Bilateral effects of unilateral visual cortex lesions in human

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Summary
We studied the vision of 12 patients with unilateral lesions of the visual cortex. All had a V1-type scotoma located in the contralateral visual fields, as expected, and visual acuity of 20/30 or better. Our aim was to test the hypothesis that they also had a visual deficit in their ipsilesional or ‘good’ visual fields. The first experiment tested the subjects’ ability to respond to transient signals presented at unpredictable temporal intervals and spatial locations amongst many spatially random and identical distracter elements. The results showed that, compared with controls, the lesion group had a significantly reduced sensitivity to signal and increased response times affecting both hemifields. In a second experiment, we tested the useful field of view (UFOV) in two of the patients under conditions of differing attention demand. Both showed bilateral constriction, compatible with the results of the first experiment. One possible explanation for the bilateral effects of unilateral occipital lobe lesions is damage to interhemispheric connections along their presplenial course, affecting the synthesis of visual information from both hemifields (i.e. the interhemispheric diaschisis effects put forth by von Monakow). The trouble is task dependent and can be construed as a global reduction in visual attention capacity. It is subtle in comparison with the contralosional V1-type scotoma that Holmes measured, yet may account for unexplained complaints of reduced performance in some patients, particularly in tasks with high visual information processing demands, such as reading and automobile driving.

Keywords: attention, corpus callosum; diaschisis; occipital lobe; signal detection; scotoma; visual cortex; visual fields

Abbreviations: A = appearance; D = disappearance; UFOV = useful field of view

Introduction
Late last century, Henschen (1893) correlated damage along the lips of the calcarine fissure in striate cortex, with visual defects in opposite fields of both eyes. This pattern reflects the reversal of the real world image by the lens and the crossing of the nasal fibres of the optic nerve. Inouye (1909) and Holmes and Lister (1916) provided confirmation and further detail. They adapted the standard perimetry techniques of Von Graefe (1856) to study soldiers with missile wounds of the occiput. The regular relationship between visual field defects and loci of brain damage defined a primary retinotopic map in calcarine cortex, or Brodmann’s (1909) area 17, now also called area V1. These studies established that the deficit caused by a unilateral occipital lobe lesion was totally restricted to the opposite visual fields. Yet, other data show that V1 and adjacent extrastriate areas function or fail in concert. Activation of human V1 in perceptual tasks produces obligatory co-activation of surrounding extrastriate cortex (Zeki et al., 1991), while lesions of extrastriate cortex alter V1 activity in human and other species (Bullier et al., 1988; Mignard and Malpelli, 1991; Shipp et al., 1994). Moreover, few if any pathological lesions affect human V1 alone. They at least disrupt the underlying white matter and U-fibre connections to adjacent occipital areas. They probably also affect interhemispheric connections along their prespinal course and even visual cortical–subcortical connections. The damage is far wider than that produced by experimental ablations in monkey (Merigan et al., 1993). Indeed, the missile wounds in Holmes cases extended as far forward as the angular gyrus of the parietal lobe (Holmes, 1918, 1945). In short, V1 dysfunction is unlikely to occur in isolation in

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human cases; extrastriate dysfunction should generally co-occur. If so, what are the behavioural correlates?

Primate extrastriate areas receive extensive visual inputs from both ipsi- and contralateral hemispheres (Rockland and Pandya, 1979; Felleman and Van Essen, 1991; Rockland, 1994). They also contain neurons with receptive fields which overlap the vertical meridian (Desimone et al., 1993) and neurons whose activity is modulated in attention tasks (Moran and Desimone, 1985; Spitzer et al., 1988; Logothetis and Schall, 1989; Schiller and Lee, 1991; Motter, 1994).

Comparative anatomical evidence suggests that similar neurons and connections exist in the human and the activity of human visual cortex appears to be attention dependent (Corbetta et al., 1991). The overall implications are that (i) unilateral human visual cortex lesions will reduce visual performance (accuracy and speed) in both hemifields, in line with the interhemispheric diaschisis effects put forward early this century (Von Monakow, 1902; Sherrington, 1906), and (ii) aspects of the deficits will be attention related. Standard perimetry procedures (such as Goldmann and Humphrey perimetry) will fail to detect such deficits, since they completely ignore speed of processing and intentionally minimize the role of attention to gain maximal estimates of sensory ability (Ball et al., 1990b). This approach is effective for gauging retina or optic nerve function, or plotting a V1-type scotoma. However, it overestimates the visual capacity in elderly or brain damaged individuals (Ball et al., 1990a). The useful visual field of view in such individuals is bilaterally constricted under conditions of increased attentional load (Ball et al., 1993).

