# NEURAL SUBSTRATES OF BLINDSIGHT IN HEMISPHERECTOMIZED SUBJECTS

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**Abstract**

Blindsight is a visual phenomenon whereby hemianopic subjects are able to process visual information in their blind visual field without awareness. Previous research demonstrating the existence of blindsight in hemianopic subjects has been criticised for the nature of the paradigms used, for the presence of methodological artefacts as well as for the possibility that spared islands of visual cortex may have sustained the phenomenon since the subjects generally had small circumscribed lesions. In order to respond to these criticisms, we have been investigating for several years now, residual visual abilities in the blind field of hemispherectomized subjects in whom a whole cerebral hemisphere has been removed or disconnected from the rest of the brain. These subjects have offered a unique opportunity to establish the existence of blindsight and to investigate its underlying neuronal mechanisms since in these cases spared islands of visual cortex cannot be evoked to explain the presence of visual abilities in the blind field. In addition, we have been using precise behavioural paradigms, strict control for potential methodological artefacts such as light scatter, fixation, criterion effects and macular sparing and we have utilized new neuroimaging techniques such as Diffusion Tensor Imaging Tractography to enhance our understanding of the phenomenon. The following article is a review of our research on the involvement of the superior colliculi in blindsight in hemispherectomized subjects.

## Introduction

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 & Krieger, 1951; Perenin & Jeannerod, 1974; Pöppel et al., 1973; Cowey & Stoerig, 1995, 1997). This visual phenomenon, whereby subjects 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 et. al 1974; Weiskrantz, 1986; Shefrin et al., 1988).

The observation that residual visual abilities vary between subjects (for example Corbetta et al., 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).

Subjects with 'Type I' blindsight demonstrate unconscious residual visual abilities that have been associated with a retinal-tectal pathway (Weiskrantz, 1989; Sahraie et al., 1997). This includes neuroendocrine responses such as melatonin suppression following exposure to a bright light (Czeisler et al., 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 (Marzi et al., 1986; Torjussen, 1978).

Subjects with 'Type II' blindsight possess some awareness of residual visual abilities such as target detection and localization by saccadic eye movements (Pöppel, Held & Frost, 1973; Weiskrantz, Warrington, Sanders & Marshall, 1974; Weiskrantz, 1989) and manual pointing (Weiskrantz, Warrington, Sanders & Marshall, 1974), movement direction detection,

relative velocity discrimination (Barbur, Ruddock & Waterfield, 1980; Blythe, Kennard & Ruddock, 1986; Blythe, Kennard & Ruddock, 1987; Weiskrantz et al., 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 & Rossetti (2005) have 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 subcategories: i) 'Action blindsight' is observed when an action is used to guess the localization of a target by pointing or saccading in the blind field; (ii) '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 & Rossetti 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; (iii) '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 dLGN (**Table1, Fig. 1**).

## Limitations of previous research

Several researchers (Campion et al., 1983; Fendrich et al., 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 as well as intra- and extra-ocular light scatter (Faubert et al., 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 subjects' 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 favour 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 et al., 2004).

Alternatively, indirect methods, which require the subject to react only to consciously perceived stimuli, have been developed to exclude methodological artefacts such as response bias. Implicit processing of a stimulus, which does not require a direct response from the subject, has been demonstrated within a field defect. For example, Zihl et al. (1980) has 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 et al., 1997) in which the simultaneous presentation of an unseen

stimulus can alter the mean reaction time to a seen stimulus (Marzi et al., 1986). With this approach, subjects 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 & Sugar, 1975¸ Rosenblatt at al., 1998) as well as 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 et al., 1992).

**Model: Hemispherectomy**

In order to eliminate the possibility that residual vision is mediated by spared striate cortex, we have conducted a series of studies on hemispherectomy subjects 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 subjects, striate cortex has been entirely ablated or deafferented such that explanations for blindsight based on spared striate cortex and lateral geniculate (LGB) or collicular projection to 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 & Marino Jr.,

2005; De Almeida et al., 2006; Fountas et al., 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.).

Hemispherectomized subjects 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 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 homologue in the intact hemisphere. Such structural integrity suggests preserved function.

## Behavioural experiments

### 1. Residual vision with awareness: Object discrimination, movement detection and localization

We tested a first group of hemispherectomized subjects in 1987 (Ptito et al.) in a pattern (2D) and an object (3D) discrimination task. The subjects 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 carried out monocularly and eye

movements were monitored through the use of Beckman EOG electrodes. Results showed that compared to a matched control group, hemispherectomized subjects 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 subjects when 3D stimuli were presented bilaterally. No discrimination was possible for any of the experimental subjects 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 subjects 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 et al., 1991). Beckman EOG electrodes were used to monitor eye movements and fixation was ensured by requiring the subject 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 subjects 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 subjects' denial of the presence of a stimulus and we were

probably measuring residual vision rather than blindsight as described at the time (Weiskrantz et al., 1974).

In a second experiment, we asked the subjects 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 subject was able to discriminate large and median differences in 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 while a single subject 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 subjects to report whether the direction 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 while the subjects obtained over 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 subjects (**Fig. 3, Table2, Table 3)**.

The positive visual functions in the blind hemifield of hemispherectomized subjects have been put into doubt by some control experiments suggesting that there may have been stray light entering the intact hemifield (King et al., 1996b). Subsequently, we showed the importance of controlling intraocular light scatter since spectral sensitivity within the blind field can be reduced

considerably and yet, high intensity stimuli can be detected probably by foveal receptors (Stoerig et al., 1996). We then presented a model that could explain the scatter properties of the eye on the visual sensitivities obtained with hemispherectomized subjects (Faubert et al., 1999).

Taking these factors into consideration and controlling for them, we nevertheless confirmed in a separate group of hemisphectomized subjects 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 et al., 1992; Wessinger et al., 1996; **Fig. 4**). A double Purkinje eye tracker was used with two hemispherectomized subjects in order 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 subjects using stabilized field mapping. An area was identified in both subjects' 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° at one field location for each subject. The areas of residual vision varied among subjects. With SE, the band was within both visual quadrants, but only above the horizontal meridian for JB. The subjects 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 subjects 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 subjects performed at chance when stimuli were outside the areas with spared detection while they were always identified correctly in each subject's seeing field (**Fig. 4** and **Table 3**).

**2. Residual vision without awareness (blindsight): Spatial Summation Effect Paradigm**

Scepticisms concerning the existence of blindsight and the methods (e.g. lax decisional criterion) remained, however. We thus decided to test four hemispherectomized subjects 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 subject's response to stimuli in the intact field. This is so because the subject 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 et al., 1997). Results showed that none of the patients were aware of stimuli (single or double) presented in their blind hemifield. Three subjects showed a spatial summation effect in their normal visual field (DR, SE, IG) and two subjects (DR and SE) showed a spatial summation effect when stimuli were presented across the vertical meridian in their blind and normal visual field despite their lack of visual awareness in their blind hemifield (**Fig. 5**). The results in subjects DR and SE are in keeping with previous studies using the spatial summation effect paradigm (Raab, 1962; Blake, Martens, & Di Gianfilippo, 1980; Marzi, et al., 1986; Minuissi, Gireli, & Marzi, 1998; Savazzi, & Marzi, 2002). We also conducted a second experiment in order to exclude the possibility that light scatter could account for the effect observed in the two hemispherectomized subjects. In this experiment the second stimulus was presented to the blind spot of normal control subjects and none of these subjects 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 subjects 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 et al., 1987; 1991), particularly for hemispherectomized subjects, 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 subjects (Leh et al., 2006a). We used the fact that electrophysiological studies indicate that the primate superior colliculus does not receive retinal input from shortwave-sensitive (S-) cones involved in colour vision, consequently rendering them colour blind to blue⁄yellow stimuli (Marrocco & Li, 1977; Schiller & Malpeli, 1977; Sumner et al., 2002; Savazzi & Marzi, 2004).

