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INVITED REVIEW

Blindsight in man and monkey

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Summary

In man and monkey, absolute cortical blindness is caused by destruction of the optic radiations and/or the primary visual cortex. It is characterized by an absence of any conscious vision, but stimuli presented inside its borders may nevertheless be processed. This unconscious vision includes neuroendocrine, reflexive, indirect and forced-choice responses which are mediated by the visual subsystems that escape the direct cerebral damage and the ensuing degeneration. While extrastriate cortical areas participate in the mediation of the forced-choice responses, a concomitant striate cortical activation does not seem to be necessary for blindsight. Whether the loss of phenomenal vision is a necessary consequnce of striate cortical destruction and whether this structure is indispensable for conscious sight are much debated questions which need to be tackled experimentally.

Keywords: cortical blindness; blindsight; residual vision; phenomenal vision; visual system

Abbreviations: dLGN = dorsal lateral geniculate nucleus; OKN = optokinetic nystagmus; PGN = pregeniculate nucleus; ROC = receiver-operating-characteristic curve

Introduction

'A survey of the recent literature indicates that the rôle of the occipital lobes in visually guided behavior in general cannot be properly evaluated as long as investigators are satisfied with studying only a few visual functions. Because certain visual functions have often not been studied at all and others only incompletely, the experimental data necessary for elucidating the character and the variety of visual disturbances in occipital cases are at present frequently lacking.' Klüver, who studied the visually guided behaviour of monkeys with surgical removal of the visual cortex, wrote this critical assessment in 1941 (p. 23). In the half century since then, the phenomenon of blindsight as a processing of visual information that is not consciously represented has been established in both man and monkey. Exploring its properties now offers a means of studying the full range of behaviour possible on the basis of unconscious vision, a task which is far from being completed. Nevertheless, from our present knowledge of what can and cannot be done with blindsight alone, we can already draw some tentative conclusions regarding the functions of conscious vision. Revealing the neuronal basis of blindsight is a means of gaining a better understanding of how the visual system works and, by exclusion, of getting a hold on the neuronal correlate of conscious vision. Indeed, whether and in what way primary visual cortex is necessary for blind and conscious vision is currently much debated. Views on the consequences of its destruction now encompass the gamut of possibilities: absence of conscious vision, absence of blindsight and absence of blindness.

Visual functions after striate cortical destruction

Background

Vascular incidents, traumata and tumours are the commonest causes of damage to the striate cortex and its geniculate afferents. Both destruction and deafferentation produce homonymous visual field defects contralateral to the side of the lesion. The field defects are perimetrically determined and clinically classified with respect to their extent, their position in the field, and their density. In a relative defect,

the patient consciously sees certain stimuli. Most commonly, high contrast stimuli that move or flicker are perceived while form and colour vision are absent (Riddoch, 1917; Holmes, 1918). In rare cases (sometimes following carbon monoxide poisoning) colour perception can be selectively spared (Rovamo *et al*., 1982; Milner and Heywood, 1989; Humphrey, 1996). Relatively impaired or amblyopic zones in which patients have conscious residual vision (not blindsight) often surround or flank absolute field defects (e.g. Holmes, 1931) where stimuli are not seen at all. In the definition of Wilbrand and Sänger (1904), absolute defects are areas where 'alle und jegliche Empfindungsqualität, also Helligkeit und Farbempfindung, ausgefallen ist und absolut nicht mehr hier empfunden wird' (p. 353). It is noteworthy that already here the lack of 'all and any sensory quality' is used to define the absoluteness of the field defect; it is not an absence of all visual function that characterizes a post-geniculate visual field defect.

Attempts to elucidate the possibility and extent of residual visual functions remaining in patients with damage to the occipital lobe were focused on two approaches. One was to use reflexive responses, notably the pupil light reflex and optokinetic nystagmus (OKN), and the other was to explore residues of conscious vision which were established by asking the patients what, if anything, they saw in response to stimulation of the defective fields (e.g. Bard, 1905; Bender and Krieger, 1951; Brindley *et al*., 1969).

The second approach was not an option in the study of animals. Here, non-verbal behavioural responses were used to uncover non-reflexive functions in monkeys with extensive occipital cortical ablations. It was on the basis of such experiments, notably those of Klüver (1941, 1942), the Pasiks (for review, *see* Pasik and Pasik, 1982), Weiskrantz and Cowey (for review, *see* Weiskrantz, 1986), and their colleagues, that similar non-verbal paradigms were introduced into the study of patients. Richards (1973) demonstrated a residual crude stereoscopic mechanism operating in cortically blind fields. That same year, Pöppel *et al*. (1973) reported that patients with absolute field defects could direct their eyes towards the approximate position of a briefly presented stimulus they had not seen. Weiskrantz and colleagues (Sanders *et al*., 1974; Weiskrantz *et al*., 1974) tested several residual visual functions that included localization and shape discrimination in a patient with a surgical ablation of primary visual cortex. They also used forced-choice methods, and coined the term 'blindsight' to account for the residual visual properties they demonstrated. Note that blindsight as we use it here is not synonymous with the term 'residual visual functions': in blindsight the stimuli are not consciously seen; in contrast, residual vision *can* be conscious.

Thus visual functions were discovered which were neither automatic, reflexive responses to visual stimulation nor residual conscious processes. Further demonstrations and increasing knowledge of the massively segregated retinofugal pathways have lent credibility to the phenomenon of 'blindsight' which has become a prime example of an 'implicit process', a non-reflexive function elicited by a stimulus that is not consciously represented.

Levels of blind visual functions

Here we shall present the evidence that demonstrates the presence of blind visual functions in patients and monkeys with destruction or deafferentation of the primary visual cortex. Four levels of visual processing will be distinguished.

Patients

Neuroendocrine responses. The lowest level of visual processing is that of neuroendocrine responses. A good example is that of melatonin suppression in response to exposure to bright light which has been elicited even in some patients blinded by retinal pathology (Czeisler *et al*., 1995). These patients show no pupillary response to light, and report not even a dim visual impression, but the neurendocrine response is still demonstrable. It is probably mediated via a small sub-population of retinal ganglion cells which continues to project directly to the hypothalamus (Moore *et al*., 1995*a*).

Reflexive responses. The reflexive responses constitute the next level of function. To a different extent they, too, remain after post-geniculate damage. The pupil continues to respond to changes in illumination (Magoun and Ranson, 1935; Bender and Krieger, 1951; Brindley *et al*., 1969) (*see* Fig. 1), to spatial (Weiskrantz, 1990) and at least in some patients to spectral information (P. Stoerig, J. L. Barbur, A. Sahraie and L. Weiskrantz, unpublished data). The photic blink reflex can be elicited when a light is flashed (Bender and Krieger, 1951; Hackley and Johnson, 1996), and the eyes move with a moving visual scene (Braak *et al*., 1971; van Hof-van Duin and Mohn, 1983; Pizzamiglio *et al*., 1984; Heide *et al*., 1990). Results of tests of OKN are controversial (for discussion *see* Verhagen *et al*., 1996). Its subcortical component (i.e. passive OKN) has been found to persist in patients with unilateral brain damage (Heide *et al*., 1990) up to, and including, hemispherectomy (van Hof-van Duin and Mohn, 1983; Braddick *et al*., 1992). In contrast, it could not be evoked in several patients with complete bilateral cortical blindness (Brindley *et al*., 1969; Perenin *et al*., 1980; Perenin, 1991), with the single exception of a patient with total cortical blindness who was studied by Braak *et al*. (1971). These prominent differences in the extent to which subcortical OKN persists in total cortical blindness have not been demonstrated in a sufficient number of patients with bilateral lesions to assess the likely roles of the extent of the primary lesion, its degenerative consequences, and additional sub-cortical damage. Apart from OKN, whose status is not clear, visual reflexes persist in the absence of functional striate cortex, although they may be sub-normal, with the pupillary constriction to light being of lesser amplitude (*see* Fig. 1),

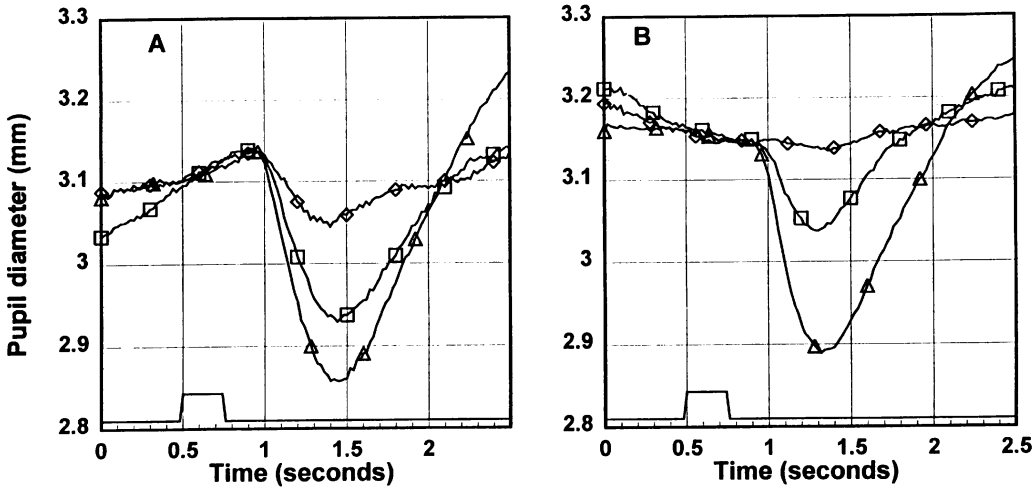

Fig. 1 Pupil light reflex traces measured with the stimulus in the intact (**A**) and cortically blind hemifield (**B**) of patient F.S. On a uniform background (27×22°, 22 cd/m^2), stimuli of 0.3, 1.5, and 3.4 contrast (increment/background) were used to elicit the pupil response. Note the reduction in amplitude, especially at lower contrast, when the response is evoked from the blind field. (Unpublished data from P. Stoerig, J. Barbur, A. Sahraie and L. Weiskrantz.)

its spatial tuning coarser and the eye-movements elicited by moving scenes asymmetric and sluggish.

Implicit processing. To demonstrate implicit processing of a stimulus presented within a field defect without requiring the observer to respond to it directly, its effect on the response to a seen stimulus in the normal visual field is examined. This represents the next level of visual function. If this response is altered in some way by the unseen stimulus, information from the blind field must have been processed implicitly. There are now several different examples. (i) Simultaneous or prior presentation of an unseen stimulus can significantly alter the mean reaction time to a seen stimulus (Marzi *et al*., 1986; Corbetta *et al*., 1990; Rafal *et al*., 1990; Cochrane, 1995). (ii) A full circle, half of which falls into the blind field, may appear more complete than a half circle which falls entirely into the good field (Warrington, 1962; Torjussen, 1976). (iii) The hue of an after image induced by fixation inside a coloured suround can change when the colour of the surround is changed, even when the change is restricted to the blind field (Pöppel, 1986). (iv) Performance in a phi-motion direction discrimination task with two consecutive stimuli flanking FS′ wedge-shaped absolute defect on its upper and lower border (*see* Fig. 2) improved for certain spatiotemporal conditions when a third and invisible stimulus was presented between the two visible ones. For certain spatiotemporal conditions patient F.S.'s performance improved even in the impaired hemifield when a third and invisible stimulus was presented between the two visible ones. Occasionally, the patient even reported an ability to infer motion instead of just detecting a temporal onset asynchrony between the stimuli (Stoerig and Fahle, 1995). (v) Unsuspected cognitive processing in blindsight was implied by the induction of an interpretational bias to an auditorily presented polysemous word (BANK) by a preceding presentation, in the blind field, of a word related to one of its meanings (RIVER/MONEY) (Marcel, 1983, 1997).

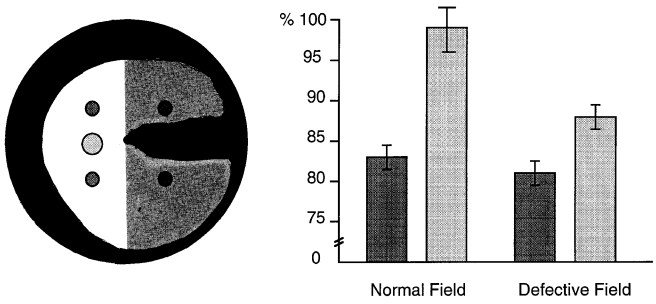

Fig. 2 Phi-motion direction discrimination with two versus three stimuli in patient F.S. *Left*: the stimulus arrangement, with either two or three stimuli aligned vertically in the intact left hemifield or in the impaired right hemifield. In the latter the top and bottom stimuli flank the absolute field defect and the central stimulus falls within it and was invisible. Although the stimuli are of equal size in both hemifields, the flanking ones appear much smaller in the defective field. *Right*: the presence of the third stimulus nevertheless improved Phi-motion direction discrimination in both hemifields. For each hemifield, the left and right bars show performance with two and three stimuli, respectively. Error bars show ±SEM. (Data from Stoerig and Fahle, 1995.)

Implicit processing of this nature can only be demonstrated if the patients retain a functional visual field because a response to a seen stimulus is used to assess the influence of an additional unseen one.

Direct responses. In contrast to implicit processing, the direct responses which constitute the highest level of functions found after striate cortical destruction require the patient to respond directly to stimuli presented in and confined to the field defect. Investigations of direct responses that commonly

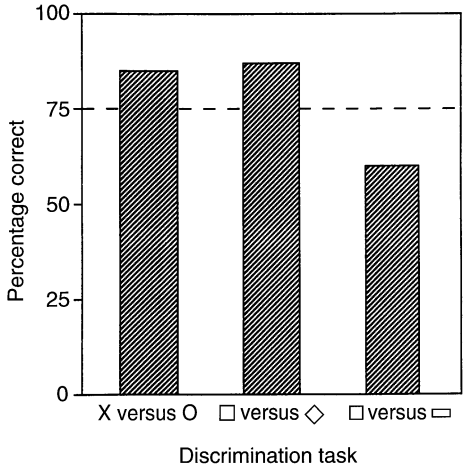

Fig. 3 Shape discrimination in patient D.B. for three stimulus pairs: X versus O, square versus diamond, and square versus rectangle. Stimuli were front projected for 150 ms at 45° eccentricity on the 45° meridian. Stimulus size was 10×8° for X and O, 7.3×7.3° for the square and diamond, and 4.4×12.2° for the rectangle. The dashed line indicates the threshold which was set at 75% correct; indiscriminate responding would yield ~50% correct. (Data taken with kind permission from Weiskrantz, 1986.)

use forced-choice methods show that some patients can localize, by hand or eye-movement, the approximate position of a stimulus presented briefly at different eccentricities in the cortically blind field (Pöppel *et al*., 1973; Weiskrantz *et al*., 1974; Perenin and Jeannerod, 1975; Blythe *et al*., 1987). Such patients can also detect stationary and moving stimuli interleaved randomly with blank trials (Stoerig *et al*., 1985; Stoerig and Pöppel, 1986; Stoerig, 1987; Stoerig and Cowey, 1989*a*, *b*, 1991; Magnussen and Mathiesen, 1989), and can discriminate stimulus orientation (Weiskrantz, 1986; Morland *et al*., 1996), target displacement (Blythe *et al*., 1986, 1987), direction of motion (Barbur *et al*., 1980; Perenin 1991) and wavelength (Stoerig, 1987; Stoerig and Cowey, 1992; Brent *et al*., 1994). Only when shapes had to be discriminated, and orientation cues were excluded by using Efron figures (figures that differ neither in orientation of their borders nor in area) did blindsight fail to reach a threshold criterion set at 75% correct performance even in the extensively tested patient D.B. (Weiskrantz, 1987). D.B. exhibited a large range of residual functions including discrimination of X and O (*see* Fig. 3); this was particularly good in same-different matches between the normal and impaired fields (Weiskrantz *et al*., 1974; Weiskrantz, 1986). Recent data extend these findings by demonstrating that a hemianopic patient's deliberate and considered verbal and manual responses to Efron-type shapes were at chance level, whereas the patient's reaching and grasping movements to the same stimuli correlated well with their shape and orientation (Perenin and Rossetti, 1996).

