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Blindsight

Introductory article

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CONTENTS

What is blindsight?

History

Experimental work on blindsight

Relevance of blindsight for consciousness and cognitive science

Patients with damage to primary visual cortex or its afferents report that they are blind in the area of the visual field corresponding to this damage. Blindsight refers to the ability demonstrated by some of these patients to perform a variety of visual tasks despite denying awareness of the stimuli to which they are responding – a dissociation between performance and consciousness.

WHAT IS BLINDSIGHT?

Blindsight is the term given to the remarkable abilities found in a small number of neurological patients who have damage affecting striate cortex, the first cortical area of the brain which normally processes visual information. Despite its rarity, the condition has profound implications for our understanding of consciousness. As a consequence of its rarity and the importance of its implications it is a condition surrounded by controversy. (See **Blindsight, Neural Basis of**)

As a result of their brain damage patients with blindsight deny being aware of visual stimuli in the area corresponding to their damage. For example, a patient with damage to the left side of striate cortex reports that he cannot see stimuli presented to the right of his direction of gaze. When tested using standard procedures these patients are classified as clinically blind in the area corresponding to their damage (that is, they have a scotoma). However, if the patients are tested in a way which forces them to make decisions about stimuli presented in their scotoma then, even though the patients deny seeing

anything and maintain that their decisions are simply guesses, they usually make the correct response to the unseen stimuli on a variety of visual tasks.

Blindsight, then, is the dissociation between awareness of visual stimuli and the ability to respond appropriately to them found in patients with damage to striate cortex or the neural connections leading directly to it. It is clear that blindsight subjects can detect whether a spot of light within their scotoma accompanies an auditory signal, whereabouts it is, and, if it is moving, in which direction and how fast it is going. The evidence for more complex residual abilities is less strong.

HISTORY

Striate cortex gets its name from a fine white line identifiable near its surface in slices of the brain. This 'stripe of Gennari', discovered in 1782, was the first evidence that the anatomy of the cortex was not uniform and hence that different areas of cortex may be specialized to serve particular functions. Striate cortex lies at the occipital pole of the brain; in humans much of it is hidden on the adjoining lateral surfaces of the cerebral hemispheres (Figure 1).

In addition to being the first identified anatomically specialized cortical area, it was also the focus of the earliest work on functional specialization. Observations of stroke patients dating back to the 1850s suggested that damage to the brain's occipital pole had specific effects on vision. Towards the

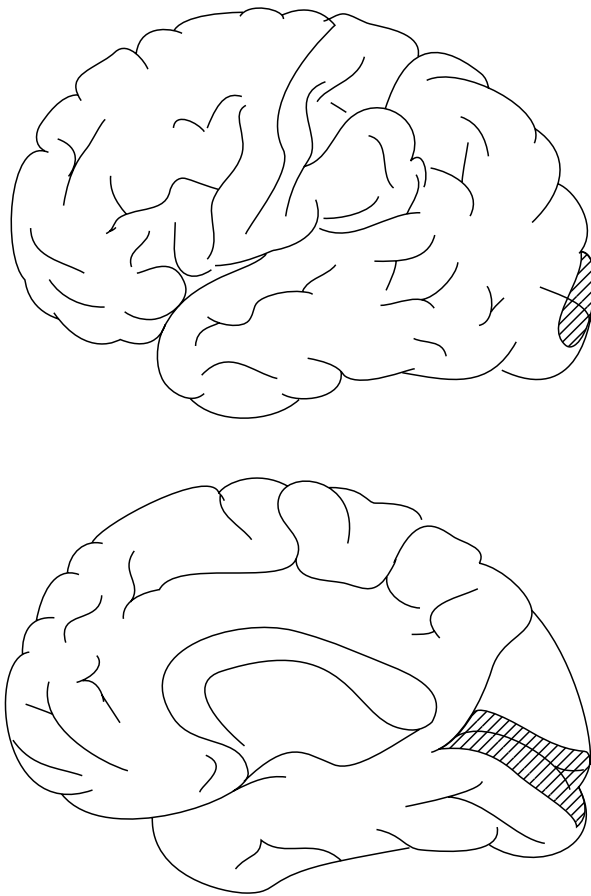


Figure 1. Lateral (upper panel) and medial (lower panel) views of the human cerebral cortex showing primary visual cortex (hatched). Note how little of primary visual cortex is exposed on the surface of the brain. Most of primary visual cortex lies on the medial surface of the brain and is therefore hidden between the two cerebral hemispheres.

