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Blindsight in hemispherectomized patients as revealed by spatial summation across the vertical meridian

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Summary

The present study provides a demonstration of blindsight in two hemispherectomy patients who showed a visual spatial summation effect across the vertical meridian despite their lack of visual awareness in one hemifield. Such an effect cannot be related to light diffusion onto the sighted hemifield because it was not present when one of the stimuli fell into the retinal blind spot of control subjects. We conclude that blindsight phenomena of the simple type described in the present study can be subserved by sub-cortical mechanisms and do not necessarily require cortical processing.

Keywords: hemispherectomy; blindsight; spatial summation; reaction time

Abbreviations: DB = double bilateral (stimuli); DBBS = double stimuli, one of which was into the blind spot; DU = double unilateral (stimuli); FSIQ = full-scale IQ; LED = light-emitting diode; LH = left hemifield; PIQ = performance IQ; RH = right hemifield; S = single (stimulus); SBS = single flash into the blind spot; VIQ = verbal IQ

Introduction

In humans, damage to the primary visual cortex produces a permanent blindness in the corresponding part of the visual field (Holmes, 1918). In the last 20 years, however, many studies have shown the existence of a wide range of often unconscious residual functions within such blind areas. These include detection and spatial localization of stationary or moving stimuli by eye or hand movements (Pöppel *et al*., 1973; Weiskrantz *et al*., 1974; Perenin and Jeannerod, 1975; Zihl, 1980; Stoerig *et al*., 1985; Blythe *et al*., 1987) as well as discriminations based on motion (Weiskrantz, 1986; Barbur *et al*., 1993), line orientation (Weiskrantz, 1987) or wavelength (Stoerig *et al*., 1987). Other evidence of unconscious visual processing includes an interaction between stimuli presented simultaneously to the blind and normal hemifields (Torjussen, 1976, 1978; Singer *et al*., 1977; Pizzamiglio *et al*., 1984; Marzi *et al*., 1986; Corbetta *et al*., 1990; Rafal *et al*., 1990). Such residual vision has been termed 'blindsight' (Weiskrantz *et al*., 1974) when the viewer does not acknowledge a visual percept and yet shows an above-chance performance. Typically, sub-cortical structures or extrastriate cortical areas have been proposed as its neural locus (*see* Weiskrantz, 1986; for a review, Cowey and Stoerig, 1991).

The existence of blindsight has been put into question because of methodological inadequacies, such as scattered light, inadvertent eye movements that bring the visual stimuli from the blind into the sighted field, a lax response criterion, or spared striate tissue (e.g. *see* Campion *et al*., 1983; Celesia *et al*., 1991; Fendrich *et al*., 1992, 1993). The possibility of such potential confounds has motivated a search for more reliable experimental paradigms. One of these is the redundant-target effect, a summation phenomenon widely known in experimental psychology (*see* Raab, 1962; Miller, 1982; Marzi *et al*., 1996; Mordkoff, *et al*., 1996), whereby the simultaneous presentation of two or more stimuli results in faster reaction time than that to a single stimulus. Using

this approach, Marzi *et al*. (1986) showed that normal subjects react more quickly to two simultaneously presented visual stimuli than to a single visual stimulus, either when the two stimuli appeared in the same hemifield or when they were presented across the vertical meridian. They found a similar effect in some hemianopic patients in whom there was a summation for stimuli presented across the vertical meridian in spite of their reporting the presence of only the stimulus appearing in the good hemifield (Marzi *et al*., 1986; Corbetta *et al*., 1990). Because the subject is supposed to respond to stimuli in the normal hemifield only and is not asked to guess about the presence of a stimulus in the hemianopic field, with this paradigm blindsight cannot be explained by the use of a lax decisional criterion in comparison to clinical perimetry (Campion *et al*., 1983).

However, the two previous studies of interfield summation with hemianopic patients (Marzi *et al*., 1986, Corbetta *et al*., 1990) could not counteract the often raised question that blindsight is mediated by remnants of striate and/or extrastriate cortex (e.g. Fendrich *et al*., 1992, 1993). To address this issue, we investigated patients who had undergone complete or partial cerebral hemispherectomy and in whom all visual cortical areas in one hemisphere were removed. Hemispherectomy subjects have previously been shown to detect and localize stationary and moving stimuli (Perenin 1978; Perenin and Jeannerod, 1978; Ptito *et al*., 1991; Braddick *et al*., 1992; King *et al*., 1996*b*), and to discriminate relative stimulus velocities in their hemianopic field (Ptito *et al*., 1991). However, negative findings as to the presence of visual abilities in the affected field of hemispherectomy patients have been reported recently (King *et al*., 1996*a*, *b*). The presence of interfield summation in such patients would indicate that this form of blindsight can be mediated by cerebral structures other than striate or extrastriate cortex.