Based on the foregoing considerations, we were motivated to test the hypothesis that unilateral visual cortex lesions produce bilateral visual deficits. To do so we studied 12 individuals with unilateral visual cortex lesions and V1-type scotomata in the contralesional fields. Experimental procedures tested the speed and accuracy of their performance for responding to visual stimuli presented to both hemifields. The first experiment required simple detection of transient visual targets. The second required stimulus discrimination and localization. We compared the results with those obtained in non-brain damaged control subjects, as described below.

**Experiment 1: detecting transient signals in a visual array**

**Subjects**

Experimental subjects were 12 patients (eight men, four women) with chronic (longer than 6 months) and strictly unilateral lesions of the visual cortex and underlying white matter (see Table 1). None had splenial lesions. All had V1-type visual field defects encompassing part or all of the hemifield contralateral to the lesion, but not ipsilateral to the lesion, as defined by Goldmann kinetic perimetry (all patients), and automated static perimetry (Patients 1, 3, 6, 9, 10 and 12) using the Humphrey 30–2 threshold test (Allergan, 1990) and the Weil 24–2 threshold test (Chron光电公司, 1990b).

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age/sex</th>
<th>Acuity</th>
<th>Side of lesion</th>
<th>Visual field defect</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68/F</td>
<td>20/20</td>
<td>L</td>
<td>RHH (FS)</td>
<td>Infra- and supra-calcarine</td>
</tr>
<tr>
<td>2</td>
<td>65/M</td>
<td>20/20</td>
<td>R</td>
<td>LUQ (FS)</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>3</td>
<td>70/F</td>
<td>20/20</td>
<td>R</td>
<td>LHH</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>4</td>
<td>60/M</td>
<td>20/20</td>
<td>R</td>
<td>LHH</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>5</td>
<td>32/M</td>
<td>20/15</td>
<td>R</td>
<td>LLQ</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>6</td>
<td>68/M</td>
<td>20/15</td>
<td>R</td>
<td>LUQ</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>7</td>
<td>53/F</td>
<td>20/20</td>
<td>R</td>
<td>LHH</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>8</td>
<td>25/F</td>
<td>20/20</td>
<td>R</td>
<td>LHH</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>9</td>
<td>65/F</td>
<td>20/20</td>
<td>L</td>
<td>RHH</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>10</td>
<td>69/M</td>
<td>20/20</td>
<td>L</td>
<td>RHH</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>11</td>
<td>65/M</td>
<td>20/30</td>
<td>L</td>
<td>RHH</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
<tr>
<td>12</td>
<td>40/M</td>
<td>20/20</td>
<td>L</td>
<td>RHH (FS)</td>
<td>Infra- and supra-calcarine, temporo-occipital junction (mesial)</td>
</tr>
</tbody>
</table>

LHH = left homonymous hemianopsia; RHH, right homonymous hemianopsia; LUQ = left upper quadrantanopsia; LLQ = left lower quadrantanopsia; FS = foveal sparing.
Method
In this experiment, we evaluated the ability of the patients to detect transient 'on' and 'off' signals presented to both hemifields using the Starry Night task (Rizzo and Robin, 1990). Performance on this task depends on a subject's visual sensory function as well as the ability to sustain visual attention over a spatial array, over time. Target events comprise the onset or appearance (A) and offset or disappearance (D) of a small (0.44 mm) white light target presented against a black background at maximal contrast (~99%) on a 14-inch diagonal monitor. The target is embedded among many identical and spatially random distracters, occurs at time \( t_n \). Any response (key press) between 100 and 2000 ms after an event was regarded as a hit. If there was no response within 2000 ms it was regarded as a miss. Any response following a hit or miss in the absence of a new event is regarded as a false positive, or false alarm. Any response within 100 ms after an event is also taken as a false positive since that response is probably initiated prior to the sensory event.

The Starry Night trials were administered at three different starting display densities of 50, 250 or 1000 elements. Each trial lasted ~5–10 min, depending on the response pattern of the subject. The sequence of A and D events was unpredictable to the subject, and was designed to keep the total number of elements in the display within ±3 elements during each trial.

All 12 occipital lobe patients participated in Experiment 1. All understood and were able to cooperate with the procedures. Controls were 12 subjects without neurological disease who were each matched in gender and in age to within 1 year of a corresponding occipital patient. The ages of the control group ranged from 25–71 years (mean age 58 years). All patients and control subjects gave their informed consent to participate in the investigation, which had approval of the local ethical committee.

