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Blindsight

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Glossary

g0005 **Contrast sensitivity function** – A function which relates the perception of image components to the reciprocal of minimum contrast energy needed for their detection. The contrast sensitivity function of a visual system represents a combination of both optical and neuronal sensitivity.

g0010 **Perimetry** – Methods for quantifying visual sensitivity. In an automated perimeter, visual targets of specific size or contrast are presented at multiple retinal locations. Visual sensitivity is then determined by subjective reports of the observer on whether s/he detected the target.

g0015 **Psychophysics** – Study of the relationship between physical parameters of a stimulus and its resultant psychological percept. The discipline was founded by Fechner in eighteenth century.

g0020 **Pupillometry** – Determining the size of the pupil aperture. Various techniques have been developed to determine the steady state pupil diameter and its fluctuations over time.

g0025 **Retinotopic** – Related to the retinal image. A topographic retinotopic representation means that each point on the retinal image is represented in a systematic fashion.

g0030 **Scotoma** – Area of total or relative visual loss within someone's field of vision.

g0035 **Signal detection theory** – An approach originally developed for engineering applications, and subsequently applied to human behavior which allows determination of subjective false alarms and bias in addition to sensitivity, not addressed in Fechnerian psychophysics.

g0040 **Spatial frequency** – Number of cyclic variations of light and dark per unit measure of space.

g0045 **Two alternative forced choice paradigm** – A psychophysical technique for determining sensitivity. In spatial version, two targets are presented simultaneously in separate spatial locations and the observer is asked to choose between the two. In the temporal version, targets are separated in time, and the time intervals often signaled via a separate modality. For example, the temporal intervals for presentation of visual targets may be separated by auditory bleeps.

g0050 **Visual field defect** – Areas of relative or absolute blindness (scotoma).

Introduction

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p0005 Put simply, blindsight refers to the ability to process certain visual stimuli presented within an area of visual field which is clinically blind, following occipital brain injury. However, the concept, theory, and practicalities of blindsight are far from simple! This area of research is exciting, complex and often controversial. Investigations into blindsight have important implications for our understanding of normal visual processing, visual awareness and consciousness, as well as plasticity and recovery of function following brain injury. Blindsight has an interesting history in terms of the links between animal and human research. It is also an area in which objective science and the study of subjective, internal phenomena meet, raising many interesting theoretical and methodological challenges.

p0010 Stroke, closed head injury, congenital abnormalities, and surgery can all cause damage to striate cortex (also know as primary visual cortex, V1 and Brodmann area 17) and result in an area of cortical blindness affecting the corresponding region of visual field. When areas of cortical blindness

(also known as visual field defects (VFDs)) are suspected, vision is tested clinically using a method called perimetry. During perimetric testing, a patient fixates (looks steadily at) a central point and pushes a buzzer each time they are aware of a small light flash presented throughout their visual field. Because lights presented within a VFD are not consciously perceived, the person will fail to push the buzzer when these lights are presented, creating a 'map' of the subjectively blind area of vision. Blindsight refers to the ability to process certain visual stimuli presented within these perimetrically blind areas of visual field under certain conditions.

p0015 Early experimental research into the impact of removal of striate cortex in nonhuman primates initially suggested survival of some visual abilities. Although some later research raised uncertainties about the limits of the lesions in some of these studies, it now seems clear that monkeys without primary visual cortex retain a range of visual functions. In contrast, initial observations suggested that human patients without primary visual cortex were phenomenally blind (i.e., they were not aware of perceiving any visual stimuli). The origins of blindsight lie in investigations of this apparent contrast between human and nonhuman primate visual systems. Unexpectedly, the use of animal methodologies to test human patients revealed that although patients stated that they were using 'guesswork' they demonstrated a range of visual abilities in the absence of striate cortex.

p0020 Although the concept of blindsight is now established, from the outset, general theory, practical issues, and criticisms have been raised and continue to be debated. Methodological issues remain vital for preventing potential experimental artifacts, selecting appropriate stimuli and experimental paradigms in order to elicit blindsight when present. Blindsight, for itself and its associated possibilities, such as insight into normal visual processing, is an area of lively activity.

p0025 One of the most intensively studied blindsight patients (DB), demonstrated detection and localization of specific visual stimuli, as well as discrimination of orientation, movement and simple form. Other visual attributes discriminated in the absence of conscious experience include; color, orientation, simple shapes, motion and the onset and offset of visual events. Implicit methods, for example, measurement of minute changes in pupil diameter (pupillometry, see also the later section on pupillometry) have also revealed evidence for processing of visual stimuli within blind areas of visual field. Physiological measures that do not rely on asking about a person's subjective experience of visual events hold promise as a potential screening tool for blindsight. To date, pupillometry has demonstrated evidence for processing of color, movement and grating stimuli in the absence of conscious visual experience. Although much of the research evidence for blindsight in humans comes from single cases or small group studies, there is now increasing evidence that blindsight may not be a rare phenomenon. The possibility that blindsight may not be as rare as once thought, raises important questions about the development of rehabilitation strategies with the aim of potentially recovering some degree of visual function.