Experiment 1

Subjects

Four patients who had undergone partial or complete cerebral hemispherectomy (one left, three right) for the relief of intractable epileptic seizures participated in this study after giving their informed consent. The experimental protocol was approved by the Research Ethics Committee of the Montreal Neurological Institute (protocol number MNIHRECO11). Case histories are described below and summarized in Table 1. MRI scans from two patients (D.R. and S.E.) and a CT scan from another patient (J.B.) are shown in Fig. 1. The work was approved by the Ethics Committee.

D.R is a right-handed woman (20 years old at the time of testing) with a left hemiparesis since birth who began suffering from epileptic seizures at the age of 5 years. Prior to surgery, CT and MRI scans of the brain revealed a severe atrophy of the right cerebral hemisphere and EEG recordings showed epileptiform activity over the right frontal–parietal–temporal regions. Cognitive testing, carried out at that time, indicated borderline intelligence scores; full-scale IQ (FSIQ), 77; verbal IQ (VIQ), 92; performance IQ (PIQ), 65. At the age of 17 years, she underwent a functional hemispherectomy which consisted in removing the temporal lobe (including the mesial structures) and a frontal–parietal corticectomy. The remaining cortical regions were left *in situ* but were disconnected from the rest of the brain by sectioning the white matter anteriorly and laterally as well as posteriorly and laterally along the falx (Fig. 1, D.R.). Subsequent neuropathological investigation revealed an inflammatory process with diffuse gliosis, consistent with chronic Rasmussen encephalitis. Follow-up assessment indicated that her level of intellectual function was in the low-average range (FSIQ 83; VIQ 87; PIQ 83). The presence of a complete contralateral hemianopia without macular sparing has been confirmed by computerized perimetry (Allergan, Humphrey); her visual acuity was 20/25.

S.E. was 28 years old at the time of testing. He is a right-handed man whose left hemiparesis was noted at birth. Seizure onset occurred at the age of 7 years; CT and MRI scans showed a porencephalic cyst occupying the right temporal–parietal–occipital regions. EEG recordings detected epileptiform activity in the right occipital cortex concomitantly with independent foci over the right temporal–parietal cortex. Cognitive testing revealed an FSIQ of 78 (VIQ 80; PIQ 79). At the age of 25 years, the porencephalic cyst was removed and a temporal–parietal–occipital lobotomy was performed sparing the anterior portion of the frontal lobe (Fig. 1, S.E.). Postoperative neuropathological examination revealed a neuronal migration disorder (cortical dysplasia). The follow-up assessment showed an increase in IQ to the average range (FSIQ 93; VIQ 90; PIQ 99) and a contralateral hemianopia without macular sparing was confirmed by computerized perimetry (Allergan, Humphrey); his visual acuity was 20/30. S.E. is one of the two patients who have been examined by Wessinger *et al*. (1996) with stabilized field mapping to eliminate artifacts from eye movements; he was found to have a band of residual vision along the vertical meridian in the hemianopic field which was generally not wider than 3.5° with the exception of a small island of vision at 6° in the upper hemifield only.

I.G. is a right-handed woman who was tested at the age of 42 years. She had a perinatal left hemiparesis and began suffering from epileptic seizures at the age of 7 years. A neuro-ophthalmological evaluation, performed at the age of 12 years, revealed a left visual hemifield loss and a skull X-ray and pneumoencephalography indicated atrophy and/or hypoplasia of the right cerebral hemisphere. EEG recordings revealed epileptiform activity localized to the frontal–parietal cortex. Cognitive testing indicated that she scored in the retarded range of intelligence (FSIQ 52; VIQ 72; PIQ 43). An anatomical hemispherectomy was performed at the age of 13 years; the entire right cerebral hemisphere was removed including the homolateral basal ganglia. Neuropathological

Table 1

Subjects	Sex	Aetiology	Symptoms	Seizures time of onset (years)	Surgery			Age at test (years)	IQ at time of testing			Visual acuity
					Type	Age (years)	Side		VIQ	PIQ	FSIQ	
D.R.	F	Rasmussen's chronic encephalitis	Seizures L-hemiparesis L-hemianopia	5	MFH	17	R	20	87	83	83	20/25
S.E.	M	Neuronal migration disorder (cortical dysplasia)	Seizures L-hemiparesis L-hemianopia	7	PH	25	R	28	90	84	99	20/30
I.G.	F	Middle cerebral artery occlusion (micropoligyria?)	Seizures L-hemiparesis L-hemianopia	7	AH	13	R	42	84	75	79	20/25
J.B.	M	Unknown (porencephalic cyst)	Seizures R-hemiparesis R-inferior quadrantanopia	5	FH	20	L	29	90	88	88	20/25

MFH = modified functional hemispherectomy; FH = functional hemispherectomy; AH = anatomical hemispherectomy; PH = partial hemispherectomy (temporo-parieto-occipital lobectomy).

