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Neural Substrates of Blindsight After Hemispherectomy

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Blindsight is a visual phenomenon whereby hemianopic patients are able to process visual information in their blind visual field without awareness. Previous research demonstrating the existence of blindsight in hemianopic patients has been criticized for the nature of the paradigms used, for the presence of methodological artifacts, and for the possibility that spared islands of visual cortex may have sustained the phenomenon because the patients generally had small circumscribed lesions. To respond to these criticisms, the authors have been investigating for several years now residual visual abilities in the blind field of hemispherectomized patients in whom a whole cerebral hemisphere has been removed or disconnected from the rest of the brain. These patients have offered a unique opportunity to establish the existence of blindsight and to investigate its underlying neuronal mechanisms because in these cases, spared islands of visual cortex cannot be evoked to explain the presence of visual abilities in the blind field. In addition, the authors have been using precise behavioral paradigms, strict control for potential methodological artifacts such as light scatter, fixation, criterion effects, and macular sparing, and they have utilized new neuroimaging techniques such as diffusion tensor imaging tractography to enhance their understanding of the phenomenon. The following article is a review of their research on the involvement of the superior colliculi in blindsight in hemispherectomized patients. NEUROSCIENTIST 13(5):506–518, 2007. DOI: 10.1177/1073858407300598

KEY WORDS *Blindsight, Superior colliculus, Hemispherectomized patients, S-cones, Spatial summation effect, Diffusion tensor imaging (DTI) tractography*

Damage to the occipital cortex has traditionally been thought to lead to permanent blindness in the contralateral visual field. The existence of residual visual functions in the blind field has, however, been observed and described in cortically blind humans and animals (Bard 1905; Riddoch 1917; Bender and Krieger 1951; Perenin and Jeannerod 1974; Pöppel and others 1973; Cowey and Stoerig 1995, 1997). This visual phenomenon, whereby patients are able to process visual information in their blind visual field without a conscious perception of the stimuli, was first coined "blindsight" by Weiskrantz (Weiskrantz and others 1974; Weiskrantz 1986; Shefrin and others 1988).

The observation that residual visual abilities vary between patients (e.g., Corbetta and others 1990) and that residual functions in the blind field may also exist with awareness led to the development of two subcategories of blindsight: "Type I" and "Type II" (Weiskrantz 1989).

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Patients with Type I blindsight demonstrate unconscious residual visual abilities that have been associated with a retinal-tectal pathway (Weiskrantz 1989; Sahraie and others 1997). This includes neuroendocrine responses such as melatonin suppression following exposure to a bright light (Czeisler and others 1995), reflexive responses as shown by pupillary reaction to changes in illumination and implicit processing whereby presentation of a stimulus in the blind field affects performance in the normal visual field (Torjussen 1978; Marzi and others 1986).

Patients with Type II blindsight possess some awareness of residual visual abilities such as target detection and localization by saccadic eye movements (Pöppel and others 1973; Weiskrantz and others 1974; Weiskrantz 1989) and manual pointing (Weiskrantz and others 1974), movement direction detection, relative velocity discrimination (Barbur and others 1980; Blythe and others 1986; Blythe and others 1987; Weiskrantz and others 1995), stimulus orientation detection (Weiskrantz 1986), and/or semantic priming from words presented in the blind field (Marcel 1998).

Because the residual visual abilities vary among individuals, Danckert and Rossetti (2005) recently put forward a new taxonomy based on the assumption that subcortical structures that were not affected by the cortical damage and the ensuing degeneration mediate blindsight. This classification system consists of three

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Table 1. Danckert and Rossetti's Classification System for Blindsight

	Action Blindsight	Attention Blindsight	Agnosopia
Residual behaviors	Grasping, pointing, saccades	Covert spatial orienting, inhibition of return, motion detection and discrimination	Wavelength and form discrimination, semantic priming
Paradigm	Direct behavior towards blind field stimuli	Forced-choice guessing, implicit processing paradigm	Forced-choice guessing
Residual visual pathways	SC–pulvinar–posterior parietal cortex (dorsal stream)	SC–pulvinar–extrastriate visual cortex (MT and dorsal stream)	Interlaminar layers of the dLGN–extrastriate visual cortex (ventral stream)

Adapted and reproduced with permission from Danckert and Rossetti, *Neuroscience and Biobehavioral Reviews* 2005.
SC = superior colliculus; dLGN = dorsal lateral geniculate nucleus.

subcategories: 1) "Action blindsight" is observed when an action is used to guess the localization of a target by pointing or saccading in the blind field. 2) "Attention blindsight" is associated with motion direction detection and implicit task interference effects of a stimulus presented in the blind visual field; here, attentional processes appear to contribute without necessarily involving a specific action. Conscious awareness of the stimulus presented in the blind visual field may or may not accompany this kind of blindsight phenomenon. Danckert and Rossetti (2005) speculate that the retinofugal pathway from the eye to the superior colliculi is involved in both action blindsight and attention blindsight, although they may differ in the regions of extrastriate cortex involved. 3) "Agnosopsia" (Zeki and Ffytche 1998) is used to describe the ability of the patient to guess the correct perceptual characteristic of the target despite being unaware of its presence in the blind field. This would include residual visual abilities that involve form or wavelength discrimination, which is presumably mediated by interlaminar layers of the dorsal lateral geniculate nucleus (dLGN) (Table 1, Fig. 1).

Limitations of Previous Research

Several researchers (Campion and others 1983; Fendrich and others 1992) suggested that residual visual functions within scotomas, whether conscious or unconscious, could be due to methodological inadequacies such as inadvertent eye movements, eccentric fixation, and intra- and extraocular light scatter (Faubert and others 1999). Furthermore, previous results on residual visual abilities contrasted with reports of patients with retrogeniculate damage who show neither blindsight nor residual vision. Individual differences have been attributed to extent, location, and age at lesion onset (an early onset makes blindsight more likely), which are not uniform across patients.

Another restricting factor is the use of forced-choice paradigms, which have been used in many studies investigating blindsight. In this approach, the patients' reaction not only depends on their sensitivity to differences between the stimuli, but it is also affected by their response criteria (bias), a tendency to consistently select one of the stimuli in favor of another independently of sensitivity, and by the fact that they are forced to guess about the presence of a stimulus in their blind visual field. For this reason, forced-choice paradigms to examine blindsight have been criticized (Cowey 2004; Ro and others 2004).

