Blindsight revisited

Lawrence Weiskrantz

Some human patients with lesions to their primary visual (striate) cortex (V1) demonstrate residual visual capacity, but without acknowledged perceptual awareness. This phenomenon has been termed blindsight. Recent results from work on blindsight patients suggest that it is unlikely to be attributable to intact residual areas (tags) of V1. Previous research has reported that blindsight patients can retain the ability to detect monochromatic light and grating stimuli, and to discriminate orientation and direction of movement in their 'blind' fields. These findings have been joined by reports that these patients also are sensitive to, and are able to discriminate, wavelength in the absence of any experience of 'colour'. This reveals that retinal pathways other than those to the striate cortex are crucially involved in vision. Conditions can be controlled for obtaining either acknowledged awareness or unawareness of discrimination of the direction of a small moving target in blindsight patients. This potentially offers the possibility to determine whether there are structures uniquely involved in visual awareness. Monkeys lacking V1 also clearly demonstrate residual visual capacity, and some evidence exists that they also experience 'blindsight'.

Address
Department of Experimental Psychology, University of Oxford, South Parks Road, Oxford, OX1 3UD, UK

Abbreviations

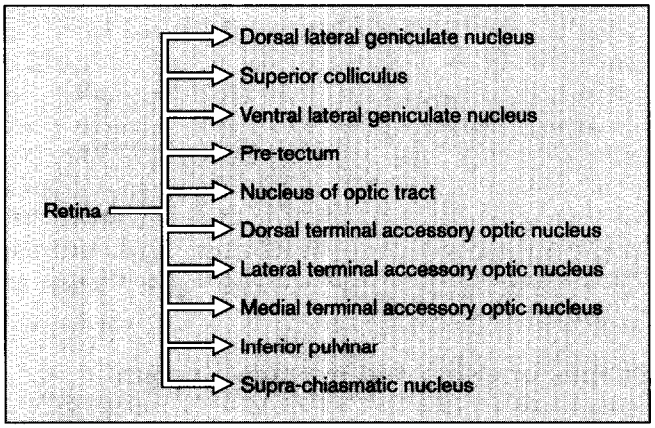
LGN	lateral geniculate nucleus
MRI	magnetic resonance imaging
PET	positron emission tomography
TEO	region inferotemporal cortex
V1	primary visual (striate) cortex

Current Opinion in Neurobiology 1996, 6:215–220

Introduction

The primate retina projects to ten different targets in the brain [1] (Figure 1). Although the pathway from retina to dorsal lateral geniculate nucleus (LGN), with a relay to V1, constitutes the largest component of the optic nerve, the remaining extrastriate pathways are not trivial — they contain five times as many fibres as the intact auditory nerve. It is not surprising, therefore, that monkeys in whom V1 has been removed can perform (with appropriate training) visual discriminations, although their capacity shows some quantitative and qualitative changes [2–8]. The paradox is that human patients in whom V1 is damaged (almost always with some additional damage) report that they are blind in the corresponding regions of their visual fields. Approximately 20 years ago, using methods originally designed to test animal vision, it was discovered that some of these patients can perform visual discriminations at a high level, such as making a saccade to a visual stimulus or a correct forced-choice response between two or more alternatives [9–11]. Nevertheless, the patients persist in saying they are 'blind' in the tested region of the field, and comment that they are 'just guessing'. This phenomenon — visual discrimination in the absence of acknowledged awareness — has been dubbed 'blindsight' [10].

Figure 1



Parallel outputs from the retina to various cerebral targets (see also [1]). Reproduced, with permission, from [33].

Blindsight is, therefore, an example of 'implicit processing' — residual functioning in the absence of explicit knowledge — that has been found in virtually every cognitive neuropsychological syndrome [12]. But early on its counter-intuitive character attracted various possible alternative explanations and suggested artifacts. These included the possibility that some light strayed into the intact portions of the visual fields (patients are typically blind only in the hemifield contralateral to the unilateral occipital damage), or that blindsight patients have poor control of their eye movements ([13]; see also [14•]). Another possibility was that the patients have a shifted response criterion for acknowledging 'seeing' [13].

