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specific synaptic connections required to generate the hippocampal theta rhythm; it was not intended to include all of the modulatory influences that undoubtedly contribute to this phenomenon.

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TINS

The neurobiology of blindsight

Alan Cowey and Petra Stoerig

Some patients can respond to visual stimuli presented within their clinically absolute visual field defects that have been caused by partial destruction of striate cortex. This puzzling phenomenon of looking, pointing, detecting and discriminating without seeing has been called blindsight, and has fascinated philosophers and neuroscientists alike as a spotlight on the nature of unconscious or covert awareness, and the means it provides of studying the visual information carried by pathways other than the major route through the striate cortex.

In 1886, Ferrier wrote that 'it is manifestly absurd to establish an antithesis between "cortical" and any other form of blindness'[1]. His verdict, generally accepted by clinical neurologists for almost a century, implies that the pathways from the eye that remain anatomically intact after a striate cortical lesion do not transmit visual information. Indeed, neurological investigations were almost unanimous in showing that 'severe lesions of the visual cortex produce complete blindness in the corresponding portions of the visual field'[2] and now, as then, asking the patients whether they perceive anything presented in the affected portions yields a firm denial. It was only when 'forced-choice' behavioural methods were applied to explore possible residual visual functions that a different picture emerged for, despite their blindness, the patients might be able to look towards stimuli presented in their field defects[3,4], to localize them by pointing[5], and to detect and discriminate movement[6–8]. One patient with complete cortical blindness was able to follow a large moving striped display with his eyes, despite disclaiming any visual sensation that might explain his visual tracking[9]. The patients can detect and discriminate flicker[6,8], orientation[4,10,11] and wavelength[12] (see Refs 13, 14 for recent reviews). Their pupils continue to respond to changes in light level, pattern and contrast[14] and, when asked to reach for visual targets, two patients adjusted their grasp so that it matched the shape and size of the unseen object[15]. Still more astonishingly, they could even use the meaning of unseen words flashed in their blind fields in order to select between pairs of words subsequently presented in the intact field[16].

Not surprisingly, these residual capacities, paradoxically demonstrable despite the patients' inability to create a conscious representation of the stimuli, were sometimes received with scepticism. Reservations included the possibility that the patients' striate cortex was merely damaged, not destroyed, or that they responded to light scattered from the stimulus onto the intact retina, or that they employed a lax criterion for detection that was very different from the one used in the normal field[17]. With at least some patients, however, all three reservations are groundless. First, blindsight can be demonstrated even in patients in whom the striate cortex[4], or even an entire cerebral hemisphere[5,10,18], has been surgically removed. Second, when a stimulus that is detected and 'identified' in blindsight is presented on the natural blind spot, it becomes undetectable – despite the fact that the optic disc normally reflects and scatters more light than the rest of the retina[11,19,20]. Third, a stimulus presented in the blind field and to which the patient is not even asked to respond can influence the response, e.g. its speed, to a companion stimulus presented in the intact field[21–23].

The realization that blindsight is a genuine phenomenon has produced an upsurge of interest in two related matters, namely the apparently indispensable role of the striate cortex in conscious visual perception, and the role in visual processing of pathways other than those involving the striate cortex. What are the pathways subserving blindsight? Is it just one pathway or might all the secondary pathways contribute, albeit in different ways, as they do in normal vision? Can these functions be used for visual field rehabilitation and, if so, what are the underlying neural mechanisms? What is it that renders striate cortex indispensable for conscious visual perception? Is it possible that the information transmitted by some pathways cannot be consciously perceived even in normal vision?