Early Experimental Evidence from Nonhuman Primate and Human Cases

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Early Human Evidence

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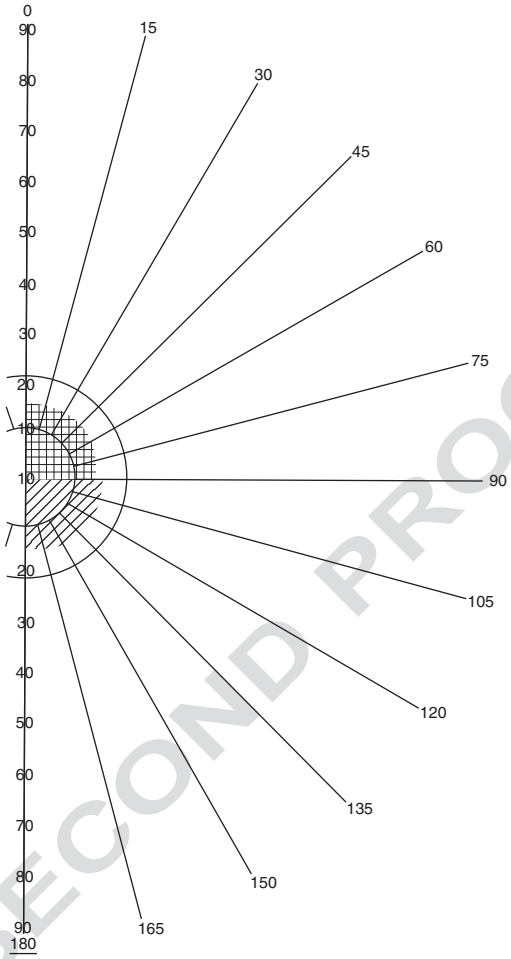
p0030 Much of the early evidence relating to VFDs was from cases of war veterans who suffered gunshot wounds to the head during the Russo-Japanese, World War I (1914–1918) and World War II (1939–1945). As the lesions caused by gunshot wounds were often fairly specific, they provided the opportunity to investigate the relationship between occipital lobe damage and resulting VFDs. Even at this time, there was disagreement about whether any visual abilities remained following destruction of striate cortex. In 1918, Gordon Holmes, a prominent ophthalmologist, published a summary of 15 cases of visual cortex and optic radiation lesions and resulting VFDs. Holmes reported a systematic, topographical representation of the contralateral hemifield of vision in the striate cortex (see **Figure 1**). Holmes concluded that scotoma were areas of total, permanent blindness. The view that destruction of unilateral striate cortex resulted in total and permanent blindness affecting the contralateral visual field and the view that bilateral destruction resulted in



complete blindness were widely held, particularly by the medical establishment. Poppelreuter claimed that no scotoma was 'absolutely' blind. He maintained that some form of rudimentary function was present and could be elicited by increasing the size or luminous intensity of the stimulus. Poppelreuter ordered different levels of visual function from: amorphous light sensitivity, size perception without definite form, amorphous form perception, perception of discrete objects, mild amblyopia, and normal vision. An investigation of gunshot cases from the World War II by Teuber revealed some discrimination between light and dark in VFDs. However, the possibility of light scattering into the intact area of visual field is difficult to rule out. Following the investigations into visual deficits resulting from gunshot wounds during the two world wars there was relatively little interest in residual visual abilities in humans as the focus shifted to nonhuman primate research.

Early Nonhuman Primate Evidence

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Perimetry is the systematic measurement of the visual field by detection of targets (usually light stimuli) presented throughout the visual field. The use of perimetry for mapping VFDs was adapted for nonhuman primates by Alan Cowey in 1963. Until this point, there were mixed reports about the extent of the impact of removal of striate

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cortex on vision in monkeys and progress was limited by the lack of a standard method of 'mapping' VFDs. The ability to study VFDs perimetrically meant it was possible to precisely define the characteristics of the deficit and relate these findings to visual organization in the monkey. Monkeys were trained to press a lever on their left when a buzzer was presented alone and a button on their right when a buzzer was presented simultaneously with a light flash from a bulb. The animal's responses could not be based on expectation as they had no means of knowing on which trial the bulb would be illuminated. If they were simply guessing there would be a high proportion of false positive responses. It was concluded that monkeys can conceal large VFDs by scanning to use intact areas of vision. One problem with thisD[of)--354(wc-255.3(guesst58.7(cgs)-578(o3por)elon3(presseN24(ca4.7(g

equated for perceived light power) targets on the basis of shape (triangle vs. circle), orientation, spatial frequency, contrast and wavelength. Despite the recovery of these abilities, the monkeys' visual sensitivity was still greatly decreased compared to preoperative levels. For example, preoperatively monkeys could discriminate all spatial frequencies between 0.5 and 32 cycles per degree compared to 0.5 and 4 cycles per degree postoperatively.

p0060 The potential importance of subcortical processing in blindsight was highlighted by comparing the effects of lesions of striate cortex and superior colliculus on detection of visual stimuli and accuracy of saccadic eye movements. Following unilateral ablation of striate cortex, monkeys gradually improved with practice at detection of light stimuli. This improvement was clearly a practice effect, as trained areas of visual field improved more than untrained areas. Following superior colliculus lesions, a midbrain nuclei with extensive multimodal processing capability and connectivity (in the absence of cortical damage), there was no evidence of detection or significant saccadic deficits. Importantly, following joint lesions of striate cortex and superior colliculus, no recovery was observed, even after 15 weeks of testing. It seemed that either striate cortex or superior colliculus was 'sufficient' for visual detection and visually guided saccades and that one or a combination of these two structures are necessary. Lesions in both (in either order) appear to result in permanent deficits in visual detection and saccade accuracy. These findings were strengthened by further research demonstrating that monkeys were able to reach accurately toward targets that were illuminated very briefly (110 ms), controlling for the possibility of discrimination on the basis of eye or head movements.

s0025 Recent Nonhuman Primate Evidence

p0065 More recently, the fascinating question of whether destriated monkeys experience phenomenal blindness within their VFDs in the same way as human patients, or whether they subjectively perceive stimuli has been investigated. Monkeys were trained to reach out and touch light stimuli when they were presented on screens in front of them. They were also trained to touch a different area when it was a 'blank' trial (i.e., no stimulus presented). In 10% of trials in which stimuli were presented within the monkey's blind field, the monkey almost always categorized the trial as a 'blank' by touching the 'blank area.' This finding suggested that monkeys were experiencing blindsight in the same way as human cases. That is, although they were capable of reaching for these stimuli when they were presented within their blind field, they classified them as eliciting no visual experience.