Our goal was to demonstrate the absence of S-cone input in the blind visual field of hemispherectomized subjects 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 either the achromatic post-receptoral pathway or the blue/yellow post-receptoral pathway which draws on S-cones while remaining invisible to the other post-receptoral 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 subjects, who had shown blindsight in previous studies reliably, were included in the study. These subjects demonstrated a spatial summation effect only to achromatic stimuli (**Fig. 6**),

suggesting that their blindsight is colour-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 superior colliculus or the contralesional pulvinar on to the remaining hemisphere. Previous studies have shown that the superior colliculus is not receiving retinal input from short-wave-sensitive cones (Marrocco & Li, 1977; Schiller & Malpeli, 1977; Sumner, Adamjee, & Mollon, 2002; Savazzi & Marzi, 2004), in contrast to the pulvinar, which receives input from all classes of colour-opponent ganglion cells (L/M as well as S-cone opponent) (Felsten et al., 1983; Cowey et al., 1994) and appears to be involved in colour processing in humans (Barrett et al., 2001). We therefore concluded from this study that blindsight is likely mediated by the superior colliculi in hemispherectomized subjects.

**Imaging studies**

**1. Functional MRI studies**

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

To investigate more directly the neural pathways involved in blindsight and/or residual vision, we conducted an fMRI experiment (Bittar et al., 1999) with three hemispherectomized subjects (JB, IG and DR) who participated in the Tomaiuolo et al. study (Tomaiuolo et al.,

1997). To the best of our knowledge, this was the first functional neuroimaging study with hemispherectomized subjects 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 et al., 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 subject (DR) who had demonstrated blindsight in previous studies. Since no significant activation within the superior colliculi or pulvinar of either the experimental or control subjects were 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 since 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 subjects with and without blindsight (Leh et al., 2006b). 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 et al., 1999; Behrens et al., 2003). First, $T_1$–weighted anatomical MRI images and diffusion weighted images were obtained. We then created seed masks on each subject's T1-weighted image, including the whole superior colliculi and used a probabilistic algorithm model for the DTI data analysis which allowed for an estimation of the most probable location of a single fiber connection (for further information see Leh et al., 2006b).

Results of this DTI tractography study demonstrated the presence of projections from the ipsi- and contralesional superior colliculus to primary visual areas, visual association areas, precentral areas/FEF and the internal capsule of the remaining hemisphere in hemispherectomized subjects with 'Type I' or 'attention-blindsight' (**example in Fig. 8A**) and an absence of these connections in hemispherectomized subjects without 'Type I' or 'attention-blindsight' (**example in Fig. 8B)**, thereby confirming our assumption (Tomaiuolo et al., 1997; Bittar et al., 1999) and that of Danckert & Rossetti (2005) that blindsight is mediated by a collicular route. Interestingly, connections from the ipsilesional superior colliculus in subjects 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.

## Discussion/ Potential Neuronal Substrate

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 subjects. This would be achieved either by a process of cortical plasticity and/or by utilization of existing neural pathways such as subcortical nuclei. Several observations have supported previous suggestions that the superior colliculus plays an important role in

blindsight (Kisvarday et al., 1991; Ptito et al., 1996; Sahraie et al., 1997; Morris, Öhman, & Dolan, 1999; de Gelder et al., 1999).

The superior colliculus is the source of two major descending tracts: tectospinal (efferent, including projections to the reticular formation, the cervical cord and the inferior colliculus; Kandel, Schwart & Jessell, 2000) and tectopontine (to the cerebellum). Its neurons are organized topographically with connections to MT (whose neurons are very sensitive to movement; Lyon, Nassi, & Callaway, 2005). Phylogenetically, the superior colliculus is older than the lateral geniculate nucleus, such that in lower mammals it is the main recipient of retinal projections. The superior colliculus also projects to the frontal eye fields (FEF; Sommer & Wurtz, 2003), K-layers of the LGN (Lachica & 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 superior colliculus (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 (Oliver et al., 2000; Rushmore & Payne, 2003; **Fig. 9**) as one dysfunctional superior colliculus can significantly influence visual awareness (Sprague, 1966; Sherman, 1977; Wallace at al., 1989; Sewards & 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 superior colliculus (**Fig. 10**) has also been reported (Ciaramitaro et al., 1997; Sherman, 1977).

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, perform wavelength discrimination, simple shape and pattern discrimination, as well as carry out velocity discrimination (see review in Ptito et al., 1996). These abilities are abolished following the additional destruction of the ipsilesional superior colliculus (Rodman et al., 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 superior colliculus (Ptito et al., 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 et al., 1989). Subsequent collicular ablation extinguishes this direction-selectivity (Rodman et al., 1990). Thus the ability to discriminate the direction of motion relies upon the integrity of not only the superior colliculus, but also of the extrastriate cortex. This would explain why hemispherectomized patients (with an absence of striate and extrastriate cortex, but a presumably intact superior colliculus) demonstrate an inability to discriminate the direction of motion in their blind field (horizontal motion or motion-in-depth; Perenin, 1991; Ptito et al., 1991; King et al., 1996).

## Conclusion/Future Directions

Advances in neuroimaging techniques, careful application of paradigms as well as strict control of methodological artifacts have enabled us to confirm the existence of blindsight with an involvement of the superior colliculi in hemispherectomized subjects. Although existing superior colliculi connections to the remaining cortical areas seem to play a pivotal role in unconscious vision, blindsight subjects remain unaware of the information 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 et al., 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, for example, Beck et al., 2001; Rees et al., 2002; Naghavi and Nyberg, 2005). Our results indicate that hemispherectomized subjects 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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## Competing Interests Statement

The authors declare that they have no competing financial interests.

Abbreviations: Diffusion Tensor Imaging (DTI)

## References


Barbur JL, Ruddock KH, Waterfield VA. 1980. Human visual responses in the absence of the geniculo-calcarine projection. Brain 103(4): 905-28.

Bard L. 1905. De la persistance des sensations lumineuses dans le champ aveugle des hemianopiques. Sem Medicale Med Soc 22: 253-255.

Barrett NA, Large MM, Smith GL, Michie PT, Karayanidis F, Kavanagh DJ, Fawdry R, Henderson D, O'Sullivan BT. 2001. Human cortical processing of colour and pattern. Hum Brain Mapp., 13(4), 213-25.