Alternatively, indirect methods, which require the patient to react only to consciously perceived stimuli, have been developed to exclude methodological artifacts such as response bias. Implicit processing of a stimulus, which does not require a direct response from the patient, has been demonstrated within a field defect. For example, Zihl and others (1980) used reflex measures and demonstrated electrical skin conductance responses to "unseen" light stimuli presented in the blind visual field.

Another indirect method used to investigate blindsight utilizes the spatial summation effect (e.g., Tomaiuolo and others 1997) in which the simultaneous presentation of an unseen stimulus can alter the mean reaction time to a seen stimulus (Marzi and others 1986). With this approach, patients show a significantly faster reaction time to two bilaterally presented stimuli, one of which is in the blind field, compared to a single one shown in the intact field.

Other important issues that have been raised to explain above-chance performances in hemianopic patients are the possibility of light scatter from the blind field into the seeing field, inadequate eye fixation, mechanisms such as cortical plasticity or reorganization of cortical functions (Smith and Sugar 1975; Rosenblatt and others 1998), and macular sparing.

In addition, among the most difficult criticisms that blindsight studies have met is the possibility that fragments or islands of intact functional striate cortex rather than extrastriate pathways are responsible for the residual visual abilities observed (Fendrich and others 1992).

Model: Hemispherectomy

To eliminate the possibility that residual vision is mediated by spared striate cortex, we have conducted a series

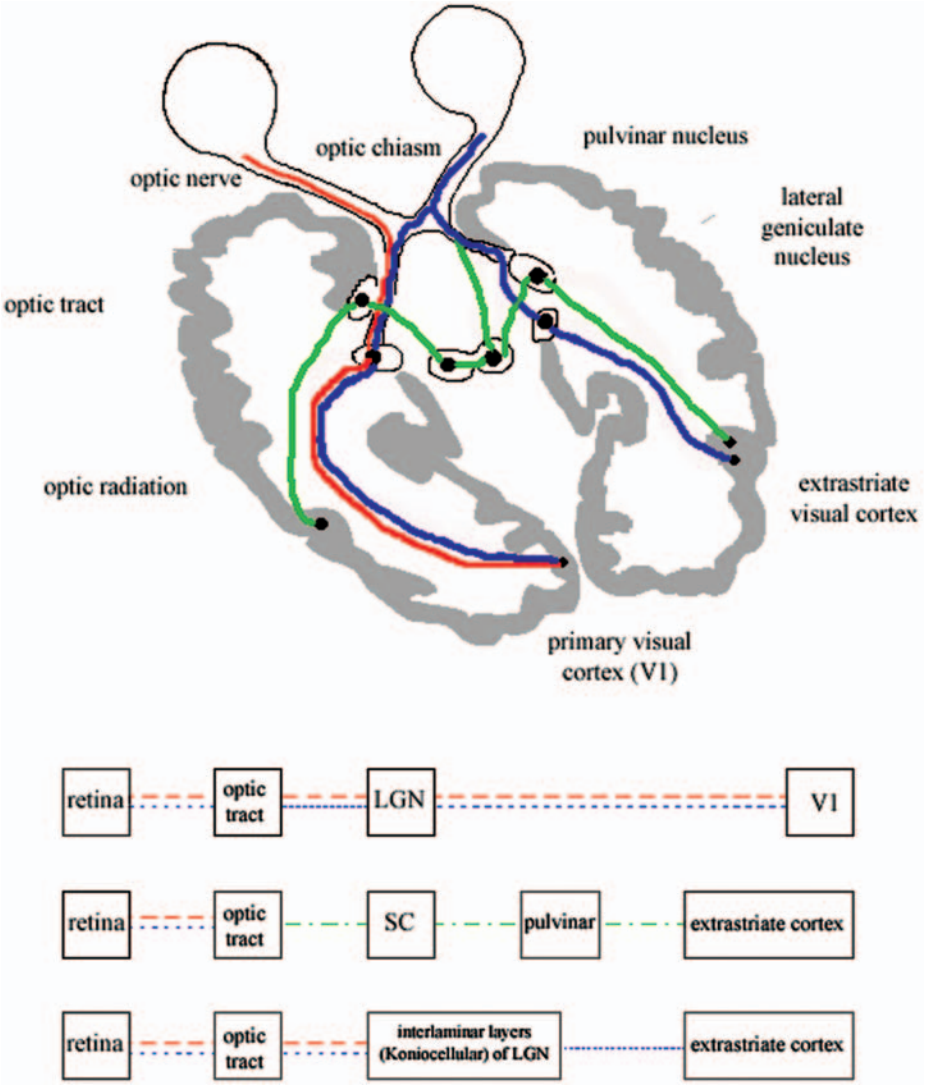

Fig. 1. Possible pathways involved in blindsight. Schematic representation of the various visual pathways from the retina to striate (V1) and extrastriate cortex. The primary geniculostriate pathway is indicated by the dashed line from the temporal hemiretina of the left eye and the widely spaced dotted line from the nasal portion of the right eye. For clarity, the two secondary pathways are shown originating from the optic tract, with the retino-tectal pathway indicated by the dashed/dotted line and the geniculostriate pathway by the closely spaced dotted line. The pathways are also represented in simple box and arrow form below the schematic. Note that recent anatomical work in the monkey has shown direct koniocellular projections to area MT (Sincich and others 2004). The possibility exists for other such pathways from the interlaminar layers of the lateral geniculate nucleus (LGN) to regions of the extrastriate cortex other than area MT. SC = superior colliculus. (Adapted and reproduced with permission from Danckert and Rossetti, *Neuroscience and Biobehavioral Reviews* 2005.)]

of studies on hemispherectomy patients who had undergone complete removal or deafferentation of a whole cerebral hemisphere. The term "hemispherectomy" describes a neurosurgical technique in which all or large amounts of cortical tissue, including the motor and sensory strip of one hemisphere, are removed or disconnected from the rest of the brain (see Fig. 2 for examples of the technique). In these patients, the striate cortex has been entirely ablated or deafferented such that explanations for blindsight based on spared striate cortex and lateral geniculate or collicular projection to the ipsilesional extrastriate cortex are inapplicable.

There are different surgical approaches to hemispherectomy, which may involve either complete removal of the cortex of one hemisphere or, alternatively, partial removal and disconnection of the residual cortex from the rest of the brain (see also De Almeida and Marino 2005; De Almeida and others 2006; Fountas and others 2006). This radical surgical technique is considered in patients with severe intractable seizure disorders originating from one side of the brain. These intractable seizures arise from diffuse lesions in a single hemisphere and have different etiologies (e.g., Rasmussen's encephalitis, Sturge-Weber syndrome, Lennox-Gastaux syndrome, porencephalic cyst, etc.).