p0070 The extent to which monkey residual visual abilities parallel those found in humans has been further investigated. The ability of monkeys with unilateral removal of striate cortex to make accurate saccades to visual targets under two different conditions has been compared. When monkeys fixated a central point and targets were presented throughout their visual field (analogous to clinical perimetry), they failed to initiate saccades to targets in their blind field, appearing blind. Yet, when the central fixation point was extinguished simultaneously with the onset of the target, they were able to successfully localize targets presented within the blind region of their visual field. There are two possible explanations for these findings. First, that the fixation offset facilitated residual visual performance by cueing the monkey to make the eye movement or, that the fixation offset disinhibited the comparatively weaker signals from the target within the VFD. Overall, these findings suggest that similar to humans, residual vision in destriated monkeys is often too weak to result in explicit responses to blind field stimulation. As a result, an external signal and/or release from contralateral inhibition may be necessary to demonstrate the spared capacities. These conclusions are consistent with human evidence showing that patients could accurately localize visual stimuli with eye movements within their blind field, yet when they were asked to indicate the presence of stimuli in their blind field during perimetry, they were unable to do so and appeared blind. When asked to guess the location of the stimuli in the presence of a signal to indicate target onset, they made accurate eye movements to the targets. So, it seems that under certain conditions, residual vision in man and in monkey can be remarkably similar. Particularly, both monkey and humans

without striate cortex seem unable to report visual sensations despite being able to localize targets under forced choice (FC) conditions.

Recent Human Evidence

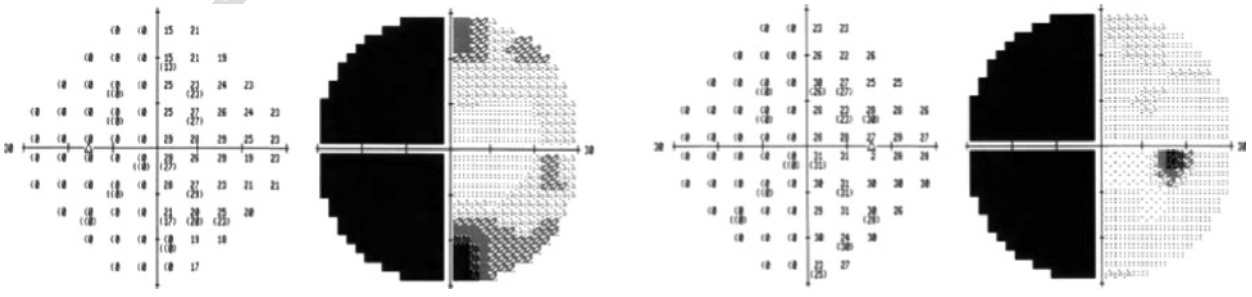
p0075 The first clear evidence that visual stimuli presented within VFDs could influence eye movements was reported in four patients who were capable of looking in the direction of a light flash in their VFD despite remaining unaware of the visual event. In contrast, patients with VFDs caused by retinal lesions did not show any association between target position and eye position.

p0080 At about the same time as this observation was published, Weiskrantz and colleagues were beginning to investigate the case of DB, a particularly interesting blindsight case. The term 'blindsight' was coined by Larry Weiskrantz for the title of a seminar presentation!

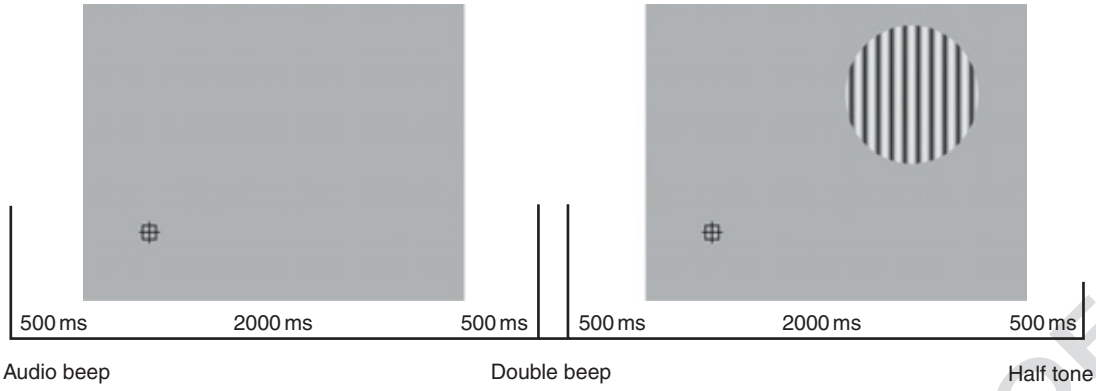
p0085 There were two crucial aspects of DB's case which acted as a catalyst in the research into residual vision following striate damage. The first was that his VFD was caused by surgical removal of a small tumor in the calcarine sulcus. So there were notes detailing the lesion dimensions, including removal of the major portion of the calcarine cortex (in which striate cortex is situated) on the medial surface of the right hemisphere. Following surgery, DB's symptoms (debilitating migraines) were much relieved, but, the majority of his left visual field was blind when tested by clinical perimetry (**Figure 2**). The special interest in DB's case came when it was noticed that DB appeared to be able to locate some objects within his blind field despite denying any conscious visual experience. The second crucial aspect of DB's case was the decision to test him using the same FC methodology as used for the previous animal experiments. Rather than reliance on the usual clinical methods, which required DB's subjective experience of his vision, for example perimetry, the use of FC methods which 'forced' DB to make decisions about visual stimuli presented within his VFD revealed a range of fascinating visual abilities.

p0090 The adoption of FC methodologies previously used in animal experiments proved crucial in uncovering DB's residual visual abilities. For example, in a temporal two alternative FC paradigm (**Figure 3**), DB was required to choose, if necessary by guessing, in which of the two time intervals a visual stimulus was presented within his VFD. Although subjectively he did not experience any visual presentations in either time interval he was 'forced' to make a choice. Over time, with sufficient numbers of trials it was possible to ascertain whether his 'guesses' were statistically different to performance on the basis of chance.