end of the nineteenth century, experiments on monkeys showed that lesions of occipital pole large enough to include all of striate cortex rendered animals blind, and it was generally agreed that the occipital lobes were indispensable for vision. From the mid-1930s, however, it became apparent that animals with lesions restricted to striate cortex and not impinging upon other parts of the occipital lobes retained some visual abilities – they could be conditioned to respond to flashes of light and could follow moving spots of light with their eyes.

Starting in the mid-1960s, Nicholas Humphrey studied a single monkey, named Helen, who had bilateral striate cortex lesions. On the basis of many years of observation, Humphrey concluded that Helen retained many (but not all) visual abilities, despite her lesion. For example, she would

routinely pick up very small objects with great precision; however, it was clear that she could not identify what these objects were until she explored them with her mouth. Helen apparently retained the ability to detect and locate visual stimuli despite her lesion, but she could no longer identify them.

Although the animal studies of Humphrey allowed the visual abilities remaining after striate cortex lesions to be identified, they could not provide any insight into the subjective nature of visual experience without striate cortex. To do so one must be able to ask a human patient lacking striate cortex to describe what they see. Such patients had been studied for many years and reported that they saw nothing in the region corresponding to their brain damage. One exception, to which we will return later, was the perception of movement. During the First World War, George Riddoch found that soldiers with injuries to the occipital cortex, although blind to stationary stimuli, reported that vigorously moving stimuli did elicit visual experience. Studies of wounded soldiers feature prominently in the history of visual neuropsychology. Careful collation of the locations of gunshot wounds and areas of lost vision in soldiers from both World Wars provided the evidence for maps of the representation of the visual field in striate cortex.

In 1973 the team of Ernst Pöppel, Richard Held, and Douglas Frost, working at Massachusetts Institute of Technology, decided to test whether soldiers (and one stroke patient) with visual scotomata as a result of damage to the visual cortex could, nevertheless, move their eyes so as to direct their gaze at spots of light presented in their regions of blindness. Pöppel, Held, and Frost were prompted to attempt this experiment by earlier work which, amongst other things, had shown intact responses of the pupil and intact optokinetic nystagmus (a slow drift of eye-gaze in one direction, interrupted by occasional flicks back in the opposite direction, induced by presentation of a continually moving pattern) in patients with occipital lesions. Since both of these responses are mediated by midbrain structures, it might be the case that neural pathways transmitting information directly from the retina to the midbrain without passing through striate cortex could support a range of simple visual abilities in these patients. As at least one circuit used in the control of eye movements is entirely subcortical, eye-movement control was a clear candidate for such a potentially spared function. Although the patients found the task puzzling, one remarking ‘how can I look at something

I haven't seen', there was a consistent relationship between the location of visual targets and the eye movements the patients produced when asked to look at the locations where they 'guessed' these targets had been presented. The appropriate behavioral response of patients to visual targets shown in this task, coupled with their complete denial of awareness of those targets, is acknowledged as the first systematic experimental demonstration of blindsight.

The term was not, however, coined until a year later when Lawrence Weiskrantz described similar work he had carried out on a patient who, as a result of surgery to alleviate pain caused by abnormalities in the blood supply to the occipital pole of the brain, had lost most of the striate cortex on one side of his brain. Weiskrantz found that not only did this patient (known as DB) move his eyes appropriately towards unseen targets, but he could also point towards target locations accurately with his finger, detect the presence of a luminance grating (a smoothly varying pattern of light and dark stripes), discriminate the orientation of lines and discriminate between the shapes 'X' and 'O' in his blind field, all while denying any visual experience. Blindsight was clearly a complex phenomenon requiring considerable work, both to evaluate the range of visual functions spared after damage to striate cortex, to determine the extent of the dissociation between behavior and visual consciousness, and to test models of the anatomical basis of residual function.