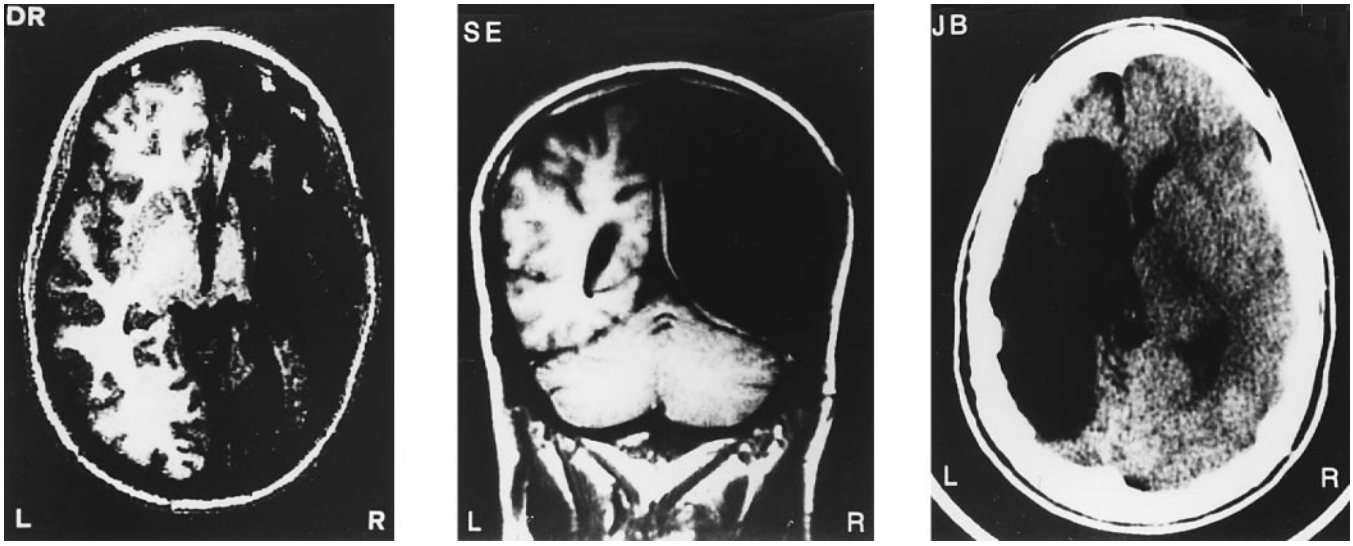

Fig. 1 Axial and coronal MRI and axial CT images showing surgical ablation in D.R., S.E. and J.B.; *see* Subjects for a description of the surgical procedures.

examination showed a large cystic cavity (porencephalic cyst) involving the frontal and temporal lobes and a severe atrophy of all layers of the neocortex with diffuse gliosis, findings consistent with a prenatal vascular accident in the territory of the right middle cerebral artery. In addition, Reil's insula was displaced, the basal ganglia were atrophic and microgyria were present in the occipital lobe. At present, I.G. functions in the low average range of intelligence (FSIQ 79; VIQ 84; PIQ 75), she has a visual acuity of 20/25 and a complete left homonymous hemianopsia without macular sparing as revealed by clinical perimetry.

J.B. is left-handed man who was tested at the age of 29 years. He has had a right hemiparesis since birth. Epileptic seizures developed at the age of 5 years and a neuro-ophthalmological evaluation performed at the age of 18 years revealed a right-sided inferior quadrantanopia. A CT scan of the brain demonstrated severe atrophy of the left hemisphere and the presence of a porencephalic cyst. EEG recordings showed multifocal epileptiform activity over the whole left hemisphere but it was more prominent over the temporal and parietal regions. Pre-operative assessment established an average range of intelligence scores (FSIQ 93; VIQ 89; PIQ 102) and speech representation in the right hemisphere, as revealed by the intracarotid sodium amytal test (Wada and Rasmussen, 1960). In order to avoid worsening of the field defect, a conservative functional hemispherectomy was initially performed. This consisted of a temporal–parietal corticectomy and disconnection of the frontal lobe from the rest of the brain. Neuropathological investigation revealed neuronal loss and moderate to severe gliosis of unknown

aetiology. Because of persisting epileptic seizures, a second surgical intervention was performed 3 months later. The functional hemispherectomy was then completed by adding a temporal lobotomy (including the mesial structures) and a partial occipital lobotomy which left the occipital pole *in situ*, but disconnected from the white matter (Fig. 1, J.B.). Neuropathological examination revealed neuronal loss in both neocortex and hippocampus. On a follow-up examination J.B. continued to score in the average range of intelligence (FSIQ 88; VIQ 90; PIQ 88). He then had a complete homonymous right-sided hemianopia without macular sparing confirmed by computerized perimetry (Allergan, Humphrey) and his visual acuity was 20/25. J.B., like S.E., has been examined with stabilized field mapping (*see* Wessinger *et al*., 1996); he was found to have only a small strip of residual vision not extending beyond 3.5° from the vertical meridian in the upper quadrant of the hemianopic field.