These objections were satisfactorily answered some time ago [11,15]. An excellent control for stray light is provided by the optic disc, the natural 'blind-spot'. A stimulus that is absolutely undetectable on the disc, and hence does not stray beyond it, is nevertheless reliable detected by blindsight subjects in the blindsight field surrounding the disc [11]. The response criteria can be varied over a range without affecting a blindsight subject's sensitivity

for detecting a light in the blindsight field [15]. Eye position has frequently been monitored continuously in a variety of tasks, and poor control of eye fixation can be confidently ruled out. Much more recently, attention has focused on another possible interpretation, that blindsight is generated by activity in small islands of intact V1. After a brief review of visual anatomical underpinnings, I will give an account of some recently studied aspects of blindsight capacity, followed by the experimental comparison of 'awareness versus unawareness', the incidence of confirmed blindsight in occipital patients, and finally, blindsight in monkeys.

Visual anatomical underpinnings

Even in the absence of V1 in the monkey, visual information can reach a large constellation of visual 'association' areas in the brain. Some of these paths arise through parallel projections from the retina to the superior colliculus, with further relays via the thalamus to the cortex [16], or by direct projections to the pulvinar and thence to the extrastriate cortex [1]. It is known that intralaminar neurons in the LGN survive removal of V1, and project to V2, V4, V5 and TEO [17–21]. Recently, a projection from the LGN to the inferotemporal cortex has been demonstrated [22••]. The LGN projections that survive V1 removal are relatively sparse in density, but are nevertheless widespread and probably encompass all extrastriate visual areas.

Islands of vision and the striate cortex

Fendrich *et al.* [23] have reported on a patient with a hemifield of blindness caused by a cerebral stroke. Like many such patients, this patient also demonstrated macular sparing. In addition, using an 'eye tracker' that ensured stability of the retinal image even if the eyes moved, the authors found a separate, tiny 1° island of vision well away from the region of macular sparing. In that tiny island, the subject displayed blindsight, that is, his detection of a visual stimulus was above chance, but he reported no awareness of it; the surrounding area was functionally 'dead'. A magnetic resonance imaging (MRI) scan showed a "minimal region of remaining striate cortex", but it is not clear whether this corresponded to the isolated island or to the macular sparing [24].

Gazzaniga and colleagues [14•,23] suggest that some intact striate cortex is a necessary prerequisite for blindsight. However, this interpretation of blindsight cannot apply to residual function in monkeys as it is found with confirmed complete removal of V1. Gazzaniga *et al.* [14•] argue that one should not generalize from monkeys to humans, despite the fact that rhesus monkeys have visual psychophysical capacities very similar to those of humans [25,26].

The results of work on a well-studied blindsight patient (known as G.Y.) are relevant to this issue. G.Y. is a 39 year old hemianopic patient who suffered damage to his left V1 as a result of a head injury from a road accident when he was eight years old. I have found [27•] that patient G.Y. has the ability to respond to a moving target and to mimic its path along different straight and curved trajectories with his hand, throughout the whole of his blind hemifield. While performing that task, G.Y.'s eye positions were continuously monitored and his control of fixation, as usual, was excellent. An explanation of G.Y.'s ability would require that a high and well-distributed concentration of many islands of cortex existed. Patient G.Y.'s MRI scan confirmed that ablation of his striate cortex was complete (with the exception of a polar region that was considered to correspond to macular sparing) [23]. In addition, a positron emission tomography (PET) study of G.Y. using moving stimuli (well outside the macular spared zone) revealed no V1 activity, although activity was seen in V5, area 7, and elsewhere [28]. Finally, G.Y. has been tested with an eye tracker using the same psychophysical parameters as in the previous study [23], and no isolated islands were found; the area of intact vision was continuous (R Kentridge, C Haywood, L Weiskrantz, unpublished data).

Thus, small islands of V1 seem unlikely to be valid as a general explanation of blindsight in humans, although they might well apply to cases such as the one reported by Fendrich *et al.* [23]. Even if residual vision does turn out to be 'patchy' after fine-grained testing, another explanation can be suggested on the basis of retinal transneuronal changes in the retina itself rather than the intact striate cortex. After striate cortex lesions, not only do most of the cells of the LGN degenerate, but there is also consequential degeneration of many of the so-called Pβ ganglion cells of the retina that normally project to the parvo-cellular layers of the LGN [29]. In contrast, the Pα ganglion cells and Pγ cells are differentially less affected; these not only have different principal projections, but their response properties are also different. Thus, part of each eye associated with the blindsight field is qualitatively different from that associated with the normal field, and is depleted of many ganglion cells [29].