EXPERIMENTAL WORK ON BLINDSIGHT

Four questions need to be addressed in the experimental study of blindsight. Apart from evaluating the anatomical basis of blindsight, the range of spared functions, and the dissociation between behavior and awareness, it is crucial to demonstrate that blindsight is a real phenomenon and that the results obtained cannot be explained by experimental artefacts which allow subjects to perform tasks using the intact portion of their visual field or in some other unintended manner.

Artefacts

Blindsight patients are quite rare. Moreover, in virtually all reported cases, visual field loss is not total. These patients therefore retain normal conscious vision in part of their visual field. The residual visual abilities of interest in blindsight are those used in response to stimuli presented in the

blind portion of the visual field. If, however, visual targets presented to a patient's scotoma also illuminate their intact visual field, then any response they make is not truly indicative of blindsight. Light from a target presented within the scotoma may reach intact areas of the visual field as it is scattered from objects in the room where testing is being conducted or as it is scattered by the internal structures of the eye.

The first of these potential artefacts is relatively easy to detect and control, the second much harder. One approach that has been taken is to use the area of visual field within the scotoma corresponding to the blind-spot in a control condition. The blind-spot is the small area of retina where photoreceptors are absent as nerve fibers from receptors throughout the rest of the eye converge to leave the eye as the optic nerve. A target presented exactly within the blind-spot could not therefore directly activate any pathway, cortical or subcortical. One would therefore expect that the subject's ability to respond appropriately to a target will be eliminated if the target is presented in the blind-spot, whether the subject has blindsight or has an undamaged cortex. If, however, the subject's response to a target depends upon light scattered to remote (and intact) portions of the visual field, it should not matter whether the target is presented over the blind-spot or an adjoining area of retina – the presence of receptors at the target location is neither here nor there. The performance of blindsight subjects does indeed fall to chance when targets are presented to the blind-spot, suggesting that residual performance in blindsight does not depend upon a scattered light artefact. This does not, however, mean that scattered light can be ignored. It may still provide cues to a subject unless steps are taken to control it. The most common of these is to use dark targets against a bright background wherever possible, and to flood the subject's intact visual field with bright light.

Light-scatter is not the only means by which information from stimuli intended to reach the scotoma alone can travel to intact regions of the visual field. The most common method of presenting stimuli to patients is with a computer display screen. Stimuli presented in one part of a computer display can produce unintended but visible effects in other parts of the display. Presentation of a bright spot, for example, can cause a small brightening in a narrow horizontal band at the same height as the spot across the entire width of the screen. Care must, therefore, be taken to mask portions of the screen visible outside a patient's scotoma when using such stimuli.

Anatomical Bases of Blindsight

The processing of visual stimuli starts in the array of interconnected photoreceptors of the retina at the back of the eye (Figure 2). The most prominent output from the retina projects to a midbrain structure called the lateral geniculate nucleus (LGN) and from there to striate cortex. This is not, however, the only output from the retina which projects to many other structures. Initially it was supposed that blindsight was mediated by such structures which controlled basic responses to light without any cortical involvement. For example, the superior colliculus can control reflexive eye movements which direct gaze towards a visual target without involving cortex. Although subcortical circuits mediate very specialized responses, blindsight patients might learn to monitor these specialized responses in the course of performing more general tasks. It might, for example, be possible to monitor the location towards which one is about to move one's eyes and use this information to choose whether or not to press a button even if the eye movement itself is suppressed. According to this

scenario, blindsight may be mediated by subcortical visual pathways.

Although the bulk of visual input to the cortex passes through the striate cortex, there are ways in which visual information can reach the cortex while bypassing the geniculo-striate route. The superior colliculus sends projections, via the pulvinar, to a number of cortical areas involved in vision (V2, V3, V4, and MT). These are parts of cortex involved in visual processing which normally receive their major input via the striate cortex. Since these areas can receive visual input in the absence of the geniculo-striate projection, it is possible that blindsight may be mediated by visual pathways outside the striate cortex.

