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# No blindsight following hemidecortication in human subjects?

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USING a guessing paradigm we measured visual sensitivity in the blind and normal half-fields of four cerebrally hemidecorticated patients. In the blind field, sensitivity was reduced by -3 log units. Stimuli which produced significant detection also evoked conscious sensations of light and colour. Control experiments showed that although Sensitivity in the blind field depended in a normal fashion on background luminance, it was independent of the luminance of a local platform, and showed no spatial summation. This residual vision can be explained by intraocular light diffusion and reflection.

Key Words: Blindsight; Hemidecortication; Humans; Blindfield; Residual vision; Light scatter

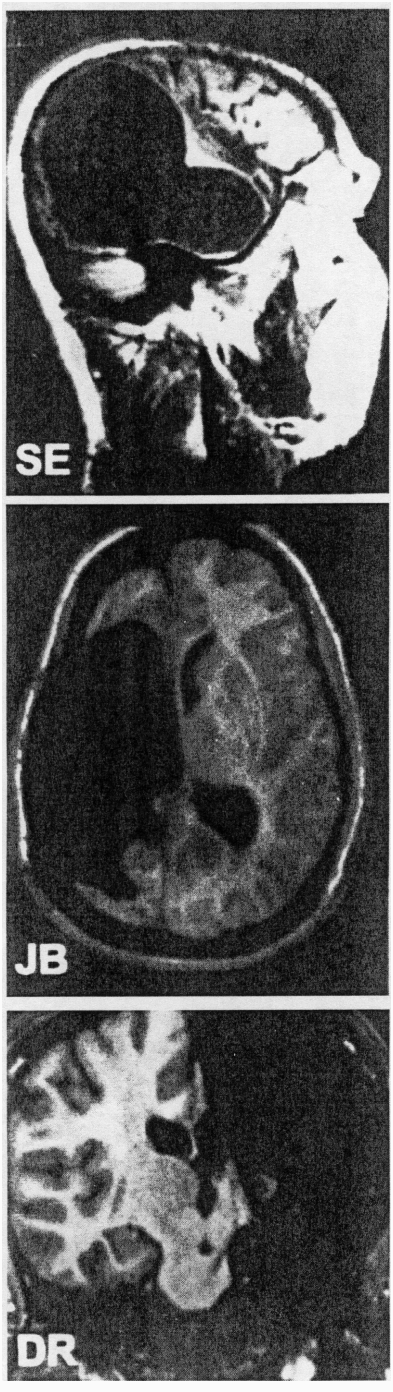
## Introduction

The residual visual functions thJt can be elicited in individuals with visual field defects caused by destruction of primary visual cortex are summarily known as blindsight' because the patients do not consciously see the stimuli they respond to. Blindsight encompasses reflexes, implicit responses (a stimulus in the field defect modifies the reaction to a stimulus in the normal field) and explicit responses. Only the latter require the patient to respond directly to a stimulus presented in the defect field. Forced-choice methods reveal an ability to detect, localize and discriminate stimuli by 'guessing' (see Refs 2,3 for recent reviews). The phenomenon has attracted attention because of its potential to elucidate the neuronal basis of conscious vision.[1–6] Extrastriate visual cortical areas which receive visual information via subcortical retinorecipient nuclei are known to mediate blindsight.' At least some extrastriate visual cortical areas, which receive visual information via subcortical retinorecipient nuclei 7, retain visual responsivity in both man[8] and monkey' with striate cortex lesions. If they were needed for blindsight responses, patients who have undergone surgical hemidecortication, an intervention which involves the removal or disconnection of all visual cortical areas of one hemisphere, should not demonstrate such responses in their blind field. The goal of the present study was therefore to investigate the residual visual abilities that can be revealed with guessing paradigms in the blind hemifield of hemidecorticated human subjects.