Processing colour in blindsight

Perhaps the most counter-intuitive finding of blindsight is the ability of some patients to process wavelength information. Stoerig and Cowey [30] found qualitatively normal spectral sensitivity functions in the blindsight fields of three patients, although quantitatively the sensitivity was reduced by up to one log unit. Interestingly, the peaks and troughs were also present at 450 nm, 525–550 nm and sometimes at 580–600 nm, assumed to reflect opponent processing. In addition, a clear Purkinje shift with dark adaption — loss of sensitivity at the red end of the spectrum — was observed. Subjects could be demonstrated to discriminate by forced-choice guessing between wavelengths [31], including relatively closely spaced wavelengths. The direct projections from the LGN to the inferotemporal cortex and other extrastriate cortical regions are plausible candidates for mediators of

such a residual capacity [22••]. It is of interest also that the subjects in these experiments reported having no experience of the stimuli.

Brent *et al.* [32••] have reported a related finding with patient G.Y.: he, too, exhibited normal spectral responses and could verbally identify colour stimuli presented to the 'blind' hemifield, although such colour naming was achieved without conscious perception of colour. Similar observations have also been made, with concomitant pupillary measurements, by Barbur and colleagues (JL Barbur, P Stoerig, L Weiskrantz, unpublished data), comparing positive evidence for colour identification 'without experience' in the blind hemifields of two subjects.

Indirect methodologies for testing blindsight

Because subjects are often uncomfortable about being asked to 'guess' about stimuli they cannot 'see', efforts have been made to develop methodologies that do not require forced-choice guessing but that allow inferences to be drawn about intactness of visual processing in the affected hemifields. Two general approaches have been adopted (see [33]). The first is to measure the influence of a stimulus in the blind hemifield on actually seen stimuli presented to the intact hemifield. This technique was pioneered many years ago by Torjussen [34], who reported completion of stimuli in the intact hemifield when, and only when, their complementary parts were presented to the blind hemifield. The technique and results have been replicated by Marcel (cf. [33]) using after-images of stimuli briefly illuminated by photo-flood flashes to control for eye movements. A related reaction time study was reported [35], in which the latency of a key-press response to a brief light-emitting diode (LED) presentation in the intact field was increased when it was shortly preceded by stimulus in the blind field. Rafal *et al.* [36] have reported a similar methodology and similar results with saccadic reaction times, but it has proved difficult to replicate the results in patients in whom no other indication of blindsight could be found (see e.g. [35]; but it was confirmed for patient G.Y. [37].

The second approach is to use reflex measures. Electrical skin conductance responses have been reported to occur as a result of 'unseen' light stimuli presented to the blind field [38]. Among these, the most promising candidate is the pupillary response, which is sensitive to spatial and spectral properties of stimuli even when the light flux or luminance is unchanged, and enables visual acuity as well as contrast sensitivity to be determined [39,40]. Some of the applications of the pupillary response to blindsight studies have been reviewed [33], and the method is currently under active investigation, especially with regard to colour and movement. Its independence from verbal subtleties also makes it useful for parallel studies with primates and with human infants [41].