p0095 Extensive testing revealed DB's ability to successfully detect and localize stimuli within his blind field by pointing, or less accurately by eye movements. He was able to discriminate an orientation difference of 10° at a position of 45° from the fixation point. He could discriminate moving from stationary stimuli and was still able to detect stimuli which appeared gradually rather than with a sudden onset. Although DB's ability to discriminate between an 'X' versus 'O' initially appeared to suggest rudimentary form discrimination, further testing showed that what appeared to be an ability to discriminate form was actually based on orientation discrimination.



f0010 **Figure 2** DB's field defect, a left hemianopia, measured using a Humphreys perimeter, 30-2 Full Threshold program. Locations marked at '<0' represent locations where the brightest stimulus (10 000 apostilbs) did not elicit a response. Reproduced from Trevethan *et al*. (2007) *Cognition*.



f0015 **Figure 3** Example of temporal two-alternative-FC paradigm in which stimulus was presented in the second time interval. The patient fixates the fixation cross to ensure that the stimulus is presented entirely within the blind visual field. Audio beeps signal the start and end of each time interval. Usually fixation is continually monitored using a video camera or eyetracker.

This example highlights some of the complexities of testing for blindsight. When he was tested with stimuli with reduced orientation cues for example, triangle versus 'X,' performance deteriorated to chance levels. He was also unable to make a 'matching' (same/different) judgment when both stimuli were presented within his blind field despite his ability to make the judgment successfully when one stimulus was presented within his blind field and one within sighted field and despite his ability to distinguish between the stimulus in his blind field when they were presented singly. Overall, the profile of DB's residual abilities was broadly consistent to that reported in monkeys without striate cortex, that is, detection and localization of certain stimuli in the absence of clear form discrimination or recognition.

p0100 Throughout testing, in response to certain stimuli, for example, stimuli with an abrupt onset, DB reported some experiences of subjective awareness, such as, 'something comes in from the screen.' This subjective awareness was not experienced as 'seeing' but as a 'feeling' that something was presented. In order to investigate DB's subjective experience he was asked to report 'aware' or 'unaware' after each experimental trial, called the 'commentary key paradigm.' For example, in a temporal two alternative FC paradigm, DB would be informed that a target would be presented in either the first or second time interval. At the end of each trial he reported in which interval he guessed the target was presented and whether he was aware or unaware, for example, 'first, unaware.' Following this approach, Weiskrantz defined two types or modes of blindsight performance. Type II performance – detection with some awareness, in the absence of 'seeing' and Type I performance – detection in the absence of acknowledged awareness. The verbal commentaries revealed that DB was more likely to report subjective experience of the stimulus when it had an abrupt onset. However, this experience was usually nonveridical and his performance actually tended to improve when he reported that he was not subjectively aware of stimuli. Interestingly, his performance was often best at times when he was tired and felt that he was not performing well.

Recent Investigations into Blindsight in DB

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p0105 More recently, DB was tested after a gap of 17 years. In addition to confirmation of his original abilities, he also reported his subjective experience of conscious visual negative afterimages in response to a range of stimuli which he was unaware of at the time of presentation, that is, unconscious stimuli producing conscious afterimages. The complementary colors and contrasts of the negative afterimages match the shapes and contrast of the original stimuli and obey Emmert's law (i.e., change size directly with projected distance). Event-related potential analysis suggested a strong anterior, left focus for the blind field presentations compared to an intact field posterior focus. The differential patterns of activity were not associated with hemispheric differences

per se as they were not found in an age-matched control participant. Frontal activation has been associated with stimuli which provoked conscious awareness in another extensively studied blindsight case, GY. In the case of GY, a functional magnetic resonance imaging investigation revealed predominantly right frontal activation associated with subjective awareness of certain stimuli. Frontal activation has also been implicated in comparisons of conscious and unconscious events in unilateral neglect and change blindness. Investigations of visual awareness in blindsight support ongoing research suggesting that visual awareness comprises and requires further processing in addition to the posterior input stage.

p0110 Further recent investigations with DB have revealed unexpected and fascinating aspects to his blindsight abilities. DB has demonstrated blindsight detection that can be superior to normal sighted vision, although still in the absence of conscious awareness. He was able to reliably detect extremely low-contrast, static grating stimuli within his blind field that he was unable to detect reliably within his sighted field. A group of age-matched control participants with normal vision were also unable to reliably detect these. DB also demonstrated form discrimination within his blind field. He reliably identified low contrast outline shapes, judging whether stimuli were the same or different and identified complex images (photographs). Although only a single case, who may well not reflect the blindsight abilities of many cases, the dramatic improvement in DB's blindsight abilities is striking. The apparent improvement in his abilities is particularly interesting as although he has participated in considerable amounts of experimental testing over the years, he has not taken part in a specific visual rehabilitation program.