## Subjects and Methods

We used forced-choice guessing responses to measure visual sensitivity in four (plus three incompletely tested) hemidecorticated patients who gave their informed consent to participate. Clinical data are summarized in Figure 1. Perimetry (Humphrey) showed all patients to have complete and dense hemianopias with macular splitting, although occasionally a stimulus (size 111) was detected close to the vertical meridian, displaced by up to 6° into the impaired field. In an independent study and using retinal stabilization, two of our patients (J.B. and S.E.) were found to have small islands of visual function up to 7° into the blind hemifield.[10] We avoided such regions of possible nasotemporal overlap by presenting visual stimuli at 20° eccentric positions in the lower hemifield, on the 225° and 315° meridians, respectively. Stimuli were generated with a monochromator (Schoeffel, GM 250) and projected onto a homogeneous white background (Ganzfeld). The patients fixated a 2°, 650 laser diode with one eye, the other eye being covered with a patch. Fixation was constantly controlled on-line with an Eye Scan system (I-Scan RK 426 and 520; resolution of 0.5°).

Detection thresholds were measured under light- and dark-adapted conditions with a 5°, 300 ms stim-




ulus presented in pseudorandom alternation with blank trials; these represented 70% of the total number of trials in a given session. The patients were asked to fixate, and to guess, on each individual presentation indicated by a sound from the shutter (UniBlitz SD-10) whether or not a stimulus had been presented. The luminance of the stimulus was increased until the patients' performance became statistically significant. The threshold values thus derived could be replicated within 0.2 log units in other sessions, and by using decreasing as well as increasing methods of limits. As we intended to measure increment threshold spectral sensitivity, we systematically varied stimulus wavelength (410–650 nm).

## Results

The resultant curves from both hemifields showed the narrowly tuned curve of rod-mediated vision when measured under dark adaptation, and the characteristic discontinuities attributed to colour-opponent processes when measured under photopic adaptation.[11] Irrespective of wavelength and clinical history, all patients showed a similar reduction of sensitivity in the blind hemifield. This reduction was in the order of up to 3 log units, and particularly evident under light-adapted conditions which are less likely to be contaminated by extraocular straylight (Fig. 2). The difference between the hemifields was much larger than the 0.5–1.5 log units observed in patients and monkeys with occipital lobe destruction.[12,13] Indeed, it matches or supercedes the difference between sensitivity in the normal field and straylight sensitivity, determined to be about 2.5 log units for patients with occipital lobe damage who respond to a halo or sensation of light from an unseen stimulus presented within the field defect.[12]

In addition, all seven patients reported phenomenal visual sensations – diffuse coloured light – emanating from the blind field whenever the performance became statistically significant. Such sensations were also mentioned in previous positive[14–17] but not negative[18–20] reports. To learn whether sensitivity was determined locally in the blind field, we conducted three control experiments with patients

**FIG.** 1. MRI scans for patients S.E., J.B. and D.R. illustrate the extent of cortical removal. **(A)** S.E., male. Neurological deficits were noted at 6 months; seizures began at age 7. At 25, a porencephalic cyst occupying the temporo-parieto-occipital regions of the left hemisphere was removed. The frontal lobe was spared. Visual acuity was 20/25. (**B**) J.B., male, had a perinatal right hemiparesis. Epileptic seizures developed at age 5. A porencephalic cyst in the atrophied left hemisphere was removed in a 2-stage functional hemispherectomy: a parieto-temporal corticectomy with a disconnection of the frontal lobe was followed 3 months later by a temporal lobectomy (including the mesial structures) and a partial occipital lobectomy which left the lobe disconnected from the white matter. Visual acuity was 20/25. (**C**) D.R., female. Neurological symptoms developed at age 5, with severe atrophy of the right hemisphere and epileptiform activity over fronto-parieto-temporal regions. At 16, she underwent a functional hemispherectomy with removal of the temporal lobe (including the mesial structures) and the fronto-parietal cortices. The remaining cortical structures were disconnected through lateral callosotomy and section of the underlying white matter. Visual acuity was 20/25. The fourth patient, F.S., female, age 30, suffered severe head trauma at 2, causing a left hemianopia and hemiparesis. At 18, a right hemispherectomy was performed, removing a large temporo-parieto-occipital porencephalic cyst. All patients had a post-operative Full Scale Intellectual Quotient (FSIQ) in the lower normal range (79–93), and showed homonymous hemianopias without macular sparing. They are presently seizure-free and unmedicated.