Awareness versus unawareness

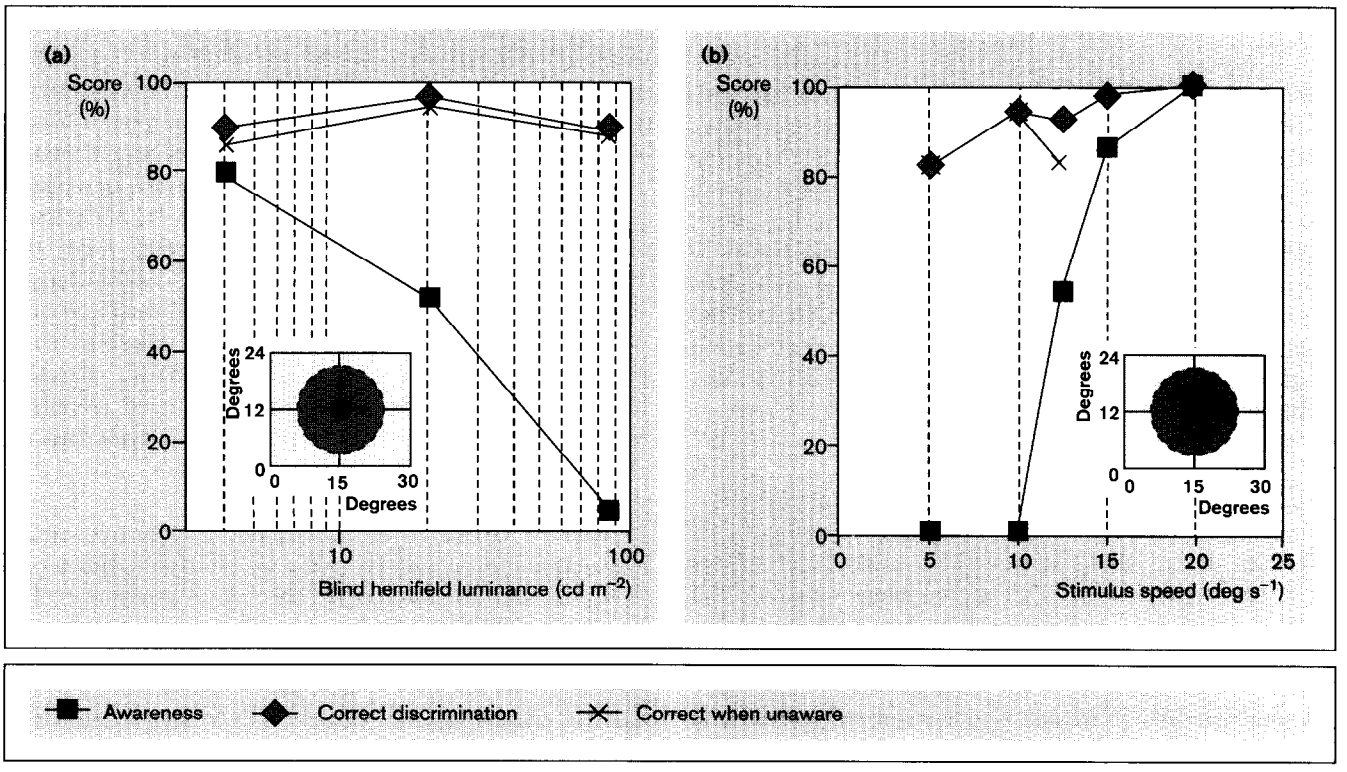
Some blindsight subjects report a kind of 'knowing' or 'awareness' with rapidly moving stimuli and with stimuli having sharp transient onset/offset [11,42,43]. A recent PET scan study of patient G.Y. as he successfully discriminated the direction of a moving bar, demonstrated activity in V3, V5, area 7 and other regions (see [27•]). It was concluded that there is "conscious visual perception without V1". The subject reported that he did not actually 'see' or 'sense' anything, but 'knew' that there was movement and its direction. Nevertheless, acknowledged conscious awareness was clearly evident, and the scan is obviously relevant to the question of whether V1 is necessary (but not whether it is sufficient) for conscious visual awareness of movement *qua* movement. (As noted above, successful blindsight responses to wavelength are devoid of awareness of colour *per se*.)

Outside a critical range of parameters for movement with 'awareness', discrimination by blindsight subjects is still possible in the complete absence of awareness. The relationship between these two modes has been recently studied in patient G.Y. [44••] using a 'commentary key paradigm' [18], in which the subject is given four keys: two keys to make a discriminative choice in a two-alternative, forced-choice situation (even by 'guessing' if necessary) concerning the direction of movement; and two other keys in which the subject signals on each trial whether he had any experience whatever, no matter how slight or effervescent (i.e. classical blindsight) [10,11]. In the blind hemifield, the range of stimulus parameters of velocity, excursion length, and luminous contrast for excellent discrimination of direction of motion of a small spot appears to be much wider for the unawareness mode than for the sharply tuned awareness mode [42] (Figure 2). Because it is possible to have matched performance levels in the blind field for the same type of movement discrimination when the subject is unaware and when he/she is aware, it should be possible with functional brain imaging to determine whether any brain activity is uniquely associated with visual awareness. Functional imaging comparing the two modes is in progress.