Beck DM, Rees G, Frith CD, Lavie N. 2001. Neural correlates of change detection and change blindness. Nat Neurosci 4: 645–50.

Behrens TEJ, Johansen-Berg H, Woolrich MW, Smith SM, Wheeler-Kingshott CAM, Boulby PA, Barjer GJ, Sillery EL, Sheehan K, Ciccarelli O, Thompson AJ, Brady JM, Matthews PM. 2003. Non-invasive mapping of connections between human thalamus and cortex using diffusion imaging. Nat Neurosci. 6: 750 - 757.

Bender MB, Krieger HP. 1951. Visual function in perimetrically blind fields. AMA Arch Neurol Psychiatry 65(1): 72-9.

Bittar RG, Ptito M, Faubert J, Dumoulin SO, Ptito A. 1999. Activation of the remaining hemisphere following stimulation of the blind hemifield in hemispherectomized subjects. NeuroImage 10: 3339-346.

Blake R, Martens W, Di Gianfilippo A. 1980. Reaction time as a measure of binocular interaction in human vision. Invest Ophthalmol. 19(8): 930-41.

Blythe IM, Bromley JM, Kennard C, Ruddock KH. 1986. Visual discrimination of target displacement remains after damage to the striate cortex in humans. Nature 320(6063):619-21.

Blythe IM, Kennard C, Ruddock KH. 1987. Residual vision in patients with retrogeniculate lesions of the visual pathways. Brain 110( Pt 4): 887-905.

Campion J, Latto R, Smith YM. 1983. Is blindsight an effect of scattered light, spared cortex and near-normal vision? Behav Brain Sci. 6:423-486.

Ciaramitaro VM, Todd WE, Rosenquist AC. 1997. Disinhibition of the superior colliculus restores orienting to visual stimuli in the hemianopic field of the cat. J Comp Neurol. 387: 568-587.

Conturo TE, Lori NF, Cull TS, Akbudak E, Snyder AZ, Shimony JS, McKinstry RC, Burton H, Raichle ME. 1999. Tracking neuronal fiber pathways in the living human brain. Proc Natl Acad Sci U S A 96(18): 10422 – 10427.

Corbetta M, Marzi CA, Tassinari G, Aglioti S. 1990. Effectiveness of different task paradigms in revealing blindsight. Brain 113: 603-616.

Cowey A. 2004. The 30th Sir Frederick Bartlett lecture. Fact, artifact, and myth about blindsight. Q. J. Exp. Psychol. A. 57A: 577-609.

Cowey A, Stoerig B, Bannister M. 1994. Retinal ganglion cells labelled from the pulvinar nucleus in macaque monkeys. Neuroscience 61(3): 691-705.

Cowey A, Stoeirg P. 1995. Blindsight in monkeys. Nature 373: 247-249.

Cowey A, Stoerig P. 1997. Visual detection in monkeys with blindsight. Neuropsychologia 35(7): 929-939.

Czeisler CA, Shanahan TL, Klerman EB, Martens H, Brotman DJ, Emens JS, Klein T, Rizzo JF 3rd. 1995. Suppression of melatonin secretion in some blind patients by exposure to bright light. N Engl J Med. 332(1):6-11

Danckert J., Rossetti Y. 2005. Blindsight in action: what can the different sub-types of blindsight tell us about the control of visually guided actions? Neurosci Biobehav Rev. 29(7): 1035-46.

De Almeida AN, Marino Jr R. 2005. The early years of hemispherectomy. Pediatr Neurosurg 41: 137-140.

De Almeida AN, Marino Jr R, Aguiar PH, Teixeira MJ. 2006. Hemispherectomy: a schematic review of the current techniques. Neurosurg Rev 29: 97-102.

de Gelder B, Vroomen J, Pourtois G, Weiskrantz L 1999. Non-conscious recognition of affect in the absence of striate cortex. Neuroreport 10(18): 3759-63.

Faubert J, Diaconu V, Ptito M, Ptito A. 1999. Residual vision in the blind field of hemidecorticated humans predicted by a diffusion scatter model and selective spectral absorption of the human eye. Vision Res. 39(1):149-57.

Fendrich R, Wessinger CM, Gazzaniga MS. 1992. Residual vision in a scotoma: implications for blindsight. Science 258(5087): 1489-91.

Felsten G, Benevento LA, Burman D. 1983. Opponent-color responses in macaque extrageniculate visual pathways: the lateral pulvinar. Brain Res. 288(1-2): 363-7.

Fountas KN, Smith JR, Robinson JS, Tamburrini G, Pietrini D, Di Rocco C. 2006. Anatomical Hemispherectomy. Childs Nerv Syst 22: 982-991.

Herter TM, Guitton D. 2004. Accurate bidirectional saccade control by a single hemicortex. Brain. 127(Pt 6):1393-402.

Kandel ER, Schwartz JH, Jessell, TM. 2000. Principles of neural science. McGrawHill, 4th edition p. 669.

King SM, Frey S, Villemure J-G, Ptito A, Azzopardi P. 1996. Perception of motion-in-depth in patients with partial or complete cerebral hemispherectomy. Behavioural Brain Research 76:169-180.

King AJ. 2004. The superior colliculus. Curr Biol 14( 9): 335-338.

Kisvarday ZF, Cowey A, Stoerig P, Somogyi P. 1991. Direct and indirect retinal input into degenerated dorsal lateral geniculate nucleus after striate cortical removal in monkey: implications of residual vision. Exp Brain Res 86(2): 271-92.

Lachica EA, Casagrande VA. 1993. The morphology of collicular and retinal axons ending on small relay (W-like) cells of the primate lateral geniculate nucleus. Vis. Neurosci. 10(3): 403-418.

Leh SE, Ptito A, Mullen KT. 2006a. Absence of S-cone input in human 'attention-blindsight' Eur J Neurosci. Eur J Neurosci. 24(10), 2954-60.

Leh SE, Johansen-Berg H, Ptito A. 2006b. Unconscious Vision: New insights into the neuronal correlate of blindsight using diffusion tractography. Brain 129 (Pt7), 1822-32.

Lyon DC, Nassi JJ, Callaway EM. 2005. Disynaptic connections from the superior colliculus to cortical area MT revealed through transynaptic labeling with rabies virus. Journal of Vision 5(8), 432.

Marcel AJ. 1998. Blindsight and shape perception: deficit of visual consciousness or of visual function? Brain 121 ( Pt 8): 1565-88.

Marrocco RT, Li RH. 1977. Monkey superior colliculus: properties of single cells and their afferent inputs. J Neurophysiol 40(4): 844-860.

Marzi CA, Tassinari G, Agliotti S, Lutzemberger L. 1986. Spatial summation across the vertical meridian in hemianopics: a test of blindsight. Neuropsychologia 24(6): 749-758.

Miniussi C, Girelli M, Marzi CA. 1998. Neural Site of the redundant target effect: electrophysiological evidence. J Cogn Neurosci 10(2): 216-230.

Morris JS, Öhman A, Dolan RJ. 1999. A subcortical pathway to the right amygdala mediating 'unseen' fear. Proc Natl Acad Sci U S A 96: 1680-1685.