Scoring of responses
Figure 1 depicts the scheme used to characterize a subject's responses on the Starry Night task. Any response between 100 and 2000 ms after an event was regarded as a hit. If there was no response within 2000 ms it was regarded as a miss. Any response following a hit or miss in the absence of a new event was taken as a false alarm or false positive. Any response within 100 ms after an event was also taken as a false positive since that response was probably initiated prior to the sensory event.

Signal detection analysis
We applied the theory of signal detection to analyse the pattern of responses on the Starry Night task. Theory of signal detection provides the most reliable index of an subject's accuracy, independent of the bias to respond (Swets and Pickett, 1982). The theory considers that all sensory observations are made against a background of noise. Sources of noise include the external environment and the nervous system itself, especially in fatigue or disease states. The
any of our dependent measures (see below) as previously testing Post hoc performance differences between the left and right fields for the occipital lobe lesion and the good field was the ipsilateral type (A versus D) and hemifield (good versus bad). In the subject factors were unilateral occipital lobe lesion and no field. For the control group, there were no significant lesion (the normal control group). The within-subject factors beta and reaction time. The between-measures were, determinations of hit and false positive rates. When beta is d’ this cut-off. It reflects a subject’s willingness to respond that signal-plus-noise, rather than noise alone, occurred at any sensory observation a subject will report ‘yes, I saw it,’ only when the sensory observation exceeds a certain magnitude, the cut-off criterion. Beta reflects the probability that signal-plus-noise, rather than noise alone, occurred at this cut-off. It reflects a subject’s willingness to respond and, like d’, can also be computed from the experimental determinations of hit and false positive rates. When beta is <1, a subject is prone to respond to noise when no signal is present.

### Statistical analysis
Statistical analyses used a mixed ANOVA. The dependent measures were, d’, beta and reaction time. The between-subject factors were unilateral occipital lobe lesion and no lesion (the normal control group). The within-subject factors were star density (50, 250, 1000 elements/display), event type (A versus D) and hemifield (good versus bad). In the lesion group, the bad hemifield was the field contralateral to the occipital lobe lesion and the good field was the ipsilateral field. For the control group, there were no significant performance differences between the left and right fields for any of our dependent measures (see below) as previously reported (Rizzo and Robin, 1990) and the right visual field was arbitrarily designated the good field. Post hoc testing was accomplished using the Tukey procedure. An a priori alpha level of 0.05 was set for all statistical tests.

### Results
**True sensitivity measure, d’**
Table 2 shows the mean and standard deviation of the d’ values for each subject group for each star density (50, 250, 500), event type (A, D) and hemifield (good, bad). Results of the ANOVA showed significant main effects for group [F(1,49) = 1217.31, P < 0.0001] and for star density [F(1,49) = 33.28, P < 0.0001]. Significant interactions were found for group-by-star density [F(2,49) = 7.23, P < 0.0009] and group-by-visual-field [F(1,49) = 45.26, P < 0.0001]. No other interactions were significant.

Figure 2A represents the group-by-field interaction. Post hoc testing showed that d’ was lower for the lesion group in each hemifield compared with the control group. As expected, the lesion group had a significantly lower average d’ for the bad field compared with the good field. No significant differences between the hemifields were found for the control group.

Figure 2B represents the group-by-star density interaction. For each star density, the lesion group had significantly lower d’ compared with controls. Within the normal group, d’ was highest in the 50 star condition and lowest in the 1000 star condition. However, there were no significant differences between the star densities within the hemianopia group, i.e. unlike normal controls the lesion group failed to show any increment in performance with decreasing star densities (Rizzo and Robin, 1990).

Figure 2C represents the main effect for event type. The results showed that there was a higher performance for A versus D events. This is a consistent finding in the Starry Night task (Rizzo and Robin, 1990; Lodge-Miller et al., 1993), and the preservation of this relationship in subjects with occipital lobe damage suggests the pattern depends on the response properties of undamaged cells elsewhere (earlier) in the visual system whose receptive fields are more sensitive to the onset that the offset of a light target (Mutlukan et al., 1992).