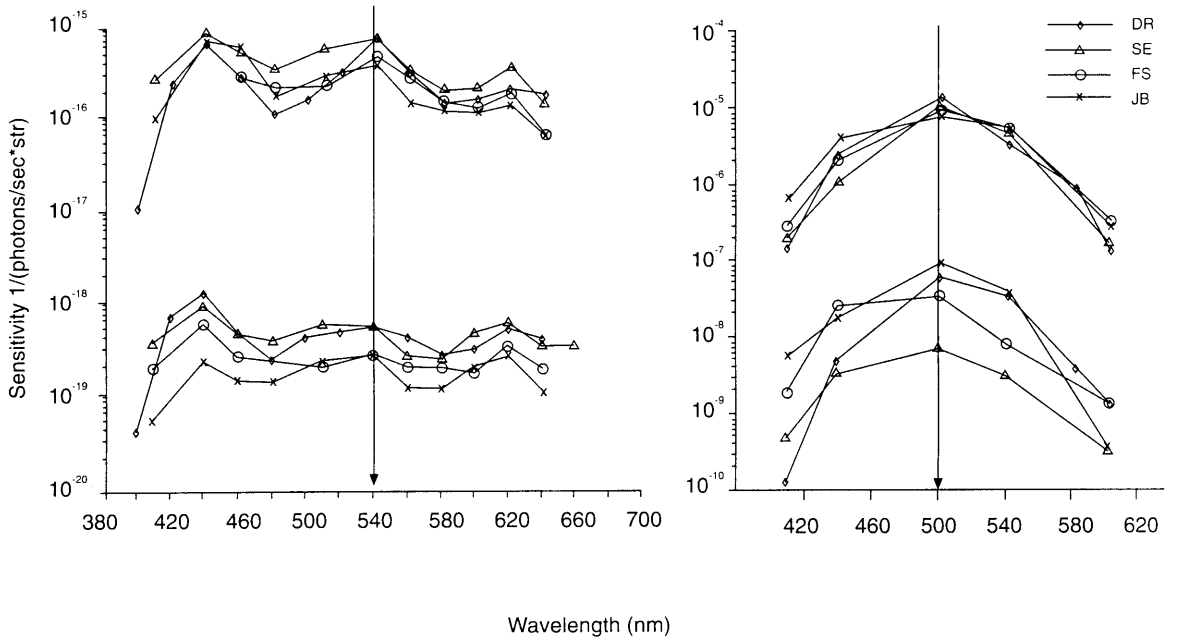



FIG. 2. Increment-threshold spectral sensitivity functions obtained under light adaptation (left) and under dark-adaptation (right) in the normal (upper set) and blind (lower set) hemifield of four hemispherectomized patients. Note the massive difference in luminance required for statistically significant detection of stimuli in the two hemifields, and the normal shape of the curves.

D.R. and S.E. In test 1, we measured the detection thresholds in S.E. as a function of retinal eccentricity on the horizontal meridian, to establish whether sensitivity decreases in the normal way with increasing retinal eccentricity, and to find functional evidence for the blind spot whose normal position we had confirmed with retino-funduscopy. The 1° stimuli that we used should have been harder to detect in this receptor-free zone, but despite testing at very closely spaced positions, sensitivity remained exactly the same. Furthermore, once the stimulus was sufficiently removed from the border of the functional visual hemifield, sensitivity remained at the same low level, showing little modulation with retinal eccentricity (Fig. 3A). We tested the spatial summation properties of this vision by comparing sensitivity for 1° and 2° diameter stimuli in S.E., and by comparing sensitivity for stimuli of different sizes at a 10° eccentric retinal position in D.R. No dependence on stimulus size was found; instead, and in contrast to the functional hemifield, sensitivity depended solely on the amount of energy emitted. In test 2, we asked S.E. to guess whether or not a stimulus had been presented at the usual 20° eccentric position in his blind field, and used stimulus luminances 1 and 0.5 log units, respectively, below his previously determined high threshold for detection. The probability of stimulus and blank trials was varied to account for response biases (signal detection procedure). We used 250 presentations in addition to the several thousand already given to determine the curves shown in Figure 2. Although the patient was well motivated he did not achieve statistically significant detection at these luminance levels which did not evoke sensations of light (Fig. 3B).

Finally, in test 3, we first measured sensitivity as a function of background luminance. We then measured it as a function of the luminance of a local 8° background superimposed on a global background held constant at 1 cd/m$^{-2}$ luminance; the 2° stimulus was presented on the 8° platform. Sensitivity decreased with increasing platform luminance in the normal hemifield, but in the blind hemifield it depended exclusively on overall adaptation (Fig. 4).