Multiplicity: pathways to vision

The known pathway from the eyes to their first target areas in the brain are shown schematically in Fig. 1. Every textbook on vision describes the projection, exceeding a million fibres per eye, to the dorsal lateral geniculate nucleus (dLGN). The projection (approximately 100 000–150 000 fibres) to the

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superior colliculus (SC) is usually mentioned as well, but the others receive scant attention, despite the fact that many of them, as judged from electrophysiological recordings, transmit information about the position, size and movement of visual stimuli (e.g. Ref. 24). Given that at least some of them appear to be involved in reflexive noncognitive responses, e.g. the ventral lateral geniculate nucleus (vLGN) in the detection of light levels and adjustments in the pupil, or the three accessory optic nuclei together with the nucleus of the optic tract (NOT) in the detection of self motion and the subsequent postural adjustments to flow fields (see Ref. 24 for review), they might seem excellent candidates for at least some residual visual functions. The pathways most often invoked in blindsight are the retinocollicular projection that reaches extrastriate cortical visual areas via the pulvinar nucleus, the direct geniculoextrastriate cortical projection and, although it can only contribute in cases of incomplete striate cortical damage, the remnants of the optic radiation and striate cortex whose imprecise topography might allow some information from the field defect to be transmitted to remaining striate cortex.

Residual visual functions in monkeys

Monkeys with striate cortical ablations have long been known to exhibit residual visual functions in their field defects (e.g. Refs 25, 26), and might help us to understand the visual role of the various pathways. However, one reason why little consideration has been given to the possible contribution of many pathways is that the problem appeared to be solved when Mohler and Wurtz[27] showed that monkeys in which a small part of the striate cortex had been removed could still direct their eyes to a spot of light presented within the visual field defect, but that following subsequent removal of the part of the superior colliculus concerned with the same region of the eye, the monkeys now behaved as if totally insensitive within the field defect. However, even earlier, the Pasiks and collaborators in their extensive studies on blindsight (see Ref. 25 for review) had demonstrated that the ability of monkeys to detect which of two small targets was illuminated survived total bilateral removal of striate cortex, but was additionally impaired more by subsequent destruction of the lateral pretectum (with interruption of the accessory optic system) than of the SC. Furthermore, the same authors showed that additional removal of extrastriate visual cortical areas similarly exacerbated the impairments of the monkeys, suggesting that nonstriate cortical input to these areas might be important, whether it comes indirectly from the colliculus or directly from the LGN. The latter possibility has recently been supported by the demonstration that saccadic eye movements to a range of visual targets in the field defect are abolished by destruction of all layers of the LGN, even though neurones in the retinotopically corresponding part of the SC retain their visual receptive fields[28].

In assessing the contribution to blindsight of these surviving interconnected subsystems, we face the problem that not only are their physiological properties only partially understood, but that they also might be altered by the degeneration caused by a striate cortical lesion.

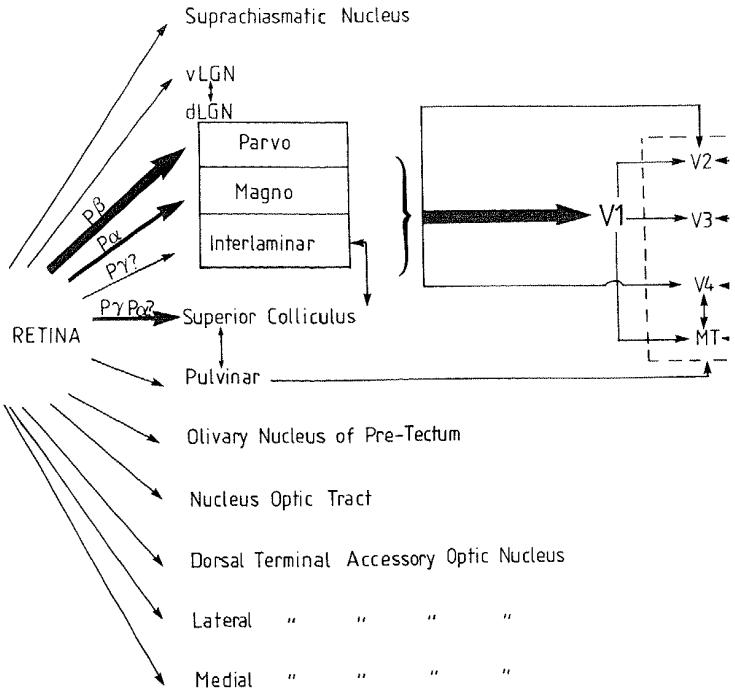

Fig. 1. *The known pathways from the eye into the brain, together with initial cortical projections. The scheme excludes the extensive further conn tions between the initial cortical visual areas and the many further visual ar The thicker arrows indicate the heaviest and most studied projections. classes of retinal ganglion cells projecting to most of the brainstem targets unknown.*