### Table 2 d’ (standard deviation) values for both subject groups across all conditions

<table>
<thead>
<tr>
<th>Stars</th>
<th>Control group</th>
<th>Lesion group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Good field</td>
<td>Bad field</td>
</tr>
<tr>
<td>50</td>
<td>4.22 (0.36)</td>
<td>4.22 (0.35)</td>
</tr>
<tr>
<td>D</td>
<td>4.06 (0.41)</td>
<td>4.07 (0.42)</td>
</tr>
<tr>
<td>250</td>
<td>3.68 (0.43)</td>
<td>3.75 (0.38)</td>
</tr>
<tr>
<td>D</td>
<td>3.40 (0.58)</td>
<td>3.46 (0.56)</td>
</tr>
<tr>
<td>1000</td>
<td>3.40 (0.81)</td>
<td>3.55 (0.95)</td>
</tr>
<tr>
<td>D</td>
<td>3.14 (0.88)</td>
<td>3.30 (0.86)</td>
</tr>
</tbody>
</table>

A = appearance events; D = disappearance events. In the occipital lesion group, the 'bad' field corresponds to the contralesional field and the 'good' field to the ipsilesional field. In the control group there were no significant performance differences between the left and right fields for any of our dependent measures and the right visual field was arbitrarily designated the good field. These conventions follow throughout.

### Table 3 Beta values (standard deviation) for both subject groups across all conditions

<table>
<thead>
<tr>
<th>Stars</th>
<th>Control group</th>
<th>Lesion group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Good field</td>
<td>Bad field</td>
</tr>
<tr>
<td>50</td>
<td>2.52 (1.39)</td>
<td>2.54 (1.27)</td>
</tr>
<tr>
<td>D</td>
<td>3.44 (1.73)</td>
<td>3.37 (1.76)</td>
</tr>
<tr>
<td>250</td>
<td>5.50 (2.84)</td>
<td>4.51 (2.49)</td>
</tr>
<tr>
<td>D</td>
<td>7.55 (3.63)</td>
<td>6.82 (3.56)</td>
</tr>
<tr>
<td>1000</td>
<td>4.17 (3.48)</td>
<td>3.89 (2.94)</td>
</tr>
<tr>
<td>D</td>
<td>4.92 (3.07)</td>
<td>5.12 (4.11)</td>
</tr>
</tbody>
</table>

Stars is the distance between the means of the noise and signal-plus-noise units, and can be derived from empirical determinations of percentage of hits and percentage of false positives. The higher the value of the calculated d’ measure, the greater the ability of a subject to detect a target signal.
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Fig. 2 A–C d': A, the group-by-field interaction; B, the group-by-star density interaction; C, the main effect for event type. (See Table 2 and text.)

Table 4 Reaction time (standard deviation) for both groups of subjects across all conditions

<table>
<thead>
<tr>
<th>Stars</th>
<th>Control group</th>
<th>Lesion group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Good field</td>
<td>Bad field</td>
</tr>
<tr>
<td>50</td>
<td>A 321 (40.89)</td>
<td>317 (37.54)</td>
</tr>
<tr>
<td></td>
<td>D 337 (47.91)</td>
<td>334 (42.14)</td>
</tr>
<tr>
<td>250</td>
<td>A 372 (60.52)</td>
<td>371 (57.89)</td>
</tr>
<tr>
<td></td>
<td>D 398 (64.66)</td>
<td>405 (71.47)</td>
</tr>
<tr>
<td>1000</td>
<td>A 377 (52.92)</td>
<td>373 (51.65)</td>
</tr>
<tr>
<td></td>
<td>D 396 (49.42)</td>
<td>390 (60.27)</td>
</tr>
</tbody>
</table>

The group-by-star density interaction is shown in Fig. 3A. Between-group comparisons showed that betas were lower for the lesion group. However, within-group comparisons showed no clear relationship between star density and response criterion. In the control group beta for the 50 stars was significantly lower than for the 250 and 1000 star conditions, but beta for the 250 star condition was significantly higher than the 50 and 1000 star conditions. In the occipital lobe group only the 250 and 1000 star condition differed significantly, with the 250 star condition having the higher beta.

The group-by-event type interaction is shown in Fig. 3B. Between-group comparisons show that the occipital group had significantly lower betas than the control group for A and D events. Beta for the A was significantly lower than for D events within the normal group, but no such difference was found within the lesion group.

Reaction time

Reaction time data for both groups of subjects for all conditions is shown in Table 4. The ANOVA showed significant main effects for group \( F(1,49) = 840.09, P < 0.0001 \) and event type \( F(1,49) = 19.72, P < 0.0001 \).
Significant interactions were found for group-by-visual field \( [F(1,49) = 16.62, \ P < 0.0001] \), group-by-star density \( [F(2,49) = 37.30, \ P < 0.0001] \), group-by-event type \( [F(1,49) = 15.33, \ P < 0.0001] \), and event type-by-visual field \( [F(1,49) = 4.24, \ P < .0411] \). No other significant interactions were found.