## Discussion

The present results suggest that the sensitivity observed in the blind field cannot be mediated locally, i.e., at the site where the visual stimulus was presented in the blind hemifield. Instead, the high luminance levels (e.g. 1000 instead of 1 cd m$^{-2}$) required to elicit significant detectability, particularly under conditions that eliminate sources of extraocular light scatter, indicate that intraocular reflection and diffusion are the most likely causes of the detection. This conclusion is in accordance with other negative findings[18–20] and accounts for the normal shape of the spectral sensitivity curves from the blind field as resulting from the normal field which shows a Purkinje-shift and colour-opponent processes, and explains the diffuse perceptions of coloured light that the patients reported.

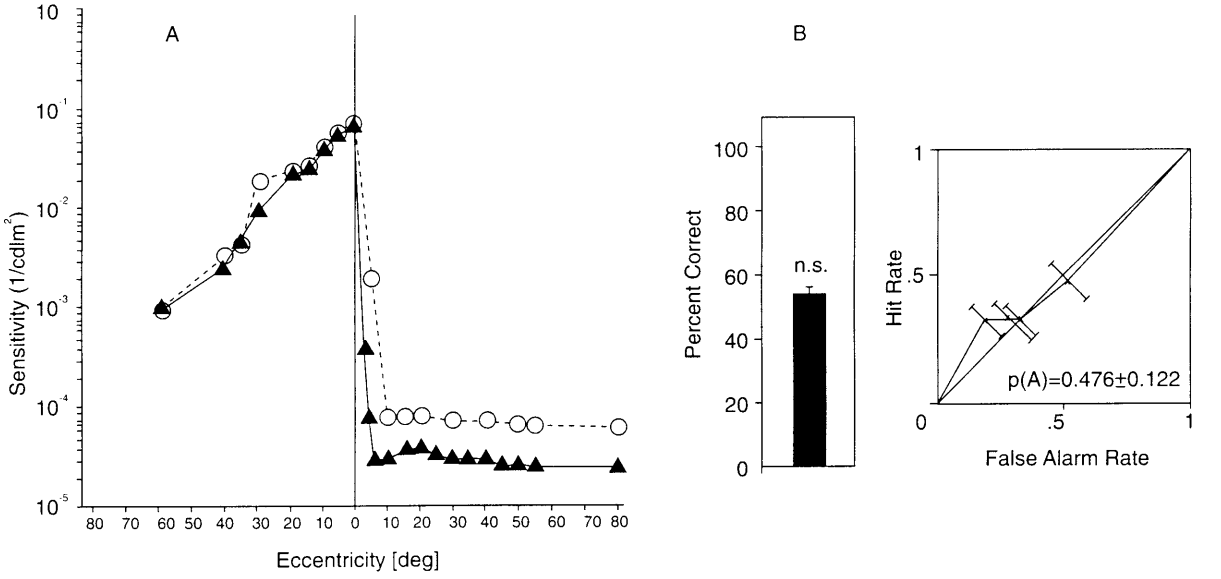



FIG. 3. (**A**) Detection thresholds for 'seen' stimuli as a function of retinal eccentricity in normal (left) and blind (right) hemifields of patient S.E. 2° (open circles) and 4° (filled triangles) stimulus sizes were used. Evidence for spatial summation is seen only in the normal hemifield. (**B**) When S.E. tried to guess the presence of a signal whose luminance was 1 and 0.5 log units below the luminance required for detection based on weak but conscious perception, his performance was at chance level.