Retinal ganglion cells: before and after striate cortical damage

The primate alpha (Pα or M) cells project to the two magnocellular layers of the dLGN[29]. Like their postsynaptic target neurones, they have spatially opponent, chromatically nonopponent (broad-band) physiological properties, i.e. the visual receptive field is circular and light, irrespective of the wavelength and has opposite effects (excitation or inhibition) in the centre and surround of the receptive field. These cells are therefore exquisitely sensitive to borders between areas of different brightness, but are much less sensitive to borders between areas of equal brightness but different colour. The primate beta (Pβ or P) cells and their targets in the four parvocellular layers of the dLGN[29] are chromatically opponent, i.e. the response to light is excitatory or inhibitory depending on its wavelength. In addition, most of these cells are spatially opponent and their small receptive field centres make them good candidates for the resolution of fine spatial details. The third class, the primate gamma (Pγ) cells, project to the midbrain[30] and are physiologically heterogeneous. Unlike the other two classes, their receptive fields often lack a clear centre–surround arrangement. They are particularly sensitive to motion, and colour opponency has not been reported[31]. However, their target cells in the SC show a wavelength bias[32] that might come (it is not yet known) from the retina and/or from the visual cortex[33]. In the normal eye, the Pα and Pγ cells each form about 10% and the Pβ cells about 80% of the retinal ganglion cell population[29,30].