The psychophysical spatiotemporal tuning curve of patient G.Y. has been determined for stationary, but temporally transient, stimuli for which patient G.Y. is typically 'aware'. The spatial tuning curve is relatively narrow, with a peak at ~1.3 cycles degree^{-1} and with temporal modulation at 10 Hz [45••]. The comparison tuning curve for the unaware mode — for stimulus of which the subject is entirely unaware — remains to be measured, but it should enable the direct determination of whether the two modes are qualitatively different in their psychophysical functions, as earlier work suggests [11].

More recently, another approach has been advanced [46•] for studying 'blindsight in normal observers', by using stimulus differences to which cells in area MT (V5) area

Figure 2



Performance by a blindsight subject (G.Y.) on a movement discrimination task in which he had to indicate, by guessing if necessary, in which of two directions a spot had moved (as indicated by the two arrows in the inset). Using independent commentary keys, G.Y. also had to indicate whether or not he had any experience whatsoever of the event. **(a)** Discrimination as a function of stimulus contrast. As contrast is decreased (by increasing background luminance), the percentage of trials on which G.Y. is aware drops sharply, but discriminative performance remains high, even when he reports no awareness. Stimulus velocity was 15 deg s^{-1}, and displacement was 20 degrees. **(b)** Similar task as for (a), but with varying stimulus velocity. At low velocities (<15 deg s^{-1}), no awareness is reported, but discrimination is good. As in (a), stimulus displacement was 20 degrees. Reproduced, with permission, from [44••].

are known to be insensitive, but to which cells in V1 remain sensitive. The subjects viewed a visual display monitor in which all stimuli elements were oriented or were moving in the same direction, except for that of the target in one of the four quadrants. The targets could be made either easy to see (aware) or difficult (and hence, unaware) by means of retinal rivalry in which the eyes were given conflicting information, or by adjusting the separation between the pairs of moving dots. The subjects could reliably detect differences of which they were unaware at a high level of performance, and their performance did not correlate with concurrent confidence ratings. Under stimulus conditions, in which the differences were made visible, a positive correlation was observed, as expected. This is not blindsight in one sense because the entire background remains actively visible to the subject—it is the inset target against the visible ground that is not consciously 'seen' but is detected. Nevertheless, this offers an interesting and promising approach.

Incidence of blindsight

It is the case that not all patients with damage to V1 have produced evidence of blindsight on test; on current evidence, only a minority do. The true incidence of blindsight is not yet known. Several possible reasons for this have been suggested. The first, and probably the most important, reason is that the location and extent of lesions across patients are not uniform. In humans, V1 lesions almost invariably encroach upon neighbouring regions, and in monkeys, residual capacity is severely degraded, as the lesions extend beyond V1. Because projections are made to visual cortical areas that neighbour V1 from the pulvinar (which receives an input both from the retina and from the superior colliculus) and from the remaining undegenerated cells of the lateral geniculate, among other sources, such potential extrastriate cortical targets can themselves be damaged by lesions that extend outside of V1. A second reason is that the age at which the damage incurred is important, especially as suggested by animal research [47••]; however, more research is needed

on humans. A third reason is that the stimulus parameters used when testing blindsight can be critical and different from those that are optimal for normal vision. It was shown clearly for patient G.Y. that when tested for detection of a Gaussian-bounded light patch under conditions that work well for normal vision, he performed consistently according to chance. But when the slope of the temporal Gaussian was increased only slightly, his performance rose to virtually perfect performance [48]. A final reason for the variable incidence is the need to conduct tests over long tiring sessions with 'forced-choice' paradigms in which the subject has no subjective awareness. This is no doubt the reason why relatively small numbers of subjects have been used, but tested very intensively. This reason is also the motivating force behind the development of indirect methods of testing.

