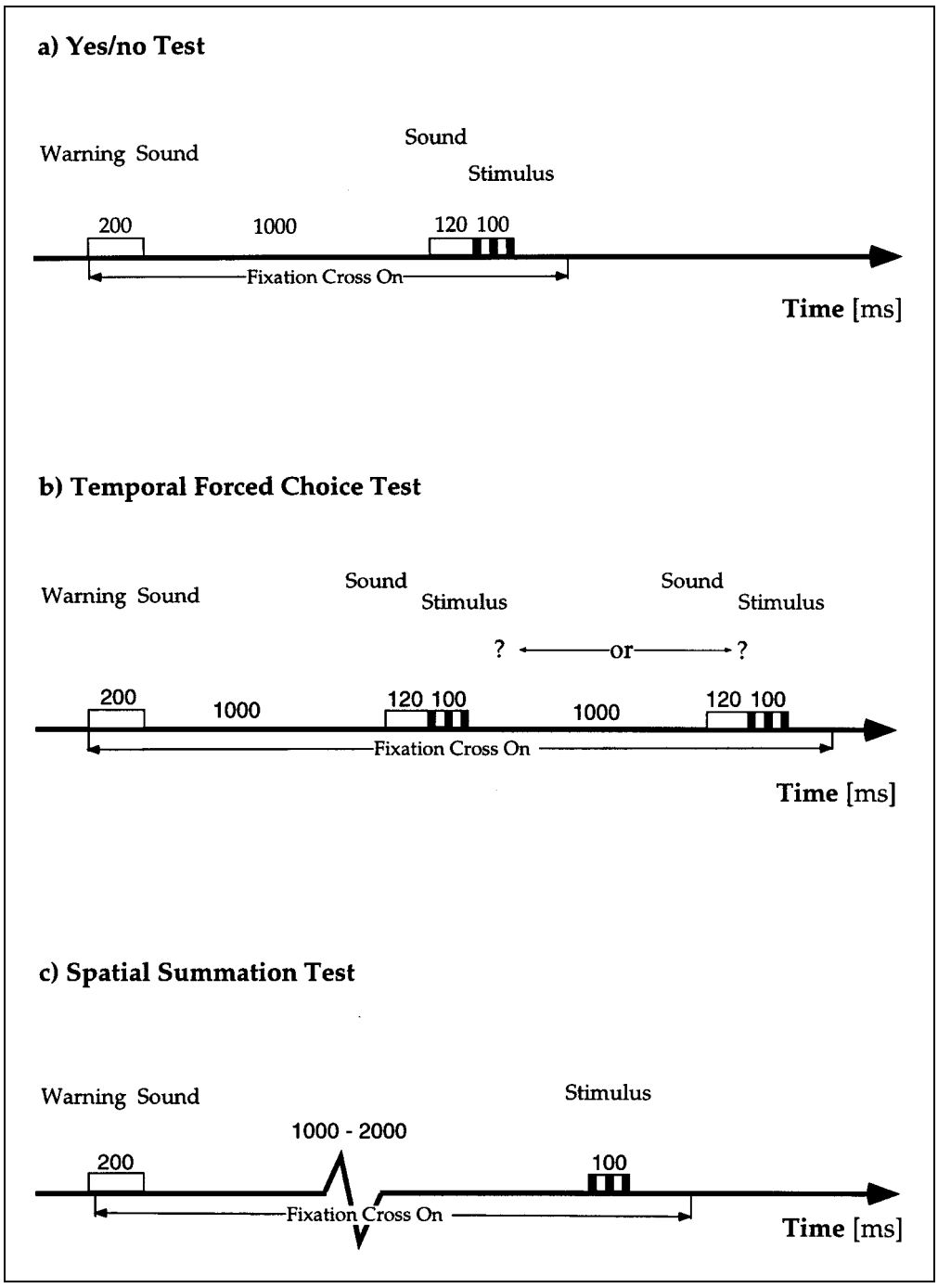

Figure 4. Timelines for the three experimental tests. (a) Yes/no test. A warning sound was followed by a sound and a stimulus. (b) Temporal forced-choice test. A warning sound was followed by two intervals that consisted of either a target stimulus or a blank, marked by a sound. (c) Spatial summation test. A warning sound was followed by a random interval and a stimulus. The time the fixation cross was on the screen is shown for each test. Numbers are times in milliseconds.

yes/no and forced-choice tests. One block consisted of 28 stimuli (same kind and same location) that were presented in the blind field only. Most locations were tested in several blocks (range 1 to 12 blocks per location) to check consistency and possible training effects. The stimulus location, however, was changed after each block of trials. In some blocks a distinctive sound after the trial indicated whether the response had been correct or not (acoustical feedback). All subjects always responded orally.

Normal Subjects

The same four locations were used as described in the yes/no test with normal subjects (see above). No blank trials were employed.

Spatial Summation Test

Procedure

After the warning sound and the fixation cross that appeared in the middle of the screen, an irregular blank foreperiod and then either a single or a double stimulus

was presented. Subjects had to respond by keypress as soon as possible after seeing the stimulus flashing on the screen (in the intact field). See Figure 4c. They always pressed the same two buttons, one with each hand. Reaction time (RT) was measured from the onset of the stimulus on the screen until the subject pressed the two response buttons. Subjects responded with both hands, pressing two different buttons as quickly as possible. Misses or trials with RT > 1000 msec were canceled and presented again at a later time. The same locations were tested in normal and clinical subjects.

Impaired Subjects

For all the impaired subjects, stimulus locations were selected close to the four corners of the screen (locations 1A, 8A, 1K, and 8K for the right eye; see Figure 1). Additional locations were chosen individually. All of them were previously used in the temporal forced-choice test. Sixty single stimuli were presented nasally (30 upper and 30 lower) in the intact area at two chosen locations (exception: subject RB, three locations; subject PC, four locations). Another 60 presentations consisted of two single stimuli, presented at the same time at an intact nasal location and at one of the 2 to 12 chosen locations in the affected visual field (subject RB, 12 locations; subject PC, 4 locations; subjects HC, AH, and AS, 5 locations; subjects PM, BG, and AN, 2 locations). Double stimuli were between 14 and 25° apart. In order to test for automatic reactions without acknowledged perception, five or six single stimuli were also presented in the affected field of four subjects (RB, HC, AH, and AS).

Normal Subjects

Four locations were chosen that were the same as those for impaired subjects with a left or right hemianopia (locations 1A, 8A, 1K, 8K for the right eye, see above). In 12 subjects the right eye was tested (right impaired control group), presenting 120 single stimuli in the upper left (60) or lower left (60) and 120 double stimuli, on the left (40), upper (40), and lower (40) locations. Similarly, in 10 subjects the left eye was tested (left impaired control group), presenting 120 single stimuli in the upper right or lower right and 120 doubles on the right, upper, and lower locations.

Data Analysis

Mann-Whitney test, chi-square test, *t* test, and analysis of variance (ANOVA) were used to compare data. An alpha of 0.05 (one-tailed) was used in all tests.

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