Localization of visual stimuli after striate cortex damage in monkeys: Parallels with human blindsight

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ABSTRACT  Blindsight is a phenomenon in which human patients with damage to striate cortex deny any visual sensation in the resultant visual field defect but can nonetheless detect and localize stimuli when persuaded to guess. Although monkeys with striate lesions have also been shown to exhibit some residual vision, it is not yet clear to what extent the residual capacities in monkeys parallel the phenomenon of human blindsight. To clarify this issue, we trained two monkeys with unilateral lesions of striate cortex to make saccadic eye movements to visual targets in both hemifields under two conditions. In the condition analogous to clinical perimetry, they failed to initiate saccades to targets presented in the contralateral hemifield and thus appeared “blind.” Only in the condition where the fixation point was turned off simultaneously with the onset of the target—signaling the animal to respond at the appropriate time—were monkeys able to localize targets contralateral to the striate lesion. These results indicate that the conditions under which residual vision is demonstrable are similar for monkeys with striate cortex damage and humans with blindsight.

Human patients with striate cortex damage show a surprising amount of visual function in the resulting field defect despite reporting no visual sensation there (1–6). This phenomenon of visual capacities in the absence of visual awareness, termed “blindsight” by Weiskrantz and colleagues (5), has attracted a great deal of attention because it offers an apparent dissociation between conscious and nonconscious visual perception. To elicit patients’ responses to stimuli presented in a “blank” portion of the visual field, investigators frequently use a forced-choice paradigm in which the subject is asked to respond by guessing at a specified time (1–4).

Under some conditions, as for human blindsight patients, monkeys with striate cortex lesions can make accurate eye movements to visual targets presented in the contralateral field (7–11). While anatomical (1, 12–14) and physiological (15–17) studies in monkeys have helped elucidate the neural mechanisms of the vision surviving striate cortex damage, it is still unclear to what extent the phenomenology of the residual vision in monkeys parallels that of human blindsight. Cowey and Stoerig (18) recently reported that although monkeys with striate lesions could reach for stimuli presented in the affected hemifield, they failed to distinguish between stimulus trials and blank trials in a signal-detection task. The authors interpreted their results as indicating that in monkeys with striate lesions, as in humans with blindsight, there is a dissociation between the ability to experience visual stimuli and the ability to respond to them. To examine further the parallels between vision after striate damage in humans and monkeys, we studied another aspect of blindsight—namely, the ability to localize visual targets under “forced-choice” and “non-forced-choice” conditions.

MATERIALS AND METHODS

Two monkeys (Macaca fascicularis) received large, unilateral, surgical lesions of striate cortex in adulthood (A1 and A2). As the monkeys are still being studied behaviorally, magnetic resonance imaging was used to assess the extent of striate damage (Fig. 1). Striate cortex removal was virtually complete in monkey A1. In monkey A2, there was some sparing of striate tissue deep within the calcarine fissure, which corresponds to the representation of part of visual space beyond approximately 25° eccentricity. Behavioral testing began 21 (A1) and 24 (A2) months after surgery.

While head-restrained and seated in a primate chair, each monkey was trained to fixate on a central point and make saccadic eye movements to visual targets (0.5° diameter, 3.1 log contrast above a scotopic background) appearing at variable times (175–3000 ms) after fixation. The animal received a juice reward for all saccades ending in an electronic error window (2–8° radius) around the target. The animal was rewarded 50 ms after the eye position fell into the error window. Failure to initiate an eye movement within 1 s of target onset was considered an error in “detection.” Targets were presented on two-thirds of the total trials; on remaining trials, no target appeared (“blank” trials), and the monkey was rewarded for maintaining fixation as soon as the fixation spot disappeared. Targets remained on for a maximum of 1 s but disappeared after 150 ms if the eye position deviated from the fixation window and did not enter the target error window (i.e., no corrective saccades could be successfully executed). Eye position was monitored using a scleral search coil (19). All testing was performed monocularly.

Monkeys were tested under conditions where the onset of the target either was unpredictable (non-forced-choice or “standard” condition) or occurred simultaneously with the offset of the fixation spot (forced-choice condition). In the standard condition, the monkeys were not provided with any signal of target appearance and had to both detect the onset of the stimulus and saccade to it. In the forced-choice condition, the offset of the fixation spot provided a signal for the animal to saccade to the target. In both conditions, the monkeys were not rewarded unless the saccade reached the electronic error window surrounding the target. For each monkey, a total of 48 target locations within the central 24° were first tested in about 3400 stimulus trials in the standard condition and then in about 1100 trials in the forced-choice condition.

In addition, the position of the natural blindspot in the field ipsilateral to the lesion was plotted as a control for scattered light (2). In the standard testing condition, neither monkey could detect visual targets when they were presented at closely spaced positions within the blindspot. Instead, both monkeys continued to fixate while the target remained on for 1 s. In the forced-choice condition, neither monkey could localize the blindspot targets. Eye movements were often initiated on

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blindspot trials when the fixation spot disappeared, but they were directed toward random positions within either hemisphere. This indicated that the extent of light scatter around the target was generally less than the size of the optic disk.