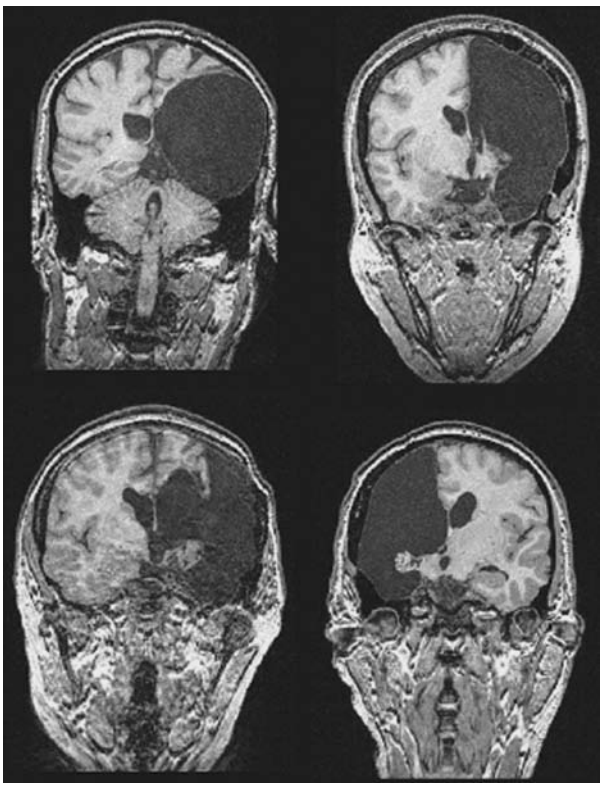

Fig. 2. Examples of anatomical MRIs of hemispherectomized patients showing three right-hemispherectomized and one left-hemispherectomized patient.

Hemispherectomized patients represent a good model for studying residual visual abilities in the blind field because all of the occipital lobe has been removed or disconnected from the rest of the brain. This leaves the patient with a contralateral visual field loss without macular sparing, and retinal pathways from the hemispherectomized side remain only to the ipsilesional superior colliculus (SC) and the contralesional pulvinar. Autopsy studies following hemispherectomy confirm these assumptions and demonstrate a retrograde degeneration of the entire thalamus on the ablated side, including the lateral geniculate body, retinal ganglion cells projecting to the midbrain, and other thalamic relay stations. In these studies (Ueki 1966), the ipsilesional colliculus remains remarkably intact, maintaining an organization and density of its seven cellular layers that are virtually indistinguishable from its homolog in the intact hemisphere. Such structural integrity suggests preserved function.

Behavioral Experiments

1. Residual Vision with Awareness: Object Discrimination, Movement Detection, and Localization

We tested a first group of hemispherectomized patients in 1987 (Ptito and others 1987) in a pattern (2D) and an object (3D) discrimination task. The patients had to indicate whether pairs of stimuli presented simultaneously in both hemifields parafoveally or at 30 degrees eccentricity were the same or different. Testing was performed monocularly, and eye movements were monitored through the use of Beckman EOG electrodes. Results showed that compared to a matched control group, hemispherectomized patients were in general impaired at discriminating 2D patterns presented simultaneously in their blind and intact visual fields. Performances improved, however, in two of the four patients when 3D stimuli were presented bilaterally. No discrimination was possible for any of the experimental patients when the two stimuli were presented in the blind field. These results led us to conclude that some complex visual abilities persist in the blind field of hemispherectomized patients and that some interfield comparisons can be carried out, suggesting that the blind field has some limited access to the intact hemisphere.

We pursued this line of research with the same four hemispherectomized patients in a study where we investigated their ability to detect and localize stationary, flashing, and moving targets at different eccentricities (Ptito and others 1991). Beckman EOG electrodes were used to monitor eye movements, and fixation was ensured by requiring the patient to look at a centrally presented row of eight randomly flickering light-emitting diodes (LEDs) superimposed at intervals of 2.5 cm and to tap on the table as soon as one of the LEDs remained on. The tapping response was picked up by a microphone and relayed to a microprocessor, which then triggered within 5 ms the presentation of the stimulus. With this rigorous control of eye fixation, we showed, as others had, that the extent and quality of the residual vision vary among patients and type of task investigated. In the first task, all could detect and localize with reasonable accuracy in their blind field a moving, flashing, or stationary stimulus presented during 150 ms. They rarely denied that a stimulus had been presented, and all experienced little difficulty in distinguishing blank control trials (absence of the visual stimulus). They were therefore aware of the presence of the stimulus without, however, specifying its nature. This contrasted with the forced-choice techniques used to circumvent the patients' denial of the presence of a stimulus, and we were probably measuring residual vision rather than blindsight as described at the time (Weiskrantz and others 1974).

In a second experiment, we asked the patients to indicate the presence or absence of a grating and, in the affirmative, to report if it was moving or not. Again, all detected without error blank trials, but individual differences with regard to performances in the blind field emerged. Whereas all were capable of detecting the presence of the grating, and two out of three could distinguish between a "rapidly" moving grating (2.6 cycles/s) and a stationary one, none could detect a slow movement (0.3 cycles/s). In the second part of this experiment, we assessed relative velocity discrimination and found a modest but still significant ability. One patient was able to discriminate large and median differences in

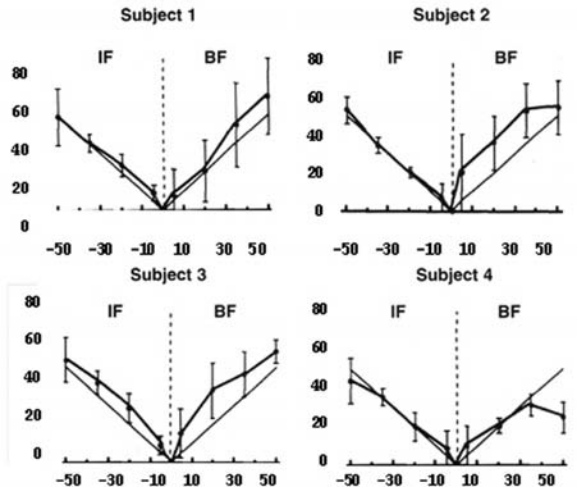

Fig. 3. Accuracy of localization of combined stationary, moving, and flashing targets for four hemispherectomized patients. Horizontal axis: target position; vertical axis: responses. IF = intact field; BF = blind field. (Adapted from Ptito and others, *Brain* 1991.)

stimulus velocity but remained at chance when the gratings moved at the same speed. In contrast, another could only detect an absence of difference between velocities, whereas a single patient remained at chance in all conditions involving his blind field. When the gratings were presented simultaneously in both hemifields, similar results were obtained.