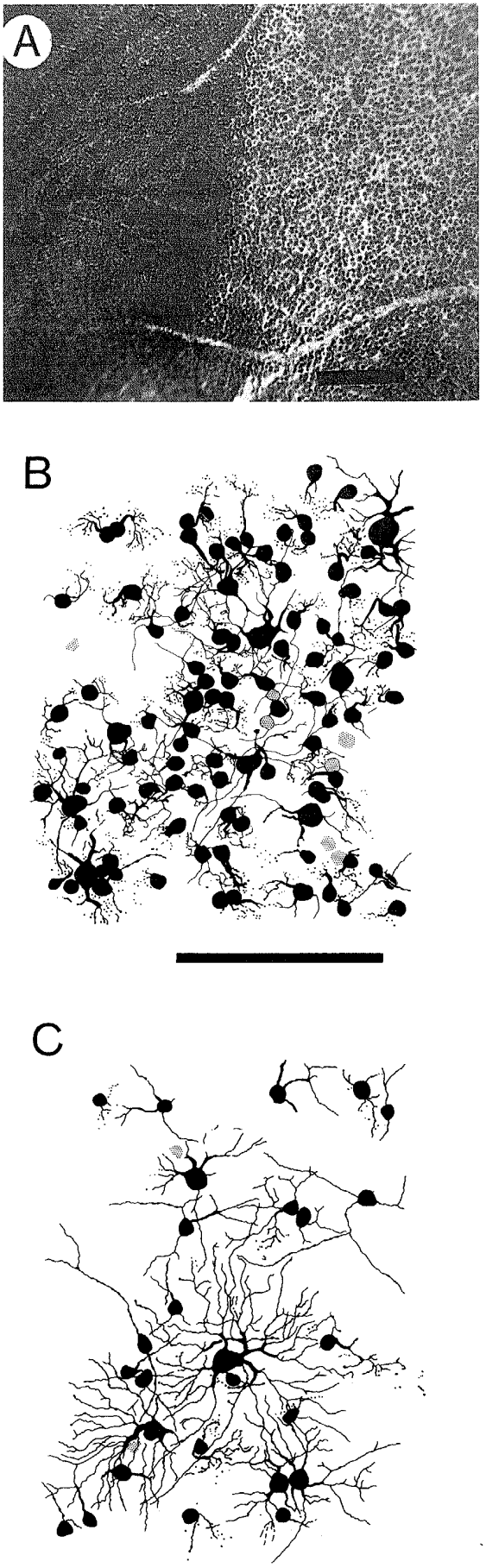

Fig. 2. (A) *Photomicrograph of the flattened Nissl-stained central retina, just above the fovea, of the right eye of a macaque monkey eight years after the left striate cortex was removed. Note the extensive loss of retinal ganglion cells to one side of the vertical retinal meridian.* **(B, C)** *Drawings of HRP-filled cells from mirror image positions in the normal (B) and degenerated (C) hemiretinae of the other eye, illustrating the huge reduction in the density of small colour-opponent Pβ cells in the degenerated half. At least half of the small cells that remain are Pγ cells, projecting to the midbrain. The shaded cells were too poorly labelled to be characterized. Scale bars: A, 1 mm; B,C, 200 μm.* (Modified, with permission, from Ref. 40.)

These numbers apply in the normal retina. Are they altered by striate cortical damage? When part of the striate cortex is destroyed, the retinotopically corresponding region of the dLGN degenerates within weeks. Transneuronally, and more slowly, the retrograde degeneration affects the retinal ganglion cell layer. Massive cell loss has been reported in humans[34] and Old World monkeys[35,36], which depends on both the age at which the lesion occurred and its duration[37–39]. Eight years after unilateral destruction of striate cortex in adolescent monkeys, the reduction ranged from 50% to 80%, the central part of the affected hemiretina being more affected than the periphery. Figure 2A shows the retina of a unilaterally destriated monkey, with the vertical meridian in the centre and the degenerated half clearly distinct from the unaffected half that projected to the other hemisphere. Also shown are drawings made from the normal (2B) and degenerated (2C) hemiretina of the animal's other retina, which was labelled with HRP from the optic nerve. They not only demonstrate the difference in density, but also the selectivity of the depletion. Classification of cells in both hemiretinae showed that only the Pβ cells are victims of transneuronal degeneration[40]. However, a number of Pβ cells, roughly equivalent to the unaffected numbers of Pα and Pγ cells, survive in apparently good health. Where do the survivors project?

Pathways to blindsight

The only well-established projection zone of Pα and Pβ cells is the dLGN, Pα cells projecting to the magnocellular and Pβ cells to the parvocellular portion (see Fig. 1). Although an eye injection followed by autoradiography showed more prominent retinal input to the magnocellular portion after long-standing removal of striate cortex[38,39], both portions degenerate massively, the population of projection neurones being reduced to less than 1% within a few months[41]. However, a small number of projection neurones survive permanently in the areas that correspond topographically to the cortical lesion. One possible reason is that they depart from the otherwise strict point-to-point projection of the dLGN onto the striate cortex, either by providing a sustaining collateral to the striate cortex remote from the primary terminal zone, or even by confining their terminals to an anomalous location. There is anatomical evidence for the first possibility in the striate cortex of monkeys[42–44] and, to test whether this is the reason for the survival, HRP was placed into the striate cortex immediately adjacent to a lesion that had been