Apparatus and procedure

The patient sat in an adjustable chair with the head restrained by a chin and forehead retainer, 57 cm in front of a black semicircular platform (diameter, 114 cm; background luminance, <1 cd/m^2) set at eye level. Because of a strabismus present in all patients, the eye ipsilateral to the brain lesion was covered with a patch and testing was carried out monocularly. Five light-emitting diodes (LEDs; size: 0.5 cm), one red and four green, were mounted on the platform. The red LED (luminance 1 cd/m^2) was used as the central fixation point and remained on throughout the experiment. Two green LEDs (40 cd/m^2) were placed 2° below the horizontal meridian at 10° and 30° in both hemifields to the right and left of the fixation point. A video camera provided an on-line image of the uncovered eye, which allowed continuous monitoring of eye fixation. In addition, eye movements were recorded with the aid of a personal computer using an infrared Pupil/Corneal Reflection Tracker (ISCAN) to ensure that no significant eye movement had occurred during stimulus presentation. Subjects were instructed to press a key with the index finger of their non-paretic hand as quickly as possible when a green LED was flashed (duration 50 ms). Visual stimulation was preceded by a 50-ms acoustic warning signal presented from 1 to 3 s before the onset of the flash. The subjects were informed that either one or two flashes would be presented on any given trial and that they were to press the key regardless of the number of stimuli, or of their position. Light scatter was minimized by flooding the entire visual field with a light intensity (8 cd/m^2) that made any light diffusion onto the intact field undetectable (Weiskrantz, 1986). Further control procedures, which will be described in the next section, used stimulus presentation into the retinal optic disc.

Each session consisted of 200 trials subdivided into three types: (i) a single stimulus (S) was flashed in the right or left hemifield either at 10° or 30° from the central-fixation LED (80 trials); (ii) double unilateral stimuli (DU), i.e. two flashes were simultaneously presented in the left or the right hemifield (40 trials); (iii) double bilateral stimuli (DB) consisted of all possible combinations of simultaneous flash presentations across the vertical meridian at different eccentricities in the left (LH) or right hemifield (RH) (i.e. 10° LH 30° RH, 10° LH 10° RH, 30° LH 30° RH, 30° LH 10° RH; 80 trials). It is important to stress that the eccentricities of stimulus presentation were extremely large in comparison with the small degree of residual vision documented by Wessinger *et al*. (1996) for two of our patients (S.E. and J.B.).

Each session, which lasted ~20 min, was repeated up to five times during one testing day. In the course of 8 weeks, the subjects underwent from nine to 12 sessions according to their availability. Prior to actual testing, all subjects were trained for 150 trials to maintain central fixation and to respond as quickly as possible to the stimulus. Fifty practice trials were administered at the beginning of each testing day. Stimulus presentation was controlled automatically with a personal computer. Trials with a reaction time <150 ms (anticipation) or >800 ms (misses or delays), as well as trials where the patient failed to maintain central fixation, were rejected. The rejection rate amounted to ~3% overall.

For statistical purposes, the median reaction times for each patient and each type of stimulus were averaged across sessions. Group analyses were performed by means of paired *t* tests. Median reaction times for each patient, session and type of stimulus were used in single-case analyses; ANOVA and *post hoc t* test were also used. Furthermore, we plotted the cumulative distribution of the reaction times to S and DB stimuli, a procedure which allows a comparison of the two conditions along the whole range of reaction times.

Results

None of the patients was aware of stimuli (single or double flash) presented in their blind hemifield. Moreover, no patient gave above-chance correct manual responses to stimuli presented to the affected hemifield. However, it should be pointed out that we did not attempt any systematic forced-choice guessing procedure by using other non-verbal responses, e.g. eyelid closure (*see* Zihl and von Cramon, 1980). In the group analysis, we found that significantly shorter reaction times were elicited in the intact field by DU presentations, compared with S presentations (with DU, 362.2 ms; with S, 381.7 ms; $t(3) = 6.619$; $P = 0.007$). By contrast, the comparison between DB presentations across the vertical meridian versus S presentations in the intact hemifield did not differ significantly (reaction time with DU, 376.9 ms; with S, 381.7 ms; $t(3) = 1.533$; n.s.), although there was a trend toward a summation effect. This latter finding indicates that spatial summation across the vertical meridian is not a consistent finding in our group of patients. Individual differences in performance among hemianopic subjects have often been reported in studies of residual vision (Weiskrantz, 1980; Marzi *et al*., 1986; Corbetta *et al*., 1990; Ptito *et al*., 1991; but *see* Kasten *et al*., 1995). We therefore