In a third experiment, we asked the patients to report whether the directions of displacement of the stimuli presented in the intact field, in the blind field, or in both fields simultaneously were the same or different. Results showed that although the patients obtained more than 90% correct responses in their intact field, none were able to discriminate direction of movement, in the blind field or in both fields simultaneously, a function associated with area MT (putative V5), absent in our patients (Fig. 3, Table 2, Table 3).

The positive visual functions in the blind hemifield of hemispherectomized patients have been put into doubt by some control experiments, suggesting that there may have been stray light entering the intact hemifield (King and others 1996). Subsequently, we showed the importance of controlling intraocular light scatter, as spectral sensitivity within the blind field can be reduced considerably and yet high intensity stimuli can be detected probably by foveal receptors (Stoerig and others 1996). We then presented a model that could explain the scatter properties of the eye on the visual sensitivities obtained with hemispherectomized patients (Faubert and others 1999).

Taking these factors into consideration and controlling for them, we nevertheless confirmed in a separate group of hemispherectomized patients the existence of residual vision with awareness in the blind field that could not be linked to light scatter, eccentric fixation, or eye movements (Fendrich and others 1992; Wessinger and others 1996) (Fig. 4). A double Purkinje eye tracker was used with two hemispherectomized patients to stabilize the stimulus displays retinally and eliminate artifacts due to eye motion. Black stimuli (<1 cd/m^2) were presented on a gray background (10 cd/m^2) to reduce light scatter. Stimulus detection and discrimination were then tested in a forced-choice paradigm within the blind visual field of the patients using stabilized field mapping. An area was identified in both patients' hemianopic field within which stimulus detection was possible. The area consisted of a horizontal band not wider than 3.5 degrees but extending up to 6 degrees at one field location for each patient. The areas of residual vision varied among patients. With SE, the band was within both visual quadrants, but only above the horizontal meridian for JB. The patients were aware of their residual vision, and mean confidence values in areas with sparing were significantly higher than in those areas without sparing. Within the areas of residual vision, both patients readily discriminated simple stimuli such as square and diamond figures and, although they were poorer at discriminating complex stimuli, they still performed above chance. Both were also able to verbally identify squares and diamonds presented within the zone of sparing, but neither could identify similarly presented complex figures. In both the discrimination and identification tasks, the patients performed at chance when stimuli were outside the areas with spared detection, while they were always identified correctly in each patient's seeing field (Fig. 4, Table 3).

2. Residual Vision without Awareness (Blindsight): Spatial Summation Effect Paradigm

Skepticisms concerning the existence of blindsight and the methods (e.g., lax decisional criterion) remained, however. We thus decided to test four hemispherectomized patients on a protocol based on the redundant-target effect, a summation phenomenon well known in experimental psychology (Raab 1962), whereby the simultaneous presentation of two or more stimuli results in a faster reaction time than to a single stimulus. This indirect procedure allowed us to observe whether unseen stimuli in the blind field can influence the patient's response to stimuli in the intact field. This is so because the patient reacts to consciously perceived stimuli in the normal visual field only and is not asked to guess whether a stimulus was presented in the blind field (Tomaiuolo and others 1997). Results showed that none of the patients were aware of stimuli (single or double) presented in their blind hemifield. Three patients showed a spatial summation effect in their normal visual field (DR, SE, IG), and two patients (DR and SE) showed a spatial summation effect when stimuli were presented across the vertical meridian in their blind and normal visual fields despite their lack of visual awareness in their blind hemifield (Fig. 5). The results in patients DR and SE are in keeping with previous studies using the spatial summation effect paradigm (Raab 1962; Blake

Table 2. Percentage of Correct Responses to Movement, Velocity Differences, and Movement Direction

		Case 1			Case 2			Case 3		
		Intact Field	Blind Field	Both Fields	Intact Field	Blind Field	Both Fields	Intact Field	Blind Field	Both Fields
Movement detection	Stationary	90	20[a]		100	65		95	93	
	Slow	30[a]	10[a]		100	35[a]		85	5[a]	
	Rapid	90	10[a]		100	65		100	95	
	Blank trials	100	100	100	100	100	100	100	100	100
Velocity differences	Same	83	20[a]	56[a]	89	30[a]	29[a]	100	94	94
	Medium	79	42[a]	41[a]	92	83	67[a]	58[a]	29[a]	42[a]
	Large	92	67[a]	25[a]	92	75	75	100	50[a]	75
Direction of movement		90	50[a]	54[a]	100	52[a]	50[a]	100	58[a]	46[a]

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a. At or below chance level.

Table 3. Percentage of Correct Responses on Discrimination and Identification of Simple and Complex Stimuli within and Outside Areas of Residual Vision in the Blind Field

	Discrimination				Identification			
	Simple		Complex		Simple		Complex	
Subject	SE	JB	SE	JB	SE	JB	SE	JB
Upfar	45	52	50	42	50	60	0	–
Upclose	93[a]	97[a]	46	77[a]	90[a]	100[a]	0	0
Downfar	50	52	54	46	61	45	0	–
Downclose	88[a]	60	63[a]	35	92[a]	55	0	0

[a]At or above chance level.
Adapted and reproduced with permission from Ptito and others, *Brain* 1991.
Upfar: Presentation in upper quadrant outside zone of sparing; Upclose: presentation in upper quadrant within zone of sparing; Downfar: presentation in lower quadrant outside zone of sparing; Downclose: presentation in lower quadrant within zone of sparing (SE only).

and others 1980; Marzi and others 1986; Miniussi and others 1998; Savazzi and Marzi 2002). We also conducted a second experiment to exclude the possibility that light scatter could account for the effect observed in the two hemispherectomized patients. In this experiment, the second stimulus was presented to the blind spot of normal control participants, and none of these patients showed a spatial summation effect.

We believe that the spatial summation effect paradigm holds great potential as an indirect method to further evaluate blindsight, as patients only have to react to the stimulus presented in their intact field, without being aware that the simultaneous presentation of another stimulus in their blind field will lower their reaction time. To date, the majority of studies investigating the spatial summation effect in blindsight have relied on the detection of simple visual stimuli, such as dots, that did not challenge the processing abilities of separate visual pathways that may be involved in blindsight.