Naghavi HR, Nyberg L. 2005. Common fronto-parietal activity in attention, memory, and consciousness: shared demands on integration? Conscious Cogn 14: 390–425.

Olivier E, Corvisier J, Pauluis Q, Hardy O. 2000. Evidence for glutamatergic tectotectal neurons in the cat superior colliculus: a comparison with GABAergic tectotectal neurons. Eur. J. Neurosci. 12: 2354-66.

Perenin MT. 1991. Discrimination of motion direction in perimetrically blind fields. Neuroreport 2(7):397-400.

Perenin MT, Jeannerod M. 1974. Residual vision in cortically blind hemiphields. Neurospychologia 13: 1-7.

Pöppel E, Frost D, Held R. 1973. Residual visual function after brain wounds involving the central visual pathways in man. Nature 243: 295-296.

Ptito A, Lassonde M, Lepore F, Ptito M. 1987. Visual discrimination in hemispherectomized patients. Neuropsychologia 25(6): 869-879.

Ptito A, Lepore F, Ptito M, Lassonde M. 1991. Target detection and movement discrimination in the blind field of hemispherectomized patients. Brain 114: 497-512.

Ptito M, Herbin M, Boire D, Ptito A. 1996. Neural bases of residual vision in hemicorticectomized monkeys. Prog Brain Res. 112: 385-404.

Raab DH. 1962. Statistical facilitation of simple reaction times. Trans N Y Acad Sci 24: 574-590.

Rees G, Kreiman G, Koch C. 2002. Neural correlates of consciousness in humans. Nat Rev Neurosci 3: 261–70.

Riddoch G. 1917 Dissociation of visual perception due to occipital injuries, with special reference to the appreciation of movement. Brain 40: 15-57.

Rodman HR, Gross CG, Albright TD. 1989. Afferent basis of visual response properties in area MT of the macaque. I. Effects of striate cortex removal. J Neurosci. 9(6): 2033-50.

Rodman HR, Gross CG, Albright TD. 1990. Afferent basis of visual response properties in area MT of the macaque. II. Effects of superior colliculus removal. J Neurosci. 10(4): 1154-64.

Ro T, Shelton D, Lee OL Chang E. 2004. Extrageniculate mediation of unconscious vision in transcranial magnetic stimulation-induced blindsight. Proc Natl Acad Sci U S A 101(26): 9933-9935.

Rosenblatt B, Vernet O, Montes JL, Andermann F, Schwartz S, Taylor LB, Villemure JG, Farmer JP. 1998. Continuous unilateral epileptiform discharge and language delay: effect of functional hemispherectomy on language acquisition. Epilepsia 39(7): 787 - 92.

Rushmore RJ, Payne BR. 2003. Bilateral impact of unilateral visual cortex lesions on the superior colliculus. Exp Brain Res 151: 542-547.

Sahraie A, Weiskrantz L, Barbur JL, Simmons A, Williams SCR, Brammer MJ. 1997. Pattern of neuronal activity with conscious and unconscious processing of signals. Proc. Natl. Acad. Sci. USA 94: 9406-9411.

Savazzi S, Marzi CA. 2002. Speeding up reaction time with invisible stimuli. Current Biology 12(5): 403 – 7.

Savazzi S, Marzi CA. 2004. The superior colliculus subserves interhemispheric neural summation in both normals and patients with a total section or agenesis of the corpus callosum. Neuropsychologia 42: 1608-1618.

Schiller PH. 1972. Some functional characteristics of the superior colliculus of the rhesus monkey. Bibl Ophthalmol. 1972;82:122-9.

Schiller PH, Malpeli JG. 1977. Properties and tectal projections of monkey retinal ganglion cells. J Neurophysiol 40(2): 428-445.

Sewards TV, Sewards M. 2000. Visual awareness due to neuronal activities in subcortical structures: a proposal. Conscious Cogn 9: 86-116.

Shefrin SL, Goodin DS, Aminoff MJ. 1988. Visual evoked potentials in the investigation of "blindsight". Neurology 38(1): 104-9.

Sherman SM. 1977. The effect of superior colliculus lesions upon the visual fields of cats with cortical ablationsJ Comp Neurol 172(2): 211-229.

Sincich LC, Park KF, Wohlgemuth MJ, Horton JC. 2004. Bypassing V1: a direct geniculate input to area MT. Nat Neurosci 7(10): 1123-1127.

Smith A, Sugar O. 1975. Development of above normal language and intelligence 21 years after left hemispherectomy. Neurology 25(9): 813 - 8.

Sommer MA, Wurtz RH. 2003. What the brain stem tells the frontal cortex. I. Oculomotor signals sent from the superior colliculus to the frontal eye field viamediodorsal thalamusJ Neurophysiol 91: 1381-1402.

Sprague JM. 1966. Interaction of cortex and superior colliculus in mediation of visually guided behavior in the cat. Science 153(743): 1544-7.

Stoerig P, Faubert J, Ptito M, Diaconu V, Ptito A. 1996. No blindsight following hemidecortication in human subjects? Neuroreport 7(12):1990-4.

Sumner P, Adamjee T, Mollon JD. 2002. Signals invisible to the collicular and magnocellular pathways can capture visual attention. Curr Biol 12: 1312-1316.

Tomaiuolo F, Ptito M, Marzi CA, Paus T, Ptito A. 1997. Blindsight in hemispherectomized patients as revealed by spatial summation across the vertical meridian. Brain 120 (Pt 5): 795-803.

Torjussen T. 1978. Visual processing in cortically blind hemifields. Neuropsychologia 16(1): 15-21.

Ueki K. 1966. Hemispherectomy in the human with special reference to the preservation of function. Prog Brain Res 21: 285-338.

Wallace SF, Rosenquist AC, Sprague, JM. 1989. Recovery from cortical blindness mediated by destruction of nontectotectal fibers in the commissure of the superior colliculusJ Comp Neurol 284(3): 429-450.

Weddell RA. 2004. Subcortical modulation of spatial attention including evidence that the Sprague effect extends to man. Brain Cogn 55: 497-506.

Weiskrantz L, Warrington EK, Sanders MD, Marshall J. 1974. Visual capacity in the hemianopic field following a restricted occipital ablation. Brain 97(4): 709-28.

Weiskrantz L. 1986. Blindsight: a case study and implications. Oxford: Clarendon Press.

Weiskrantz L, Barbur JL, Sahraie A. 1995. Parameters affecting conscious versus unconscious visual discrimination with damage to the visual cortex (V1). Proc Natl Acad Sci U S A 92(13): 6122-6.

Weiskrantz L. 1989. nsciousness and commentaries. In: Hameroff S, Kaszniak A & Scott A. (Eds.), Towards a Science of Consciousness II—The Second Tucson Discussion and Debates. (MIT Press, Cambridge, 371–377).

Weiskrantz L, Cowey A, Barbur JL. 1999. Differential papillary constriction and awareness in the absence of striate cortex. Brain 122: 1533-1538.

Wessinger CM, Fendrich R, Ptito A, Villemure JG, Gazzaniga MS. 1996. Residual vision with awareness in the field contralateral to a partial or complete functional hemispherectomy. Neuropsychologia 34(11):1129-37.