Paradigms derived from signal detection theory (Green and Swets, 1968; Swets and Pickett, 1982) have occasionally been used in the study of direct responses because they provide a means to account for oberver bias and allow a careful analysis of detectability and discriminability. In a study by Stoerig *et al*. (1985) receiver-operating-characteristic curves (ROCs) were determined at five positions in the cortically blind field of patient K.K., each one based on 2500 presentations. A shift in criterion was introduced by varying the ratio of target-to-blank trials. The resultant data points (five per curve) were replotted on a double probability scale to test whether the assumptions that underlie the use of parametric detectability indices [such as d' and d(A)] were satisfied. The distribution of ROC-points indicated a different variance for target and blank conditions, and the authors concluded that non-parametric measures, like the area under the curve [$P(A)$] or the percent correct value, provided indices less fraught with assumptions about the underlying distributions. We have since restricted ourselves to the use of these (e.g. Stoerig and Pöppel, 1986; Stoerig, 1987; Stoerig and Cowey, 1989*b*, 1992).

The lengthy testing required to aquire sufficient data for signal detection analysis has hindered its general application; Weiskrantz (1990) dubbed this approach 'heroic'. For similar reasons, attempts to measure sensitivity (as opposed to detectability and discriminability) in the cortically blind field are comparatively rare; after all, determining sensitivity amounts to nothing less than measuring detectability for an unknown number of luminance values. The thresholds that have, nevertheless, been measured were all elevated when compared with those of the normal visual field, although the elevation can be surprisingly small. The threshold for orientation discrimination was found to be ~ 10° as compared with 2–3° at the corresponding position in the normal half-field of patient D.B. (Weiskrantz, 1986). Wavelength discrimination thresholds ranged between 20 and 30 nm, depending on the individual and the part of the spectrum tested, as compared with a few nm at the correspondingly eccentric control position in the patients and in normal observers (Stoerig and Cowey, 1992). Grating acuity was 15.8 cycles/degree as compared with 20 cycles/degree (Weiskrantz, 1986), and as shown in Figs 4 and 5, increment threshold sensitivity was reduced by ~0.4 to 1.5 log units (Stoerig and Cowey, 1989*a*, 1991; Stoerig, 1993*a*). Depending on variables such as retinal position, size, colour, onset time and type, speed of the stimulus and level of adaptation, the patients' performance ranges from chance level (e.g. Hess and Pointer, 1989; Stoerig, 1987) through moderate but statistically significant (e.g. Magnussen and Mathiesen, 1989; Stoerig and Cowey, 1989*b*) and up to 100% correct (Perenin, 1991; Weiskrantz *et al*., 1991, 1995; Barbur *et al*., 1994).

The small threshold elevation for detection or discrimination makes it implausible that the detection is based on stray light from the stimulus in the blind field. However, when grossly supra-threshold stimuli are used, this possibility must be considered. It is impossible now to assess the extent to which such effects may have contaminated the residual conscious sensitivity that was reported by early investigators such as Bard (1905); the patients' attribution of a light source to the blind field does not suffice to rule

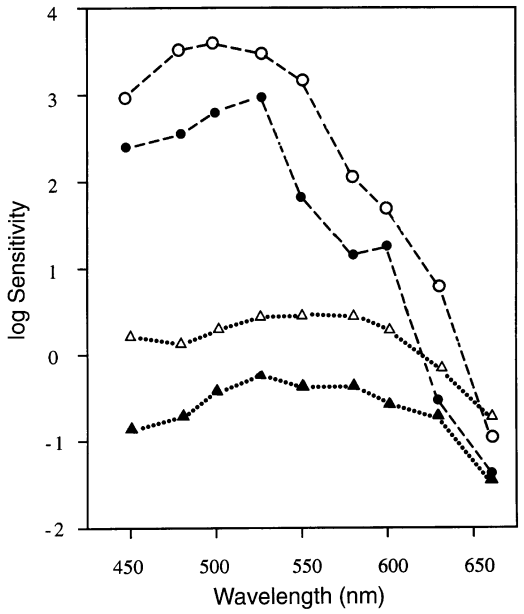

Fig. 4 Spectral sensitivity curves from the normal (open symbols) and cortically blind hemifield (filled symbols) of patient B.R. Note the clear Purkinje-shift when adaptation is changed from dark (upper two curves) to light (lower two curves), and the small loss of sensitivity in the blind field. Stimuli were 116′ in diameter, and presented at 10° eccentricity on the upper oblique meridians (45 and 135°, respectively). (Data from Stoerig and Cowey, 1991.)

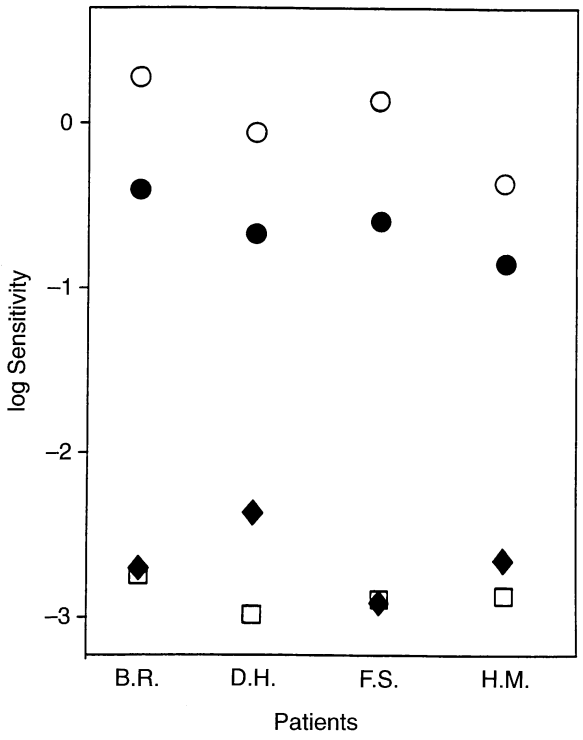

Fig. 5 Sensitivity (log) in four patients for 116′, 200 ms stimuli presented at 10° (patients B.R., D.H. and F.S.) and 30° eccentricity (patient H.M.). Sensitivity in the normal hemifield (open circles); blindsight sensitivity in the cortically blind field (filled circles); stray light sensitivity with the target presented on the natural blind spot in the scotoma (closed diamonds); stray light sensitivity with the target presented at the position used for measuring blindsight sensitivity in the scotoma (open squares). Note the small difference between normal and blindsight sensitivity but the vastly greater difference between both these and the detection of scattered light.

out such effects. Many of the more recent investigators have therefore introduced a variety of control tasks. One of the most convincing examples is to present the stimulus within the normal blind spot; provided the stimulus is smaller than the blind spot, detection (Stoerig *et al*., 1985; Stoerig and Pöppel, 1986; Weiskrantz, 1986, 1987; Stoerig and Cowey, 1989*a*; Stoerig, 1993*a*) and localization (Pöppel *et al*., 1973) fail. This receptor-free zone can also be used to measure stray light sensitivity, which then allows an assessment of its possible effects. We have used this procedure, asking patients not to guess whether or not a target has been presented, but instead to base their decision on any weak percept of light or of a halo emanating from an unseen stimulus. An example is given in Fig. 5. It demonstrates that sensitivity for a 2° circular target detected by stray light is 2–3 log units lower than sensitivity measured under identical conditions at a corresponding position in the normal field. In the specially designed Tübinger perimeter we have used for testing the patients, only 2° white stimuli could be made intense enough to be detected on the basis of stray light under light-adapted conditions in the whole field. In light-adapted conditions where performance is less likely to be contaminated by stray light the difference between sensitivity in the normal field and stray light sensitivity in the blind spot was ~3 log units (Stoerig, 1993*a*). The difference between sensitivity in blindsight as determined by forced-choice guessing, and stray light sensitivity was between 1.5 and 3 log units. Only a very small difference was found between stray light sensitivity determined by presenting the stimulus in the blind spot, and presenting it in the blind field but instructing the patient not to guess but to wait for some weak percept that would indicate stimulus presentation. As the optic disc is normally more reflective than the normal retina, this could indicate a change in reflectance caused by transneuronal retrograde degeneration (*see* below).

Monkeys

Neuroendocrine responses. While neuroendocrine responses have not been measured in monkeys with surgical ablation of the striate cortex, rapid reflexive responses have.

Reflexive responses. Several investigations reported that cortically blind monkeys retained a blink reflex and appropriate pupillary change in response to a bright light but the reports were unconvincing until Klüver (1936, 1941, 1942) demonstrated them in monkeys in which all or nearly all striate cortex was subsequently shown to have been removed. He was even able to show that frontal stimulation,

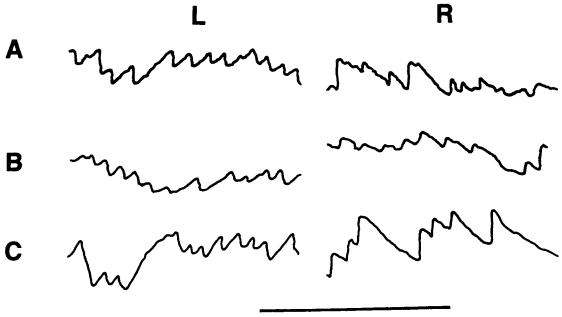

Fig. 6 OKN in a monkey. An upward deflection indicates an eye movement to the left. L and R indicate the direction of rotation of the vertical black and white stripes of the optokinetic drum surrounding the head. (**A**) OKN in a normal monkey; (**B**) 4 months and 2 months after removing the left and right striate cortex, respectively; (**C**) 5 and 3 months. Stripe width was ~7.5° and angular speed between 25 and 50°/s. Scale bar represents 5 s. (Adapted from Braak and van Vliet, 1963.)

presumably stimulating the central retina, was more effective than lateral stimulation, even though the latter is more likely to fall in any spared peripheral vision (1936). Klüver also noted an absence of the blink response to a threatening gesture; however, in some presentations of a large and contrasty looming stimulus King and Cowey (1992) could elicit an avoidance response. The photic blink reflex was subsequently studied by Pasik and Pasik (1964, 1982) who demonstrated its independence of the pupil by paralysing the latter. They also showed a clear relationship between stimulus intensity and the occurrence of blinking (probability of blinking >0.9 with the most intense stimuli) and that blinking was not an artefact of heat from bright lights.

OKN was first thoroughly investigated by the Pasiks (Pasik and Pasik, 1964, 1982), with a greater range of stimulus conditions than those used by Braak and van Vliet (1963), who were the first to show that 6 months after histologically verified total removal of striate cortex, OKN was 'not essentially different from before the operation' (*see* Fig. 6). According to the Pasiks, OKN was present as little as 1 week after bilateral removal of striate cortex, with substantial recovery after 1 month, especially for stimulus velocities of 22–45°/s. The peak frequency of response was roughly halved, and OKN was abolished at velocities of 80–90°/s. Like ter Braak and van Vliet, they noted after-nystagmus. Flicker-induced nystagmus in which monocular stimulation provokes nystagmus in the direction of the stimulated eye, was essentially normal (Pasik *et al*., 1970).

Implicit processes. To the best of our knowledge, implicit processes have not previously been investigated with indirect methods in monkeys. We therefore include an example of one of our own unpublished experiments in which one normal and three hemianopic monkeys were trained to touch a start light near the centre of a visual display unit. This action extinguished the start light and led to the presentation of a target light in the normal left hemifield on every trial. In order to obtain a peanut or raisin, the monkey had to touch the target within its presentation time of 1 s. The target was presented immediately upon extinction of the start light, or with a delay of up to 500 ms. On half the trials, randomly selected, a second stimulus was presented in the hemianopic field at the same time as the target or preceding it by up to 500 ms. Figure 7 shows that, as in some patients with blindsight, the unseen target slowed the reaction time to the seen target, especially at the longer delays.

Direct responses. From the time of Klüver's investigations (Klüver, 1936, 1941, 1942) it was clear that monkeys with extensive or complete removal of striate cortex were able to respond directly to visual stimuli in the corresponding parts of the visual field. Like patients with blindsight, monkeys show direct responses. They can localize visual stimuli in the cortically blind field, making manual (Keating, 1975; Feinberg *et al*., 1978) as well as saccadic responses (Mohler and Wurtz, 1977; Segraves *et al*., 1987). They can detect targets (Pasik and Pasik, 1973) and discriminate between stimuli differing in luminous flux (Klüver, 1941; Schilder *et al*., 1971), brightness (Schilder *et al*., 1971) and orientation (Keating, 1975). In contrast, they show no evidence of detecting visual stimuli in field defects caused by retinal lesions, even though the usual opportunities for detecting scattered light are present (Cowey, 1967).

Wavelength discrimination was reported by Schilder *et al*. (1972), Pasik and Pasik (1980) and Keating (1979) who showed that five monkeys relearned a red versus green discrimination despite substantial and randomized variation in the intensity of the broad-band stimuli following total removal of striate cortex (subsequently verified histologically). After additional removal of extra-striate areas that included areas V2, V3, V4, TEO, and even caudal IT, four of the monkeys reached at least 80% correct, and those who were switched to blue versus green achieved 70–85% correct. In contrast, Humphrey (1974) found no evidence that what to a normal observer would be red and green were discriminable irrespective of intensity for his monkey Helen. By varying the 'brightness' of the green Humphrey found an equivalence point where Helen's discrimination collapsed. Curiously, this equivalence point resembled a mesopic rather than a photopic match when the targets were on a white background, but was characteristic of photopic vision when tested in a different way by detecting a green spot on a red surround.

Results from Malmo (1966) and Leporé *et al*. (1975) add to this puzzle by showing that after total removal of striate cortex, peak spectral sensitivity under light-adaptation is almost the same as under dark-adaptation, i.e. at ~500 nm. This indication of a loss of cone function is in contrast to the clear indication of a Purkinje-shift we could measure in our hemianopic monkeys (*see* Fig. 8). A difference between uni- and bilaterally destriate monkeys could underly the latter difference as the monkeys of Malmo (1966) and Leporé *et al*. (1975) had extensive bilateral lesions, whereas our three

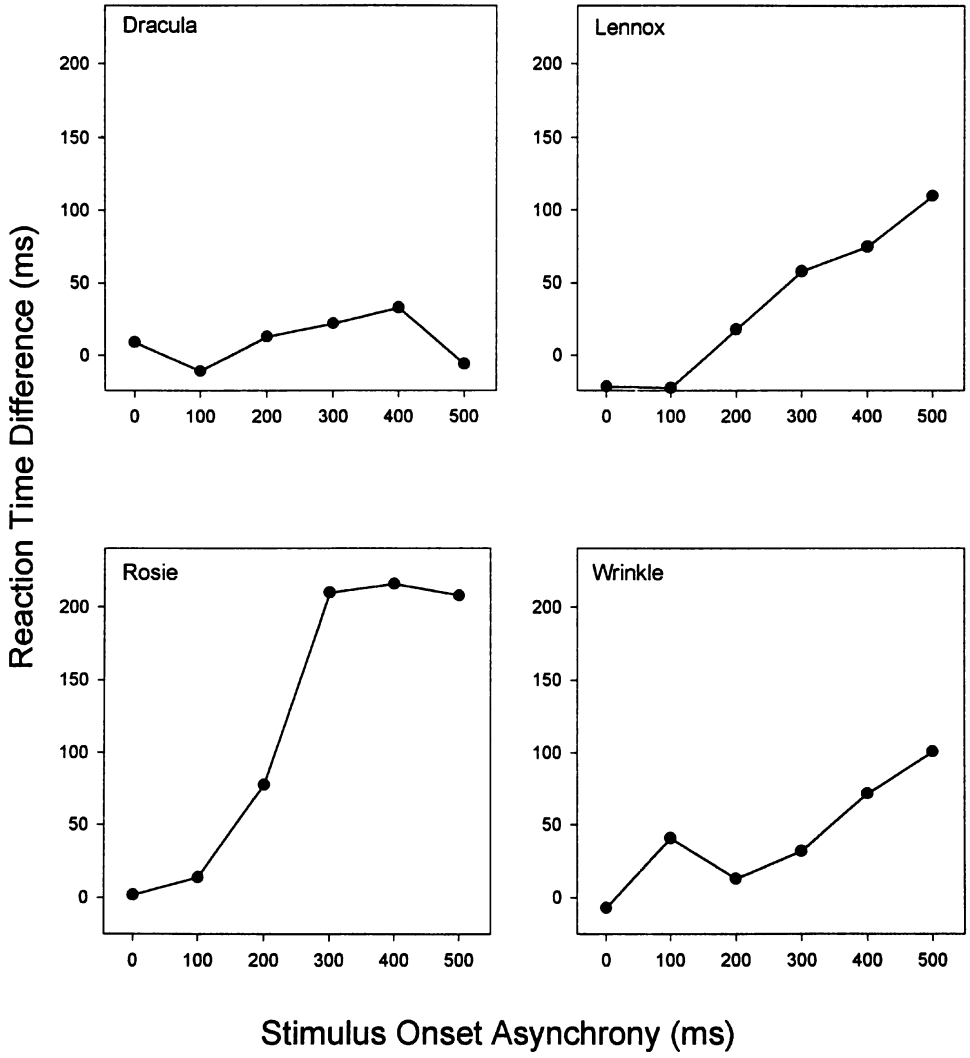

Fig. 7 The effect on reaction time to a target (4°, 1 s, 20 cd/m^2, 10° eccentricity) in the normal left hemifield of presenting a similar but 40 cd/m^2 target in the right hemifield of a normal monkey (Rosie) and three monkeys with a right-sided hemianopia (Dracula, Lennox and Wrinkle). The monkeys were rewarded for touching the target in the normal left hemifield which appeared on every trial. On half the trials, an additional (irrelevant) target appeared on the right, coinciding with the left target or preceding it by up to 500 ms. This target in the hemianopic field slowed the reaction time to the left target in all monkeys, whether normal or hemianopic. (Our own unpublished data, *see* text for details.)

monkeys had striate cortex removed unilaterally and without extensive damage to extrastriate cortex. However, even if this difference explained the Purkinje-shift in the hemianopic monkeys, it cannot explain the differences in wavelength discrimination because all animals had bilateral lesions, with those who performed best having the largest lesions (Keating, 1979).