The group-by-visual field interaction is shown in Fig. 4A. For each hemifield, the control group had significantly faster reaction times than the lesion group. Within the lesion group reaction times in the good field were significantly faster than in the bad field. No significant differences between fields were found within the control group. These results mirror the findings for \( d' \) described above.

The group-by-star density interaction is shown in Fig. 4B. For all star densities the control group had significantly faster reaction times than the hemianopia group. Within the control group reaction times for the 50 star condition were significantly faster than for the 250 and 1000 star condition. Within the lesion group, reaction times were significantly faster for the 50 star condition than the 1000 star condition, but no other differences reached significance.

The group-by-event interaction is shown in Fig. 4C. The control group had significantly faster reaction times than the occipital lobe lesion group for both A and D events. Within-subject comparisons showed that for both groups reaction times for A were significantly faster than for D events, again mimicking the results for \( d' \).

The event type-by-field interaction is shown in Fig. 4D. Within the good field, reaction times for A were significantly faster than for D. In the bad field no significant differences between A and D were found. There were no significant differences between fields for either A or D.

**Comment**

The optimal strategy while performing the Starry Night task is to remain fixated on the small cross in the centre of the experimental display according to the instructions. There is no advantage in normal subjects searching around the display since target events are equally likely to occur in any region. However, patients with hemianopic visual field loss might adopt a different strategy (Chedru et al., 1973; Meienberg et al., 1981; Ishiai et al., 1987). For example, Ishiai et al. (1987) found that patients with left or right hemianopia (but without neglect) tended to fixate more often and for longer total durations upon portions of displays located toward the side of their visual field defect, whereas normals and hemineglect patients tended to look more in the centre. The experimental display was a horizontal line or rectangle subtending the central 20° of vision. The results appear to differ from those of Rizzo and Hurtig (1992), who found similar fixation patterns in normal and hemianopic observers viewing picture faces and scenes (1992). However, suppose...
the hemianopic subjects in the current study were biased to look away from the centrally located fixation cross on the Starry Night task and toward the blind field (presumably to improve the ability to detect events in the aberrant field). Would such preferential looking lead to a decrease in performance in the ‘good’ field to account for our result? A possible explanatory mechanism would be the projection of visual targets to more eccentric locations in the good field served by less sensitive areas of the retina.

We addressed the hypothetical effects of an eccentric gaze preference in five normal controls by comparing their performance: (i) fixating the centre of the Starry Night display according to standard procedures versus (ii) fixating 75% of the way across the display so that the majority of the display fell into one hemifield throughout the entire procedure (250 star condition). In other words, we attempted to simulate the fixation preference reported by Ishiai et al. (1987) in their left and right homonymous hemianopic (non-hemineglect) patients (depicted in their fig. 1A). A repeated measures ANOVA was performed on the data. The results showed that subjects were not different in terms of accuracy when fixating the centre of the display compared with fixating 75% across the display (d' central fixation = 3.62; d' off-central fixation = 3.63; P > 0.7). There were also no significant differences for reaction time (reaction time central fixation = 352 ms; off-central fixation reaction time = 356 ms; P > 0.7).

In another attempt to evaluate the role of search strategy on performance, we restricted all sensory events in the Starry Night display to the ‘good’ fields of two occipital lesion patients (Patient 5 and Patient J.A., a 69-year-old man with a left occipital lobe lesion, who is not included in the current study because he was tested in the acute phase of injury). The subjects were made aware that no targets would be presented into their hemianopic fields. This adjustment should have eliminated any incentive to ‘cheat’ by looking toward the aberrant field. Patient 5 showed d' = 2.36, reaction time = 476 ms for targets presented to both hemifields, and d' = 2.14, reaction time = 456 ms for events presented in the ‘good’ field only. J.A. showed d' = 1.86, reaction time = 522 ms for targets presented to both hemifields and d' = 1.85, reaction time = 517 ms for targets presented to the ‘good’ field only. Thus, performance in the ‘good’ field in these two patients was abnormal whether targets were directed to both fields or to the ‘good’ field alone. These and the aforementioned observations in normal subjects suggest that preferential looking is unlikely to account for our findings of visual loss in the ipsilesional fields in the current study.