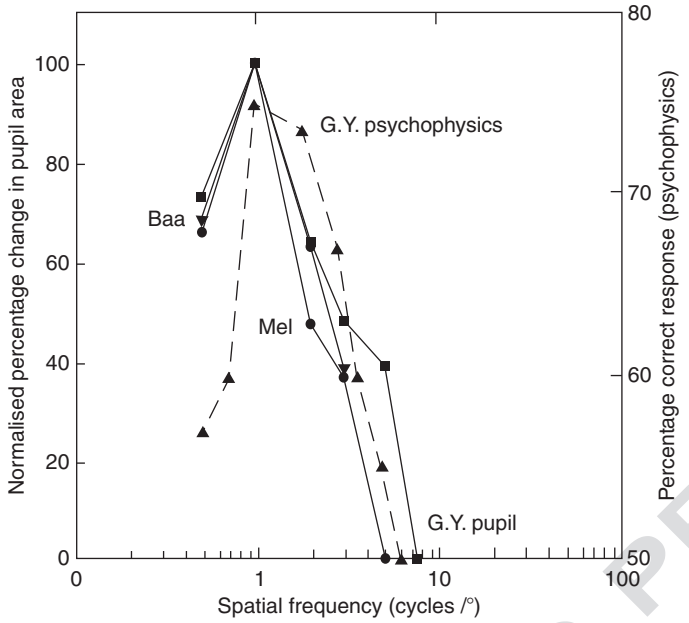
s0040 Implicit Processing

s0045 Pupillometry

p0115 Psychophysical techniques have been called the 'heroic method' of testing for blindsight because of the reliance on considerable effort, cooperation and time on the part of both the participant and the experimenter. Although the results of psychophysical investigations have brought great advances in the investigation of blindsight, the value of indirect and objective testing methods is clear. Pupillometry is the measurement of minute fluctuations in pupil diameter in response to a stimulus. It is an objective method which can be used in conjunction with subjective methods which may also offer greater insight into the mechanisms mediating blindsight. The retina, optic nerves, optic chiasm and optic tracts are all shared by both visual processing and pupillary control. Consequently damage to these areas affects both vision and pupil responses, enabling the use of pupil responses as an objective indicator of visual pathway function. It has been demonstrated that pupil responses to sinusoidal gratings resemble contrast sensitivity functions for foveal (central) and peripheral presentations. This suggests that pupil responses could be used as an objective measure of visual acuity. Measurable blindfield pupil grating responses (PGRs) have been reported in GY which were similar to those measured in two monkeys without striate cortex. The blindfield PGRs suggested a narrowly tuned channel for spatial vision with a peak sensitivity at 1 cycle per degree and a cutoff around 7–8 cycles per degree. There was close correspondence between the residual spatial channel in GY, measured objectively by pupillometry, and the psychophysically determined data (**Figure 4**). Blind field pupil responses to gratings of equal space-averaged luminance (i.e., no difference in illumination) and colored stimuli in GY have been demonstrated in the absence of reported awareness. The finding that pupil responses can be measured in the absence of acknowledged awareness raises possibilities for the further development of pupillometry as a potential screening technique for blindsight. Measurement of reliable pupil responses in DB as well as in a group of blindsight cases (unpublished data) supports this potential development. In terms of the mechanisms mediating pupil responses, the narrowing of the response profile following a cortical lesion can be viewed as direct evidence of cortical involvement in the generation of stimulus specific pupil responses.

Other Examples of Implicit Processing s0050

p0120 Implicit processing within a VFD occurs when a stimulus presented in a blind area of visual field



f0020 **Figure 4** Psychophysical and pupillometric data for blind field stimulus presentation in a hemianope (GY) and the pupil responses in two Monkeys with unilateral striate lesions. Reproduced from Weiskrantz *et al*. (1998) *Brain*.

can be shown to exert an effect on the response of the observer despite their failure to 'see' or be aware of it. There are several different methods of investigating implicit processing including, 'completion' and the 'redundant target effect.' The 'completion' phenomenon occurs when a patient with a hemianopia is shown a stimulus, such as an outline circle, with its center in line with the fixation point (so half of the circle is within their blind field) yet they report seeing a full circle. If the half-circle within the blind field was shown alone, no stimulus was reported. In the 'redundant target effect,' simultaneous or prior presentation of an unseen stimulus influences reaction times to a seen target.

s0055 How Common is Blindsight?

p0125 Initially, it seemed that across a random sample of patients with VFDs resulting from cortical damage, blindsight would only be found in a minority. An early study reported evidence of residual function (responses to movement and localization by pointing or eye movements) in 5 out of 25 patients. One of these patients was GY, who is another extensively studied blindsight case.

p0130 There are several reasons why it was thought that evidence of blindsight would be unusual or difficult to clearly demonstrate. First, lesions affecting occipital cortex are typically variable and as V1 is buried deep in the medial aspect of the brain, lesions restricted to V1 alone (rather than including visual association areas) are likely to be fairly uncommon. The age at which damage occurs may also influence the likelihood of demonstrating blindsight as this has already been shown in animal research. It is also important to consider that many of the ways of testing for blindsight can seem strange! Asking someone to repeatedly guess about something that they cannot consciously see can be very frustrating and tiring and not all participants are willing and able to do this. Another absolutely crucial consideration is the choice of stimulus parameters as parameters that may be suitable for testing normal vision may not apply to the blind field. A more recent larger-scale investigation found eight out of ten cases tested in a psychophysical investigation of spatial frequency sensitivity showed evidence for blindsight. The results of an ongoing study into visual training following cortical blindness has demonstrated evidence for blindsight in 20 out of 23 cases tested to date.

The Importance of Specific Stimulus Parameters

Closely related to the issue of incidence of blindsight is the issue of stimulus parameters. Systematic investigation into the factors that affected visual sensitivity in the case of GY began to reveal the importance of stimulus parameters in determining whether or not blindsight may be demonstrated. Despite previous demonstrations of blindsight in GY, when he was retested by a different group of researchers, no evidence of blindsight was reported. It later emerged that the choice of stimulus parameters for the experiments was crucial. In the negative study, the diameter of the visual stimulus was 8.4°, whereas an increase in size to 13.2° resulted in near perfect performance. Similar findings were also found with the temporal characteristics of the stimuli as well as stimulus contrast (e.g., chance levels of detection at a contrast of 15% compared to 98% correct performance at a contrast of 84%). These findings suggested that in GY, these changes to stimulus parameters resulted in a dramatic improvement in performance. These results raise important questions about the incidence of blindsight and detection/screening methods as they clearly demonstrate how such abilities could be overlooked during testing. As mentioned previously, a more recent, larger-scale, systematic investigation found evidence for blindsight in 20 out of 23 cases tested. In these cases, blindsight was characterized by sensitivity to a narrow-range of low spatial frequencies (<4 cycles per degree) and temporal frequencies between 5 and 20 Hz. Stimulus size and contrast were also important for successful detection.