Equally controversial are the results of shape or pattern discrimination. Klüver (1941) showed that a destriated monkey could discriminate between a luminous square and 76 small circles of the same total area and flux, choosing the circle on 90% of trials. Klüver's conclusion that 'the topographical aspects of the stimulus configuration may become effective in determining the reactions' (p. 39) was confirmed by Weiskrantz (1963) who came to regard the total length of contour as one of the discriminable features, as part of his general view that differences in total retinal ganglionic activity were the basis for much of the discrimination. That shape discrimination is, nevertheless, not an easy task to master in a cortically blind field was shown in the extensive series of experiments by the Pasiks and their collaborators [Pasik *et al.*, 1969; *see* Pasik and Pasik (1982) for review]. Macaque monkeys without any striate cortex but with postoperative experience and success with a wide range of visual tasks required several thousand trials in order to relearn to discriminate between a circle and a triangle at 90% correct. Although the monkeys were subsequently unaffected in a range of control conditions where changes were introduced in the luminance or luminous flux of the two shapes, their performance was disrupted by

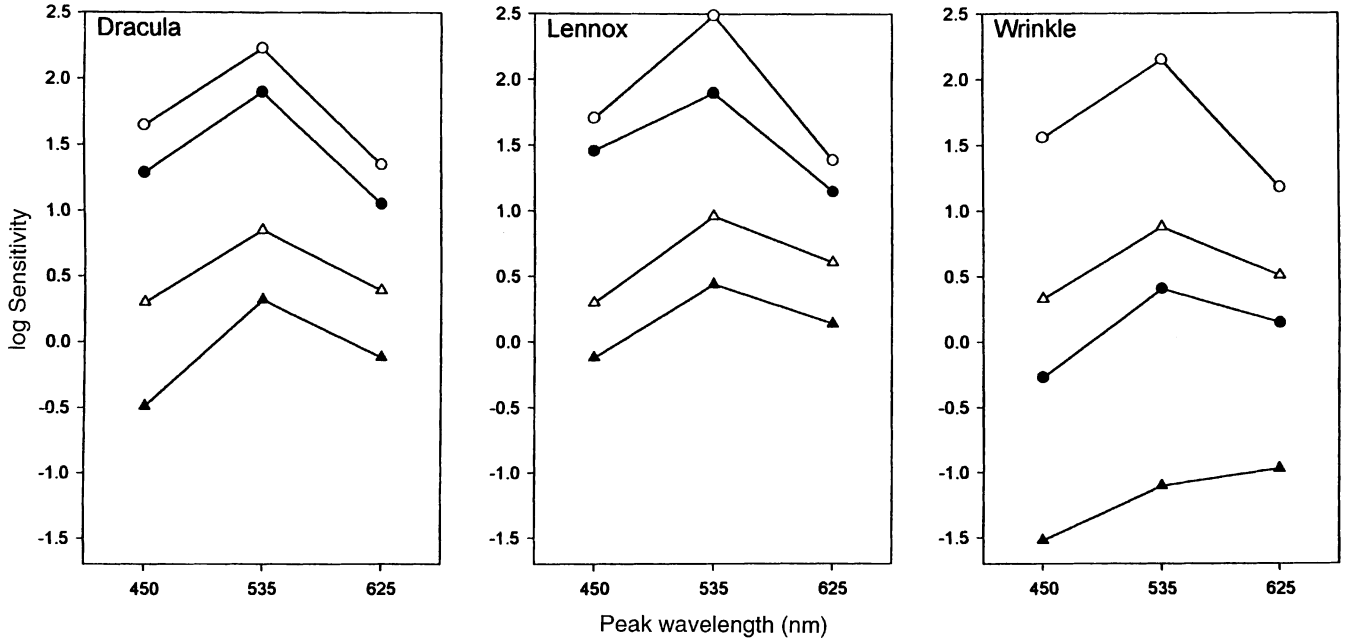

Fig. 8 Spectral sensitivity in the seeing (open symbols) and in the blind fields (filled symbols) of three hemianopic monkeys. Peak wavelength refers to the peak of the the broad-band spectral emissions of the three phosphors of a visual display unit. Note that the reduction in sensitivity in the blind field is as little as 0.4 log units, with the exception of monkey Wrinkle. Note also the Purkinje shift: when dark adapted (circles) the animals are relatively less sensitive to long wavelengths in both hemifields than when light adapted (triangles) (Our own unpublished data, *see* text for details.)

changing the size of the triangle or by inverting it. The difficulty of pattern discrimination was also demonstrated by Dineen and Keating (1981) when only three of their five monkeys succeeded in responding at better than 80% correct to the rewarded luminous flux equated pattern, where amount of contour, shape of sub-elements and number of corners were systematically varied. Finally, Humphrey (1970, 1974) showed that the destriated monkey Helen could discriminate between several targets that differed only in shape. However, when the discriminability of each stimulus from some standard stimulus was tested, Humphrey found that different shapes were indiscriminable when matched for salience (roughly, the extent of a stimulus' discriminability from a standard): a 10 mm diameter red circle was indiscriminable from a 10×5 mm black rectangle, and a circle was indiscriminable from a triangle (Humphrey, 1974).

Whether this should be seen as evidence against genuine shape discrimination in bilaterally destriate monkeys (interestingly, it has never been studied in the field defects of monkeys with unilateral or extensively incomplete bilateral lesions) or whether it only demonstrates that the presence of more than one stimulus difference overtaxes the system, remains open. Certainly the monkeys show no evidence of visually recognizing complex objects (such as fruit or model snakes) in their cortically blind visual fields even after many years of experience (Cowey and Weiskrantz, 1963; Humphrey, 1974).

The diminution in detection and discrimination sensitivity in the monkeys is of the same order of magnitude as in the patients: Orientation discrimination thresholds are around 8° in bilaterally destriated monkeys, as compared with about 1° in normal animals (Pasik and Pasik, 1980) (Fig. 9). The highest resolvable spatial frequency at maximum contrast was reduced from ~40 to 12 cycles/degree (Miller *et al*., 1980) (Fig. 9). Sensitivity to the contrast rather than the upper spatial frequency of resolvable gratings, arguably a better guide to visual function, was reduced from ~100 to 4 at 2 cycles/degree (Miller *et al*, 1980, and Fig. 9). Difference thresholds (Weber fractions) for targets of different luminance but identical area were 0.2–0.3, i.e. approximately a threefold elevation (Klüver, 1941).

Increment thresholds were increased by 0.5 to 1.6 log units for achromatic (Leporé *et al*., 1976; Cowey and Stoerig, 1995) as well as chromatic stimuli (Cowey and Stoerig, 1995). The level of performance can approach 100% correct for supra-threshold targets (e.g. Cowey and Stoerig, 1995) (Fig. 10).

The striking similarities between the species are in contrast to the old conjectures of unbridgeable gaps between man and the other animals, often based on the doctrine of encephalization of function (for debate, *see* Weiskrantz, 1972), and provide the rationale for discussion of the anatomical and physiological underpinnings of blindsight on the basis of material which stems primarily from monkeys.

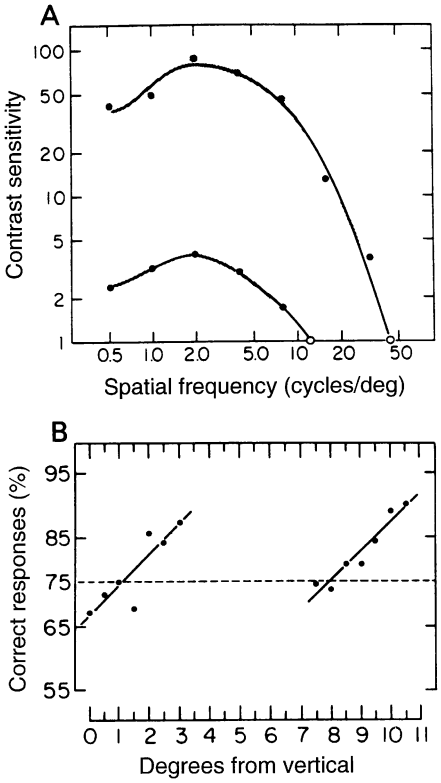

Fig. 9 (**A**) Contast sensitivity of four macaque monkeys before (top curve) and after (bottom curve) removing all striate cortex. Filled circles give group means. Open circles show the extrapolated upper limit of spatial resolution. (**B**) Percentage correct responses as a function of orientation difference between a vertical and an inclined bar. Results are means for two monkeys before (*left*) and after (*right*) removal of striate cortex. Threshold for 75% correct was 1.4° versus 7.5°, respectively. [**A** reproduced from Miller *et al.* (1980), with permission from the American Physiological Society and **B** reproduced from Pasik and Pasik (1982) with permission from Academic Press, Orlando, USA.]

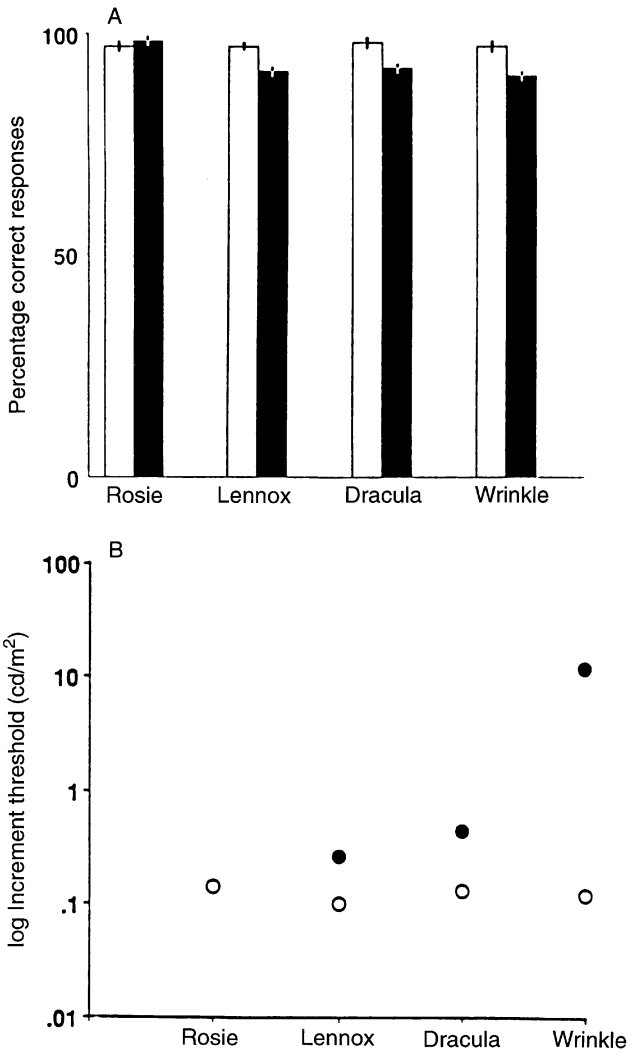

Fig. 10 (**A**) Percentage correct detection in a normal monkey (Rosie) and three hemianopic monkeys for 200 ms, 2° targets presented at a lateral eccentricity of ~20°. Stimulus luminance was adjusted to 0.7 log units above the threshold shown below. All monkeys perform better than 90% correct in both hemifields. Open columns, normal left field; filled columns, (blind) right field; bars indicate SEM. (Cowey and Stoerig, 1995; reproduced with permission.) (**B**) Binocular increment threshold luminance in the normal left (open circles) and hemianopic right (filled circles) hemifields for 75% correct performance, using the same 200 ms 2° target. Normal monkey Rosie's sensitivity was the same in both hemifields. (Adapted from Cowey and Stoerig, 1997.)

The functional anatomy of blindsight

Monkeys

Anatomical consequences of the lesion

The residual visual functions that have been demonstrated in the visual field defects caused by striate cortical destruction must be mediated by the visual pathways that survive the degenerative consequences of the lesion. This residual system receives its retinal input from the ganglion cells which escape transneuronal retrograde degeneration (Van Buren, 1963; Cowey, 1974). In normal monkeys the ratio of Pα : Pβ : Pγ ganglion cells is about 1 : 8 : 1, at least in the central 30° of the retina (Perry and Cowey, 1984; Perry *et al.*, 1984). Although all three classes contribute to the surviving population their ratio becomes roughly 1 : 1 : 1 as a result of the striking and, as far as is known, selective death of Pβ cells (Cowey *et al.*, 1989; Weller and Kaas, 1989; Niida *et al.*, 1990). The surviving ganglion cells continue to project to the retinorecipient nuclei of the midbrain and diencephalon. A decrease in innervation density has been reported for the retinal projection to the dorsal lateral geniculate nucleus, which loses the vast majority of its projection neurons (van Buren, 1963; Miahailovic *et al.*, 1971), and for the projection to the olivary nucleus of the pretectum (Dineen *et al.*, 1982); in contrast, the projection to the pregeniculate nucleus hypertrophies (Dineen *et al.*, 1982) (*see* Fig. 11). Projections to the superior colliculus (Dineen *et al.*, 1982) and pulvinar nucleus appear to be unchanged (Cowey *et al.*, 1994) (*see*

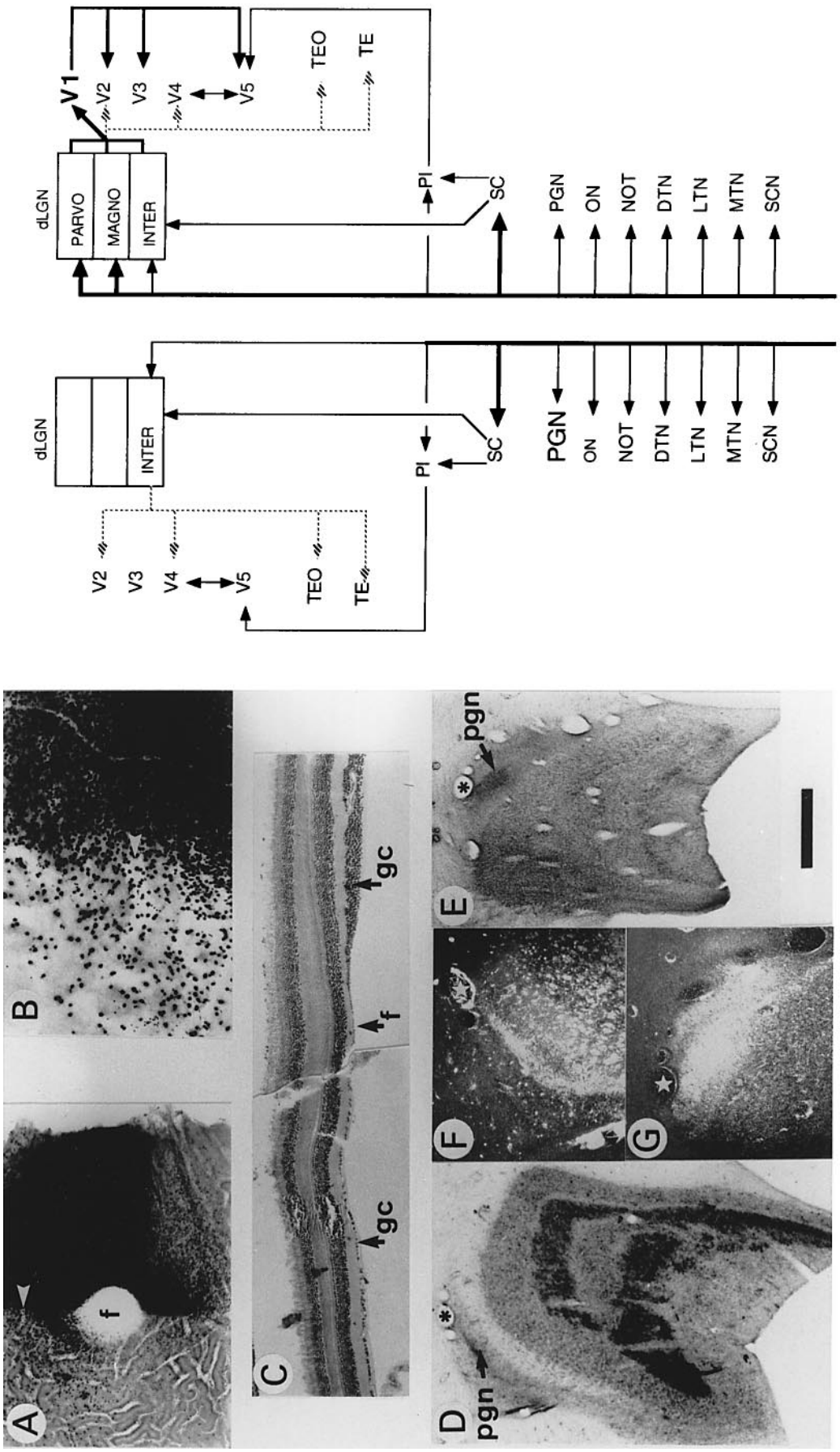
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Fig. 11). Note, however, that present tract tracing methods are likely to reveal only prominent changes.