Zeki S, Ffytche DH. 1998. The Riddoch syndrome: insights into the neurobiology of conscious vision. Exp. Brain Res. 121: 25-45.

Zihl J, Tretter F, Singer W. 1980. Phasic electrodermal responses after visual stimulation in the cortically blind hemifield. Behav Brain Res 1: 197-203.

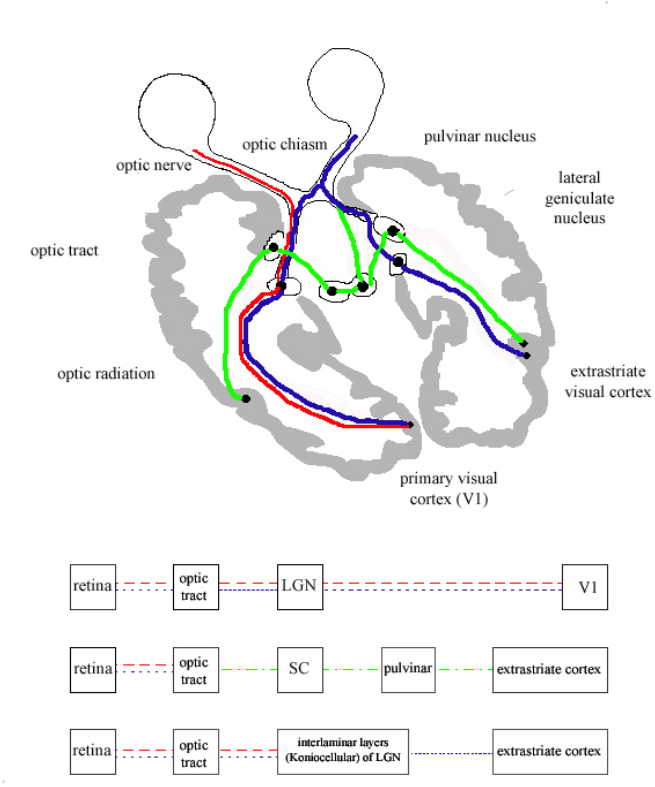


**Fig. 1. Possible pathways involved in blindsight.**

**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 et al., 2004). The possibility exists for other such pathways from the interlaminar layers of the LGN to regions of extrastriate cortex other than area MT. (Adapted and reproduced with permission from Danckert & Rossetti, Neuroscience and Biobehavioral Reviews 2005).

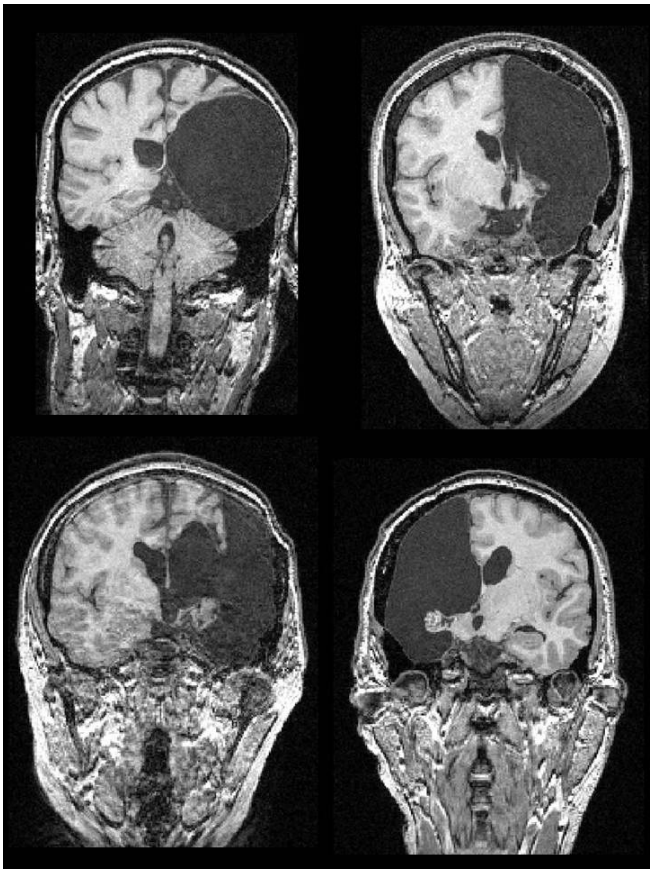

**Fig. 2. Examples of anatomical MRIs of hemispherectomized subjects**

**Fig. 2. Examples of anatomical MRIs of hemispherectomized subjects** showing three right-hemispherectomized and one left-hemispherectomized subject.

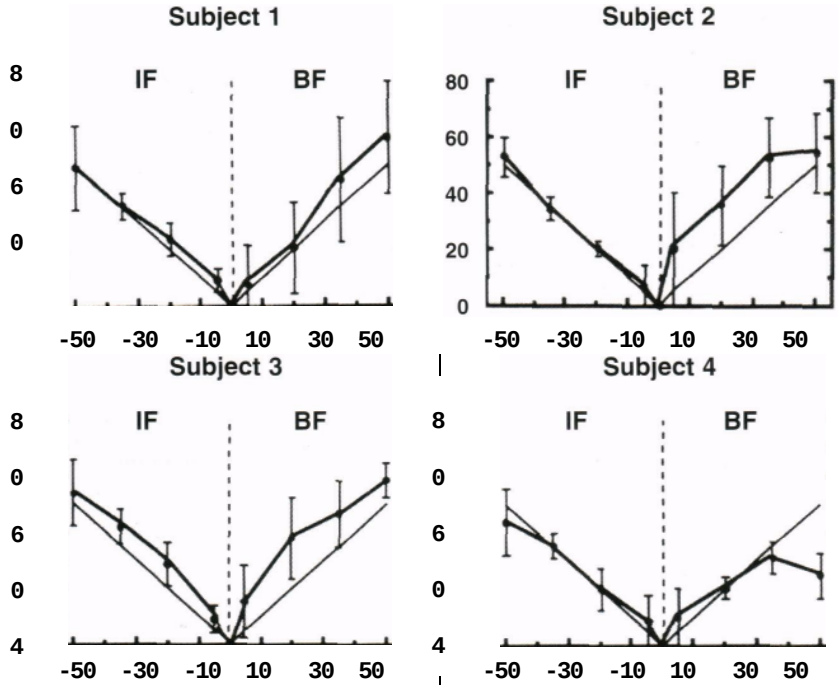


**Fig. 3. Accuracy of localization of combined stationary, moving and flashing targets for four hemispherectomized subjects.**