Criticism and Controls – Does Blindsight Exist and is it just Normal Degraded Vision?

From the outset, once details of blindsight within clinically (perimetrically) blind scotoma were reported, a number of criticisms and questions about experimental controls were raised and continue to be discussed. Indeed, some researchers concluded that an adequate case for blindsight had not been made, and some still resolutely hold this position. In the light of continued research, in part generated in response to criticism, this view now seems extreme. However, a number of important methodological issues require discussion for a full understanding of the research evidence for blindsight. At times, a range of criticisms has been leveled against research into blindsight, both in general and in the case of specific experimental findings. In terms of experimental methodology, artifacts as a result of poor fixation and stray light either from within the eye (intraocular) or from visual stimuli (extraocular) require consideration. In terms of the theoretical explanations for experimental findings a number of issues have been highlighted:

Is blindsight simply degraded/near threshold normal vision?

Is blindsight only the result of differences in decision criteria?

Is blindsight mediated by residual 'islands of V1'?

A number of control methods have been used by experimenters which include fixation monitoring, stimuli of brief duration, use of the optic disk as a control presentation position, use of various luminance masking methods, and comparison with patients with pregeniculate lesions.

Experimental Methodology

One of the first criticisms raised in discussions of blindsight is the possibility that the patient had faulty eye fixation and/or made inadvertent, unmonitored saccades toward the visual target. If a patient moves his/her eyes, visual performance loses its scientific interest as intact areas of visual field are used. Clearly, rigorous controls are required to ensure accurate fixation. Apart from the early investigations, eye fixation is usually controlled throughout testing by an infrared camera. Stimuli presented for brief durations shorter than the saccadic onset latency (minimum time needed to initiate an eye movement to a visual target) can also rule out the possibility of eye movements accounting for performance.

Clearly, if blindsight is shown to be attributed to stray light scattering into intact areas of visual field or within surfaces of the eye, the results of blindsight experiments would not reveal anything novel about visual processing and would not be of

particular scientific interest. For this reason, the question of whether discriminable light energy has been scattered into intact parts of visual field or within surfaces of the eye is extremely important. A number of experiments have been carried out to investigate the potential effect of light scatter on performance. Taken as a whole, the implications of the investigations into light scatter in blindsight suggest light scatter can be successfully controlled experimentally and cannot be used as an adequate explanation for many cases of residual visual processing following striate cortex damage.

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Is Blindsight Mediated by Residual 'Islands of V1'?

p0155 It follows that, if blindsight is mediated by residual 'islands' or 'tags' of striate cortex, the study of blindsight would be limited to the study of degraded vision and would not advance knowledge of visual function or consciousness greatly. This point has been raised by some researchers and there is experimental evidence suggesting that in some cases, visual abilities following striate cortex damage may be mediated by some residual islands of striate cortex. More recently it has been acknowledged that variation between cases is likely and some cases of blindsight may be mediated by such remnants of functioning striate cortex. Despite this caveat, there are a number of reasons why this explanation is unlikely to apply to all cases of blindsight. The idea that blindsight abilities are mediated exclusively by residual 'islands' of remaining striate cortex cannot apply to the considerable body of animal evidence in which the completeness of the lesion and the absence of striate cortex can be confirmed as well as the cases in which the entire hemisphere was removed. The role of subcortical mechanisms was also underlined by evidence that, in two monkeys, following unilateral striate cortex lesions, the ability to detect and saccade toward light targets gradually recovered unless there was an additional superior colliculus lesion. Although it is not appropriate to assume a complete 'mapping' of the monkey visual system onto the human visual system, the close similarities between these two systems, which have been demonstrated anatomically and behaviorally, suggests the applicability of the animal evidence in relation to investigations of blindsight in humans.

p0160 Evidence from patients who have undergone complete removal of all cortex from one hemisphere (hemispherectomy), usually as treatment for severe epilepsy, is very relevant as it can help to determine whether subcortical pathways alone can sustain blindsight in the absence of cortical input. Evidence for successful localization, pattern discrimination and motion detection has been reported. Some researchers have presented evidence suggesting that light scatter may have been a factor in some of these experiments. Other evidence, reporting interactions between the intact and impaired hemifield cannot be explained on the basis of light scatter and instead point toward potential subcortical mediation in some cases.

p0165 The range of blindsight abilities demonstrated in the case of GY, for example, his ability to follow the path of motion of a small spot moved through a range of trajectories throughout different areas of his VFD, cannot be explained based on small islands of vision within the damaged striate cortex (these capabilities would require a considerable number of islands!) There is also substantial anatomical evidence from a high-resolution computed tomography scan, magnetic resonance imaging (MRI) scan as well as functional imaging (PET and functional magnetic resonance imaging (fMRI)) which do not show any residual 'tags' of striate cortex, only an area of spared tissue situated at the occipital pole which is consistent with GY's area of macular sparing shown by perimetry. Functional imaging data from another frequently tested case, FS, suggests that blindsight is not dependent on islands of preserved tissue in striate cortex. When a large, flickering stimulus was presented to assess visual responsiveness in V1, activation was found in the contralesional (opposite side to lesion) visual cortex and ipsilesional (same side as lesion) extrastriate cortex. Crucially, no stimulus-related MRI changes were shown in striate cortex on the side of FS's brain injury.