The role of extrastriate visual cortex

Information from the degenerated retina can reach extrastriate visual areas either directly from retino-recipient subcortical nuclei, dorsal lateral geniculate nucleus (dLGN) (Yukie and Iwai, 1981; Cowey and Stoerig, 1989) and inferior pulvinar or indirectly via other retinorecipient structures (pregeniculate nucleus, olivary nucleus, accessory optic nuclei and nucleus of the optic tract). All of these project to other mid-brain structures, notably the superior colliculus, which in turn project to the dLGN and to the inferior pulvinar.

Physiological studies in anaesthetized monkeys, whose striate cortex was ablated or reversibly inactivated, indicate that the extrastriate cortical areas that form the dorsal stream retain much of their visual responsiveness (for review, *see* Bullier *et al*., 1993). In contrast, cells in the ventral stream that respond to stimulation of the blind field are only rarely encountered, despite the anatomically demonstrated sparse but widespread direct projections of a sub-set of dLGN cells to areas V2, V4, TEO, and IT. The ventral stream thus appears to be more dependent on striate cortex for its visual responsiveness. However, general anaesthesia could influence the results, and a sparse distributed population of cells like those receiving its input directly from the dLGN could elude single cell recording. Furthermore, the immediate effects of removing or inactivating striate cortex on such a sparse cortical innervation might not be a good guide to the role of an extrastriate visual pathway in a conscious alert subject months or years after striate cortical damage.

Visual responses have been recorded in dorsal extrastriate cortical areas of monkeys who had no functional striate cortex on that side (Rodman *et al*., 1989, 1990), and residual visual functions involving direct responses have been demonstrated in monkeys with complete removal of striate cortex. Therefore islands of spared striate cortex can neither explain the remaining cortical responsiveness nor the presence of residual visual functions in the affected hemifield of unilaterally destriated monkeys.

The role of extrastriate cortex for blindsight has also been addressed in behavioural investigations. The effects of including extensive ablation of extra-striate as well as striate cortex were examined in two monkeys in which the lobectomy was made at a caudal level such as to include what are now known as areas V2, V3, V4 and perhaps V5 (MT) (Pasik and Pasik, 1971). Both monkeys were substantially more impaired than those with less involvement of extrastriate cortex, requiring twice as many trials (6000 as opposed to 3000) to select, eventually, the more intense of two targets of equal size. When required to relearn discrimination between two targets of very different area but equivalent flux (small bright versus large dim) they still failed to reach criterion after 6000 trials, whereas monkeys with less extensive extrastriate removal relearned the task in about 600 trials.

Comparing the effects of striate removal alone and striate plus extrastriate cortical removal, Keating (1975) found that lesions including areas OA and OB left the monkeys unable to discriminate objects or to even locate and retrieve them accurately; even the retrieval of moving targets seemed more impaired in these animals. Devastating consequences of extensive extrastriate lesions alone (i.e. sparing striate cortex) have been described by Keating and Horel (1972) and Nakamura and Mishkin (1986), and explain the turn for the worse observed in the visual capacities of monkeys with lesions extending far beyond striate cortex. Klüver's surgical procedure would have put his monkeys into this group, with the extrastriate cortical damage even including dorsal parietal

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Fig. 11 (**A**) Photomicrograph of flat-mounted retina of a macaque monkey in which the upper half of the left optic nerve was labelled with horseradish peroxidase 4 years after removing the left striate cortex at the age of 5.5 years. The normal nasal hemiretina is densely stained, in comparison with the retrogradely transneuronally degenerated temporal hemiretina to the left of the fovea (f). (**B**) Higher power photomicrograph centred on the white arrowhead, also shown in **A**. The loss of ganglion cells is substantial. (**C**) Horizontal section (10 μm) in the vicinity of the fovea, through the other eye of the same monkey. The degenerated ganglion cell layer of the nasal hemiretina, normally slightly thicker than the temporal hemiretina, has been reduced to a monolayer. The section had cracked to one side of the fovea and has been realigned. (**D** and **E**) Sections of the normal and degenerated dLGN of a macaque monkey in which the striate cortex was removed unilaterally at the age of 18 months and the animal was perfused 22 months later, following an injection of [^{3}H]proline into the eye contralateral to the ablation. Dark bands, prominent in **D** but faint in **E**, show by autoradiography, labelling of magnocellular and parvocellular layers. The pregeniculate nucleus (PGN) is both relatively and absolutely larger on the degenerated side. (**F** and **G**) Dark-field photomicrographs of the PGN showing the more prominent input to the degenerated side, some but not all of which is attributable to the normally greater labelled contralateral input. Star and asterix in **D** and **F** mark a corresponding capillary, as they do in **E** and **B**. Scale bar represents 1 mm for **A**, **D** and **E**; 250 μm for **B** and **C**; 625 μm for **F** and **G**. The schematic diagram shows known inputs from the eye to retinorecipient nuclei, and some of the subsequent forward projections. The right half of the figure shows the normal condition, the left half the effect of removing the striate cortex (V1). The cortical pathways exclude further projections from V2, V3, V4, V5, etc. For example, parts of the inferior pulvinar also project to V2, V3 and V4. Following removal of striate cortex the PGN expands and the ON contracts, indicated by the size of the lettering. With respect to geniculo-cortical pathways the dotted lines indicate relatively sparse projections. SCN = suprachiasmatic nucleus; MTN, LTN, DTN = medial, lateral and dorsal terminal accessory optic nuclei; NOT = nucleus of optic tract; ON = olivary nucleus. PGN = pregeniculate nucleus; SC = superior colliculus; PI = inferior pulvinar. (Our own unpublished material.)

visual areas in both the superior and inferior parietal lobules. The effects of unilateral hemidecortication on visually guided behaviour that would further elucidate the role of extrastriate cortex have hardly been assessed. One notable exception (Tusa *et al*., 1986) showed that this procedure abolished saccades to visual targets in the hemianopic field.

The extent to which extrastriate cortex participates in the processing of visual information from the cortically blind field obviously depends on the extent to which it is damaged; this is highly variable, even in monkeys where the lesion is deliberately inflicted. It also depends on the age at which the damage occurs, with lesions in infancy sparing more visual functions (Moore *et al*., 1995*b*; Ptito *et al*., 1996), possibly by preserving or prompting greater direct innervation of extrastriate cortical areas, as has been shown in cats (Payne and Cornwell, 1994). Finally, it depends on the residual function tested.

The role of subcortical nuclei

The question as to which sub-cortical nuclei are involved in which function was addressed by the Pasiks (1965, 1971) who studied the relearning of light versus no-light discrimination after bilateral removal of striate cortex, then examined the effects of additional bilateral sub-cortical lesions. To their surprise, lesions of the inferior pulvinar, or superior colliculi, or medial pretectum had no or only slight effects when compared with those of the lateral pretectum plus the accessory optic tract (and probably involving indirectly all three of its terminal nuclei). Following the latter lesion, three monkeys failed to relearn this simple discrimination in 6000 trials. Thus, while light versus no-light discrimination seems to depend on lateral pretectum and possibly the accessory optic system, localization in the cortically blind field depends on the superior colliculus. Mohler and Wurtz (1977) demonstrated that the ability to make saccadic eye movements to small brief targets, presented within a field defect produced by a partial striate lesion is abolished by a subsequent lesion confined to the retinotopically corresponding part of the superior colliculus. A similar conclusion was reached for manual localization (Solomon *et al*., 1981). Physiological evidence that the superior colliculi may be involved in motion processing was provided by Rodman *et al.* (1990) who found that as many as half of the neurons in extrastriate area MT (or V5) retained normal directional sensitivity to visual stimuli after the striate cortex representing that part of visual space had been removed. But when the appropriate part of the retinal representation in the superior colliculus was additionally removed, their sensitivity was abolished. The superior colliculus is also implicated in the mediation of the visual functions that survive hemidecortication, because it shows much less degeneration than the dLGN, and preserved metabolic activity, at least following hemidecortication in infant monkeys (Ptito *et al*., 1996).

The role of the unlesioned hemisphere is difficult to assess because the vast majority of tests were carried out in monkeys with bilateral ablation. The visual functions of our monkeys who are hemianopic are rather similar to those of bilaterally lesioned monkeys reported in the literature, with the possible exception of OKN and photopic spectral sensitivity. The OKN exhibits deficits in bilaterally destriate animals, but recovers in unilaterally destriate monkeys even following subsequent resections of almost the entire cerebral hemisphere (Pasik *et al*., 1959). However, combined cortical and accessory optic lesions disrupted OKN (Pasik and Pasik, 1973). Whether our evidence for a Purkinje-shift depends on the survival of a normal visual hemifield which could determine and set the adaptation level, or whether it is the limited extent of the occipital lesion, or some other critical feature, is presently unknown. Interestingly, however, our own unpublished results on wavelength discrimination are as puzzling as those of investigators who studied it in monkeys with complete cortical blindness.

Together, the results imply that whether and which cortical and sub-cortical structures are involved depends on the visual function tested, i.e. on both the stimulus presented and the response measured, and it depends on the age at which the lesion was incurred. There is no good evidence that the retinofugal projection to the superior colliculus is the sole provider of the visual information processed in blindsight.

Patients

In patients we have almost no evidence with regard to the involvement of subcortical structures in the mediation of blindsight function. This section will therefore be restricted to evidence of cortical involvement.

Unilateral occipital damage

Patient F.S. was recently studied with functional MRI (fMRI) performed while the central visual field was stimulated with an array of flickering red lights subtending ~20°×12°. The scans showed no activation in the ipsilesional striate cortex which was deafferented by a traumatic lesion affecting left temporo-parietal areas and invading the optic radiation. In contrast, the contralesional striate cortex was strongly activated (*see* Fig. 12).

A PET-study of patient G.Y., who also suffered a traumatic lesion which, in contrast to F.S.'s, was largely restricted to striate cortex, similarly showed no activation within the lesioned striate cortex when a bar was moved through the affected hemifield; instead, extrastriate cortical activation in areas assumed to be involved in motion processing were responsive (Barbur *et al*., 1993). Extrastriate cortical visual activation was observed in both patients and is in close agreement with the physiological results obtained in monkeys.

G.Y. and F.S. are currently the most extensively studied patients with post-geniculate visual field defects, and show a broad range of residual visual functions (for G.Y. *see* Barbur *et al*., 1980; Hess and Pointer, 1989; Brent *et al*.,

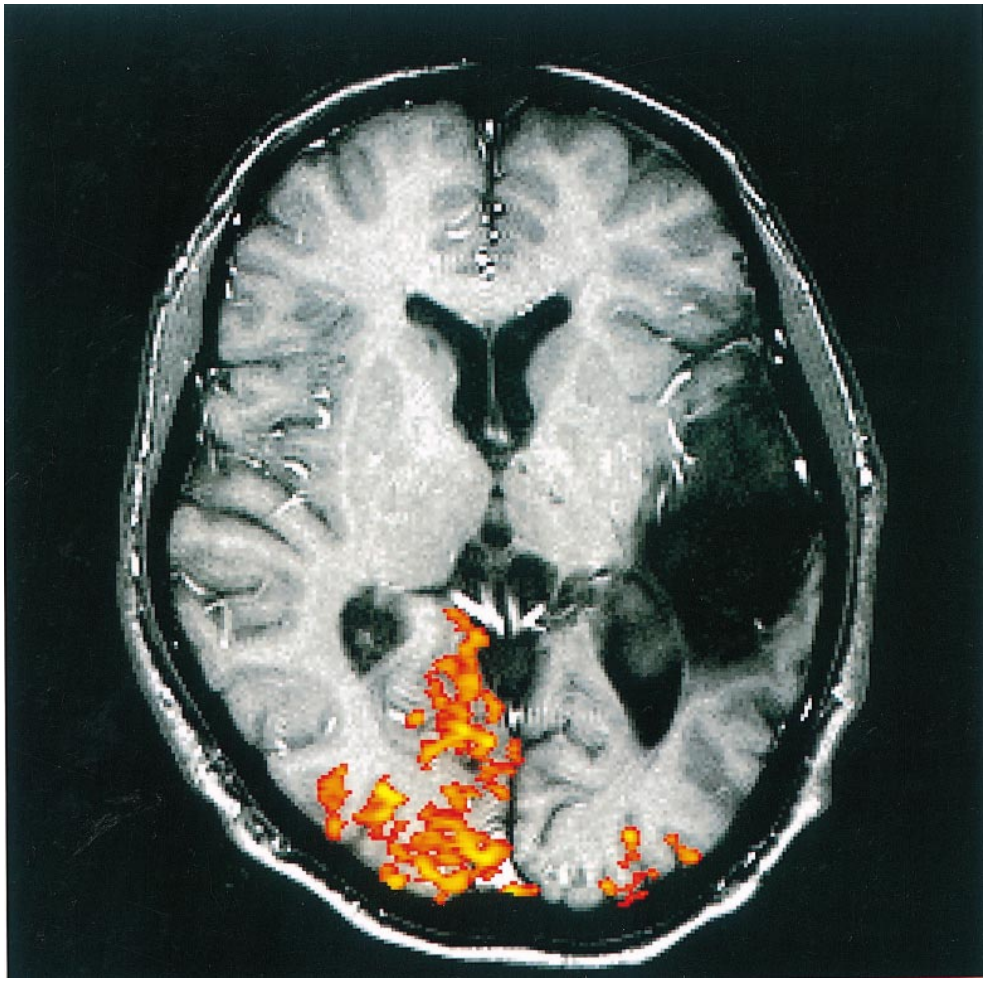

Fig. 12 The fMRI that sampled the calcarine cortex by 4 mm sections showed no activation in the deafferented part of striate cortex of patient F.S. in response to a binocularly presented 10 Hz optic fibre array which subtended about 20°×12°; the control condition was darkness. The colour-coded activation map was superimposed onto a flow-sensitized anatomical MRI. fMRI was performed by dynamic acquisition of FLASH MRI. Data evaluation was based on pixel-by-pixel temporal correlation of MRI signal intensity changes with a reference waveform reflecting the stimulation protocol corrected for haemodynamic latencies. Further analysis employed individualized thresholding of correlation maps (Kleinschmidt *et al*., 1995); thus the activation map comprises areas with maximum correlation coefficients above 0.6 complemented by neighbouring pixels exceeding a coefficient of 0.3. (Unpublished data from P. Stoerig, A. Kleinschmidt and J. Frahm.)

1994; Weiskrantz *et al*., 1995; Morland *et al*., 1996; for F.S. *see* Pöppel, 1985, 1986; Stoerig, 1987, 1993). Nevertheless, neither showed any evidence for visual responsiveness in lesioned or deafferented striate cortex. Together with other arguments concerning the nature of their visual function (Stoerig, 1993*b*; Weiskrantz, 1996), this result is incompatible with assertions that residues of striate cortex, in the form of functional 'islands' in lesioned V1 tissue that correspond to islands of blindsight, are both responsible and indispensable for blindsight (Campion *et al*., 1983; Celesia *et al*., 1991; Fendrich *et al*., 1992). Moreover, the lack of activation in G.Y.'s lesioned striate cortex, which we have recently confirmed using fMRI with its superior spatial resolution (R. Goebel, P. Stoerig, L. Muckli and W. Singer, unpublished data), is particularly interesting because G.Y., in contrast to F.S. who has blindsight, actually has (conscious) residual vision (*see* below for discussion).