Finally, we note that the topographic pattern of the functional deficit in the ‘good’ field, is not apparent from our reporting of d' and reaction time. However, inspection of the spatial location of target events associated with misses or prolonged reaction time in the ‘good’ field in the Starry Night display shows a pattern that is not mirror symmetrical to the V1-pattern contralesional field loss. For example, a lesion producing a contralateral quadrantanopia (Patients 2, 5 and 6) affected performance in all three remaining quadrants. Thus, the performance deficits we measured appear to represent more ‘global’ or ‘diffuse’ effects, rather than specific topographical effects. Such global effects can be viewed in terms of a limited capacity attention model (Broadbent, 1958) in which damage to a portion of the visual cortical pathways reduces the information processing capacity and efficiency of the whole system (see Discussion). Moreover, the magnitude of this visual loss may be related to the size (‘dose’) of the lesion. This is because patients with occipital lesions causing quadrantanopia (Patients 2, 5 and 6) performed better in the ipsilesional field [d' = 2.15 (0.81); reaction time 448 ms (27.1)] than those with hemianopic field loss (Patients 1, 3, 4 and 7–12) [d' = 1.51 (1.18); reaction time 559 ms (130.8)], which was generally associated with greater damage to visual cortex and white matter.

Experiment 2: measuring the useful field of view

Subjects

Two of the experimental subjects whom we studied in Experiment 1 (Patients 1 and 2), were studied in further detail in Experiment 2. Both patients showed normal performance on a broad battery of tests of intellectual function. Subject 1 had unilateral left occipital lobe damage (Fig. 5A) causing V1-type visual field defects in upper and lower quadrants of the right hemifield (see Fig. 6, top). Subject 2 had a unilateral right occipital lobe lesion (Fig. 5B) that caused a homonymous left upper quadrant V1-type scotoma that affected the central 3–8 degrees of vision (Fig. 6, bottom).

Method

Assessment of the useful field of view (UFOV) used the Visual Attention Analyser, Model 2000 (Visual Resources, Inc., Bowling Green, Ky, USA). This microprocessor-based instrument uses three subtests which provide a reliable measure of UFOV size, expressed in terms of the percentage reduction (0–90%) of a maximum 35° radius field (Ball et al., 1990b). Briefly, in the first subtest, which is designed to assess speed of visual processing, subjects are required to identify a target of varying duration, presented at fixation. This target is the silhouette of a car or a truck. The second subtest, designed to assess the ability to divide attention, also requires the identification of the central target, as well as the localization of a simultaneously presented peripheral target presented at one of 24 possible locations. These stimuli were presented 10, 20 or 30° out from fixation along one of eight radially equidistant imaginary spokes (coinciding with the horizontal, vertical and oblique axes). The duration of the display was varied to measure speed of visual processing for this task. The third subtest was identical to the second...
Results

Subjects 1 and 2 both showed a pathological reduction in the UFOV. Neither had difficulty with the first subtest; however, both subjects performed poorly when the visual processing demands of the task were increased. Subject 1 showed a reduction of the UFOV, of 27.5% on subtest 2 of divided attention and of 30% on subtest 3 of selective attention, for a total UFOV reduction of 57.5%. Subject 2 had less damage to visual cortex and white matter connections and a total UFOV reduction of 30% due entirely to his defective performance on subtest 3. The reduction in the UFOV in Subjects 1 and 2 was not predicted on the basis of the standard visual field examinations and was not attributable to any preferential looking effect: the required identification of a central fixation target at the same time as peripheral targets were presented eliminated this possibility. The measured UFOV loss was also not simply the result of presenting targets into the subjects' scotoma. On the contrary, plotting the locations of the errors upon which the UFOV calculations depend showed that the major component of the UFOV reduction in these subjects was due to impaired response to targets presented outside the known VI-type scotoma including in the ipsilesional (good) field. These results are compatible with the findings obtained by very different means in Experiment 1 on the Starry Night task.

Discussion

We find that unilateral visual cortex lesions are associated with visual processing deficits outside the fields of the classically defined VI-type scotoma and extending into the supposedly normal hemifield, the ipsilesional field. These results are compatible with our introductory hypothesis that unilateral occipital lobe lesions produce bilateral visual deficits.

Compared with controls, the patients with unilateral occipital lesions were more willing to report a signal when none was present. This bias to respond was evident in
Experiment 1 showing reduced beta rates. Reduced beta (and increased false positive) rates can reflect the efforts of patients to maximize their hit rates and thus their apparent visual performance (uncorrected by theory of signal detection calculations) in the face of sensory difficulties. Alternatively, they could be due to increased ‘noise’ in the damaged nervous system of the patients with unilateral occipital lobe lesions. In essence, the subjects were reacting to mirages. The findings underscore the role of visual cortex in separating signal form noise (Baker et al., 1991; Rizzo et al., 1995) and facilitating accurate perceptual decisions (Salzman and Newsome, 1994).