Decision Criterion

s0080

p0170 Blindsight can be demonstrated in the form of a dissociation between visual performance in two different paradigms/tasks, namely clinical perimetry and FC tasks. In humans, the apparent discrepancy between an area of clinically blind visual field and the ability to make some form of

visual discrimination was only revealed by the implementation of 'animal type' FC methodologies. The basis of clinical perimetry is a 'yes-no' (yn) task in which one of two possible stimuli (target or blank) is presented on each trial and the participant's task is to judge which one was presented. This task allows the participant the freedom to say 'blank' or 'no stimulus' on every trial presentation if they wish to do so. Consistently replying with 'no' is what one would expect if they are subjectively unaware of visual stimuli throughout the task. In an *m* Alternative FC task (*m*AFC), each of *m* different stimuli is presented at every trial and the participant has to judge which of m intervals contained a specified stimulus, either in a temporal or spatial (i.e., localization) interval. This paradigm ensures that the participant is effectively forced to make a judgment, for example, between two temporal intervals, as the option to judge 'no stimulus' simply does not exist. This facilitates the revelation of above-chance detection or discrimination performance where it exists, in the absence of subjective awareness (generally required for yn task). Blindsight is characterized by the dissociation in response between the two paradigms, for example, performance levels of *c.* 0% detection in yn paradigm compared to >90% correct discrimination in the FC task (despite the denial of subjective awareness throughout testing). One of the criticisms of blindsight theory is that the apparent dissociations between yn responding in perimetry and FC performance could result from the use of different decision/response criteria in the two tasks. The implication of this assertion is that the distinction between blindsight and normal, near-threshold vision would not be clear, which implies that the study of blindsight would not add anything to the understanding of mechanisms of visual awareness that could not be drawn from the investigation of normal participants operating at the lower limits of their vision.

p0175 According to signal detection theory (SDT) the judgment of a participant in a detection or discrimination task depends not only on his/her sensitivity (d') but also on his/her response criterion/bias (the tendency to select one or other of the stimuli, irrespective of sensitivity). In SDT, sensitivity is calculated independently of response bias and vice versa. However, in the majority of blindsight research percent correct is used to denote performance which, it can be argued, represents performance accurately only in the absence of response bias. In contrast, 2AFC tasks are criterion free as any bias reflects a bias to one or other interval rather than to one or other stimulus. This raises the potential issue of whether the dissociation reported in blindsight between perimetry and FC tasks is due to a difference in response criteria between the two tasks. This has been tested directly in GY during yn and FC detection of static and moving stimuli. GY's response criterion differed significantly between yn and FC responding, and the difference was sufficient to result in a blindsight-type dissociation with bias-sensitive measures of performance. When measured independently of bias, GY's sensitivity to static targets was greater in the FC compared to the yn task (in contrast to normal control participants), but GY's sensitivity to moving targets did not differ. These results suggested that differences in response criterion could account for dissociations between yn and FC detection of motion stimuli, but not for static target presentations. This may explain the trend for blindsight cases to report increased awareness in response to motion stimuli. Importantly, these results also suggest that blindsight is not qualitatively the same as normal, near-threshold vision and that the neural mechanisms for pattern and motion-detection in blindsight may differ.

p0180 The question of decision criterion/response bias has also been addressed by altering the proportion of stimuli to blank trials in perimetry. Despite variations in response criterion, the patient's ability to detect stimuli remained essentially impervious to such variations, again suggesting that the discrepancy between subjective awareness and detection ability cannot be attributed to a difference in response criterion.

Is Blindsight Normal, Degraded Vision? s0085

p0185 One of the objections to blindsight as a phenomenon is the argument that it is qualitatively the same as normal vision but quantitatively weaker. This issue is important in relation to the mechanisms mediating blindsight as well as potentially providing an insight into the mechanisms associated

with visual awareness. Participants have been reported to localize very briefly presented targets by apparently guessing as they reported being unaware of the stimuli. It has also been reported that when participants are asked to report/guess the position of a mixture of subjectively visible or invisible targets (choice of four locations), they accurately report the position of the targets despite remaining subjectively unaware of it and rating their confidence as poor. Despite these reports of localization in the absence of awareness in participants with intact vision, these findings have been difficult to replicate. Other studies have reported high correlations between localization and confidence. Although the investigation of perception in the apparent absence of awareness in participants with intact vision may have a role in blindsight research, the claim that blindsight is normal, degraded vision does not appear to be supported.

What is Mediating Blindsight?

The geniculostriate pathway (retina – dorsal lateral geniculate nucleus – primary visual cortex), the pathway disrupted by damage to primary visual cortex appears to make the largest contribution to conscious vision. To date, around ten different pathways from the eye to different regions of the brain have been discovered (**Figure 5**), providing a number of possible alternative routes for the processing of visual information in the absence of the geniculostriate pathway. A number of neuroimaging studies have been carried out, some highlighting the role of the superior colliculus in mediating the unconscious (Type I) visual capacity. In addition to the potential role of other visual pathways, there is also evidence for the contribution of primary visual cortex from the intact hemisphere is some cases. The potential role of extrastriate cortex on the same side of the brain as the lesion has also been highlighted. Recently, the use of transcranial magnetic stimulation to simulate blindsight in observers with normal vision by temporarily disrupting visual cortical electrical activity has provided further insights into the processes underlying unconscious visual perception in blindsight.