We hypothesized that the superior colliculi are likely implicated in blindsight (e.g., Ptito and others 1987, 1991), particularly for hemispherectomized patients, and we recently utilized the color vision properties of collicular cells to demonstrate the involvement of this structure in the residual visual abilities of hemispherectomized patients (Leh, Ptito, and Mullen 2006). We used the fact that electrophysiological studies indicate that the primate SC does not receive retinal input from shortwave-sensitive (S-) cones involved in color vision, consequently rendering them color blind to blue/yellow stimuli (Marrocco and Li 1977; Schiller and Malpeli 1977; Sumner and others 2002; Savazzi and Marzi 2004).

Our goal was to demonstrate the absence of S-cone input in the blind visual field of hemispherectomized patients with blindsight using psychophysical methods. We designed a computer-based reaction time test using achromatic black/white and blue/yellow stimuli. These two stimuli types were designed and calibrated to isolate

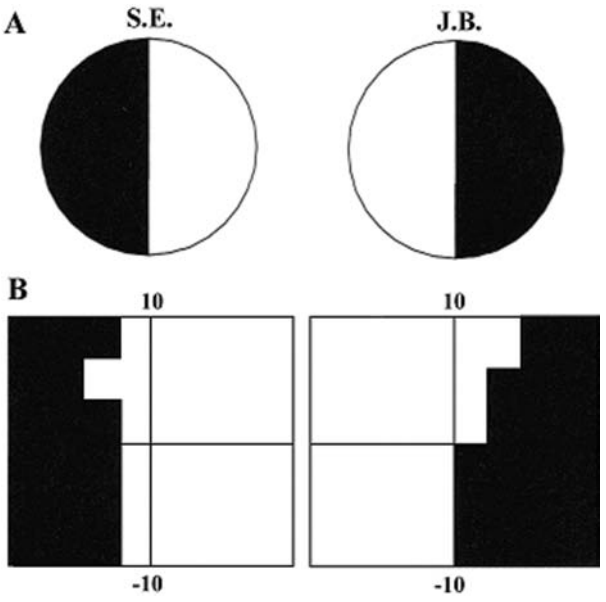

Fig. 4. Schematic representations of (*A*) perimetric test results of patients SE and JB showing contralateral hemianopia without macular sparing and (*B*) stabilized visual field detection results for SE and JB. (Adapted and modified from Wessinger and others, *Neuropsychologia* 1996.)

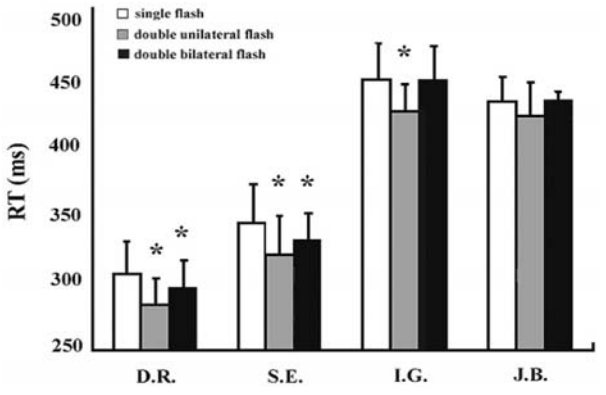

Fig. 5. Mean reaction times (RT) for two hemispherectomy patients and a normal control patient who showed a spatial summation effect. *Statistically significant spatial summation effect (one single flash compared to double unilateral presentations in intact field and double bilateral presentations; $P = .05$). (Adapted and modified from Tomaiuolo and others, *Brain* 1997.)

either the achromatic postreceptoral pathway or the blue/yellow postreceptoral pathway, which draws on S-cones while remaining invisible to the other postreceptoral pathways. Eye movements were closely monitored with an eye-tracking device, and stimuli were modulated about a uniform white background of the same luminance and chromaticity. Three hemispherectomized patients, who had shown blindsight in previous studies reliably, were included in the study. These patients

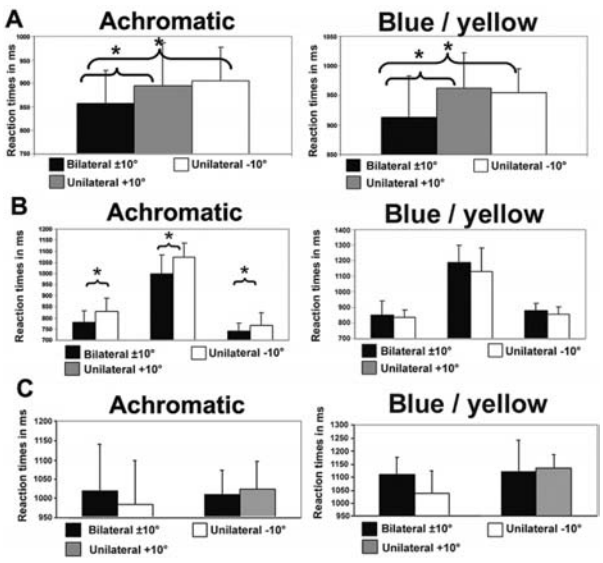

Fig. 6. Achromatic versus blue/yellow spatial summation effect in (*A*) normal individuals. A significant spatial summation effect was observed independently of color (N = 16, $F(1, 15) = 23.37$; $P < .001$). *B*, Hemispherectomized patients with blindsight (N = 3, DR, LF, SE). A spatial summation effect was observed for achromatic stimuli (N = 2, DR: $t \leq 0.001$, $df = 24$; LF: $t \leq 0.05$, $df = 24$; SE: $t \leq 0.05$, $df = 24$) but not for blue/yellow stimuli (DR: $t = 0.36$, $df = 24$; LF: $t = 0.73$, $df = 24$; SE: $t \leq 0.5$, $df = 24$). *C*, Hemispherectomized patients without blindsight (FD, JB). No spatial summation effect was observed for either achromatic or blue/yellow stimuli (achromatic: FD: $t = 0.20$, $df = 24$; JB: $t = 0.61$, $df = 24$; blue/yellow: FD: $t = 0.14$, $df = 24$; JB: $t = 0.34$, $df = 24$). Note that all individuals were tested with the right eye, while the left eye was occluded. *Significant. (Adapted and reproduced with permission from Leh and others, *European Journal of Neuroscience* 2006.)

demonstrated a spatial summation effect only to achromatic stimuli (Fig. 6), suggesting that their blindsight is color blind specifically to blue/yellow stimuli and is not receiving input from retinal S-cones.