Hemidecortication

In contrast to striate cortex, extrastriate cortex may indeed be necessary, at least for the direct (forced choice) blindsight responses. Lower-level reflexive responses such as the pupillary light response and the photic blink response persist even in coma patients (Keane, 1979), and require no cortical activation. A pupillary light reflex (Weiskrantz, 1990) as well as some subcortical OKN have been demonstrated in hemispherectomized patients (Van Hof-van Duin and Mohn, 1983; Braddick *et al*., 1992), indicating that neither requires functional cortex in the hemisphere subserving the blind

field. In contrast, explicit blindsight functions could depend on the presence of functional ipsilateral extrastriate cortical areas. This hypothesis is supported by the negative findings of three groups who independently studied a variety of explicit functions in the blind hemifields of patients with unilateral cerebral decortication. Two patients, tested for their ability to discriminate the direction of visual motion, performed at chance level (Perenin, 1991). Another five patients performed at chance level in a variety of tasks except when demonstrably detecting stray light with the intact hemifield (King *et al.*, 1996) (*see* Fig. 13), and yet another four (plus three incompletely tested) patients showed a reduction, and other properties, of sensitivity in the blind field indicative of detection based on stray light (Stoerig *et al.*, 1996*b*). Should these results, which disagree with other published data (Perenin, 1978; Perenin and Jeannerod, 1978; Ptito *et al.*, 1987, 1991) but agree with the loss of saccadic localization in hemidecorticated monkeys (Tusa *et al.*, 1986), prove to be the rule rather than the exception, they would imply that explicit functions actually depend on extrastriate cortex. The depleted residual visual system that escapes degeneration after hemidecortication (Walker, 1938; Peacock and Combs, 1965; Ueki, 1966; Ptito *et al.*, 1996) would then be capable of mediating only the lower level responses, possibly up to the indirect level (Tomaiuolo *et al.*, 1994).

Bilateral occipital damage

In normal observers, fMRI has demonstrated extensive bilateral activation in higher extrastriate cortical areas in response to unilateral stimulation (e.g. Mendola *et al.*, 1996). Should any blindsight function following unilateral destruction of striate cortex depend on processes in the contralesional hemisphere, it should be absent, weakened, or altered in bilateral as compared with unilateral cases. However, with the already mentioned possible exceptions (OKN and Purkinje-shift), there is little evidence of marked differences between unilaterally and bilaterally damaged patients, and the same is true of monkeys. Contour discrimination, wavelength discrimination, localization, grasping for moving objects have all been demonstrated in bilaterally destriated monkeys. Complete cortical blindness is fortunately rare in human patients, but those who have been tested exhibited explicit visual functions such as motion direction discrimination (Perenin, 1991) (Fig. 14), localization and (at least temporarily) crude wavelength discrimination (Perenin *et al.*, 1980). If the bilateral damage is incomplete, not causing total cortical blindness but sparing some 'seeing' field, the patients demonstrate 'normal' blindsight in the impaired field (e.g. Stoerig, 1987) including OKN (Heide *et al.*, 1990). If the bilateral damage causes a relative cortical blindness, i.e. allowing some conscious residual vision, the patients may demonstrate such remarkable visual abilities as navigation in unfamiliar surroundings (Ceccaldi, 1992; Mestre, 1992).

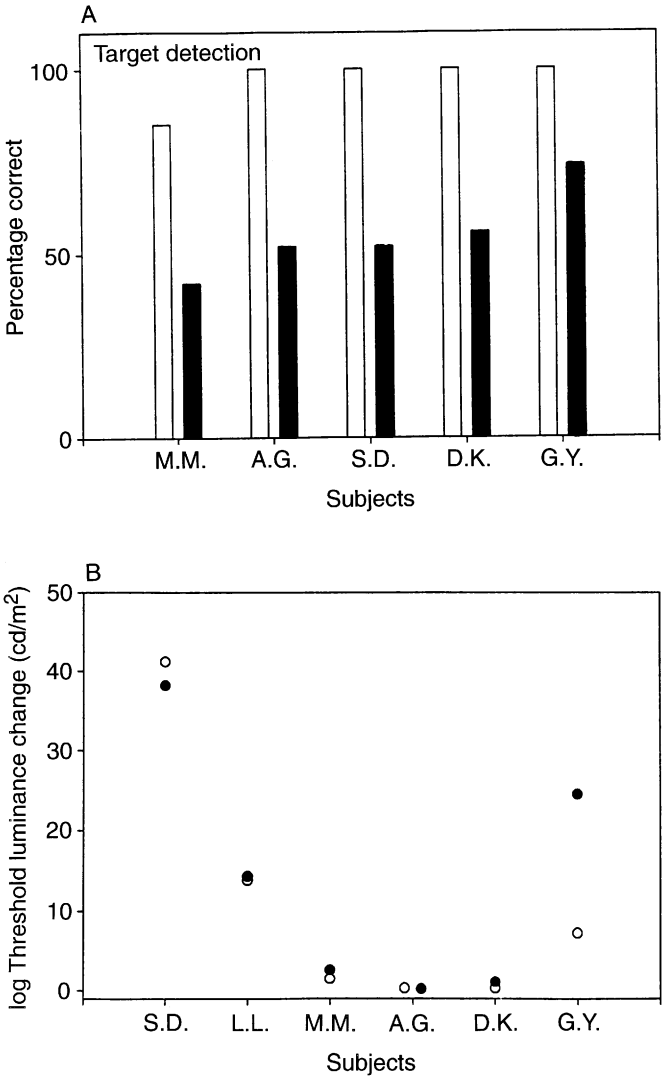

Fig. 13 (**A**) Detection performance measured in the blind temporal hemifield and intact nasal hemifield using a two alternative forced-choice paradigm in four patients with total cerebral hemispherectomy. The target (5° black disc, 9.8 cd/m^2, 200 ms, 48° from midline, on a 76°×56° background of 84.4 cd/m^2) was presented in one of two intervals. After 50 training trials a further 50 trials were given, the results of which are shown. Performance in the intact field was so good that only 20 trials were given. Performance based on chance would be 50% correct. For comparison, data of patient G.Y. who had suffered an occipital lobe lesion are also given. (**B**) Performance of the same subjects, plus one other with hemispherectomy, on a task in which the increment or decrement luminance intensity threshold was measured for 75% correct detection, using a 2AFC paradigm and an adaptive staircase procedure. The stimulus was presented in the temporal hemianopic field of one eye. Open circles, unrestricted view of the display; closed circles, half patch over the temporal half of the viewing eye obscured any direct view of the display. In all five hemispherectomized patients the threshold was almost the same with and without the patch, indicating that the threshold for detecting light scattered on to the intact hemiretina was being measured. In contrast, patient G.Y. had a much lower threshold without the half-patch, i.e. his detection threshold in this condition is not based on light scattered into his normal field. The large differences among subjects with respect to the threshold values is attributable to the deliberate variation in level of adaptation, size of stimulus, and whether an increment or decrement was used [*see* King *et al.* (1996) from where the data are taken, for further details.]

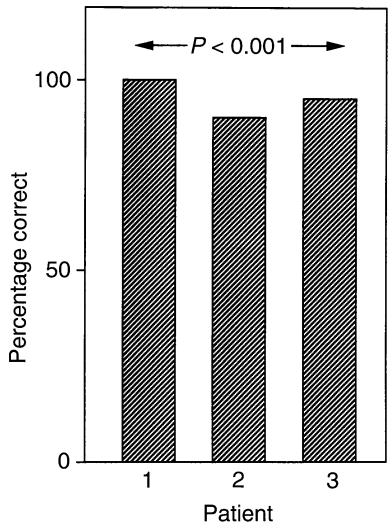

Fig. 14 Motion direction discrimination measured in three patients with bilateral cortical blindness. Horizontal motion was produced in a rotating cylinder covered with vertical black and white stripes in patients 1 and 2, while a moving random-dot pattern (5–8° dark spots on white background, contrast 80%, 30°/s, duration 2 s) projected on a hemicylindrical screen was used in patient 3. Number of trials was 40, 20 and 30, respectively. No OKN was elicited even with durations of 15 s. (Data taken with kind permission from Perenin, 1991.)

The function of blindsight

Visual functions can persist in patients rendered blind by lesions which destroy almost the entire retinal input to the brain. The melatonin suppression in response to exposure to bright light found in patients rendered blind by retinal damage (Czeisler *et al*., 1995) is one example. It demonstrates that a sparse population of retinal ganglion cells which escape damage and project directly to the hypothalamus (Moore *et al*., 1995*a*), can mediate the response in the absence of any other sign of visual processing, and through this response exert influence on the patient's circadian rhythm. In view of the massively divergent projections from the retina into the central visual system (*see* Fig. 11) it is not surprising that lesions in structures as distant from the retina as the primary visual cortex spare a much larger set of visual functions. They were summarized above.

These other pathways and functions can also influence behaviour, although their relevance, particularly that of the higher ones, is less obvious in patients with circumscribed visual field defects who retain normal vision in the remaining visual field. Although the blind field may, for instance, contribute to the stabilization of posture, outside the laboratory the patients use the normal visual field for seemingly all purposes of navigation, prehension, and judgement. How potentially useful the blind functions are would be better demonstrated in patients with complete bilateral cortical blindness. Fortunately, these are rare; unfortunately, their everyday impairment has hardly been studied, and only cortically blind children have been trained to use what may or may not be blindsight to guide their

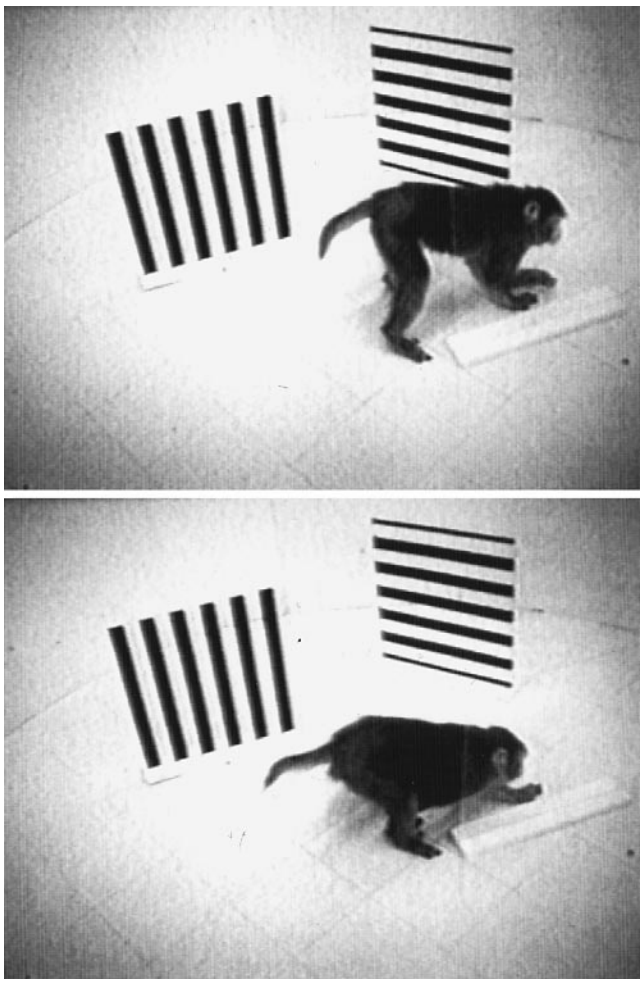

Fig. 15 The bilaterally destriated monkey Helen roamed freely among the objects in the test arena. She would, however, bump into the obstacle made of transparent perspex, as shown, revealing that her navigation was not based on non-visual cues. (Photographs taken from a film by N. Humphrey, and published with his kind permission.)

behaviour (Werth and Möhrenschlager, 1996). Note that rehabilitation aimed at enlarging the residues of the 'seeing' field is another matter because it attempts to train conscious vision, not blindsight (*see* below).

In monkeys, the situation is different. Helen, to cite the most thoroughly studied case with quasi-complete bilateral destruction of striate cortex, was able to orient towards, follow, grasp, detect, localize and discriminate visual objects. Apart from her excellent abilities in formal tests she could move about freely, would not bump into objects and obstacles (unless they were made of clear perspex, demonstrating the visual nature of the navigation, *see* Fig. 15), and often appeared normal in her spontaneous visually guided behaviour as long as she was not alarmed (Humphrey, 1974, 1992).

On learning blindsight

Like other monkeys with occipital lobectomy, Helen had to be taught to use her remaining visual abilities. That learning

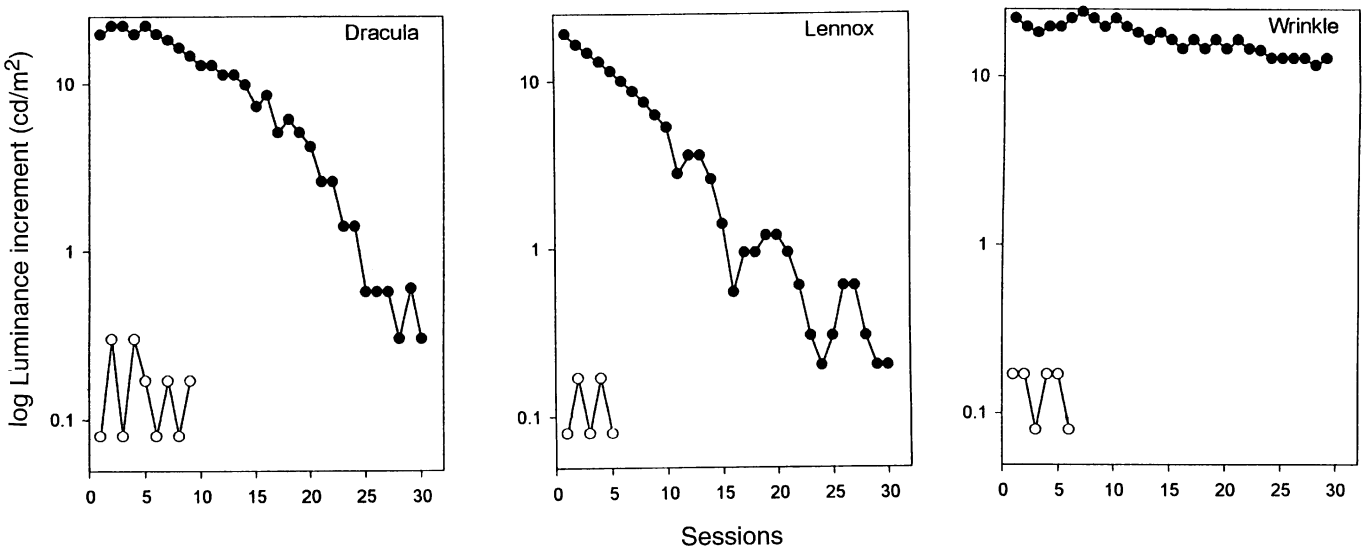

Fig. 16 'Learning curves' for detecting a 200 ms 2° green target in the normal left (open circles) and hemianopic right (closed circles) visual field of three monkeys. Each point represents the result of a single testing session of at least 100 trials. Across sessions the target intensity was reduced until the monkey performed at worse than 80% correct; then intensity was systematically altered using a staircase procedure until performance was stable at about 75% correct. In each case the graph begins at the first session at which the animal failed to score better than 80% correct. Note how rapidly the final increment threshold was reached in the normal field, and how slowly in the hemianopic field. Testing sessions with red and blue targets were interleaved with those for green but are not shown. A similar protracted improvement in performance in the hemianopic field was found for red and for blue. (Our own unpublished data, *see* text for details.)

is important, and does not occur without prompting, was pointed out by Weiskrantz and Cowey (1970), who noted that the scotoma which was behaviourally assessed in a specially designed monkey perimeter appeared to shrink with training. Summarizing their findings, they say: '. . . the ability of every animal to detect a given stimulus intensity within its field defect improved from the first postoperative testing session 1 week after surgery to the end of daily testing 6 to 9 months later . . . The gradual improvement probably did not occur spontaneously, because one animal who was not tested for 2 years postoperatively showed an unchanged picture on retesting, although it did then show gradual improvement with subsequent testing' (Weiskrantz and Cowey, 1970, pp. 243–4). We have observed precisely the same phenomenon with respect to measurements of spectral sensitivity in normal and hemianopic visual fields in monkeys. Detection thresholds in the normal field are rapidly established; those in the hemianopic field do not stabilize until weeks or months of practice (*see* Fig. 16).