Our findings of abnormal visual performance in Experiment 1 on the Starry Night task as well as in Experiment 2 on the UFOV task, parallel those of Hess and Pointer (1989), who found spatial and temporal contrast sensitivity deficits in the sighted fields in patients with homonymous hemianopia. Taken together, the results of the two studies are compatible with the existence of long-range interactions between different portions of the same hemifield, and between the two visual hemifields. The existence of such long-range links may underpin phenomena such as colour constancy, figure ground separation (Desimone et al., 1993), visual memory and imagery (Doty et al., 1994; Lewine et al., 1994; Ringo et al., 1994) and underlie why the world looks seamless, even though its primary visual cortical representation is split between the hemispheres.
Possible underlying mechanisms

Retinal connections

One explanation for visual deficits in the fields ipsilateral to a unilateral occipital lobe lesion begins at the level of the retina. In some primate species it has been suggested that the foveal or macular representation has a bilateral representation in geniculostriate pathways. Bunt and Minkler (1977) and Leventhal et al. (1988) found that ipsilaterally and contralaterally projecting retinal ganglion cells are mixed in a vertical strip that ranges in width from 1–3°. Thus ipsilaterally projecting cells around the fovea can generate 2–3° of a bilateral representation. By this means, damage to area V1 should affect inputs from both halves of the retina. However, we believe there is insufficient evidence to support such a mechanism in our subjects. First, the ipsilesional deficits we measured in both Experiments 1 and 2 extended far outside the central 1–3° in which such a representation has been supposed to exist. Indeed, the deficits we detected extended at least as far as 35° eccentric to fixation. Secondly, the type of transient stimuli we used, particularly in Experiment 1 on the Starry Night task, were not likely to have depended strongly on P beta cells, the class that convey a bilateral foveal representation to V1 (Leventhal, 1988). P beta cells are the retinal cells which contribute to a 'P'-pathway in primates that sends relays to visual cortex via parvocellular layers 3–6 of the lateral geniculate nucleus. The P-pathway, which may correspond to a psychophysical 'sustained' or colour-opponent channel is not sensitive to transient signals such as we presented (Kulikowski and Tolhurst, 1973). Thirdly, no anatomical study that we know has yet demonstrated a bilateral foveal representation in V1 in human.

Damage to connectional systems

An alternative explanation of bilateral deficits following a unilateral occipital lobe lesion is damage to white matter interfering with the many connections between V1 and other areas. Diaschisis effects (‘diascheisis’), in Von Monakow’s (1902) framework, were thought to represent a loosening of the nexus between neuron chains comprising a reflex arc. In Sherrington’s (1906) interpretation, the conducting cells (‘schaltzellen’) failed to perform their normal function as connecting elements. Such explanations were originally used to explain the phenomena of spinal shock but can also be applied to cerebral deficits.

Damage to several connectional systems, such as callosal and feedback, may explain the observed effects in the current study. In general, the two areas 17 do not communicate directly with each other across the corpus callosum and, in macaque monkey, callosal connections enter only a short distance into 17 (Gould et al., 1987). However, a substantial interhemispheric exchange is evident between portions of areas V2, V4 and MT thought to correspond to areas 18 and 19 in human (Cusick and Kaas, 1988; Felleman and Van Essen, 1991). There are also heterotopic connections (fibres which link non-corresponding visual areas across the callosum) reported from area V2 to MT and V4. None of our patients had overt callosal damage, yet it is quite possible to disrupt interhemispheric connections along their precallosal course (Dejerine, 1892; Damasio and Damasio, 1983).

While the pattern of callosal connections is not well known in the human, comparative anatomical studies suggest that human occipital lesions would typically affect connections resembling those demonstrated in non-human primates (Burkhalter and Bernardo, 1989; Clarke and Miklossy, 1990). Feedback connections to V1 from V2 (area 18) (Rockland and Pandya, 1979) might also be compromised by white matter damage. The overall result of damaging these and other systems (e.g. visual cortical–subcortical) could be to disturb the synthesis of information from other vision areas including from both hemispheres, with, as reported here, resultant disturbances in the ipsilesional field and in the contralateral field outside the expected scotoma. Functional neuroimaging using PET has demonstrated long range effects of unilateral cerebral damage in the cerebellum (Bogsdur et al., 1990; Di Fiero et al., 1990) and we hypothesize that similar effects will be found in the human visual cortex opposite to a unilateral occipital lobe lesion to correlate with the current findings. Similar functional considerations may also apply in the somatosensory system based on the patterns of ipsi- and contralateral connectivity of S1 and S2 (Manzoni et al., 1986).