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

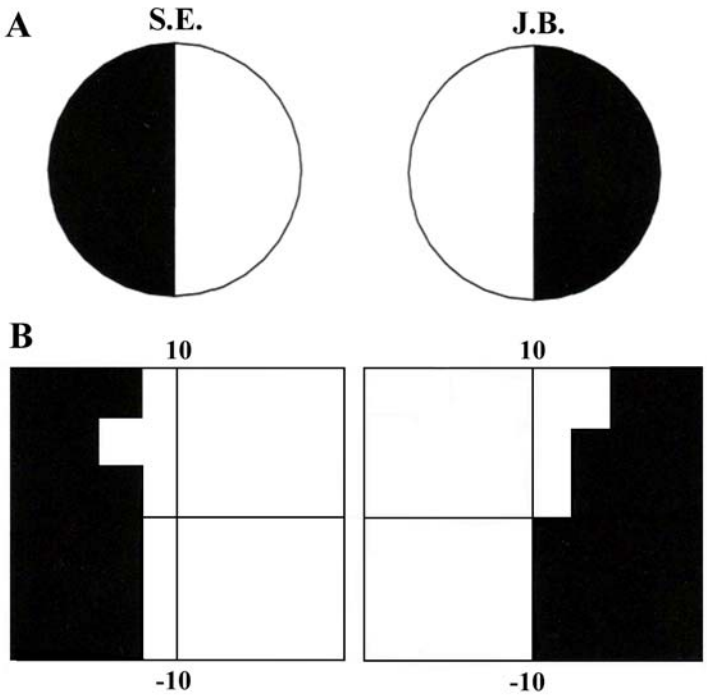


**Fig. 4. Schematic representations of:**

**Fig. 4. Schematic representations of:** **A** perimetric test results of subjects 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 et al., Neuropsychologia 1996)

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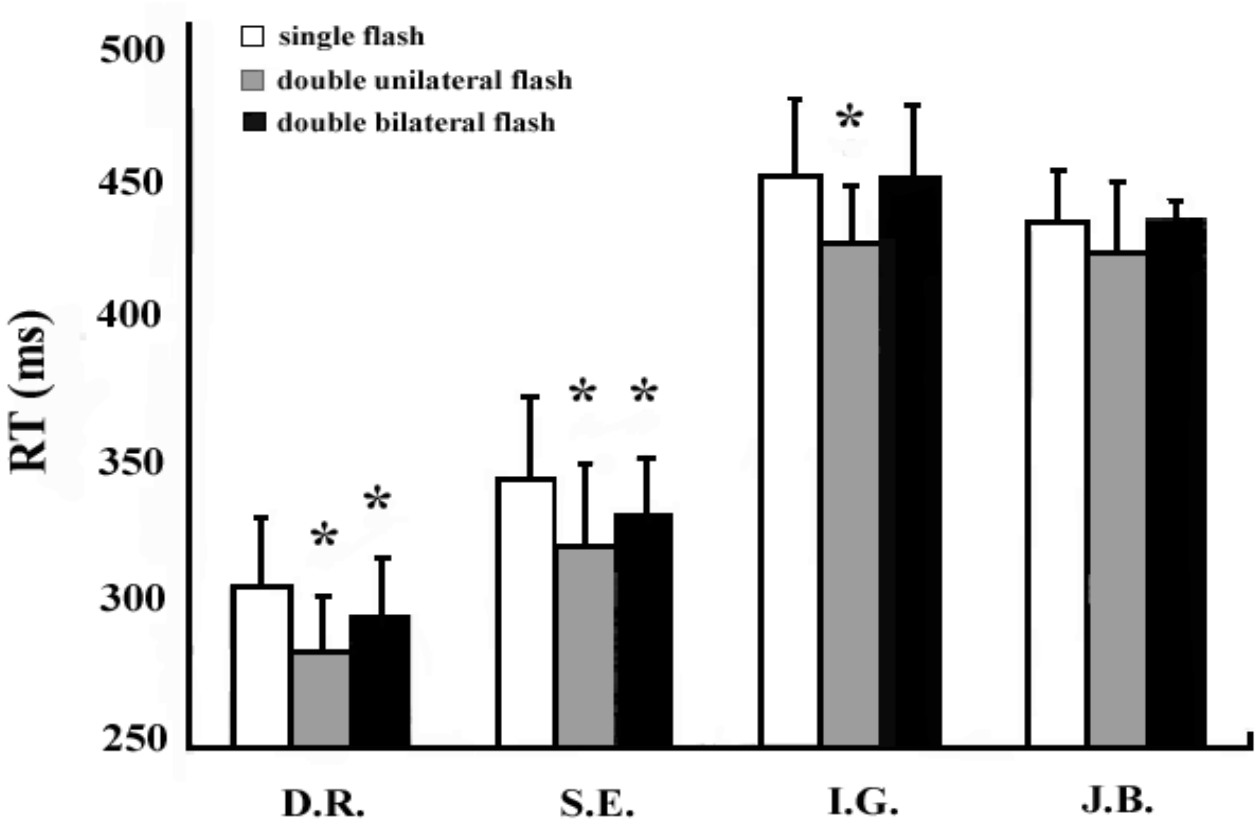


**Fig. 5. Mean reaction times (RT) for two hemispherectomy subjects and a normal control subject who showed a spatial summation effect.**

**Fig. 5. Mean reaction times (RT) for two hemispherectomy subjects and a normal control subject 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; $P = 0.05$). (Adapted and modified from Tomaiuolo et al., Brain 1997)

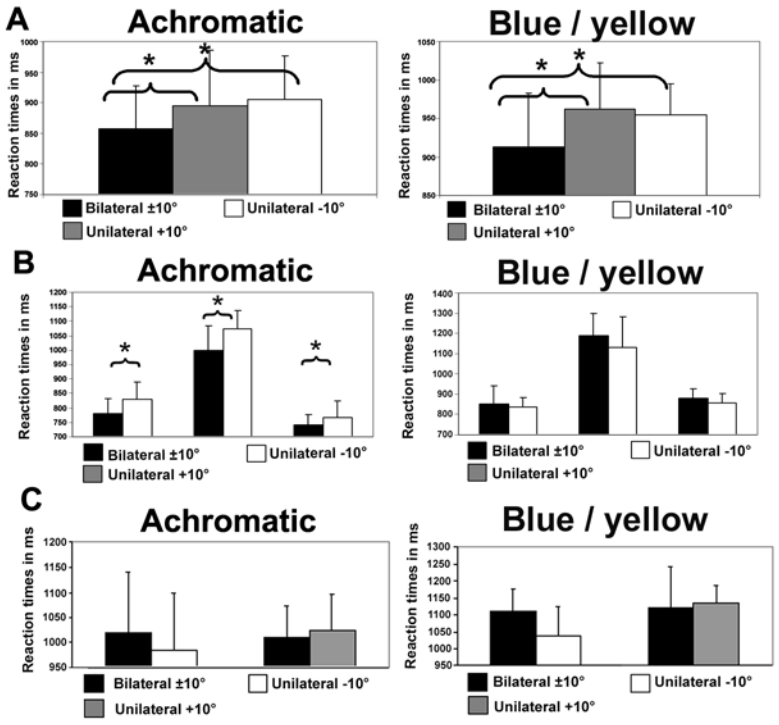


**Fig. 6. Achromatic versus Blue/Yellow Spatial Summation Effect**

**Fig. 6. Achromatic versus Blue/Yellow Spatial Summation Effect** in: **A normal subjects.** A significant spatial summation effect was observed independently of color (N=16, F (1, 15)=23.37; P<0.001). **; B hemispherectomized subjects with blindsight.** (N=3, DR, LF, SE). A spatial summation effect was observed for achromatic stimuli (N=2, DR: t≤ 0.001, df=24; LF: t≤ 0.05, df=24; SE: t≤0.05, df=24), but not for blue/yellow stimuli (DR: t=0.36, df=24; LF: t=0.73, df=24; SE: t≤0.5, df=24)**; C hemispherectomized subjects 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 subjects were tested with the right eye, while the left eye was occluded. (Adapted and reproduced with permission from Leh et al., European Journal of Neuroscience 2006)