Studies in human patients confirm the trainability of blindsight (e.g. Zihl, 1980; Bridgeman and Staggs, 1982; Zihl and Werth, 1984). Indeed, patient F.S. whom we have frequently cited, presents an extreme case of blindsight learning. For several years he showed no statistically significant detection or discrimination in our (P.S.'s) hands (e.g. Stoerig and Pöppel, 1986, patient F; Stoerig, 1987, case 8) but eventually his performance began to improve, and his sensitivity has become as good as we have yet seen, being reduced by no more than 0.3 log units under optimal conditions (Stoerig, 1993*a*, case 4; and *see* Fig. 5).

Learning of this kind requires determination and persistence. This is uncommon in studies of patients; their time is too valuable, and the tests are usually performed without extensive prior training. It is more common in behavioural studies with monkeys who have little choice, require extensive teaching to learn the required responses, and can be tested daily for months or years. The doggedness of the examiner makes a difference as well; with 8 years, Humphrey holds the undisputed record. However, we are getting closer, having now worked with the same monkeys for 6 years, and with some patients for over 10 years. This information is not just anecdotal; if protracted learning is involved, only long-term investigations can reveal the extent of the residual capacities.

The incidence of blindsight

If slow learning is involved, differences in the assessment of whether or not a patient or monkey exhibits blindsight must influence the results, and consequently the estimated incidence of the phenomenon. In monkeys the incidence is high. There are very few cases in which a monkey failed to reach criterion, e.g. two of the five monkeys tested on shape discrimination by Dineen and Keating (1981). Estimates in patients range from one (or four, depending on the criterion) out of 20 (Marzi *et al*., 1986) to 14 out of 22 (Weiskrantz, 1980) to six (or eight, again depending on the criterion) out of 10 (Stoerig, 1987).

Obviously the incidence will depend on several variables, including which function is tested, and what the exact

conditions are. As monkeys are slowly taught to respond to stimuli in their cortically blind field, tasks getting increasingly difficult, they have a different basis from which to start than patients, who are often just tested in one particular test. Lesion factors such as the age at lesion, its position and extent will play a part. In addition, the amount of retrograde degeneration in the retina varies markedly among individual monkeys (our observations) and may vary similarly in patients; more degeneration in the retina and elsewhere could well entail less residual function. The pattern of functions could also vary in accordance with the amount of damage to specialized extrastriate cortical areas. While all these factors apply to both species, more variation may be expected in patients who suffer 'natural' lesions than in monkeys, where the lesion can be restricted to striate cortex with only slight involvement of extrastriate visual areas. However, if extreme cases such as those who have no functional visual cortex left in the lesioned hemisphere are excluded, and if no additional subcortical damage complicates the picture, all of the remaining patient and monkey populations may be able to learn to use blindsight.

Visual field restitution

It is unlikely that we will soon learn whether or not this radical suggestion is true because, hitherto, attempts at training have been aimed at enlarging the seeing part of the visual field. Positive results have been reported by several groups (e.g. Zihl and von Cramon, 1985; Schmielau, 1989; Kerkhoff *et al*., 1994; Kasten *et al*., 1996). Others found an enhancement of the blind functions and/or an improvement of the strategies used to explore space with the remaining functional field, but no enlargement of its seeing portion (Bach-y-Rita, 1983; Balliet *et al*., 1985; Pommerenke and Markowitsch, 1989). According to the positive reports, the visual field enlargement follows certain rules: (i) it depends on where in the visual field the training takes place, being restricted to the trained part; (ii) it commences on the border with the seeing field, shrinking the absolute defect; (iii) it does not depend on the time that has elapsed since the lesion, nor on the age at lesion. While the periphery of the affected visual field is not the first to show functional improvement with training, training commonly being concentrated in the central visual field, such a pattern has repeatedly been observed in spontaneous recovery (Riddoch, 1917). It has also been observed in blindsight patients who during extended periods participated in tests of their visual functions without undergoing any formal training (Stoerig, 1997). Patient D.B. who was studied for several years by Weiskrantz and colleagues (*see* Weiskrantz, 1986) even recovered conscious vision in a quadrant that formed part of his initial incomplete hemianopia, indicating that its striate cortical representation may not have been removed during surgery. Whether or not an enlargement of the seeing field is possible, with or without training, in cases who have lost all their primary visual cortex in one hemisphere, is one of the important questions that need to be addressed.

What is lacking in blindsight?

If patients and monkeys with occipital lesions are capable of using visual information to steer their behaviour to such an extent, what do they lack? Is it just that form discrimination is absent, thresholds are elevated, and the information is treated in some coarser fashion? Or is the absence of conscious vision that characterizes the cortically blind field functionally important as well? How do blindsight patients describe this blindness, how do monkeys indicate that they, too, do not consciously *see* the stimuli they respond to?

'A different kind of nothing': the patients' reports

Only because the patients claimed not to see anything when their visual field defects were stimulated, did the early investigators hold on to the view that all but reflexive visual function is impossible after striate cortical destruction. The lack of a visual experience in response to stimulation of the cortically blind field has been confirmed in many reports on blindsight. The patients 'never reported seeing any targets during the experimental procedure' and, finding the task puzzling, one commented: 'How can I look at something that I haven't seen?' (Pöppel *et al*., 1973, p. 295). The patients 'consistently, repeatedly and firmly said that they did not experience anything' (in relation to stimulus presentation) (Stoerig and Cowey, 1992, p. 431); 'no form of visual impression was ever reported in the hemianopic field' (Magnussen and Mathiesen, 1989, p. 727); 'none of the patients exposed to visual rotation in the blind field was aware of anything happening on this side' (Pizzamiglio *et al*., 1984, p. 96); the patient 'insisted that he saw nothing' (Weiskrantz *et al*., 1974, p. 4); 'none of the subjects ever saw the bars when the stimuli were delivered into their blind field' (Richards, 1973, p. 338), 'over and over [the patient] claimed that he had no sensation whatsoever' (Stoerig *et al*., 1985, p. 596). Patient F.S. to whom we have referred repeatedly has been asked innumerable times 'Do you see anything?'; he always said 'No'. When probed further to find out what makes him say 'yes' in one and 'no' in another trial (concerning target detection), he was happy to conclude that 'maybe it is a different kind of nothing'.

This is what defines the cortically blind field: an absence of any and all sensation, as pointed out by Wilbrand and Sänger (1904). Is the same true in monkeys with occipital lobe destruction? Do they experience the same absence of (conscious) vision?

'A stimulus is no stimulus': the monkeys' reports

Monkeys with striate cortical ablation exhibit a large repertoire of residual visual functions in the affected part of

their visual field. Note that the response-options monkeys have been given have commonly been limited to choosing a spatial position: for target detection they had to touch the illuminated one of a pair of bulbs (Pasik and Pasik, 1971); in tests of wavelength discrimination, they had to pull-in the food-well in front of the rewarded (say red) stimulus (Schilder *et al*., 1972) or to reach below the positive stimulus (Keating, 1979); in spatial frequency or contour or salience discrimination, they had to respond to the grating and not the spatially homogenous stimulus of equal mean luminance (Miller *et al*., 1980; Weiskrantz, 1963) or to the pattern they found most salient (Humphrey, 1974); in visual field perimetry they had to press one lever when a light appeared and a different lever on blank trials (Cowey, 1963); in measurements of their residual sensitivity they had to touch the position at which a stimulus was presented (Cowey and Stoerig, 1995). None of these approaches could elucidate whether or not the monkeys experienced their hemianopic fields as blind, as the patients do.

Working long-term with our three hemianopic monkeys, we had already established their excellent ability to localize stimuli in the impaired hemifield, and had identified the luminance required for localization at ~100% correct (*see* Figs 8 and 10). To see how they would categorize such supra-threshold stimuli, we then introduced a signal-detection paradigm in which the monkeys responded to a stimulus in the normal hemifield by pressing its position, and to a blank trial by touching a constantly present square on the screen that indicated 'no stimulus'. Having mastered this new task, how would they respond to detectable visual targets in the hemianopic field?

All three unilaterally destriated monkeys touched the no-stimulus area when a stimulus that yielded >90% correct performance in the localization paradigm was presented in the hemianopic field (Fig. 17). As they responded in this fashion when the stimulus in the normal hemifield was only 0.3 log above detection threshold set at 75% correct and therefore very dim, we interpret their indicating 'blank trial' in the hemianopic field as evidence for 'phenomenal' blindness, another incidence of species similarity (Cowey and Stoerig, 1995, 1997).

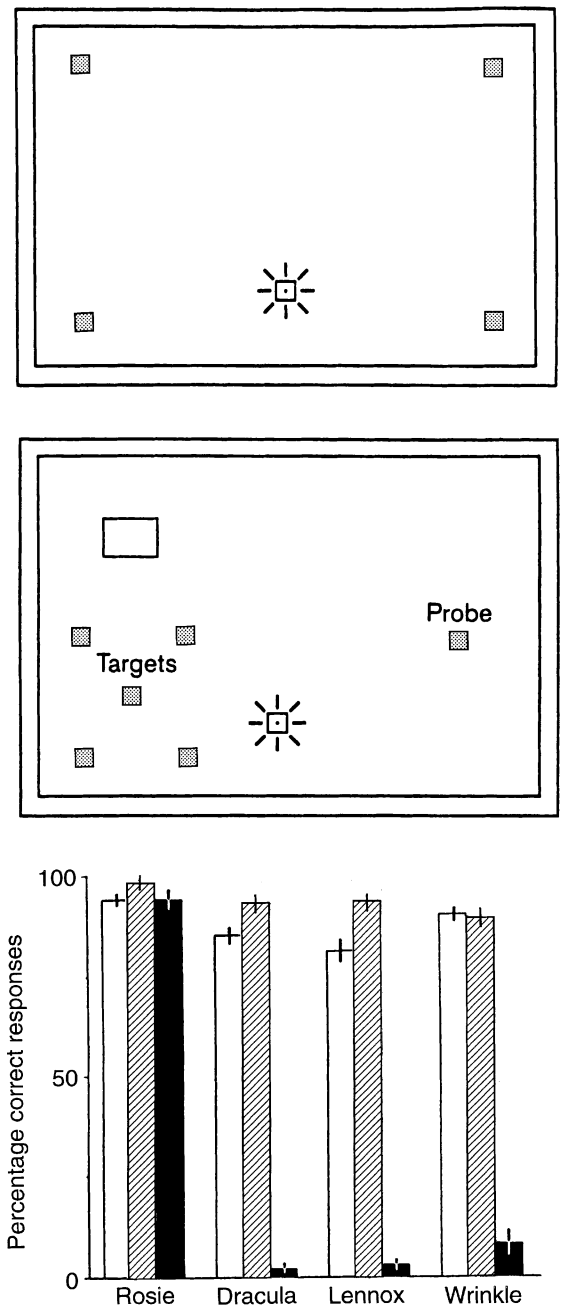

Fig. 17 *Top*: display used to measure detection thresholds. To start a trial, the monkey touched the central light, which was instantaneously followed by a brief 2° flash in one of the four corners of the display. Detection thresholds in the normal and hemianopic field and percentage correct with stimuli that are supra-threshold are shown in Fig. 9. *Centre*: display used to test the responses to stimuli that were detected in either hemifield at better than 90% correct in a signal-detection paradigm. The monkey starts each trial as before. On half the trials, no stimulus follows the pressing of the start light, and the permanently present outlined rectangle must be touched. On most of the other trials, a light appears in one of the five positions in the normal left hemifield and the monkey touches it for a reward. Occasionally the light appears in the right hemianopic field (probe). *Bottom*: the histograms (open, left target; cross-hatched, blank; filled, probe) show that the three hemianopic monkeys, but not the unoperated monkey, respond to probe trials as if they were blanks, i.e. they almost always press the rectangle. [Reprinted from Cowey and Stoerig (1995) with permission from Macmillan Magazines Ltd.]

Consequences of the loss of phenomenal vision

Cortical blindness implies an absence of visual sensation. From the patients' reports and the monkeys' behaviour we conclude that blindsight is characterized by the presence of a repertoire of behaviourally demonstrable visual functions which include implicit and forced-choice responses to stimuli that are not consciously represented. Not seeing the stimuli, as claimed by both patients and monkeys, means that the visual information about contrast, intensity, reflectance and wavelength is not represented in its phenomenal form as borders, brightness, texture and colour. If qualia (the sensations of brightness, texture, colour) are available, vision is 'phenomenal'; if they are absent, vision is 'blind'. The

presence of phenomenal vision distinguishes between cortical blindness and psychic blindness or agnosia; it is absent in the former and present in the latter. As patients with severe visual agnosia lack both object vision and recognition, but still see qualia (consciously, phenomenally), phenomenal vision appears to be the lowest form of conscious vision (Stoerig, 1996).

The commentaries of blindsight patients indicate that they have not only lost phenomenal vision, but also all consciously accessible record of their performance. They cannot judge its quality, they cannot even distinguish between the highly significant performance shown in response to visual stimuli in their blind field and the chance level performance shown in response to stimuli confined to their blind spot (our own observations). 'When shown his results he [patient D.B.] expressed great surprise, and reiterated that he was only guessing' (Weiskrantz *et al*., 1974, p. 4); the patients 'actually believed their performance was completely random' (Pöppel *et al*., 1973, p. 296); the patient 'had no awareness of her above-chance performance' (Magnussen and Mathiesen, 1989, p. 727); the patient 'claimed he was only guessing, and could hardly believe that his performance was above chance' (Stoerig *et al*., 1985; p. 596).

This lack of conscious (as opposed to behaviourally demonstrable but unconscious) access to the processed visual information could well be a consequence of the loss of phenomenal representation. This hypothesis is in accordance with a recent result showing that normal subjects who were unable to see a stimulus in the test field could localize it with high accuracy, but rated their performance as poor (Kolb and Braun, 1995). This approach may open avenues to studying the effects of making stimuli sub-phenomenal in normal observers, thereby ruling out that the loss of conscious access to one's performance is an effect of the patients' lesions which is independent of the loss of phenomenal vision.

At present we do not know whether our blindsighted monkeys also experience themselves as guessing. If monkeys lose conscious access as patients do, the monkey Helen's easily provoked insecurity could be seen as resulting from this lack of a conscious informedness of her visual capacities: 'When she was running around a room she generally seemed as confident as any normal monkey. But the least upset and she would go to pieces: an unexpected noise, or even the presence of an unfamiliar person in the room was enough to reduce her to a state of blind confusion' (Humphrey, 1992, p. 89).

In addition to the insecurity which can reasonably be assumed to result from having-to-guess, Stoerig (1996) has argued that the loss of a phenomenal representation may entail an inability both to construct visual objects and to consciously recognize them for what they are and what they mean. The absence of true form discrimination in blindsight (Weiskrantz, 1987; *see* Fig. 3) favours the notion that cortical blindness encompasses agnosia, as do Humphrey's observations of the super-blindsighted monkey Helen who was 'a monkey who in a sense sees everything but recognizes nothing.' (Humphrey, 1970, p. 334). While agnosia is a higher-order 'psychic' blindness (Seelenblindheit) which spares phenomenal vision and can leave patients with normal visual acuity, brightness discrimination, motion processing, and colour vision (Benson and Greenberg, 1969), cortical blindness (Rindenblindheit) would consequently always include agnosia. This suggestion agrees with William James' original definition: 'Hemianopic disturbance comes from lesion of either [occipital lobe], and total blindness, sensorial as well as psychic, from destruction of both' (James, 1890, p. 47).

If this hypothesis gained support from further evidence, a function not just for conscious vision as such but for its particularly mysterious phenomenal level would follow: Without it, neither conscious recognition nor conscious records are possible.

Phenomenal vision in cortically blind visual fields

Phenomenal vision not generated by direct ocular stimulation

Does the loss of conscious vision that is caused by striate cortical destruction and deafferentation extend to other forms of phenomenal vision? In addition to the coloured, textured, moving objects that constitute our external visual world, we phenomenally see phosphenes and chromatosphenes, after-images and visual dreams, imagined and hallucinated images. These different phenomenal images can be produced in a variety of ways which range from electrical or magnetic stimulation to voluntary or involuntary endogenous activation. Whether, and to what extent, these images disappear in cortically blind visual fields has not been extensively investigated. However, three results are relevant to this issue.