Potentially important effects of inter-hemispheric connections on the activity of neurons in the early visual association cortex have been demonstrated in the monkey. Specifically, Desimone et al. (1993) found that the receptive fields of neurons in simian V4 have large suppressive regions that span the vertical meridian. Moreover, these ipsilateral suppressive regions were abolished by callosal section. Yet, because V2 has large callosal connections that span several degrees near both vertical and horizontal meridian (Merigan et al., 1993), we suggest that dysfunction as early as V2 may also alter processing in the ipsilesional field.

In particular, Merigan et al. (1993) placed unilateral ibotenic acid lesions in portions of simian V2 serving the central 3–7° of the visual field. Under conditions of controlled fixation they found abnormal spatial discriminations with preservation of acuity and contrast sensitivity, far different from the profound dysfunction produced by comparable V1 injections [in disagreement with Horton and Hoyt (1991) who predicted V1-like deficits in the fields of a V2/V3 scotoma]. However, contrary to the expectations of the current study, the V2 deficits reported by Merigan et al. (1993) were contralateral only. We see three plausible reasons why no ipsilesional deficits were found. First, ibotenic acid is primarily a neuronal toxin that spares white matter connections, does not cause retrograde degeneration, and causes less dysfunction than destructive lesions of human or monkey brain. Secondly, the lesions in the study of Merigan et al. (1993) were relatively small and highly selective. They
did not trespass beyond the boundaries of V2 and indeed affected only a small portion of that map. Thirdly, the presentation of stimuli in the Merigan study followed the tack of most conventional clinical perimetric tasks employed in human patients, which, as previously mentioned, minimize speed of processing and attention factors to gain maximal estimates of sensory ability, and thereby fail to measure attention related deficits.

**Nature of the ipsilesional deficit**

Connectional fibres are an inherent source of intra- and interhemispheric conduction delays, particularly for complex tasks performed under time constraints (Ringo et al., 1994). Simulated damage to such fibres can produce global effects in a network of neural units. The result is reduced accuracy and increased processing time (Ringo et al., 1994), resembling the global (nontopographic) performance effects we observed in our patients (see Comment).

Concerning the nature of the functional loss in the ipsilesional fields, this is a topic that requires further exploration. The results of the current study show very basic defects for detecting the occurrence of visual transients and for identifying and localizing simple shapes under varying attention demands. Hess and Pointer (1989) reported abnormal spatial and temporal contrast sensitivity. A more recent study of a woman with a right occipito-temporal topectomy for the treatment of visual epileptic phenomena (Nawrot et al., 1996) found an ipsilesional deficit of second order motion perception that later resolved. We anticipate that the number and variety of such reports will increase as different testing strategies are adopted.

Aspects of the deficit reported here resemble those reported in humans with bilateral lesions in dorsal portions of areas 18 and 19 (which may contain portions of human V2/V3). In particular, the latter patients showed a reduced ability to detect signals in a field bereft of a V1-type scotoma (Rizzo and Hurtig, 1987; Rizzo and Robin, 1990), as reported in the current study. The lesions in the latter patients presumably affected attention-related neurons (and connections) similar to those identified in the prestriate regions of the monkey (Moran and Desimone, 1985; Spitzer et al., 1988; Motter, 1994), and subsequently produced measurable deficits in visual attention or vigilance. However, because the lesions were bilateral, an ipsilesional component of the deficit could not be evaluated.

**Possible sequelae of the defect**

The bilateral visual performance deficits we measured in this study are subtle compared with the dense V1-type deficits measured by standard perimetry procedures. They may, however, plausibly translate to real-life difficulty and help account for some unexplained symptoms in patients with unilateral occipital lobe lesions. This includes complaints of tired eyes; things looking different, blurred or funny; and a tendency to shun tasks with high visual information processing demands such as reading or automobile driving (particularly in heavy traffic, complex intersections, poor weather or low light). Interestingly, the degree of UFOV loss demonstrated in Patient 1 does appear to predict car crashes even in the absence of a classically defined V1-type visual field deficit (Ball et al., 1993). A practical implication for the perimetry task is that more sophisticated techniques, that involve the role of attention, are needed to define the true functional capacity in the supposedly ‘intact’ visual fields of brain-damaged patients.

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