Monkeys and blindsight

Blindsight research, as indicated in the Introduction, grew out of animal research showing that monkeys without V1 have a substantial visual capacity. But do they have blindsight? That is, are their discriminations made without visual experience? A recent study by Cowey and Stoerig [49••] addresses the question directly (see also [50•], which draws a human–monkey blindsight comparison). The rationale, which is also a property of the commentary key paradigm, requires not only a discriminative choice, but an independent response that classifies the discrimination. It was confirmed, first, that monkeys with unilateral striate removal have an excellent capacity to locate light patches in their 'blind' field. The animals were then trained on a random light/no light discrimination in their intact field, requiring a press of a separate panel on 'no light' trials. The question was whether probe lights presented into the blind field would be treated as a 'light' or 'no light'. The animals consistently pressed the 'no light' panel, just as a human blindsight subject would do. Thus, the research is moving full circle back to its starting point.

Conclusions

The loss of V1 in monkeys or humans does not necessarily remove the capacity for visual discriminations; however, if the damage also extends well outside V1 — as often is the case clinically — visual capacity may be degraded or absent. Anatomical work on animals suggest that the age at which the lesion occurs may be important. Intact tags of striate cortex may account for residual visual function in some patients, but evidence suggests that this is unlikely to be generally applicable to all cases, and it can not be an explanation of the results from work on monkeys. In human patients with V1 damage, qualitatively normal spectral sensitivity and a capacity for colour discrimination have been reported, in the absence of any acknowledged awareness, which complements earlier evidence for detection of monochromatic lights, discrimination of orientation and simple shapes without awareness.

Some patients also have a kind of 'awareness' for rapidly moving or sharply transient stimuli, but are unaware of the stimuli outside a sharply tuned set of parameters. This, in principle, offers the attractive possibility of comparing functional images for the aware and unaware modes for the same kind of discriminations at matched levels of performance. Indirect quantitative methods of testing for blindsight, such as interactions between intact and damaged hemifields and pupillometry, are being developed. Finally, a monkey homologue of blindsight appears to exist; monkeys with V1 damage, like human blindsight subjects, detect and locate visual stimuli very well, but classify them as being 'no light'.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

1. Cowey A, Stoerig P: **The neurobiology of blindsight.** *Trends Neurosci* 1991, **14**:140–145.
2. Cowey A, Weiskrantz L: **A perimetric study of visual field defects in monkeys.** *Q J Exp Psychol* 1963, **15**:91–115.
3. 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**:419–435.
4. Miller M, Pasik P, Pasik T: **Extrageniculate vision in the monkey. VII. Contrast sensitivity functions.** *J Neurophysiol* 1980, **43**:1510–1526.
5. Schilder P, Pasik P, Pasik T: **Extrageniculate vision in the monkey. III. Circle vs triangle and 'red vs green' discrimination.** *Exp Brain Res* 1972, **14**:436–448.
6. Humphrey NK: **Vision in a monkey without striate cortex: a case study.** *Perception* 1974, **3**:241–255.
7. Weiskrantz L, Cowey A, Passingham C: **Spatial responses to brief stimuli by monkeys with striate cortex ablations.** *Brain* 1977, **100**:655–670.
8. Weiskrantz L: **Behavioural analysis of the monkey's visual nervous system.** *Proc R Soc Lond [Biol]* 1972, **182**:427–455.
9. Pöppel E, Held R, Frost D: **Residual visual function after brain wounds involving the central visual pathways in man.** *Nature* 1973, **243**:295–296.
10. Weiskrantz L, Warrington EK, Sanders MD, Marshal J: **Visual capacity in the hemianopic field following a restricted occipital ablation.** *Brain* 1974, **97**:709–728.
11. Weiskrantz L: *Blindsight: A Case Study and Implications.* Oxford: Oxford University Press; 1986.
12. Weiskrantz L: **Disconnected awareness for detecting, processing, and remembering in neurological patients.** *J R Soc Med* 1991, **84**:466–470.
13. Campion J, Latto R, Smith YM: **Is blindsight an effect of scattered light, spared cortex, and near-threshold vision?** *Behav Brain Sci* 1983, **6**:423–448.
14. • Gazzaniga MS, Fendrich R, Wessinger CM: **Blindsight reconsidered.** *Curr Directions Psychol Sci* 1994, **3**:93–95.
A convenient review for a general psychology audience of evidence that suggests to the authors the notion that blindsight may be attributable to intact tags of V1. It is based on their finding reported in *Science* (see [23]) of a hemianopic patient in whom they found an isolated small island of blindsight function.
15. Stoerig P, Hubner M, Pöppel E: **Signal detection analysis of residual vision in a field defect due to a post-geniculate lesion.** *Neuropsychologia* 1985, **23**:589–599.
16. Rodman HT, Gross CG, Albright TD: **Afferent basis of visual response properties in area MT of the macaque. I. Effects of striate cortex removal.** *J Neurophysiol* 1989, **9**:2033–2050.