The first regards visual imagery. Farah *et al*. (1992) measured the angle of the mind's eye in a patient who had to undergo unilateral occipital lobectomy. Using Kosslyn's (1978) method for estimating the angular extent of the imagery field, they had the patient judge the distance between herself and imagined objects such as cats, cars and bananas at the point at which they completely filled the internal, mental 'screen'. When comparing the results from before and after the lobectomy, they found that the imagery field had shrunk in tandem with the visual field.

The second result regards after-images. Bender and Kahn (1949) attempted to induce after-images in a patient with a quadrantanopia caused by cerebral infarcts. The after-images that followed central fixation of the stimulus field were stimulus-dependent. With a large square stimulus, the after-image appeared larger and more complete than the seen part of the figure. When instead the large square was sub-divided into four smaller squares, no completion was observed, and the one square that fell into the patient's blind field did not elicit an after-image. Marcel confirmed these results with

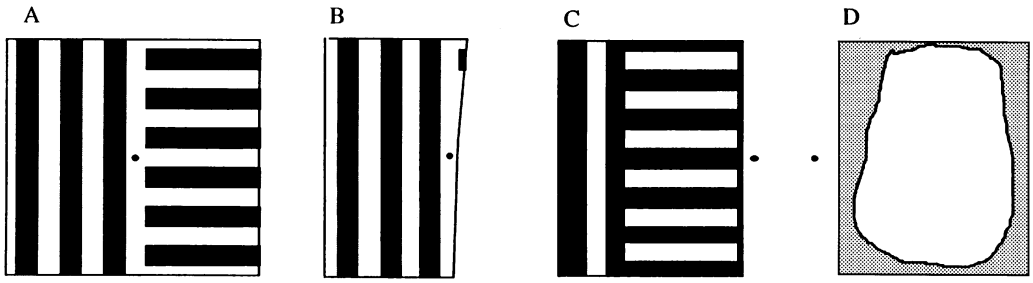

Fig. 18 After-images induced in patient F.S. **A** shows the stimulus field which was 13° in diameter viewed monocularly from 65 cm distance. Black and white represent red and green stripes. (**B**) F.S.'s percept of the stimulus upon central fixation. The after-image that followed central fixation was complementary of the original percept, but occasionally showed completion. (**C**) The after-image resulting when F.S. fixated to the right of the stimulus which then fell entirely into the good hemifield. Its lateral extent appears shrunk, but structure and colour were preserved. (**D**) The after-image that was induced on some trials from the stimulus confined to the field defect. Again, the image is somewhat shrunk but in contrast to the 'normal' after-image, it has lost both colour and structure, and appears as an undefined brightish blob against the white background. (Our own unpublished data, *see* text for details.)

two patients (Marcel, 1983, p. 276; 1997) as did our current investigation of this phenomenon with four patients. All four—they included FS and GY—occasionally reported completion of the after-image induced by a centrally presented stimulus that fell into both normal and cortically blind parts of the visual field. However FS, and only FS, in some instances reported an after-image when only the blind field was stimulated. It lacked both the colour and the structure of the after-image seen in the normal field, and appeared as a grayish blob (*see* Fig. 18). Neither GY nor another patient with an almost complete hemianopia (RH) experienced after-images in this condition.

The third result regards visual hallucinations. Encompassing the full range, from simple phosphenes to geometric colour patterns, to complex scenes featuring several people, they have long been known to occur in patients with complete hemianopia (Seguin, 1886). Customarily, they appear very soon after the cerebral lesion, grow complex within hours or days and disappear within a matter of weeks, to reappear (if at all) in periods of exarcerbated stress. Presumably, they are a consequence of pathological hyper-excitation of the temporal visual cortex which can be caused by occipital lobe lesions (Gloning *et al.*, 1967; Kölmel, 1985). It follows that phenomenal images can, for a limited period of time, appear in the absence of all ipsilesional striate cortex if the extrastriate cortical areas in the temporal lobe become sufficiently activated.

Externally generated phenomenal vision in visual field defects

Is there any evidence that even crude low-level phenomenal images can be caused by visual stimulation after complete destruction of striate cortex? It has been known since the earliest investigations that cortical scotomata can be absolute as well as relative, the latter granting qualitatively reduced phenomenal representations of salient stimulus features, most commonly fast motion and high contrast. Colour vision was commonly lost, and white objects appeared indistinct. That such residual perception often returns after a period of absolute blindness was explained by the 'local cerebral concussion' caused by gunshot wounds and supposed to cause a transient blind field (Holmes, 1918). Relative or amblyopic defects were seen as indicative of incomplete lesions of the primary visual cortex, while absolute defects assumedly produced complete blindness in the corresponding part of the visual field.

This view can accommodate an enlargement of the functional visual field and a recovery from transient blindness, because damaged as opposed to destroyed striate cortical tissue can return to a state of (commonly diminished) function. Can it also accommodate the case of patient G.Y.?

Patient G.Y. suffered a craniocerebral trauma at age 8 years which caused an extended period of 'semi coma'. The lesion destroyed the left primary visual cortex but did not extensively invade the surrounding extrastriate cortical areas. According to his recollection, the resultant hemianopia with small macular sparing in the lower quadrant was absolute at first. However, in a report published 16 years later, it was noted that G.Y. was aware of salient visual events, such as fast moving stimuli in the hemianopic field (Barbur *et al.*, 1980). In the 16 years since, G.Y. has taken part in numerous studies of his residual vision, and has improved with respect to both what he can detect and discriminate and what he is aware of. Compared with the normal hemifield, the loss of sensitivity in the hemianopic field is presently (1996) no larger than ~0.5 log; measured under identical conditions 3 years earlier it was 1.5–2.0 log (P. Stoerig and L. Weiskrantz, unpublished data). In addition, the 'percepts' he reports are much less transient now, and can be evoked reliably over several hours of testing. They still appear to be of very low quality. The patient has occasionally described them in visual terms, for instance as 'like black on black' (personal communication to A.C.), but he still insists that the use of visual terms is for lack of a better alternative because in fact

he does not see the stimulus. He tries hard to find an appropriate description, but then again finds that 'it's impossible, like trying to explain seeing to a blind person' (personal communication to P.S.).

GY can be aware of certain types of stimuli. Fast transients are particularly effective (Weiskrantz *et al*., 1995), whereas colours are invisible and can only be guessed. While this is similar in other patients who have had relative field defects from the onset, or who have developed areas of relative blindess in the course of prolonged testing as described earlier, GY is different in that his left striate cortex is entirely destroyed, or almost so. On our fMRI scans we found no clear evidence even of tissue spared at the occipital pole and corresponding to his macular sparing (R. Goebel, P. Stoerig, L. Muckli and W. Singer, unpublished data). It is therefore possible, but in view of the limits of the technology no more than possible, that GY has developed a type of conscious vision in the absence of ipsilesional striate cortex. Should this be confirmed, GY could teach us the extent of visual function possible after early unilateral destruction of V1.

The extent to which this visual function involves ipsilesional striate cortex, contralesional extrastriate cortex, or even contralesional striate cortex remains to be studied. As long as some functional striate cortex remains, it could conceivably participate in the mediation of visual information from non-topographically corresponding spatial positions. This hypothesis has gained support from a recent report showing that after a few months of wearing prisms that laterally reversed the visual field, the monkey's striate cortical cells now responded to ipsi- as well as to contralateral visual field stimulation (Sugita, 1996) These cells were not selective for either orientation or direction of motion, but responded well to flashes. In view of these extraordinary findings it is feasible that any surviving striate cortex might participate in the processing of information from the blind parts of the visual field. We must await an unequivocal demonstration of conscious vision in the absence of all striate cortex.

Conclusion

The evidence shows that the effects of striate cortical destruction are very similar in man and monkey. The residual visual functions can be studied behaviourally in both species with experimental paradigms that differentiate visually guided behaviour, phenomenal vision, and self-assessment. Maybe in another 55 years it will be possible to plot their functional links and dissociations in a manner that would satisfy Klüver's critical spirit.

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References

Bach-y-Rita P. Controlling variables eliminates hemianopsia rehabilitation results. Behav Brain Sci 1983; 6: 448.

Balliet R, Blood KMT, Bach-y-Rita P. Visual field rehabilitation in the cortically blind? J Neurol Neurosurg Psychiatry 1985; 48: 1113–24.

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

Barbur JL, Watson JD, Frackowiak RSJ, Zeki S. Conscious visual perception without V1. Brain 1993; 116: 1293–302.

Barbur JL, Harlow AJ, Weiskrantz L. Spatial and temporal response properties of residual vision in a case of hemianopia. Philos Trans R Soc Lond B Biol Sci 1994; 343: 157–66.

Bard L. De la persistance des sensations lumineuses dans le champ aveugle des hemianopsiques. Sem Med 1905; 22: 253–5.

Bender MB, Kahn RL. After-imagery in defective fields of vision. J Neurol Neurosurg Psychiatry 1949; 12: 196–204.

Bender MB, Krieger HP. Visual function in perimetrically blind fields. Arch Neurol Psychiatry 1951; 65: 72–9.

Benson DF, Greenberg JP. Visual form agnosia. Arch Neurol 1969; 20: 82–9.

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

Blythe IM, Kennard C, Ruddock KH. Residual vision in patients with retrogeniculate lesions of the visual pathways. Brain 1987; 110: 887–905.

Braak JW ter, Vilet AGM van. Subcortical optokinetic nystagmus in the monkey. Psychiat Neurol Neurochir 1963; 66: 277–83.

Braak JWG ter, Schenk VWG, Vliet AGM van. Visual reactions in a case of long-lasting cortical blindness. J Neurol Neurosurg Psychiatry 1971; 34: 140–7.

Braddick O, Atkinson J, Hood B, Harkness W, Jackson G, Vargha-Khadem F. Possible blindsight in infants lacking one cerebral hemisphere. Nature 1992; 360: 461–3.

Brent PJ, Kennard C, Ruddock KH. Residual colour vision in a human hemianope: spectral responses and colour discrimination. Proc R Soc Lond B Biol Sci 1994; 256: 219–25.

Bridgeman B, Staggs D. Plasticity in human blindsight. Vision Res 1982; 22: 1199–203.

Brindley GS, Gautier-Smith PC, Lewin W. Cortical blindness and the functions of the non-geniculate fibres of the optic tracts. J Neurol Neurosurg Psychiatry 1969; 32: 259–64.

Bullier J, Girard P, Salin P-A. The role of area 17 in the transfer of information to extrastriate visual cortex. In: Peters A, Rockland KS, editors. Cerebral cortex, Vol.10. New York: Plenum Press, 1994: 301–30.

Campion J, Latto R, Smith YM. Is blindsight an effect of scattered light, spared cortex, and near-threshold vision? Behav Brain Sci 1983; 6: 423–86.

Ceccaldi M, Mestre D, Brouchon M, Balzamo M, Poncet M. Autonomie déambulatoire et perception visuelle du movement dans un cas de cécité corticale quasi totale. Rev Neurol (Paris) 1992; 148: 343–9.

Celesia GG, Bushnell D, Toleikis SC, Brigell MG. Cortical blindness and residual vision. Is the 'second' visual system in humans capable of more than rudimentary visual perception? Neurology 1991;41; 862–9.

Cochrane KA. Some tests of residual visual functioning in humans with damage to the striate cortex [Ph.D.thesis]. Oxford: Oxford University, 1995.

Corbetta M, Marzi CA, Tassinari G, Aglioti S. Effectiveness of different task paradigms in revealing blindsight. Brain 1990; 113: 603–16.

Cowey A. The basis of a method of perimetry with monkeys. Q J Exp Psychol 1963; 15: 81–90.

Cowey A. Perimetric study of field defects in monkeys after cortical and retinal ablations. Q J Exp Psychol 1967; 19: 232–45.

Cowey A. Atrophy of retinal ganglion cells after removal of striate cortex in a rhesus monkey. Perception 1974; 3: 257–60.

Cowey A, Stoerig P. Projection patterns of surviving neurons in the dorsal lateral geniculate nucleus following discrete lesions of striate cortex: implications for residual vision. Exp Brain Res 1989; 75: 631–8.

Cowey A, Stoerig P. Blindsight in monkeys [see comments]. Nature 1995; 373: 247–9. Comment in: Nature 1995; 373: 195.

Cowey A, Stoerig P. Visual detection in monkeys with blindsight. Neuropsychologia 1997. In press.

Cowey A, Weiskrantz L. A perimetric study of visual field defects in monkeys. Q J Exp Psychol 1963; 15: 91–115.

Cowey A, Stoerig P, Perry VH. Transneuronal retrograde degeneration of retinal ganglion cells after damage to striate cortex in macaque monkeys: selective loss of Pβ cells. Neuroscience 1989; 29: 65–80.

Cowey A, Stoerig P, Bannister M. Retinal ganglion cells labelled from the pulvinar nucleus in macaque monkeys. Neuroscience 1994; 61: 691–705.

Czeisler CA, Shanahan TL, Klerman EB, Martens H, Brotman DJ, Emens JS, et al. Suppression of melatonin secretion in some blind patients by exposure to bright light [see comments]. N Engl J Med 1995; 322: 6–11.

Dineen J, Keating EG. The primate visual system after bilateral removal of striate cortex. Survival of complex pattern vision. Exp Brain Res 1981; 41: 338–45.

Dineen J, Hendrickson A, Keating EG. Alterations of retinal inputs following striate cortex removal in adult monkey. Exp Brain Res 1982; 47: 446–56.

Farah MJ, Soso MJ, Dasheiff RM. Visual angle of the mind's eye before and after unilateral occipital lobectomy. J Exp Psychol Hum Percept Perform 1992; 18: 241–6.

Feinberg TE, Pasik T, Pasik P. Extrageniculostriate vision in the monkey. VI. Visually guided accurate reaching behavior. Brain Res 1978; 152: 422–8.

Fendrich R, Wessinger CM, Gazzaniga MS. Residual vision in a scotoma. Implications for blindsight [see comments]. Science 1992; 258: 1489–91. Comment in: Science 1992; 258: 1438–9, Comment in: Science 1993; 261: 493–4.

Gloning I, Gloning K, Hoff H. Über optische Halluzinationen. Wien Z Nervheilk 1967; 25: 1–19.

Green DM, Swets JA. Signal detection theory and psychophysics. New York: John Wiley, 1966.

Hackley SA, Johnson LN. Distinct early and late subcomponents of the photic blink reflex: I. Response characteristics in patients with retrogeniculate lesions. Psychophysiology 1996; 33: 239–51.

Heide W, Koenig E, Dichgans J. Optokinetic nystagmus, self-motion sensation and their after effects in patients with occipito-parietal lesions. Clin Vis Sci 1990; 5: 145–56.

Hess RF, Pointer JS. Spatial and temporal contrast sensitivity in hemianopia. Brain 1989; 112: 871–94.

Holmes G. Disturbances of vision by cerebral lesions. Br J Ophthalmol 1918; 2: 353–84.

Holmes G. A contribution to the cortical representation of vision. Brain 1931; 54: 470–9.

Humphrey NK. What the frog's eye tells the monkey's brain. Brain Behav Evol 1970; 3: 324–37.

Humphrey NK. Vision in a monkey without striate cortex: a case study. Perception 1974; 3: 241–55.

Humphrey N. A history of the mind. New York: Simon and Schuster, 1992.

Humphrey GK. The McCollough effect: misperception and reality. In: Walsh W, Kulikowski J, editors: Visual constancies: why things look as they do. Cambridge: Cambridge University Press, 1996. In press.

James W. The principles of psychology. London: Macmillan, 1980.

Kasten E, Schmielau F, Behrens-Baumann W, Wüst S, Sabel BA. Grundlagen der Behandelbarkeit cerebral verursachter Gesichtsfelddefekte. In: Kasten E, Kreutz MR, Sabel BA, editors. Jahrbuch der Medizinischen Psychology 12. Göttingen: Hogrefe, 1996: 165–186.

Keane JR. Blinking to sudden illumination. A brain stem reflex present in neocortical death. Arch Neurol 1979; 36: 52–3.

Keating EG. Effects of prestriate and striate lesions on the monkey's ability to locate and discriminate visual forms. Exp Neurol 1975; 47: 16–25.

Keating EG. Rudimentary color vision in the monkey after removal of striate and preoccipital cortex. Brain Res 1979; 179: 379–84.

Keating EG, Horel JA. Effects of prestriate and striate lesions on performance of simple visual tasks. Exp Neurol 1972; 35: 322–36.