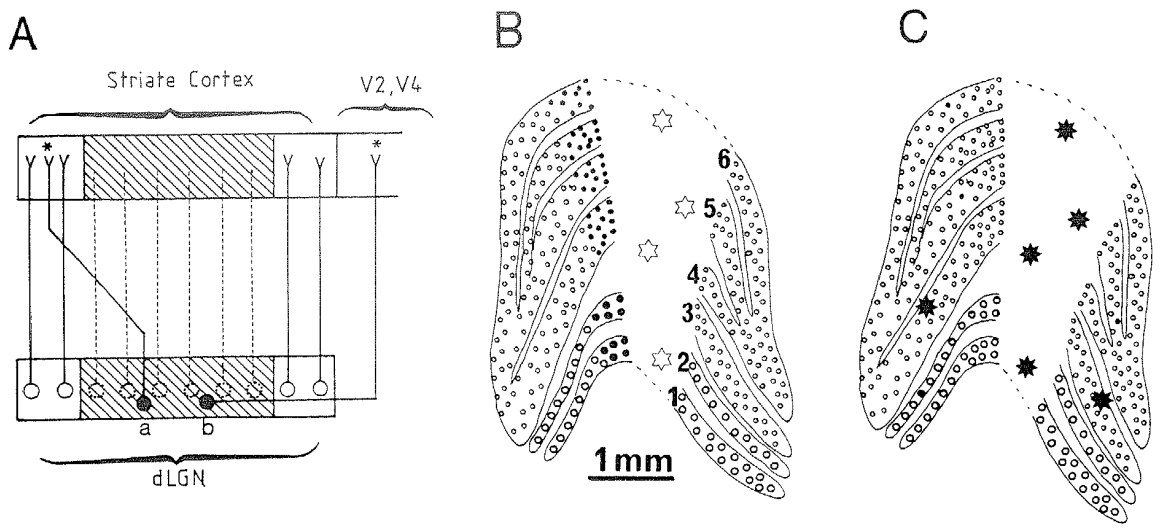

Fig. 3. (A) *A diagram of the predominantly point-to-point orderly projection of neurones from the dLGN to the striate cortex. The striped regions indicate a cortical lesion and the resulting area of degeneration in the dLGN. If projection neurone* **a** *survives because it projects to an anomalous position in the striate cortex, it should be possible to label it from that position (asterisk). If neurone* **b** *survives because it projects to extrastriate cortex, it should be possible to label it from there (asterisk in V4).* **(B)** *A diagram of the result of placing HRP into the striate cortex adjacent to a long-standing lesion. No projection neurones (stars) are labelled within the degenerated region. The label (black cells) is confined to normal parvocellular and magnocellular regions in the appropriate topographical position.* **(C)** *A diagram of the results of placing HRP in V4. Scattered surviving projection neurones (black stars) are now labelled throughout the nucleus, including the degenerated region. Numbers in (B) indicate layers: 1–2, magnocellular; 3–6, parvocellular.*

inflicted several years earlier[45]. As shown schematically in Fig. 3, the neurones alongside the degenerated sector of the dLGN showed dense retrograde labelling, but none of the surviving neurones *within* the area of degeneration was stained. This pattern was found at all levels of the dLGN in three monkeys, showing that the overlap that exists in the normal geniculostriate cortical projection is not responsible for the survival of the remaining dLGN projection neurones.

What other projection preserves them? The dLGN was thought to project only to the striate cortex, until a series of papers showed that a few thousand of its projection neurones projected directly to extrastriate visual cortex[46–49]; it could be these neurones that survive destruction of the striate cortex. This possibility was examined by placing HRP in extrastriate cortex, chiefly visual area V4 of the prelunate gyrus, following long-standing removal of part or all of the striate cortex in one hemisphere[45,46,50]. As shown in Fig. 3, many of the scattered large neurones in the degenerated dLGN (up to 50%) were retrogradely labelled[45], implying that it is indeed their direct projection to extrastriate cortex that sustains them. After cortical damage in infancy, they even have conspicuously enlarged cell bodies and dendrites that suggest an enlarged terminal area in V4 and a greater retinorecipient zone in the dLGN[50].

This direct geniculocortical projection could therefore mediate residual visual functions, provided it received direct or indirect retinal input. This problem was first addressed by Dineen *et al.*[51], who identified both retinal and nonretinal synaptic terminals within the degenerated dLGN on the basis of ultrastructural criteria. Unfortunately it was not possible to establish whether the postsynaptic targets of these surviving terminals included either surviving projection neurones or interneurones. In a recent re-examination of this problem, surviving dLGN projection neurones were retrogradely labelled by injections of HRP into visual area V4, while retinothalamic terminals were anterogradely labelled by an intraocular injection of HRP[52], so that retinogeniculate terminals could be identified both on the basis of their characteristic ultrastructure[53] and by the presence of HRP reaction product in the terminal boutons. In the normal dLGN, the great majority of retinal terminals were in direct synaptic contact with the dendrites of projection neurones. By contrast, in the degenerated dLGN, every one of 184 identified terminals was in contact with the dendrites of a GABA-immunopositive interneurone and it was these interneurones that were presynaptic to the surviving projection neurones. In other words, there is anatomical evidence for a direct retinal input to a degenerated dLGN. In addition, we also found that the retrogradely labelled projection neurones received synaptic inputs that are characteristic of excitatory synapses and of the projection from the SC[54]. It follows that there is both a direct and indirect route from the eye to a degenerated dLGN and on to extrastriate cortex. The physiological properties of this projection might be rather unusual.