17. Benevento LA, Yoshida K: **The afferent and efferent organization of the lateral geniculo-striate pathways in the macaque monkey.** *J Comp Neurol* 1981, **203**:455–474.

18. Fries W: **The projection from the lateral geniculate nucleus to the prestriate cortex of the macaque monkey.** *Proc Natl Acad Sci USA* 1981, **213**:73–80.

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

20. Webster MJ, Bachevalier J, Ungerleider LG: **Subcortical connections of inferior temporal areas TE and TEO in macaque monkeys.** *J Comp Neurol* 1993, **335**:73–91.

21. 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**:631–638.

22. •• Hernandez-Gonzalez CC, Reinoso-Suarez F: **The lateral geniculate nucleus projects to the inferior temporal cortex in the macaque monkey.** *Neuroreport* 1994, **5**:2692–2696.
A clear demonstration, by retrograde transport, that the dorsal LGN of the monkey projects to the inferotemporal cortex. The authors also provide a very convenient review of the other projections of the LGN to visual cortical areas, which are now thought to be widespread.

23. Fendrich R, Wessinger CM, Gazzaniga MS: **Residual vision in a scotoma: implications for blindsight.** *Science* 1992, **258**:1489–1491.

24. Stoerig P: **Sources of blindsight.** *Science* 1993, **261**:493.

25. De Valois RL, Morgan HC, Polson MC, Mead WR, Hull EM: **Psychophysical studies of monkey vision. I. Macaque luminosity and color vision tests.** *Vision Res* 1974, **14**:53–67.

26. De Valois RL, Morgan HC, Snodderly DM: **Psychophysical studies of monkey vision. III. Spatial luminance contrast tests of macaque and human observers.** *Vision Res* 1974, **14**:75–81.

27. • Weiskrantz L: **Blindsight: not an island unto itself.** *Curr Directions Psychol Sci* 1995, **4**:146–151.
A useful review for a general psychology audience, demonstrating why islands of intact V1 are unlikely to provide a general explanation of residual visual function in human blindsight patients, and certainly not in monkeys.

28. Barbur JL, Watson JDG, Frackowiak RSJ, Zeki S: **Conscious visual perception without V1.** *Brain* 1993, **116**:1293–1302.

29. 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.

30. Stoerig P, Cowey A: **Wavelength sensitivity in blindsight.** *Nature* 1989, **342**:916–918.

31. Stoerig P, Cowey A: **Wavelength discrimination in blindsight.** *Brain* 1992, **115**:425–444.

32. •• Brent PJ, Kennard C, Ruddock KH: **Residual colour vision in a human hemianope: spectral responses and colour discrimination.** *Proc R Soc Lond [Biol]* 1994, **256**:219–225.
This study confirms that normal spectral sensitivity can be found in the affected hemifield of a blindsight subject (see [28]). The investigators also investigated the subject's ability to identify verbally colour stimuli presented to his 'blind' hemifield, and found that, with forced-choice guessing, he achieved a high proportion of correct responses to large stimuli. Colour naming was achieved without conscious perception of colour.

33. Weiskrantz L: **Outlooks for blindsight: explicit methodologies for implicit processes (Ferrier Lecture).** *Proc R Soc Lond [Biol]* 1990, **239**:247–278.

34. Torjussen T: **Residual function in cortically blind hemifields.** *Neuropsychologia* 1978, **16**:15–21.

35. Marzi CA, Tassinari G, Aglioti S, Lutzemberger L: **Spatial summation across the vertical meridian in hemianopics: a test of blindsight.** *Neuropsychologia* 1986, **30**:783–795.