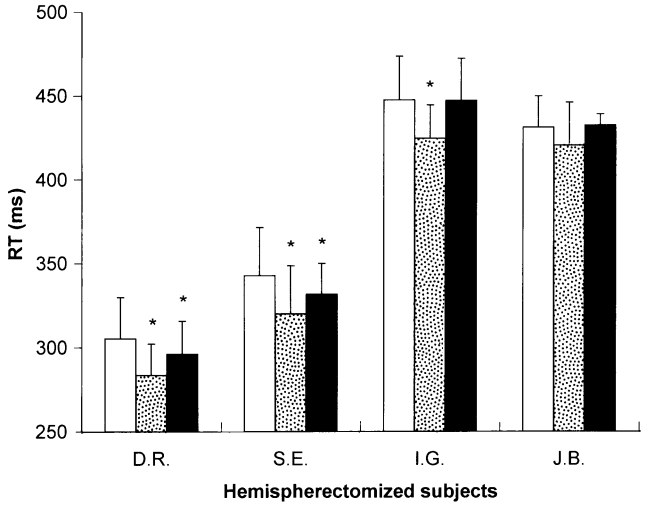

Fig. 2 Mean reaction times (RT) for the four hemispherectomized patients in the three conditions of stimulus presentation. Open, shaded and fillled bars show results from single (S), double unilateral flash (DU) and double bilateral double (DB) flash experiments, respectively. Asterisks mark a statistically significant difference in the crucial comparison between double bilateral flash presentations and single flash presentations in the sighted hemifield. Such a difference is significant in D.R. and S.E. but not in I.G. and J.B. However, note that the latter patients have considerably slower RTs than the former. In D.R., S.E. and I.G. the difference between single flash and double flash presentation in the sighted field is reliable while such is not the case in J.B.

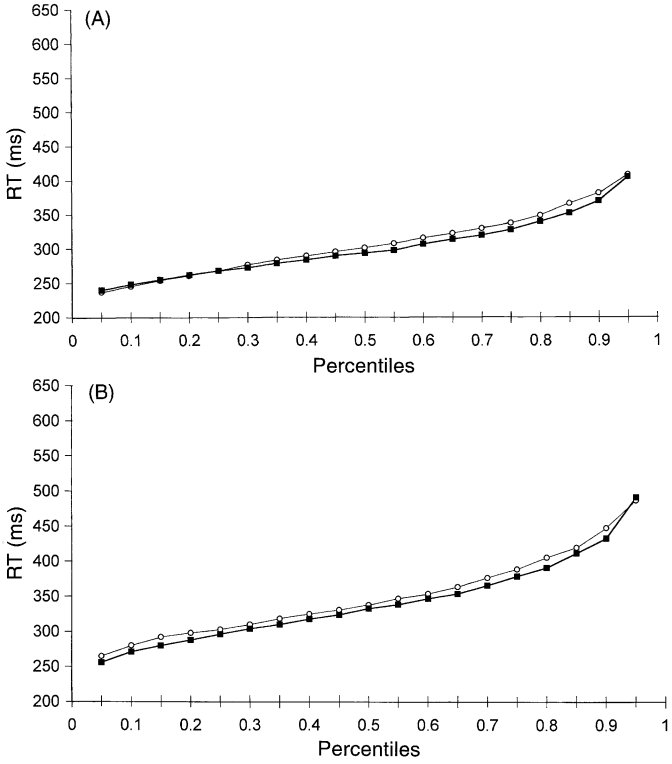

Fig. 3 Cumulative frequency distributions of reaction times from the two patients (D.R., **A**; S. E., **B**) showing interfield summation (D.R. top graph and S.E. bottom graph). Note that there is practically no overlap between reaction times to single flashes in the sighted field (S, open symbols) and those to double flashes across the vertical meridian (DB, filled symbols). The latter are consistently faster throughout the whole distribution.

decided to carry out a single case analysis by comparing the performance of each subject in the three experimental conditions (S versus DU versus DB). The results illustrated in Fig. 2 indicate that in all patients, but J.B., reaction times were significantly shorter in the DU presentation compared with the S presentation in the intact hemifield [reaction times: for D.R., with S 305.5 ms, with DU 283.6 ms, $t(11) = 4.52$, $P = 0.001$; for S.E., with S 342.7 ms, with DU 320.0 ms, $t(11) = 2.92$, $P = 0.014$); for I.G., with S 447. 5 ms, with DU 424.5 ms, $t(8) = 3.37$, $P = 0.01$].