After a hemispherectomy, visual information cannot be processed by geniculo-extrastriate pathways; consequently, visual information from the blind visual field can only be processed via either the ipsilesional SC or the contralesional pulvinar on to the remaining hemisphere. Previous studies have shown that the SC is not receiving retinal input from S-cones (Marrocco and Li 1977; Schiller and Malpeli 1977; Sumner and others 2002; Savazzi and Marzi 2004), in contrast to the pulvinar, which receives input from all classes of color-opponent ganglion cells (L/M as well as S-cone opponent) (Felsten and others 1983; Cowey and others 1994) and appears to be involved in color processing in humans (Barrett and others 2001). We therefore concluded from this study that blindsight is likely mediated by the superior colliculi in hemispherectomized patients.

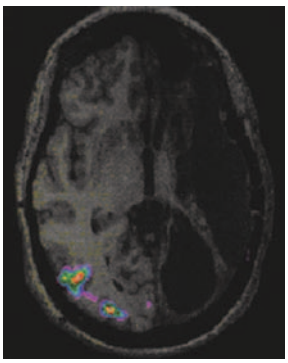

Fig. 7. Blind (*left*) hemifield stimulation in a right hemispherectomized patient (DR) shown to possess blindsight. Note ipsilesional extrastriate activation foci in areas V5 and V3/V3A. (Adapted and reproduced with permission from Bittar and others, *Neuroimage* 1999.)

Imaging Studies

1. Functional MRI Studies

The results we have been discussing in hemispherectomized patients strengthen previous observations that individual differences among patients exist. Whereas some demonstrate total blindness, others experience under certain experimental conditions residual visual abilities with some awareness (Type II blindsight; Ptito and others 1987; Ptito and others 1991; Wessinger and others 1996), whereas others show unconscious visual abilities (Type I blindsight; Tomaiuolo and others 1997; Herter and Guitton 1998, 2004).

To investigate more directly the neural pathways involved in blindsight and/or residual vision, we conducted an fMRI experiment (Bittar and others 1999) with three hemispherectomized patients (JB, IG, and DR) who participated in the Tomaiuolo and others study (1997). To the best of our knowledge, this was the first functional neuroimaging study with hemispherectomized patients aiming to visualize the cerebral regions involved in blindsight. Computer-generated randomly moving dots were presented in the baseline condition. For the activation condition, we designed black-and-white semicircular gratings, which were moving in opposite directions on a dynamic random-dot background to prevent Lambertian intraocular scatter and exclude the possibility that blindsight is due to intraocular light scatter (Faubert and others 1999). These stimuli were presented unilaterally on a background of randomly moving dots in the blind visual field. An activation minus baseline subtraction showed activation of the ipsilateral occipital lobe (V5/MT: x = –48, y = –75, z = –2; V3/V3A: x = –12, y = –87, z = 16; x = –24, y = –86, z = –24) (Fig. 7) in a hemispherectomized patient (DR) who had demonstrated blindsight in previous studies. Inasmuch as no significant activation within the superior colliculi or pulvinar of either the experimental or control patients was seen, likely because of the limited resolution of the apparatus, we speculated that the remaining hemisphere contributes to these residual functions in the blind hemifield in conjunction with ipsilateral subcortical structures because the activated areas are known to have connections with these regions.

2. Diffusion Tensor Imaging Tractography

The advent of a relatively new neuroimaging technique, diffusion tensor imaging (DTI) tractography, has allowed us to investigate specifically superior colliculi connectivity in hemispherectomized patients with and without blindsight (Leh, Johansen-Berg, and Ptito 2006). With this innovative approach, fiber tracts can be visualized by sensitizing the MRI signal to the random motion (diffusion) of water molecules to provide local measures of the magnitude of water diffusion. The data can then be used for further computational analysis, to reconstruct white matter fiber tracts three-dimensionally in vivo, allowing assessment of connectivity between different regions (Conturo and others 1999; Behrens and others 2003). First, T1-weighted anatomical MRI images and diffusion-weighted images were obtained. We then created seed masks on each patient's T1-weighted image, including the whole superior colliculi, and used a probabilistic algorithm model for the DTI data analysis that allowed for an estimation of the most probable location of a single fiber connection (for further information, see Leh, Johansen-Berg, and Ptito 2006).

Results of this DTI tractography study demonstrated the presence of projections from the ipsi- and contralesional SC to primary visual areas, visual association areas, precentral areas/FEF (frontal eye field), and the internal capsule of the remaining hemisphere in hemispherectomized patients with Type I or attention blindsight (example in Fig. 8*A*) and an absence of these connections in hemispherectomized patients without Type I or attention blindsight (example in Fig. 8*B*), thereby confirming our assumption (Tomaiuolo and others 1997; Bittar and others 1999) and that of Danckert and Rossetti (2005) that blindsight is mediated by a collicular route. Interestingly, connections from the ipsilesional SC in patients with Type I or attention blindsight, which crossed at the level of the SC, were more prominent than the crossed projections seen in healthy controls.

Potential Neuronal Substrates

The results so far are consistent with the possibility that the remaining hemisphere plays a role in the mediation of blindsight and/or residual visual abilities in the blind field of hemispherectomized patients. This would be achieved either by a process of cortical plasticity and/or by utilization of existing neural pathways such as those traversing subcortical nuclei. Several observations have supported previous suggestions that the SC plays an important role in blindsight (Kisvarday and others 1991; Ptito and others 1996; Sahraie and others 1997; de Gelder and others 1999; Morris, Öhman, and Dolan 1999).