36. 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**:118–121.

37. Cochrane KA: **Some tests of residual visual functioning in humans with damage to the striate cortex [PhD thesis].** Oxford: University of Oxford; 1995.

38. Zihl J, Tretter F, Singer W: **Phasic electrodermal responses after visual stimulation in the cortically blind hemifield.** *Behav Brain Res* 1980, **1**:197–203.

39. Barbur JL, Forsyth PM: **Can the pupil response be used as a measure of the visual input associated with the geniculo-striate pathway?** *Clin Vision Sci* 1986, **1**:107–111.

40. Barbur JL, Thomson WD: **Pupil response as an objective measure of visual acuity.** *Ophthalmic Physiol Opt* 1987, **7**:425–429.

41. Cocker KD, Moseley MJ, Bissenden JG, Fielder AR: **Visual acuity and pupillary responses to spatial structure in infants.** *Invest Ophthalmol Vis Sci* 1994, **35**:2620–2625.

42. Blyth IM, Bromley JM, Kennard C, Ruddock KH: **Visual discrimination of target displacement remains after damage to the striate cortex in humans.** *Nature* 1986, **320**:619–621.

43. Blyth IM, Bromley JM, Kennard C, Ruddock KH: **Residual vision in patients with retrogeniculate lesions of the visual pathways.** *Brain* 1987, **110**:887–905.

44. •• 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**:6122–6126.
A study in which a blindsight subject had to discriminate movement direction, by guessing if necessary, and also indicate on each trial whether he was aware of any visual event. Velocity, excursion length, orientation differences, and contrast were varied, yielding a relatively narrowly tuned awareness mode. Outside that range, excellent performance without any acknowledged awareness was observed.

45. •• Barbur JL, Harlow AJ, Weiskrantz L: **Spatial and temporal response properties of residual vision in a case of hemianopia.** *Philos Trans R Soc Lond [Biol]* 1994, **343**:157–166.
The spatiotemporal tuning curve for patient G.Y. was determined for a stationary monochromatic light patch, either structured or unstructured, with Gaussian-bounded spatial boundary. With structuring, a relatively sharply tuned channel was found, with a peak at 1.3 cycles degree^{-1}. A unstructured luminance channel was also found with temporal modulation. The subject was aware of the stimuli. The unaware channels remain to be determined.

46. • Kolb FC, Braun J: **Blindsight in normal observers.** *Nature* 1995, **377**:336–338.
An intriguing possible homologue of blindsight in normal subjects. Target stimuli were used for which it was assumed that cortical area MT would be insensitive, but for which V1 would be sensitive; this approach enables this particular type of V1 output to be disconnected, but allows subcortically mediated pathways to operate. The subjects had to guess in which quadrant of the display the target fell. They could do this although they had no awareness of the target and there is no correlation between their success and their confidence ratings on each trial. With visible stimuli, a clear correlation with confidence rating was observed.

47. •• Payne BR: **System-wide repercussions of damage to the immature visual cortex.** *Trends Neurosci* 1994, **17**:126–130.
A very good review of the effects of age on visual capacity following visual cortical lesions in the cat, to which the author has contributed very important work. This review is informative behaviourally, anatomically, and conceptually.

48. Weiskrantz L, Harlow AJ, Barbur JL: **Factors affecting visual sensitivity in a hemianopic subject.** *Brain* 1991, **114**:2269–2282.

49. •• Cowey A, Stoerig P: **Blindsight in monkeys.** *Nature* 1995, **373**:247–249.
An important 'first' demonstration of a homologue of blindsight in the monkey. The authors show that monkeys with unilateral V1 removal can detect and localize stimuli in their affected hemifields despite being classified by the animals as 'non-lights'.

50. • 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* 1995, **92**:8215–8218.
A study that draws a parallel between the behaviour of monkeys with unilateral V1 lesions and human blindsight in patients with unilateral occipital lobe lesions. It was found that in conditions analogous to clinical perimetry, in which human subjects are found to be 'blind' in their affected hemifields, the monkeys failed to initiate saccades to the targets. It was necessary to signal the occurrence of the target by turning off the fixation point for the monkeys to saccade to its position.