* significant

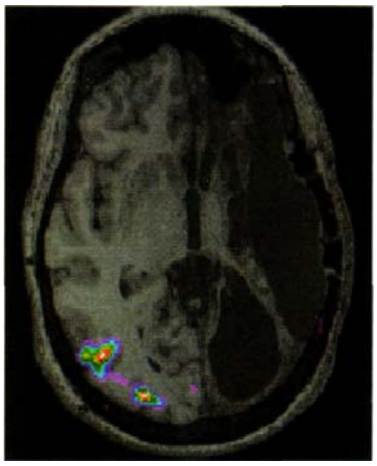

**Fig. 7. Blind (left) hemifield stimulation in a right hemispherectomized subject (D.R.) shown to possess blindsight.**

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

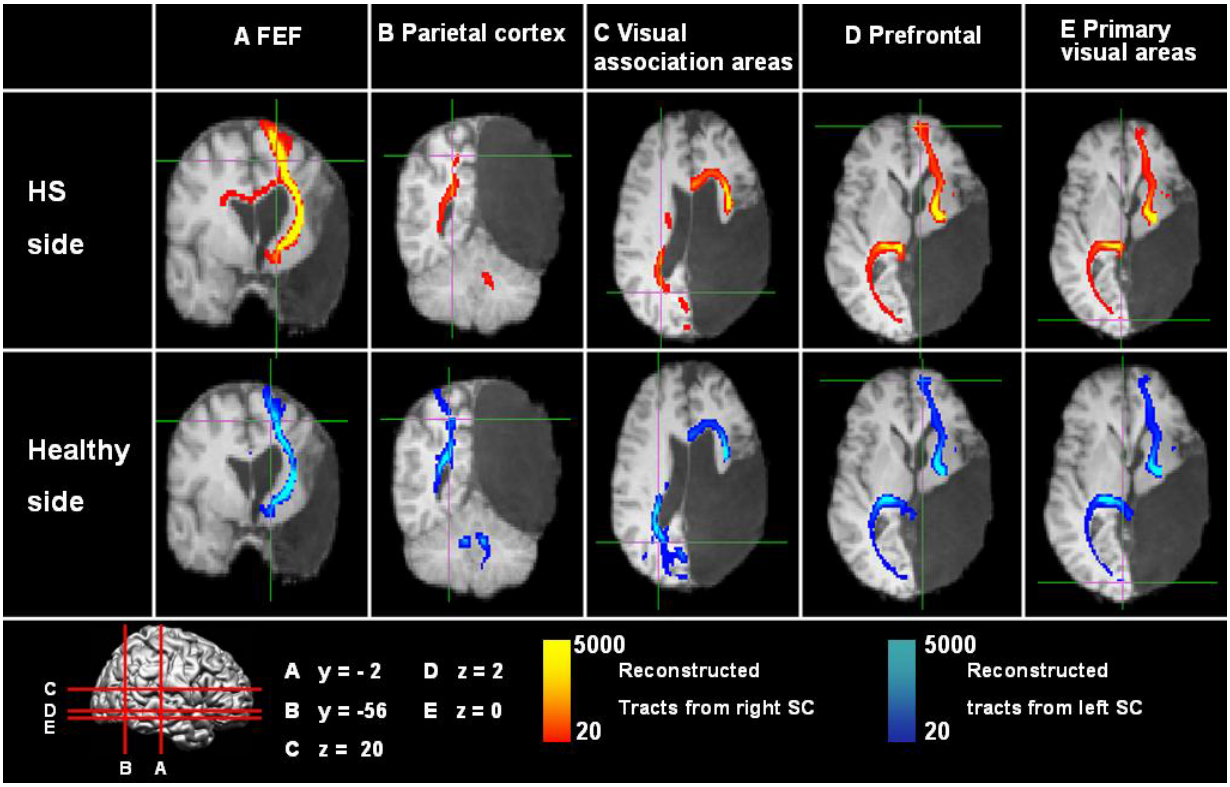


**Fig. 8a. Diffusion Tensor Imaging Tractography in a hemispherectomized subject (S.E.) with 'Type I'-blindsight ('attention-blindsight').**

**Fig. 8a. Diffusion Tensor Imaging Tractography in a hemispherectomized subject (S.E.) 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 colour 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. S.E. showed strong connections from the ipsi- and contralesional superior colliculus to an area close to the FEF (**Fig. 8A;** x = 18, y = -2, z = 50), to parieto-occipital areas (**Fig. 8B**; x = -20, y = -56, z = 48), to visual association areas (**Fig. 8C**; x = -4, y = -90, z = -22) and to primary visual areas (**Fig.8E**; x = -2, y = -90, z = 0). S.E. also showed projections from the ipsi- and contralesional superior colliculi to spared prefrontal areas on the hemispherectomized side (**Fig. 8D**; x = 12, y = 64, z = 2). (Adapted and reproduced with permission from Leh et al., Brain 2006)

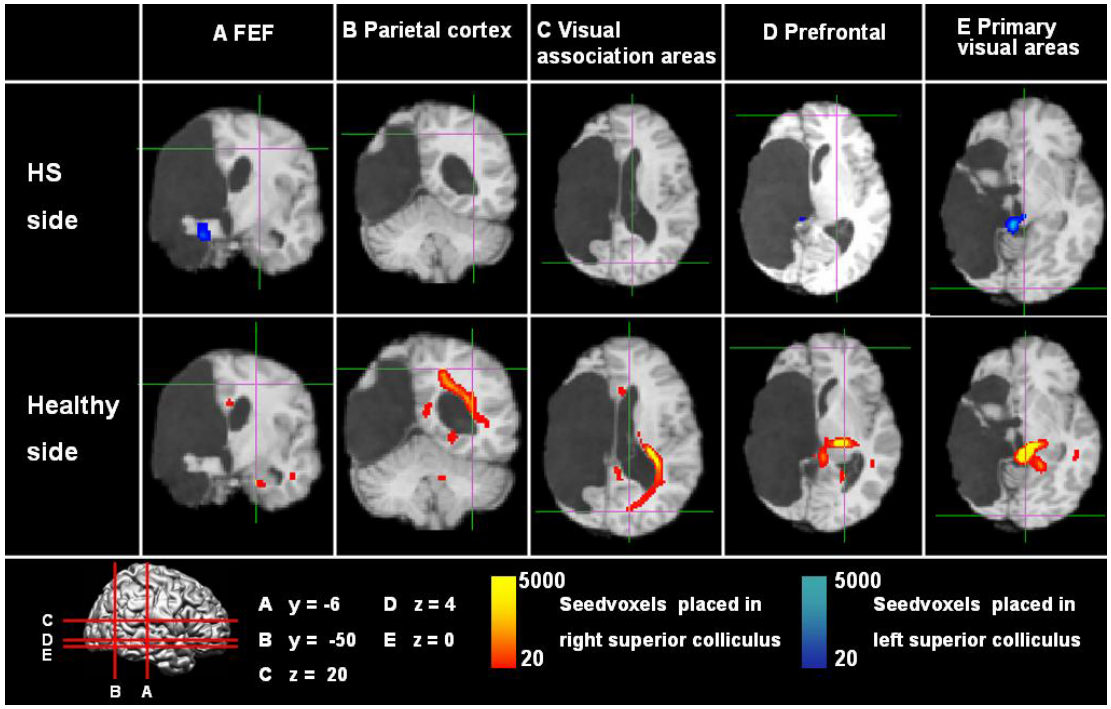


**Fig. 8b. Diffusion Tensor Imaging Tractography in a hemispherectomized subject (J.B.) without 'Type I'-blindsight ('attention-blindsight').**