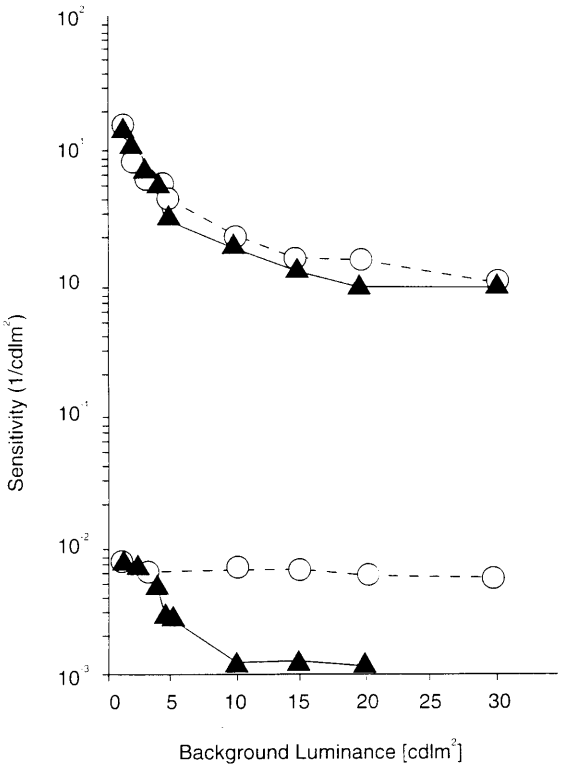



FIG. 4. Sensitivity for a 'seen' stimulus depends on the global luminance of the homogeneous background in both hemifields (filled triangles). A local background superimposed on the background behind the stimulus (open circles) exerts a similar influence on sensitivity in the normal field (upper curves), but has no effect on sensitivity in the blind field (lower curves) which is determined by the overall background illumination of 1 cd $m^{-2}$. Together (with Fig. 3) the data indicate that thresholds based on seeing and thresholds based on guessing are identical in the blind field, and that the sensitivity is not mediated locally in the blind field but determined by light diffused and reflected in the eye.

Ablation of ipsilesional extrastriate cortex thus seems to abolish the residual sensitivity that can be revealed with forced-choice methods in patients with striate cortical damage. Nevertheless, hemidecorticated patients may still possess reflexive functions; indeed, photic blink and pupil light reflexes,[2] optokinetic nystagmus[21] and even a reduction of reaction time to a stimulus in the functional hemifield caused by an additional one in the blind hemifield[22] have been demonstrated, indicating that lower functions can be mediated by the degenerated subcortical system that survives hemidecortication.[23,24] Recent anatomical studies carried out on hemispherectomized monkeys have indeed indicated that the dorsal lateral geniculate nucleus (dLGN) ipsilateral to the lesion is severely atrophied and gliotic[25] whereas the ipsilateral superior colliculus (SC) undergoes minor reduction and little gliosis. In fact, its metabolic activity evidenced by cytochrome oxydase histochemistry is comparable to that of the contralateral SC. Moreover, the retinae show a dramatic loss of the medium size ganglion cells (X-like) that project to the dLGN and a preservation of large (Y-like) and small (W-like) ganglion cells that send axons to the SC.[26] In the light of the functions attributed to these two channels, namely processing of stimulus and motion localization, it is reasonable to assume that the preservation of the collicular system would enable the patients to detect motion, and localize visual stimuli presented in their blind hemifield as previously demonstrated.[15–17] The quasi-complete disappearance of the dLGN observed in monkeys[25]

and on postmortem examination of hemispherectomized patients (reviewed in Ref. 25) argues against any type of higher visual functions such as the detection or discrimination of colour. Indeed, our patients showed no evidence of detecting unseen stimuli by guessing.

## Conclusion

The present results indicate that the residual visual abilities (e.g. spectral sensitivity) observed in the blind hemifield of patients who have undergone the removal of one cerebral hemisphere are probably due to intraocular light scatter. When the visual stimulus was presented well within the periphery in the blind hemifield, detection was achieved at the central fixation spot. We argue that blindsight defined as an unconscious processing revealed with guessing paradigms and demonstrated in patients and monkeys with lesions of the occipital lobe does not exist in patients with hemidecortication. Whenever the hemidecorticated patients showed evidence of statistically significant stimulus detection, they were aware of the stimulus. The control experiments showed that both awareness and detection are likely to be based on intraocular light scatter and reflection.

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**General Summary**

Blindsight is a perceptual phenomenon which has been described in patients with occipital damage. Some of these patients can respond to visual stimuli presented in their blind hemifield, although they are unaware of their presence. This phenomenon has been attributed to visual pathways other than the major route to the striate cortex. In hemidecorticated patients, all the extrastriate cortical areas have been removed and they offer an ideal model in which to study the contribution of subcortical stuctures to residual vision in the blindfield. In this study, we show that hemidecorticated patients are different from those with occipital damage in the sense that they do not show blindsight as revealed by forced choice guessing. The detectability we could measure in the blind hemifield of hemidecorticated patients is based on intraocular light scatter and reflection.