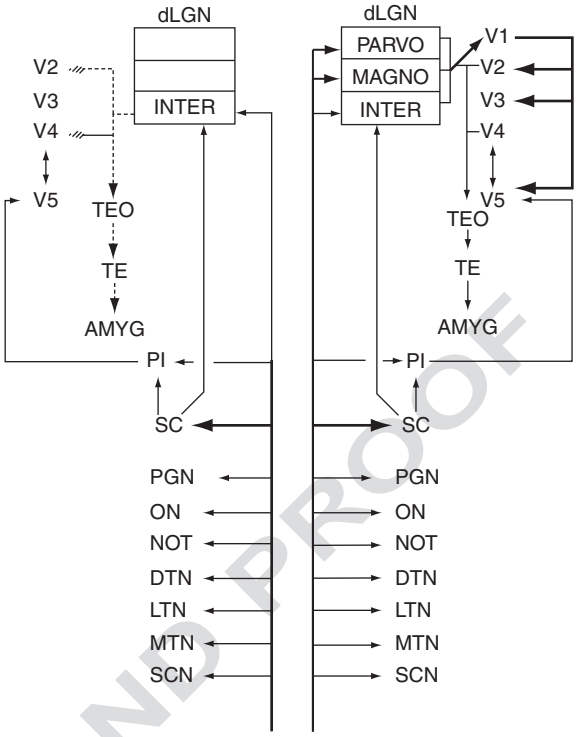

Figure 5 Schematic diagram of the ten established pathways from the eye to their retinorecipient targets in the brain. Only some of the forward projections from there are shown. The right side of the diagram shows the normal arrangement, and the left side the effect of removing striate cortex, V1. Thicker lines and arrows indicate heavier projections. For simplicity the many onward cortical pathways from V2, V3, V4, and so on are not shown, except those destined for the amygdala. Note especially on the degenerated side that the visual input to the ventral processing stream is severely impoverished but that the pregeniculate nucleus has expanded whereas the olivary nucleus has contracted. Labeling from bottom upward: SCN, suprachiasmatic nucleus; MTN, LTN, and DTN, medial, lateral, and dorsal terminal accessory optic nuclei; NOT, nucleus of the optic tract; PGN, pregeniculate nucleus; ON, olivary nucleus; SC, superior colliculus; PI, inferior pulvinar; dLGN, dorsal lateral geniculate nucleus. Reproduced from Cowey A (2004) The 30th Sir Frederick Bartlett lecture. Fact, artefact, and myth about blindsight. *Quarterly Journal of Experimental Psychology, A* 57(4): 577–609.

What Does Blindsight Tell us about Conscious Awareness?

Blindsight has highlighted that although primary visual cortex is important for conscious visual awareness, consciousness itself cannot be localized

to one brain area. Neuroimaging evidence highlights the likely interaction between several different brain regions, particularly several frontal areas. fMRI has been used to compare brain activity in GY when performing visual discriminations with and without conscious awareness. Comparison between 'aware' and 'unaware' modes of processing revealed that rather than an isolated 'center' for visual awareness there was a shift in neuronal activity from cortical areas, particularly dorsolateral prefrontal areas and extrastriate area 18 (both hemispheres) when GY was aware of the stimuli; to subcortical activity including activation of the superior colliculus and right medial and orbital areas when he was unaware of the stimulus. So, the areas that appear to be differentially involved in visual awareness are located far from the classic areas of visual cortex, supporting the view that visual consciousness involves frontal sites of activity. Imaging evidence from participants with normal vision reveals activation associated with visual awareness in multiple extrastriate ventral, parietal, and prefrontal cortical areas. There is also interest in the potential importance of connectivity between regions involved in visual awareness.

Visual Rehabilitation and Blindsight

p0200 As already described, early expectations for recovery of vision following V1 damage in humans were limited. Rehabilitation strategies have focused on compensatory techniques rather than attempting the restoration of vision within the blind field: for example, use of prisms in spectacles in order to superimpose a limited part of blind field image onto the intact field. Another way that patients can benefit from saccadic training involves learning to scan more efficiently and regularly into the blind field. This technique has been used on computer screens as well as large scale patterns presented in the immediate environment and has shown some success in a number of cases. Following the success of saccadic training in nonhuman primates, extensive research has been carried out with the aim of shrinking the field defect by training patients to saccade to light stimuli presented on the sighted/blind field boundaries. Repeated stimulation of such boundaries using light targets (without the saccadic task) has also been shown to result in improved visual sensitivity. Nevertheless, intrinsic to all studies that rely on stimulation of sighted/blind field borders, there are some methodological concerns that small gaze shifts or eccentric fixation may contaminate some of the data, exaggerating the extent of the recovery. More recent studies have shown that visual stimulation deep within the field defect, using stimuli which are optimally configured to elicit blindsight performance (low spatial and high temporal frequency structures), can lead to significant increases in visual sensitivity. In some cases after months of stimulation, blindsight performance may be manifest. The rate of recovery increases if positive feedback is provided after each successful detection. Research is continuing into these recent, exciting developments. It will be interesting to continue to explore which factors are important in facilitating this recovery of function.

p0205 It is thought that the recovery of function following repeated stimulation may be mediated via two possible channels which may not be mutually exclusive. The signals within surviving neurones after brain injury may be strengthened and therefore their response rises to above threshold levels for further processing in other brain regions. Alternatively the signals along alternative pathways (outlined above) may be utilized, leading to successful detection. Further research is needed to establish which one (or both) of the above-postulated mechanisms is likely to mediate the improvement.

p0210 Blindsight is an area which appears to attract strong views and opinions. It is an area where theories and practices of science are proposed, refuted and modified. The focus of much of the human research into blindsight on single case studies or small number of cases provides important examples of the possibilities, but also raises questions about the extent to which these findings can be generalized. There is much more to be discovered, confirmed, disproved and understood fully. It is hoped that the reader will wish to be critical, curious and energetic in discovering more about blindsight!

See also: Neural Basis of Perceptual Awareness (00054); Perception: Implicit, Subliminal (00058).

Suggested Readings

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Biographical Sketch

Ceri T Trevethan graduated with a first class honours MA degree in psychology from the University of Aberdeen in 2000. Ceri subsequently obtained a PhD in psychology in 2004, investigating the spatial and temporal characteristics of blindsight performance. During this time she also worked as a research assistant with Arash Sahraie on a project investigating rehabilitation of visual deficits following brain injury. This was followed by a 2-year BBSRC funded postdoctoral fellowship. Ceri is currently a trainee clinical psychologist with National Health Service, Grampian and an honorary research fellow at athe University of Aberdeen.

Arash Sahraie obtained a PhD in optics and vision sciences from City University, London in 1993 under supervision of Prof. John Barbur. He then continued with 5-year postdoctoral assistantship to Profs. John Barbur and Larry Weiskrantz before moving to University of Aberdeen, Scotland in 1998. He is currently professor in vision sciences at College of Life Sciences and Medicine, University of Aberdeen. Arash Sahraie has a long-standing research interest in neuropsychology of vision and rehabilitation of visual deficits following brain injury.