**Fig. 8b. Diffusion Tensor Imaging Tractography in a hemispherectomized subject (J.B.) without 'Type I'-blindsight ('attention-blindsight').** The saturation of the colour (intensity of the colour scale) indicates the voxel value in the connectivity distribution, which represents the number of samples that passed through the voxel: the lighter the colour of the tract (yellow or light blue) the higher the number of probable fibres 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. (Adapted and reproduced with permission from Leh et al., Brain 2006)

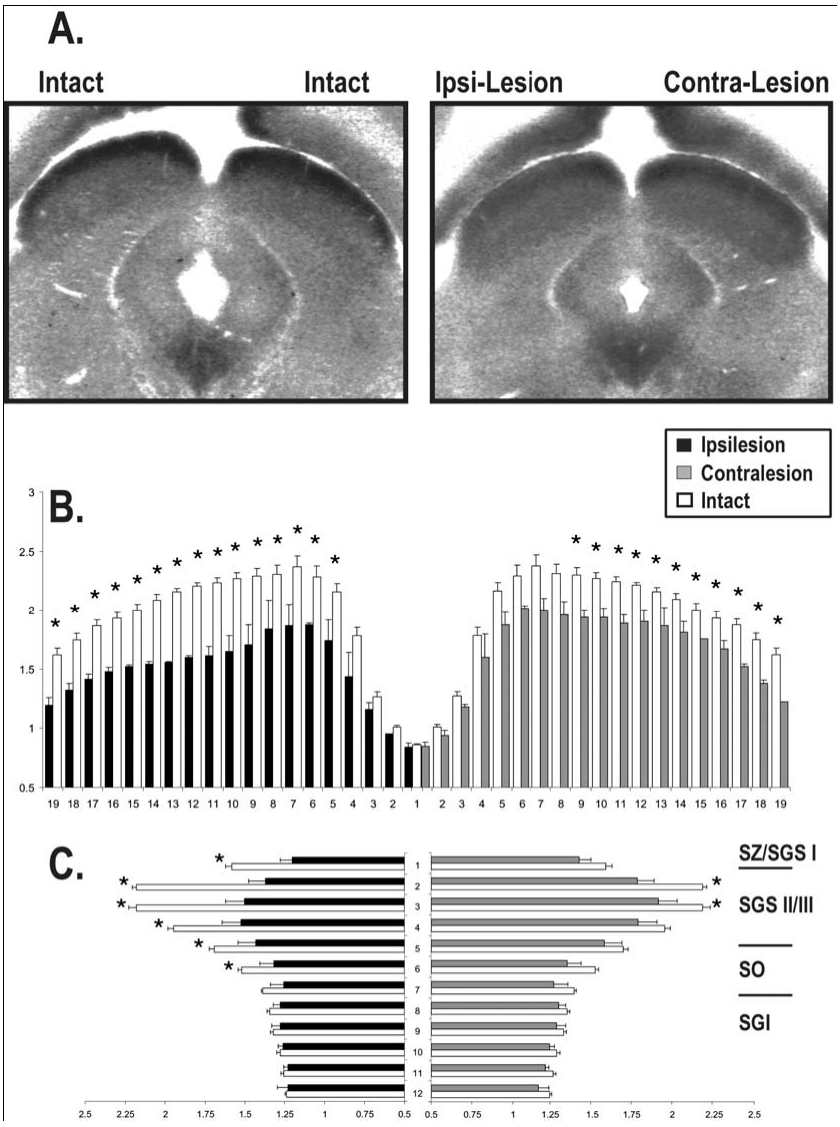


**Fig. 9. Effect of a visual cortex lesion on the ipsi- and contralateral superior colliculus in the cat.**

**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 & Payne, Exp Brain Res 2003).

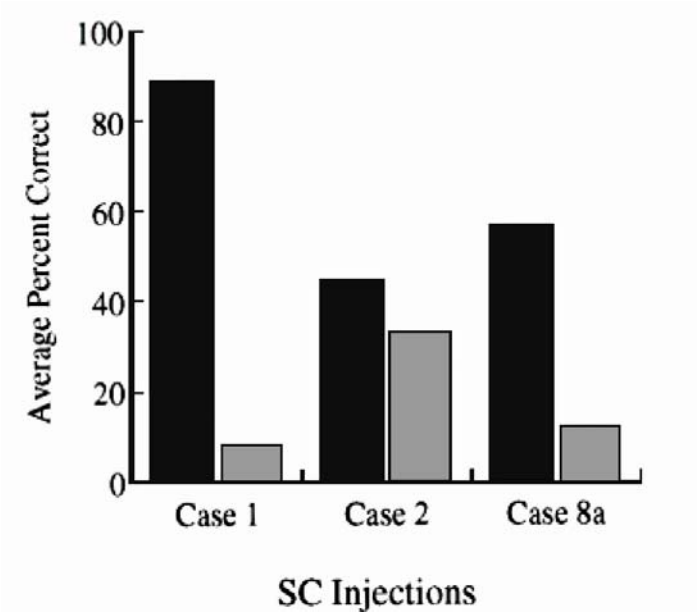


**Fig. 10. Recovery of the Central Versus Peripheral Portions of the Hemifield.**

**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 SC. Average percent correct responsiveness is collapsed across the central hemifield, visual stimuli presented 0–45° from midline (black bars), and the peripheral hemifield, visual stimuli presented from 45–90° from midline (shaded bars). Data reflect injection 1 for case 1, injection 2 for case 2, and injection 2 for case 8a. (Adapted and reproduced with permission from Ciaramitaro, Todd & Rosenquist, The Journal of Comparative Neurology 1997).

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

**Table 1. Danckert & Rossetti's classification system for blindsight.**

**Table 1. Danckert & Rossetti's classification system for blindsight.** (Adapted and reproduced with permission from Danckert &Rossetti, Neuroscience and Biobehavioral Reviews 2005)

| | | 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] |

[a] At or below chance level.

**Table 2. Percentage of correct responses to movement, velocity differences and movement direction.**

**Table 2. Percentage of correct responses to movement, velocity differences and movement direction.** (Adapted and reproduced with permission from Ptito et al, Brain 1991)

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

**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.**

**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.** 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). [a] At or above chance level. (Adapted and reproduced with permission from Ptito et al, Brain 1991).