The crucial comparison between reaction times elicited in the DB presentation (simultaneous display in the normal and blind hemifield) and the S presentation, revealed that two patients yielded significantly faster responses in the former than in the latter condition [Fig. 2: for D.R., reaction times with S 305.3 ms, with DB 296.1 ms, $t(11) = 3.05$; $P = 0.011$; for S.E., with S 342.7 ms, with DB 331.7 ms, $t(11) = 2.31$; $P = 0.041$]. The other two patients, J.B. and I.G., did not show significantly faster reaction times when a second flash was simultaneously presented in their blind hemifield. The latter patients were also much slower overall than the former and one (J.B.) did not show a reliable summation effect in his seeing hemifield.

Figure 3 shows the reaction times cumulative frequency distribution for the two patients showing reliable interfield summation (D.R. and S.E.). It is clear from inspection of this figure, that the distribution of reaction times in the two conditions of stimulus presentation are well differentiated, confirming the summation effect with bilateral stimuli across the vertical meridian. In contrast, an almost complete overlap between the distributions is present for the other two patients (I.G. and J.B); *see* Fig. 4.

Experiment 2

This experiment was carried out with four normal control subjects in order to confirm that light scatter could not be a factor in the results obtained with the experimental subjects.

Subjects

Four normal right-handed volunteers participated in the experiment, two males and two females, ranging in age between 22 and 33 years. All had normal vision and visual acuity ranging between 20/30 and 25/25. Informed consent was obtained from each subject.

Apparatus and procedure

The experimental apparatus and procedure were the same as previously used for the patients in Experiment 1. In addition, a green LED (40 cd/m^2) was positioned to stimulate the blind

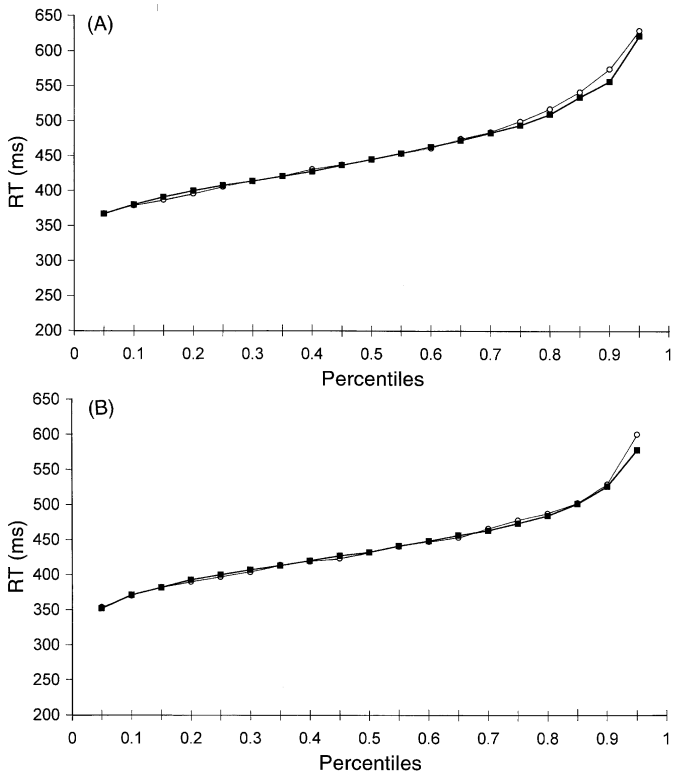

Fig. 4 Cumulative frequency distributions for reaction times of the two patients (I.G., **A**; J.B., **B**) not showing interfield summation. Note the substantial overlap between the reaction times for the S and the DB conditions.

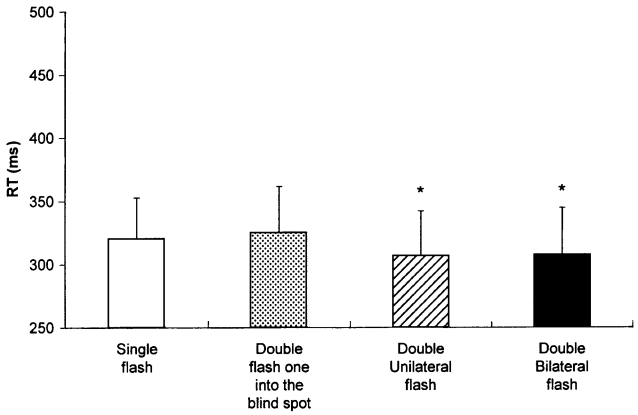

Fig. 5 Mean reaction times of the four normal subjects tested in Experiment 2. The asterisks indicate a statistically significant difference between reaction times to double flashs in the sighted field and a single flash in the same field and another significant difference between a bilateral presentation across the vertical meridian a the single flash presentation. NB. There is no difference between single flash presentations into the sighted field and a bilateral presentation with one of the flashes presented in the blind spot of one eye.