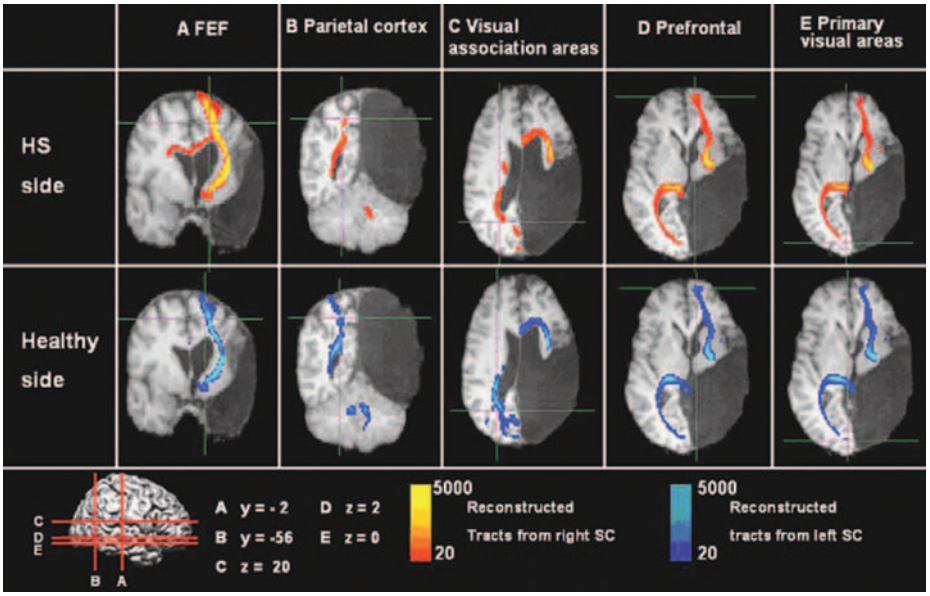

Fig. 8*A*. Diffusion tensor imaging tractography in a hemispherectomized patient (SE) with "Type I" blindsight ("attention blindsight"). Illustration shows reconstructed right (red hues) and left (blue hues) superior colliculi tracts. The saturation of the color (intensity of the color scale) indicates the voxel value in the connectivity distribution, which represents the number of samples that passed through the voxel: the lighter the color of the tract (yellow or light blue), the higher the probability of a pathway passing through this voxel. SE showed strong connections from the ipsi- and contralesional superior colliculus to an area close to the frontal eye field (FEF) (*A*: $x = 18$, $y = -2$, $z = 50$), to parieto-occipital areas (*B*: $x = -20$, $y = -56$, $z = 48$), to visual association areas (*C*: $x = -4$, $y = -90$, $z = -22$), and to primary visual areas (*E*: $x = -2$, $y = -90$, $z = 0$). SE also showed projections from the ipsi- and contralesional superior colliculi to spared prefrontal areas on the hemispherectomized side (*D*: $x = 12$, $y = 64$, $z = 2$). (Adapted and reproduced with permission from Leh and others, *Brain* 2006.)

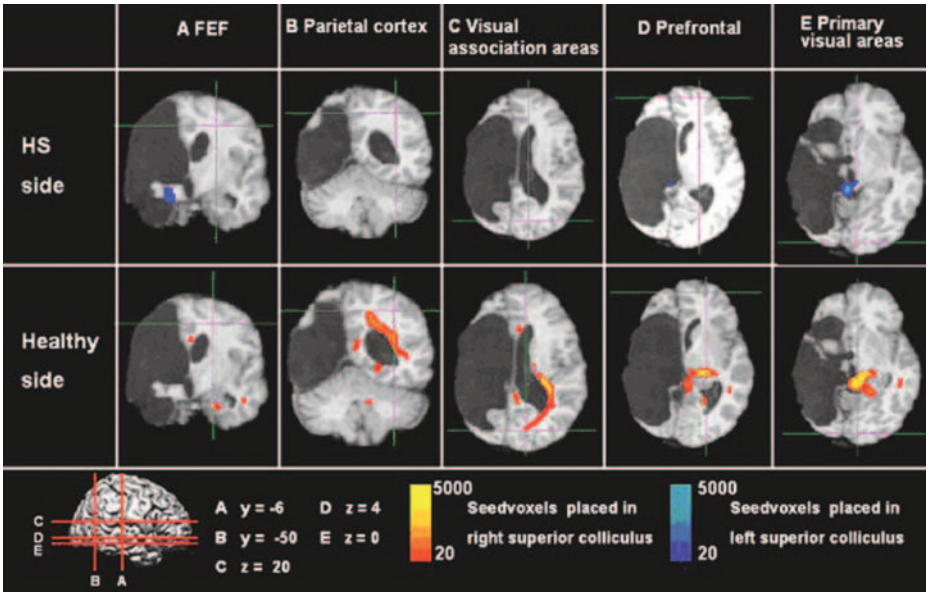

Fig. 8*B*. Diffusion tensor imaging tractography in a hemispherectomized patient (JB) without "Type I" blindsight ("attention blindsight"). The saturation of the color (intensity of the color scale) indicates the voxel value in the connectivity distribution, which represents the number of samples that passed through the voxel: the lighter the color of the tract (yellow or light blue), the higher the number of probable fibers passing through this voxel. Reconstructed superior colliculi tracts demonstrate almost no connections from the ipsilesional superior colliculus, and projections between the contralesional superior colliculus and other cortical areas suggest degeneration of both superior colliculi. FEF = frontal eye field. (Adapted and reproduced with permission from Leh and others, *Brain* 2006.)

The SC is the source of two major descending tracts: tectospinal (efferent, including projections to the reticular formation, the cervical cord, and the inferior colliculus; Kandel and others 2000, p. 669) and tectopontine (to the cerebellum). Its neurons are organized topographically with connections to MT (whose neurons are very sensitive to movement; Lyon and others 2005). Phylogenetically, the SC is older than the lateral geniculate nucleus (LGN), such that in lower mammals, it is the main recipient of retinal projections. The SC also projects to the frontal eye fields (FEFs; Sommer and Wurtz 2003), K-layers of the LGN (Lachica and Casagrande 1993), and pulvinar. Similar but weaker retino-collicular projections also exist in humans and were demonstrated in a recent single case study in which visual orientation was restored in a left-sided neglect patient after an additional lesion of the contralesional SC (Weddell 2004).

Anatomical and lesion studies in animals further support a role of subcortical pathways in blindsight. Excitatory and inhibitory intercollicular connections were demonstrated in the cat (Olivier and others 2000; Rushmore and Payne 2003) (Fig. 9), as one dysfunctional SC can significantly influence visual awareness (Sprague 1966; Sherman 1977; Wallace and others 1989; Sewards and Sewards 2000; Weddell 2004) and modulate the activity of the contralateral partner (Rushmore and Payne 2003). Restoration of visual responses in the blind visual field after injection of a GABA antagonist (bicuculline methiodide) into the contralateral SC (Fig. 10) has also been reported (Sherman 1977; Ciaramitaro and others 1997).

In monkeys, the superior colliculi receive direct input from both the retina and the striate cortex and contain a complete representation of the visual field (Schiller 1972). Destriated monkeys can localize visual stimuli in the blind hemifield and perform wavelength discrimination and simple shape and pattern discrimination, as well as perform velocity discrimination (see review in Ptito and others 1996). These abilities are abolished following the additional destruction of the ipsilesional SC (Rodman and others 1990). In hemispherectomized infant monkeys who could detect stimuli in their blind hemifield, anatomical and histochemical studies reveal transneuronal retrograde degeneration of many retinal ganglion cells, a large reduction in volume of the ipsilesional dLGN, but only a very slight reduction in volume of the ipsilesional SC (Ptito and others 1996).