Although the other secondary pathways have not been explicitly studied after striate cortical damage, the projection to the SC appears anatomically normal, and the same is to be expected from the pathways to the NOT and accessory optic system. Whether this is coupled with a physiological normality has yet to be established.

Function: neurophysiology and behaviour

In primates it is not known which class of retinal ganglion cell provides the input to all the target zones shown in Fig. 1. Speculating on the basis of what is

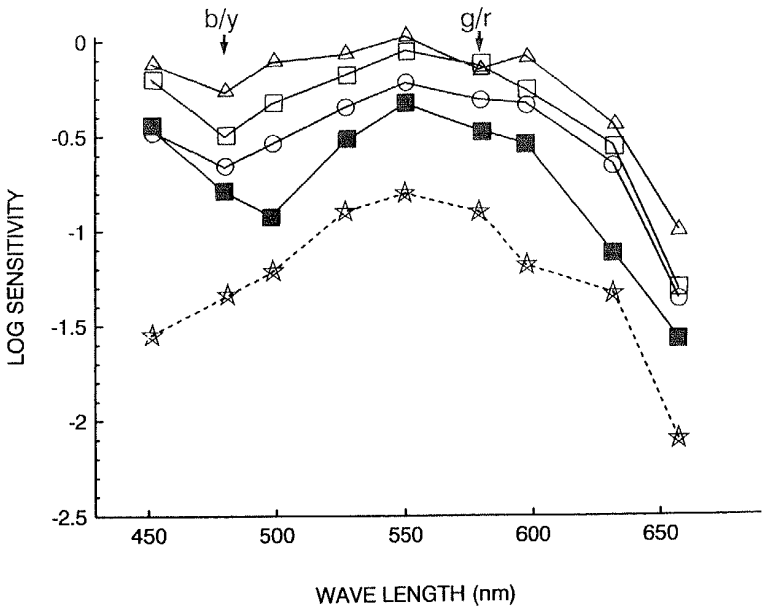

Fig. 4. *The four curves at the top show monocular light-adapted, increment-threshold spectral sensitivity at an eccentricity of 10°, measured by presenting narrow-band 2°, 200 ms coloured stimuli on a white 32 cd m^{-2} background. Note the peaks and troughs, the positions of which are characteristic of blue–yellow (b/y) and green–red (g/r) colour-opponent processing. The top two curves are from the nasal (△) and temporal (□) field of a normal observer. The bottom two curves show sensitivity in the normal nasal hemifield (○) and the blind hemifield (■) of a patient with blindsight. The bottom curve is from the same normal subject but, in order to stimulate the broad-band luminance channel preferentially, measurements were made at an eccentricity of 30° with a smaller target (44') flickering at 5 Hz. Note that this curve shows no increase in sensitivity at the blue end of the spectrum. For ease of comparison, the luminance curve has been lowered vertically by 0.7 log units.* (Modified from Refs 60, 67.)

known from anatomy, physiology and behaviour in other mammals, and from clinical work, input from Pα retinal ganglion cells appears almost omnipresent and input from Pγ cells is possibly almost as widely distributed (although it is weak in the dLGN, being restricted to the interlaminar zones). The Pβ cells seem to be the most restricted class; their only well-established target is the parvocellular dLGN layers, although further inputs to the vLGN and to the pulvinar nucleus, in whose retinorecipient zone colour-opponent cells have been reported[55], are conceivable.

Relating the functional properties of ganglion cell classes to these pathways and to blindsight, it is likely that the anatomically normal Pα and Pγ cell populations, together with their numerous targets, mediate a number of residual visual functions. As in normal vision[56], they must certainly be involved in the processing of motion in visual field defects[6–9]. In addition, Pα and Pγ pathways are probably involved in stimulus localization. For saccadic localization especially, the SC and its projections, including that to the interlaminar dLGN layers (see Ref. 57 for review) seem to play a prominent role (see, for example, Refs 58, 59).