spot of the right eye of each subject (~12.5 cm lateral to the fixation point and on the same horizontal axis as the other green LEDs). Eye position was monitored as described above. All testing was carried out with the right eye. Each subject was tested in a single session that comprised 400 trials involving five types of stimuli: a single flash at 10° or 30° eccentricity in the right or in the left hemifield (S, 160 trials); a single flash into the blind spot (SBS, 40 trials); a double unilateral flash (DU, 60 trials); a double bilateral flash (DB, 60 trials); a double bilateral flash, one of which was into the blind spot (DBBS; 80 trials). The subject's task was to press a key with the index finger as quickly as possible following stimulus onset. The session lasted ~45 min. Before formal testing, the subjects were allowed 50 training trials.

For statistical purposes, the median reaction times for each subject and type of stimulus were calculated. Group analyses were performed by means of paired *t* tests.

Results

None of the subjects ever responded to the flashes presented in their blind spot, indicating that light scatter was at most restricted to an area not larger than the blind spot (~5°). This conclusion is further supported by the absence of a difference between responses in the S versus DBBS presentations where one of the flashes was delivered into the blind spot (with DBBS 325.2 ms; with S 320.4 ms; $t = 0.78$; not significant). Both DU and DB presentations elicited faster reaction times than S presentations [Fig. 5: with DU 307.0 ms; with S 320.4 ms; $t(3) = 6.79$; $P = 0.007$; with DB 308.0 ms; with S=320.4 ms; $t(3) = 3.88$; $P = 0.03$]. No difference was found between reaction times to double flashes presented either across or within hemifields. These results are consistent with those previously obtained by Marzi *et al.* (1986).

Discussion

The experimental paradigm used in this study allowed us to assess unconscious visual sensitivity in the hemianopic field in patients with partial or complete hemispherectomy. This was achieved by measuring reaction times to flashes presented to the normal hemifield and to the simultaneous appearance of a second flash in the hemianopic field. Two out of four patients (D.R. and S.E.) showed faster reaction times to double-flash (DB) than to single (S) flashes, despite their unawareness of the flashes presented in the hemianopic field. The other two patients did not show this effect. However, in J.B. we did not see the expected summation effect even when the flashes were presented within his normal hemifield, a result which raises the possibility of a general perceptual impairment. The other patient (I.G.) showed the summation effect in her intact field, but not across the vertical meridian. In this patient, the large extent of the lesion that also included the basal ganglia could, perhaps, explain this negative result. It should be also pointed out that the two subjects who did not show a summation effect were those with overall slower reaction times (*see* Fig. 2). One might therefore speculate that they suffer from a general vigilance impairment that could increase response variability and mask summation effects. This is not an unlikely possibility since reaction

times are reliable indicators of perceptual processing only when subjects are performing at their best.

Before discussing the possible neural substrates of the summation effect, let us comment on some methodological issues. In interpreting our results, we can rule out the possible effect of light scatter for the following reasons. First, the patients never responded to flashes, either single or double, when they were presented in the hemianopic field. Secondly, our control subjects did not respond to a single flash presented within the blind spot using the same apparatus and stimulation parameters as those employed in the patient study. Furthermore, no significant difference was found in reaction times between that to a single stimulus and those to a pair of flashes with one of the stimuli presented in the blind spot. With respect to the possibility of an inadequate eye fixation, we monitored eye movements both on-line and off-line and were able to ascertain that the subjects were indeed fixating centrally during stimulus presentation. Moreover, the very brief duration of the stimuli and the absence of responses to flashes presented in the hemianopic field make it very unlikely that the stimuli might have fallen into the patients' sighted field. Further, one of the patients showing blindsight, namely S.E., was one of the two subjects whose visual field was assessed recently by Wessinger *et al*. (1996) with stabilized mapping to avoid eye motion artifacts and found to lack residual vision beyond a narrow strip close to the vertical meridian, i.e. very far from the inner limit of our stimulus presentations. Finally, possible criterion effects were minimized in the present experimental paradigm because the subjects were not required to guess about a visual stimulus of which they were unaware as in other paradigms used to assess blindsight; they were simply asked to respond to a stimulus presented to their sighted hemifield. Such a paradigm effectively eliminates the influence of a pre-selected response criterion as in a forced-choice decision task. We therefore believe that the observed spatial-summation effect is reliable. Our results are at odds with a recent study (King *et al*., 1996*a*) in which, among a large series of psychophysical tests, the authors have tested their hemispherectomy patients for interfield effects on reaction times to simple visual stimuli. In contrast to our present study, they did not find reliable evidence of interfield spatial summation but such a discrepancy may be explained by the largely different number of trials in the two studies [40 for each condition of stimulus presentation in the King *et al*. (1996*a*) study and at least 1800 for each condition in the present study]. Moreover, it is interesting to point out that the mean reaction time of the patients of King *et al*. (1996*a*, *b*) was slower than that of our patients showing interfield summation and approximately similar to that of our patients not showing such an effect.