Primate area MT contains a large contingency of direction-selective neurons, and these neurons remain direction-selective following ablation of the striate cortex (Rodman and others 1989). Subsequent collicular ablation extinguishes this direction selectivity (Rodman and others 1990). Thus, the ability to discriminate the direction of motion relies on the integrity of not only the SC but also the extrastriate cortex. This would explain why hemispherectomized patients (with an absence of striate and extrastriate cortex but a presumably intact SC) demonstrate an inability to discriminate the direction of motion in their blind field (horizontal motion or motion-in-depth; Perenin 1991; Ptito and others 1991; King and others 1996).

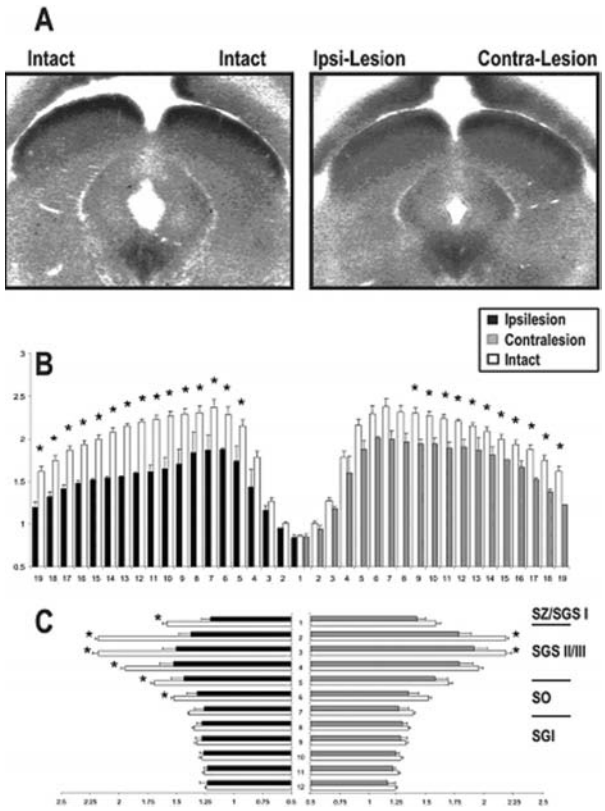

Fig. 9. Effect of a visual cortex lesion on the ipsi- and contralateral superior colliculus in the cat. *A–C*, Impact of unilateral primary visual cortex lesion on 2-deoxyglucose (2DG) uptake in the superior colliculus (SC). *A*, Autoradiographs of the SC from an intact (*left*) and a unilaterally lesioned (*right*) cat. *B*, Quantitative data from medial-lateral analysis of 2DG uptake in the stratum griseum superficiale (SGS). 1–19 represent measurements at medial to lateral sample sites shown. Black bars = ipsilesional SC; gray bars = contralesional SC; open bars = intact levels of 2DG uptake. *C*, Translaminar analysis through the superior colliculus. Conventions as in *B* (SZ = stratum zonale; SGSI = stratum griseum superficiale sublamina I; SGSII/III = SGS sublaminae II and III; SO = stratum opticum; SGI = stratum griseum intermediale). Error bars represent standard error of the mean. (Adapted and reproduced with permission from Rushmore and Payne, *Exp Brain Res* 2003.)

Conclusions and Future Directions

Advances in neuroimaging techniques, careful application of paradigms, and strict control of methodological artifacts have enabled us to confirm the existence of blindsight with an involvement of the superior colliculi in hemispherectomized patients. Although existing superior colliculi connections to the remaining cortical areas seem to play a pivotal role in unconscious vision, blindsight patients remain unaware of the information

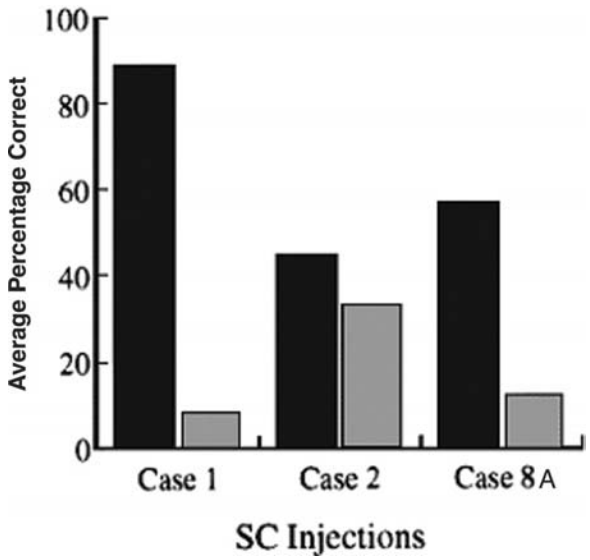

Fig. 10. Recovery of the central versus peripheral portions of the hemifield. Visual perimetry data for the period marking the beginning of recovery (criterion: average of 33% correct responsiveness to stimuli in the previously "hemianopic" field) are shown for the three cases that recovered subsequent to bicuculline methiodide injection in the superior colliculus (SC). Average percentage correct responsiveness is collapsed across the central hemifield, visual stimuli presented 0 to 45 degrees from midline (black bars), and the peripheral hemifield, visual stimuli presented from 45 to 90 degrees from midline (shaded bars). Data reflect injection 1 for case 1, injection 2 for case 2, and injection 2 for case 8*A*. (Adapted and reproduced with permission from Ciaramitaro, Todd, and Rosenquist, *The Journal of Comparative Neurology* 1997.)

processed in their blind visual field. One possibility for the absence of awareness may lie in the lack of synchronicity in cerebral activation. The human visual pathways process information simultaneously and yet are able to work independently of each other (as is the case following a circumscribed lesion in a visual cortical area) (Rees and others 2002; Naghavi and Nyberg 2005). For conscious perception, however, a specific synchronized activation pattern of different cortical areas involving ventral, parietal, and frontal visual areas is believed to be crucial (see, e.g., Beck and others 2001; Rees and others 2002; Naghavi and Nyberg 2005). Our results indicate that hemispherectomized patients with Type I or attention blindsight are able to enhance visual performance in their blind field but remain unaware of visual processing presumably because they are unable to access a more complex synchronous cortical activation pattern involving higher top-down mechanisms necessary for conscious vision.

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