Kerkhoff G, Münssinger U, Meier EK. Neurovisual rehabilitation in cerebral blindness [see comments]. Arch Neurol 1994; 51: 474–81. Comment in: Arch Neurol 1995; 52: 651–2.

King SM, Cowey A. Defensive responses to looming visual stimuli in monkeys with unilateral striate cortex ablation. Neuropsychologia 1992; 30: 1017–24.

King SM, Azzopardi P, Cowey A, Oxbury J, Oxbury S. The role of light scatter in the residual visual sensitivity of patients with complete cerebral hemispherectomy. Vis Neurosci 1996; 13: 1–13.

Kleinschmidt A, Requardt R, Merboldt KD, Frahm J. On the use of temporal correlation coefficients for magnetic resonance mapping of functional brain activation. Individualized thresholds and spatial response delineation. Int J Imag Syst Technol 1995; 6; 238–44.

Klüver H. An analysis of the effects of the removal of the occipital lobes in monkeys. J Psychol 1936; 2: 49–61.

Klüver H. Visual functions after removal of the occipital lobes. J Psychol 1949; 11: 23–45.

Klüver H. Functional significance of the geniculo-striate system. Biol Symp 1942; 3: 253–99.

Kolb FC, Braun J. Blindsight in normal observers [see comment]. Nature 1995; 377; 336–8. Comment in: Nature 1995; 377: 290.

Kölmel HW. Complex visual hallucinations in the hemianopic field. J Neurol Neurosurg Psychiatry 1985; 48: 293–38.

Kosslyn SM. Measuring the visual angle of the mind's eye. Cognit Psychol 1978; 10: 356–89.

Leporè F, Cardu B, Rasmussen T, Malmo RB. Rod and cone sensitivity in destriate monkeys. Brain Res 1975; 93: 203–21.

Leporé F, Ptito M, Cardu B, Dumont M. Effects of striatectomy and colliculectomy on achromatic thresholds in the monkey. Physiol Behav 1976; 16: 285–91.

Magnussen S, Mathiesen T. Detection of moving and stationary gratings in the absence of striate cortex. Neuropsychologia 1989; 27: 725–8.

Magoun HW, Ranson SW. The central path of the light reflex. Arch Ophthalmol 1935; 13: 791–811.

Malmo RB. Effects of striate cortex ablation on intensity discrimination and spectral intensity distribution in the rhesus monkey. Neuropsychologia 1966; 4: 9–26.

Marcel AJ. Conscious and unconscious perception: an approach to the relations between phenomenal experience and perceptual processes. Cognit Psychol 1983; 15: 238–300.

Marcel AJ. Blindsight and shape perception: deficit of visual consciousness or of visual function? Brain 1997; 120. In press.

Marzi CA, Tassinari G, Aglioti S, Lutzemberger L. Spatial summation across the vertical meridian in hemianopics: a test of blindsight. Neuropsychologia 1986; 24: 749–58.

Mendola JD, Dale AM, Liu AK, Reppas JB, Sereno MI, Rosen BR, et al. Topography of the ipsilateral representation in human visual cortex revealed by fMRI. Invest Ophthalmol Vis Sci 1996; 37: 4969.

Mestre DR, Brouchon M, Ceccaldi M, Poncet M. Perception of optical flow in cortical blindness: a case report. Neuropsychologia 1992; 30: 783–95.

Mihailovic LT, Cupic D, Dekleva N. Changes in the numbers of neurons and glial cells in the lateral geniculate nucleus of the monkey during retrograde cell degeneration. J Comp Neurol 1971; 142: 223–9.

Miller M, Pasik P, Pasik T. Extrageniculostriate vision in the monkey. VII. Contrast sensitivity functions. J Neurophysiol 1980; 43: 1510–26.

Milner AD, Heywood CA. A disorder of lightness discrimination in a case of visual form agnosia. Cortex 1989; 25: 489–94.

Mohler CW, Wurtz RH. Role of striate cortex and superior colliculus in visual guidance of saccadic eye movements in monkeys. J Neurophysiol 1977; 40:74–94.

Moore RY, Speh JC, Card JP. The retinohypothalamic tract originates from a distinct subset of retinal ganglion cells. J Comp Neurol 1995a; 352: 351–66.

Moore T, Rodman HR, Repp AB, Gross CG. Localization of visual stimuli after striate cortex damage in monkeys: parallels with human blindsight. Proc Natl Acad Sci USA 1995b; 92: 8215–8.

Morland AB, Ogilvie JA, Ruddock KH, Wright JR. Orientation discrimination is impaired in the absence of the striate cortical contribution to human vision. Proc R Soc Lond B Biol Sci 1996; 263: 633–40.

Nakamura RK, Mishkin M. Chronic 'blindness' following lesions of nonvisual cortex in the monkey. Exp Brain Res 1986; 63: 173–84.

Niida T, Mukuno K, Ishikawa S, Iwai E. Transneuronal retrograde degeneration in adult monkey retina following ablation the striate cortex. In: Iwai E, Mishkin M, editors. Vision, memory, and the temporal lobe. New York: Elsevier 1990; 369–75.

Pasik P, Pasik T. Occulomotor function in monkeys with lesions of the cerebrum and the superior colliculi. In: Bender MB, editor. The occulomotor system. New York: Hoeber 1964: 40–80.

Pasik P, Pasik T. Visual behavior of monkeys with occipital lobe lesions. In: Disturbances of the occipital lobes. Proceedings VIII. International Congress of Neurology, 1965; 3; 127–30.

Pasik T, Pasik P. The visual world of monkeys deprived of striate cortex: effective stimulus parameters and the importance of the accessory optic system. Vision Res 1971; Suppl 3: 419–35.

Pasik T, Pasik P. Extrageniculostriate vision in the monkey. IV. Critical structures for light vs. no-light discrimination. Brain Res 1973; 56: 165–82.

Pasik P, Pasik T. Extrageniculostriate vision in primates. In: Lessell S, van Dalen JTW, editors. Current neuro-ophthalmology 1. Amsterdam: Elsevier 1980: 95–119.

Pasik P, Pasik T. Visual function in monkeys after total removal of visual cerebral cortex. Contrib Sens Physiol 1982; 7: 147–200.

Pasik P, Pasik T, Krieger HP. Effects of cerebral lesions upon

optokinetic nystagmus in monkeys. J Neurophysiol 1959; 22: 297–304.

Pasik P, Pasik T, Schilder P. Extrageniculostriate vision in the monkey: discrimination of luminous flux-equated figures. Exp Neurol 1969; 24: 421–37.

Pasik P, Pasik T, Valciukas JA. Nystagmus induced by stationary repetitive light flashes in monkeys. Brain Res 1970; 19: 313–7.

Payne BR, Cornwell P. System-wide repercussions of damage to the immature visual cortex. [Review]. Trends Neurosci 1994; 17; 126–30.

Peacock JH, Combs CM. Retrograde cell degeneration in diencephalic and other structures after hemidecortication of rhesus monkeys. Exp Neurol 1965; 11: 367–99.

Perenin MT. Visual function within the hemianopic field following early cerebral hemidecortication in man. II. Pattern discrimination. Neuropsychologia 1978; 16: 697–708.

Perenin MT. Discrimination of motion direction in perimetrically blind fields. Neuroreport 1991; 2: 397–400.

Perenin MT, Jeannerod M. Residual vision in cortically blind hemifields. Neuropsychologia 1975; 13: 1–7.

Perenin MT, Jeannerod M . Visual function within the hemianopic field following early cerebral hemidecortication in man. I. Spatial localization. Neuropsychologia 1978; 16: 1–13.

Perenin MT, Rossetti Y. Grasping without form discrimination in a hemianopic field. Neuroreport 1996; 7: 793–7.

Perenin MT, Ruel J, Hecaen H. Residual visual capacities in a case of cortical blindness. Cortex 1980; 16: 605–12.

Perry VH, Cowey A. Retinal ganglion cells that project to the superior colliculus and pretectum in the macaque monkey. Neuroscience 1984; 12: 1125–37.

Perry VH, Oehler R, Cowey A. Retinal ganglion cells that project to the dorsal lateral geniculate nucleus in the macaque monkey. Neuroscience 1984; 12: 1101–23..

Pizzamiglio L, Antonucci G, Francia A. Response of the cortically blind hemifields to a moving visual scene. Cortex 1984; 20: 89–99.

Pommerenke K, Markowitsch JJ. Rehabilitation training of homonymous visual field defects in patients with postgeniculate damage of the visual system. Restorat Neurol Neurosci 1989; 1: 47–63.

Pöppel E. Bridging a neuronal gap. Naturwissenschaften 1985; 72: 599–600.

Pöppel E. Long-range colour-generating interactions across the retina. Nature 1986; 320: 523–5.

Pöppel E, Held R, Frost D. Residual visual function after brain wounds involving the central visual pathways in man [letter]. Nature 1978; 243: 295–6.

Ptito A, Lassonde M, Lepore F, Ptito M. Visual discrimination in hemispherectomized subjects. Neuropsychologia 1987; 25: 869–79.

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

Ptito M, Herbin M, Boire D, Ptito A. Neural bases of residual vision in hemspherectomized monkeys. Prog Brain Res 1996; 112: 381–400.

Rafal R, Smith W, Krantz J, Cohen A, Brennan C. Extrageniculate vision in hemianopic humans: saccade inhibition by signals in the blind field. Science 1990; 250: 118–21.

Richards W. Visual processing in scotomata. Exp Brain Res 1973; 17: 333–47

Riddoch G. Dissociation of visual perceptions due to occipital injuries, with especial reference to appreciation of movement. Brain 1917; 40: 15–57.

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

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

Rovamo J, Hyvärinen L, Hari R. Human vision without luminance-contrast system: selective recovery of the red-green colour-contrast system from acquired blindness. Doc Ophthalmol Proc Ser 1982; 33: 457–66.

Sanders MD, Warrington EK, Marshall J, Weiskrantz L. 'Blindsight': vision in a field defect. Lancet 1974; 1: 707–8.

Schilder P, Pasik T, Pasik P. Extrageniculostriate vision in the monkey. II. Demonstration of brightness discrimination. Brain Res 1971; 32; 383–98.

Schilder P, Pasik P, Pasik T. Extrageniculostriate vision in the monkey. III. Circle vs triangle and 'red vs green' discrimination. Exp Brain Res 1972; 14: 436–48.

Schmielau F. Restitution visueller Funktionen bei hirnverletzten Patienten: Effizienz lokalisationsspezifischer sensorischer und sensomotorischer Rehabilitationsmaβnahmen. In: Jacobi P, editor. Jahrbuch der medizinischen Psychologie 2. Leipzig: Hogrefe 1989: 115–26.

Segraves MA, Goldberg ME, Deng SY, Bruce CJ, Ungerleider L, Mishkin M. The role of striate cortex in the guidance of eye movements in the monkey. J Neurosci 1987; 7: 3040–58.

Seguin EG. A contribution of the pathology of hemianopsis of central origin (cortex-hemianopsia). J Nerv Ment Dis 1886; 13: 1–38.

Solomon SJ, Pasik T, Pasik P. Extrageniculostriate vision in the monkey. VIII. Critical structures for spatial localization. Exp Brain Res 1981; 44: 259–70.

Stoerig P. Chromaticity and achromaticity: Evidence for a functional differentiation in visual field defects. Brain 1987; 110: 869–86.

Stoerig P. Spatial summation in blindsight. Vis Neurosci 1993*a*; 10: 1141–9.

Stoerig P. Sources of blindsight. Science 1993b; 261: 493–4.

Stoerig P. Varieties of vision: from blind responses to conscious recognition. Trends Neurosci 1996; 19: 401–6.

Stoerig P. Blindsight. In: Huber A, Koempf D, editors. Klinische Neuroophthalmologie. Stuttgart: Thieme-Verlag, 1997. In press.

Stoerig P, Cowey A. Residual target detection as a function of stimulus size. Brain 1989b; 112: 1123–39.

Stoerig P, Cowey A. Wavelength sensitivity in blindsight. Nature 1989a; 342: 916–8.

Stoerig P, Cowey A. Increment-threshold spectral sensitivity in blindsight. Brain 1991; 114: 1487–512.

Stoerig P, Cowey A. Wavelength discrimination in blindsight. Brain 1992; 115: 425–44.

Stoerig P, Fahle M. Apparent motion across a scotoma: an implicit test of blindsight. Eur J Neurosci 1995. Suppl 8: 76.

Stoerig P, Pöppel E. Eccentricity-dependent residual target detection in visual field defects. Exp Brain Res 1986; 64: 469–75.

Stoerig P, Hübner M, Pöppel E. Signal detection analysis of residual vision in a field defect due to a post-geniculate lesion. Neuropsychologia 1985; 23: 589–99.

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

Sugita Y. Global plasticity in adult visual cortex following reversal of visual input. Nature 1996; 380: 523–6.

Swets JA, Pickett RM. Evaluation of diagnostic systems. New York: Academic Press: 1982.

Tomaiuolo F, Ptito A, Paus T, Ptito M. Spatial summation across the vertical meridian after complete or partial hemispherectomy [abstract]. Soc Neurosci Abstr 1994; 20: 1579.

Torjussen T. Residual function in cortically blind hemifields. Scand J Psychol 1976; 17: 320–3.

Tusa RJ, Zee DS, Herdman SJ. Effect of unilateral cerebral cortical lesions on ocular motor behavior in monkeys: saccades and quick phases. J Neurophysiol 1986; 56: 1590–625.

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

van Buren JM. Trans-synaptic retrograde degeneration in the visual system of primates. J Neurol Neurosurg Psychiatry 1963; 26: 402–9.

van Hof-van Duin J, Mohn G. Optokinetic and spontaneous nystagmus in children with neurological disorders. Behav Brain Res 1983; 10: 163–75.

Verhagen WIM, Huygen PLM, Mulleners WM. Lack of optokinetic nystagmus and visual motion perception in acquired cortical blindness. Neurophthalmology 1997. In press.

Walker AE. The primate thalamus. Chicago: University of Chicago Press, 1938.

Warrington EK. The completion of visual forms across hemianopic field defects. J Neurol Neurosurg Psychiatry 1962; 25: 208–17.

Weiskrantz L. Contour discrimination in a young monkey with striate cortex ablation. Neuropsychologia 1963; 1: 145–64.

Weiskrantz L. Behavioural analysis of the monkey's visual nervous system. [Review]. Proc R Soc Lond B Biol Sci 1972; 182: 427–55.

Weiskrantz L. Varieties of residual experience. Q J Exp Psychol 1980; 32: 365–86.

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

Weiskrantz L. Residual vision in a scotoma: a follow-up study of 'form' discrimination. Brain 1987; 110: 77–92.

Weiskrantz L. Outlooks for blindsight: explicit methodologies for implicit processes. [Review]. Proc R Soc Lond B Biol Sci 1990; 239: 247–78.

Weiskrantz L. Blindsight revisited. Curr Opin Neurobiol 1996; 6: 215–20.

Weiskrantz L, Cowey A. Filling in the scotoma: a study of residual vision after striate cortex lesions in monkeys. In: Stellar E, Sprague JM, editors. Progress in physiological psychology 3. New York: Academic Press 1970: 237–60.

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

Weiskrantz L, Harlow A, Barbur JL. Factors affecting visual sensitivity in a hemianopic subject. Brain 1991; 114: 2269–82.

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

Weller RE, Kaas JH. Parameters affecting the loss of ganglion cells of the retina following ablations of striate cortex in primates. Vis Neurosci 1989; 3: 327–42.

Werth R, Möhrenschlager M. Spontanerholung und Wiederherstellung von Sehfunktionen bei cerebral blinden Kindern. In: Kasten E, Kreutz MR, Sabel BA, editors. Jahrbuch der Medizinischen Psychologie 12. Göttingen: Hogrefe, 1996: 195–203.

Wilbrand H, Sänger A. Die Neurologie des Auges, Vol.III. Wiesbaden: J.F.Bergmann, 1900.

Yukie M, Iwai E. Direct projection from the dorsal lateral geniculate nucleus to the prestriate cortex in macaque monkeys. J Comp Neurol 1981; 201: 81–97.

Zihl J. 'Blindsight': improvement of visually guided eye movements by systematic practice in patients with cerebral blindness. Neuropsychologia 1980; 18: 71–7.

Zihl J, von Cramon D. Visual field recovery from scotoma in patients with postgeniculate damage. Brain 1985; 108: 335–65.

Zihl J, Werth R. Contributions to the study of 'blindsight' II. The role of specific practice for saccadic localization in patients with postgeniculate visual field defects. Neuropsychologia 1984; 22: 13–22.

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