In principle, the present summation effect we observed might be subserved by a neural mechanism operating at a motor or a visual level. A detailed discussion of such mechanisms is beyond the scope of this study. Nevertheless, there is recent event-related potential evidence of an absence of a neural summation effect in the motor component (Mordkoff *et al*., 1996) while a recent event-related potential study in our laboratory has shown that the interfield summation effects which are present take place at the level of cortical extrastriate visual areas (Miniussi *et al*., 1996).

The question remains as to which cerebral structures could mediate the residual vision. The fact that hemispherectomized patients who have a complete absence or a disconnection of occipital cortex on one side still show residual vision, without awareness in their blind field, suggests a role of sub-cortical structures. In these patients a retinal-collicular projection remains, which might mediate visual functions in the absence or disconnection of a whole cerebral hemisphere. Several studies support this possibility. Following hemispherectomy in humans, Ueki (1966) found a retrograde degeneration of the entire thalamus and preservation of the superior colliculi. In the monkey, the superior colliculi receive direct input from the retina as well as from the striate cortex and they contain a complete representation of the visual field (Schiller, 1972). Anatomical studies using anterograde transport of HRP (horseradish peroxidase) and quantitative cytoarchitecture and cytochrome oxidase activity have shown retrograde degeneration of the geniculo-striate system and remarkable survival of the collicular system after neonatal hemidecortication in the vervet monkey (Ptito *et al*., 1996).

Residual detection abilities disappear in striatectomized monkeys following destruction of the ipsilateral superior colliculus (Mohler and Wurtz, 1977). The collicular projection has been implicated in the mediation of saccadic and manual localization of targets in hemianopic fields by patients with occipital lobe lesions (Weiskrantz *et al*., 1986; Cowey and Stoerig 1991). Hemispherectomized patients have also shown localization ability in their blind field, indicating that the extrastriate visual cortex of the hemisphere contralateral to the hemianopic field is not necessary for localization (Ptito *et al*., 1991; Stoerig and Cowey, 1993).

That the superior colliculus may be involved in the spatial summation effect across the vertical meridian in hemispherectomized subjects is further supported by studies showing interactions between the intact and blind hemifields in patients with restricted occipital lesions. Pöppel and Richards (1974) tested two hemianopic patients who had small scotomata in the opposite visual field. They found residual visual function in the hemianopic field, but only in the mirror-symmetric position corresponding to the location of the scotomata in the opposite field. Singer *et al*. (1977) reported that, in normal subjects, threshold elevation for repetitive light flashes in one half field could be reset by stimulating a mirror-symmetric position in the contralateral visual field. Such an interfield interaction was also found in hemianopic patients when the mirror-symmetric position in the blind field was stimulated. Zihl and von Cramon (1979) confirmed this finding by demonstrating that a patient with a congenital malformation of the right superior colliculus did not exhibit threshold elevation when stimulated repeatedly in the left visual field, while stimulation in the right led to the expected increase in threshold. This was interpreted as

suggesting that threshold elevation occurs as a consequence of collicular adaptation and that the mirror-symmetrically organized interhemispheric interaction was mediated at collicular levels. Rafal *et al*. (1990) have demonstrated that distractor signals in the blind half of the visual field could inhibit saccades towards targets in the intact visual field, a result they attribute to the retino-collicular pathway.

Finally, further evidence in favour of a sub-cortical mediation of the summation effect can be found in recent data gathered by Reuter-Lorenz *et al*. (1995) and by Marzi *et al*. (1997) in split brain subjects. These authors have independently demonstrated the presence of an interfield summation effect despite the absence of the corpus callosum. In the absence of the corpus callosum and other cortical commissures, the redundant target effect could only have been subserved by sub-cortical (probably collicular) commissural mechanisms.

In summary, we believe that the unconscious summation effect across hemifields that we have observed in patients in whom a whole cerebral hemisphere has been removed or deafferented is likely to be subserved by the retino-collicular pathway. This is not in contradiction with our recent event-related potential findings (Miniussi *et al*., 1996) because the extrastriate areas that have been shown likely to subserve the summation effect may well receive a subcortical input indirectly from the superior colliculus. Since the geniculo-striate system shows massive retrograde degeneration following hemispherectomy in man and monkeys (Ueki 1966; Ptito *et al*., 1996), it is not surprising that other visual functions such as discriminations based on wavelength, form and direction of motion that are unlikely to be subserved by the collicular vision, may no longer be possible (Faubert *et al*., 1995; King *et al*., 1996*a*, *b*).

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