What about the depleted but still substantial population of colour-opponent Pβ ganglion cells? In normal vision, Pβ cells and their pathways are known to be important for the resolution of fine spatial detail, and for processing wavelength information. It is known from psychophysical studies that certain experimental conditions – e.g. white photopic background, long presentation time, large stimulus – favour the colour-opponent channels[59], which presumably correspond to the Pβ pathways. Using such conditions in experiments on wavelength processing in blindsight has recently revealed evidence for colour-opponent processes in the field defects[60]. Figure 4 shows the photopic spectral sensitivity curves of a patient with a unilateral visual field defect, compared with those of a normal observer. It can be seen that the curve from the blind field is only moderately reduced in sensitivity, and that the characteristic peaks and troughs are present, indicating inhibitory interactions in the red–green and blue–yellow opponent channels[60]. In addition, such patients could discriminate, by forced choice, targets of different colours whose luminance was matched on the basis of spectral sensitivity curves measured under identical conditions[61]. The curve for the blind field certainly does not resemble that of the broad-band luminance channel, shown at the bottom of Fig. 4 and measured in the normal observer by using a small flickering stimulus in the peripheral retina. These results suggest that the Pβ cells and their pathways also contribute to blindsight, and that adaptation level, the response requirements and the nature of the visual stimulus collectively determine which subsystem responds preferentially, as is the case in normal vision.

Whether the participation of extrastriate visual cortical areas – via the few surviving projection neurones in the dLGN and/or the pulvinar nucleus – is necessary for wavelength processing in blindsight is still unclear, but might be revealed by testing spectral sensitivity and colour discrimination in hemispherectomized patients. Extrastriate cortical areas might also contribute to other aspects of blindsight, because sensitivity to motion *per se* is preserved after hemispherectomy, whereas sensitivity to its direction, present in field defects from circumscribed striate cortical damage, is lost. But which extrastriate areas remain functional after striate cortical damage? Visual sensitivity in area V2 in anaesthetized monkeys is temporarily abolished by cooling the striate cortex[62]; again under anaesthesia, the inferotemporal cortex is visually unresponsive after striate cortical ablation[63], and the same was said about area MT (cited in Ref. 63). However, recent reports show that about 50% of the neurones in MT, referred to as the motion area because most of its cells are tuned to the direction and velocity of moving stimuli, retain their stimulus selectivity even when the striate cortex of the same hemisphere has been surgically removed or reversibly inactivated by cooling[64]. When the SC is additionally removed, MT can no longer be activated by visual stimuli[65]. Other extrastriate visual areas, including V4, where colour and wavelength tuning are common, have not been studied.

The blind in blindsight

Blindsight reveals the kinds of visual processing that are possible in the extrageniculostriate cortical system. The range of functions subserved by the field defect is much richer than was expected. In addition the sensitivity in the blind field is astonishingly high,

particularly in subjects who have participated in experiments over a long period of time (see Fig. 4). The increase in sensitivity over time indicates that training of visual function in field defects might be effective, but the conditions under which this will enhance the patients' reflexive functioning in a visual world, or even lead to a recovery of perception, are poorly understood.

In view of the remarkable sensitivity in the blind field, the major difference from the normal field appears to be the absence of a conscious percept of the visual stimuli. Unawareness cannot be attributed to a general lack of cortical participation, as it has been shown that some extrastriate visual areas might contribute to some aspects of blindsight, such as detection of motion and its direction. Is it possible that the other anatomically numerous, nonstriate cortical projections to extrastriate cortex are functionally disrupted or inhibited by neurochemical alterations caused by the lesion? Or is it that they cannot by themselves create a conscious representation of a stimulus, i.e. is the primary visual cortex indispensable for visual consciousness? The progressive complexity of receptive field properties and their increasing resemblance to our percepts with hierarchical levels of processing in visual cortical areas (e.g. Ref. 66) makes this a difficult hypothesis to entertain, unless the hierarchy depends crucially on striate cortical input. In this context it would be especially interesting to know whether the monkeys that have contributed so much to our understanding of the anatomy and physiology of the residual visual functions experience the same dissociation between function and experience as the patients.

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