RESULTS

Fig. 2 summarizes the frequency of detection errors made in the standard condition in the hemifields ipsilateral and contralateral to the striate cortex lesion for both monkeys. A detection error was defined as a failure of the monkey to initiate an eye movement away from the fixation spot during the presentation of a target. Both monkeys appeared hemianopic—that is, they failed to initiate saccades to targets presented in the hemifield contralateral to the striate lesion and continued fixating, even though failure to respond to a target never resulted in reward. By contrast, both monkeys consistently initiated saccades to targets presented in the ipsilateral hemifield.

By removing the fixation spot simultaneously with target onset, the forced-choice condition was intended to signal or “force” the animal to initiate an eye movement and attempt to localize the target. Each point that was tested in the standard condition was tested again in the forced-choice condition. In this condition, the monkeys quickly learned to make eye movements whenever the fixation spot was turned off. Moreover, the monkeys’ saccades indicated that they had information about the location of the same visual targets they did not respond to in the standard condition. Fig. 3 shows examples of localization accuracy of saccades to four targets at 12° eccentricity, two in each half field. Note that saccade endpoints cluster around each target in both hemifields. The points in the contralateral field were previously undetected in the standard condition.

Fig. 4 summarizes localization accuracy for all test points. In the ipsilateral visual field the angle of the saccades, as expected, was highly correlated with the angular position of the target \( r = 0.99, P < 0.001 \); monkey A1; \( r = 0.99, P < 0.001 \), monkey A2) and the angular gain of the saccades (slope of regression line) was close to 1 (slope = 1.02 and 0.81 for A1 and A2, respectively). In the hemifield contralateral to the lesion, target position also reliably predicted the direction of saccades (r = 0.54, P < 0.001, monkey A1; r = 0.94, P < 0.001, monkey A2; slope = 0.44, A1, and 0.76, A2) particularly for monkey A2, for which the slopes of the regression lines in both hemifields did not differ significantly (t test for slopes, P > 0.10). The localization accuracy for monkey A2 was also significantly better than that of A1 (ANOVA; F = 3.8, P < 0.007).

DISCUSSION

These results show that some monkeys with striate cortex lesions behave as if blind in the hemifield contralateral to the lesion when trained to saccade to visual targets and no additional cue is provided about the onset of the target. However, when given a signal to saccade to these targets, they do so with precision and accuracy. Our results are similar to those of Zihl and Werthe (20), who reported that two human patients without striate cortex could accurately localize visual
stimuli with eye movements. In standard perimetry, both of their patients were instructed to indicate the appearance of visual stimuli in the field contralateral to the striate cortex damage but could not and appeared blind. Yet when they were asked to guess the location of stimuli and were provided a signal at target onset, their saccades to these targets were accurate. Our results in monkeys provide a parallel to human blindsight, in which some patients with striate cortex damage are unable to report visual sensations in the corresponding field defect but can localize visual targets under forced-choice conditions.

An interesting possibility we have considered is that the presence of the fixation point and active fixation by the animal in the standard condition may have prevented or reduced the number of responses to contralateral visual targets. Fixation offset may play two different roles: it may cue the monkey to make an eye movement, and it may also disinhibit comparatively weaker signals from targets within the scotoma. This disinhibition would be consistent with the observation that the visual responses of some neurons in extrastriate cortex, recorded in awake monkeys, are attenuated by fixation of a fixation spot (21, 22). Weak signals elicited by stimulation of the visual field corresponding to a striate lesion may not be sufficient to give rise to an oculomotor response away from an

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**Fig. 2.** Errors in detecting visual targets presented systematically throughout the central 24° of the visual field for monkey A1 and monkey A2 in the standard condition. Each circle represents one test point. The field contralateral to the lesion is to the right for both plots. Points were positioned in polar coordinates (every 6° eccentricity, every 30° θ) in both hemifields from 6 to 24° eccentricity. Points in the contralateral hemifield were tested on 48 trials each; ipsilateral points were tested on 96 trials each. The central square represents the fixation point error window (2° radius).

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**Fig. 3.** Localization of visual targets in ipsilateral and contralateral (blind) hemifields in the forced-choice condition for monkey A2. The task was the same as in the standard condition except that the fixation spot was turned off to signal target onset. Targets (filled circles) were 12° from the center of gaze at polar angles 30°, 150°, 210°, and 330° (see Fig. 2). The final positions of all eye movements that left the fixation window and then clustered within a 2° radius for at least 50 ms were counted as endpoints. Mean endpoints from the initial eight saccades to each target are represented by open squares (30°), diamonds (150°), circles (210°), and triangles (330°). The two contralateral hemifield points were previously undetected in the standard test condition.

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**Fig. 4.** Accuracy of saccades to targets in the ipsilateral (open symbols) and contralateral (filled symbols) fields at varying polar angles for both monkeys in the forced-choice condition. Each point on the graphs represents mean endpoints for eye movements made within 1 s of target presentation. A total of 128 (A1) and 123 (A2) saccades to the ipsilateral field and 84 (A1) and 104 (A2) saccades to the contralateral field were obtained from 160 target trials in each hemifield. On remaining trials, the monkey either did not initiate eye movements or did not make eye movements with discrete endpoints. Error bars denote SEM across eccentricity, 6–24°.
actively fixated target. In any case, it seems likely that in monkeys with unilateral striate cortex lesions, as well as in human patients with blindsight, the residual vision is often too weak to evoke explicit responses to blind field stimulation. Thus external signals and/or release from contralateral inhibition are necessary to demonstrate the spared capacities.

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