In addition to mediation by subcortical or extrastriate cortical routes, there remains the possibility that damage to striate cortex in blindsight patients is not, in fact, complete. Rather than demonstrating that circuits other than the major geniculo-striate route support visual function but do not give rise to visual awareness, residual visual function in blindsight would then essentially be a demonstration that the magnitude of stimulation required to

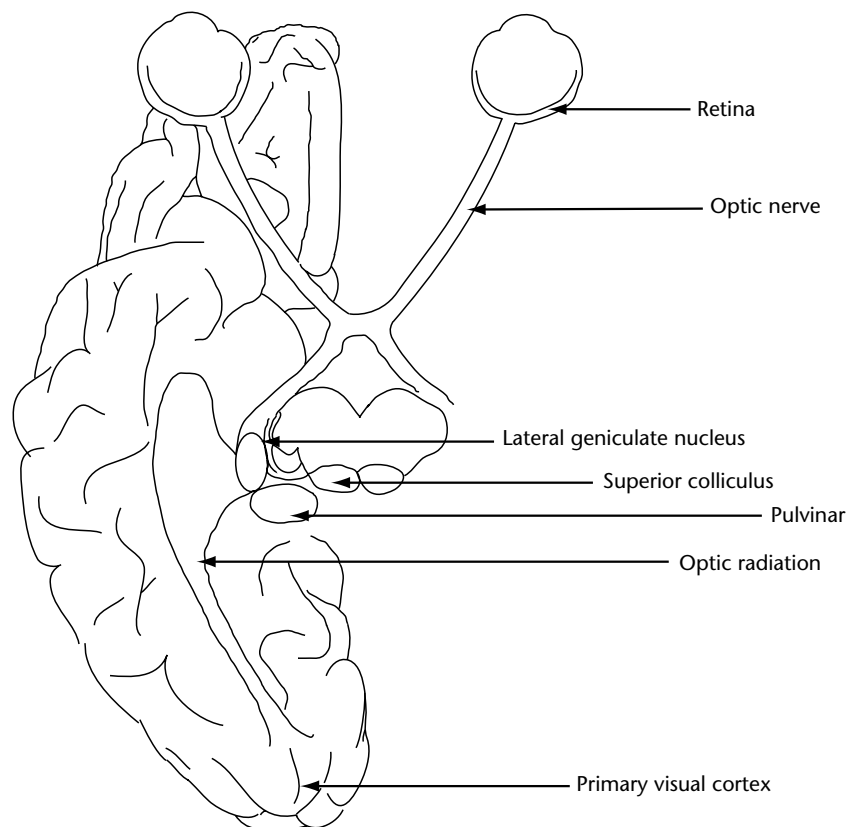


Figure 2. A basal view of the human brain showing major components of the visual system which may be involved in the mediation of blindsight.

evoke awareness from the geniculo-striate system is greater than that required to support simple behavioral responses. Blindsight would not, under these circumstances, be a particularly special phenomenon since it would differ little from the abilities of normal subjects when presented with stimuli near the limits of their visual abilities (e.g., very faint or very short duration stimuli).

A number of studies have been made of patients who have had small spared regions of striate cortex surrounded by damage. These patients did not experience stimuli falling in these spared regions, yet, as in blindsight, they could perform simple visual discriminations in these regions. Can such spared cortex explain the apparently extensive region of blindsight found in other blindsight subjects? Patches of residual vision surrounded by areas of complete blindness might not be revealed in most studies if random eye movements fortuitously brought stimuli into a region of the retina which activated a patch of spared cortex. If, however, one ensures that eye movements cannot affect the location in the cortex which a visual stimulus potentially activates, then any patchiness should become apparent. It is possible to do this by using eye-movement measurements to yoke stimulus position to the direction of gaze. A study using this technique in a patient with blindsight covering a large proportion of one visual field did not reveal patches of residual vision surrounded by blindness, suggesting that an explanation of all blindsight in terms of islands of spared cortex is untenable.

Diffuse, as opposed to patchy, subtotal damage is harder to detect behaviorally. The undamaged neurons in a diffusely damaged region of cortex should, however, still be metabolically active. Functional neuroimaging, which detects changes in blood flow or blood oxygen levels indicative of metabolic activity, has not revealed activity in the striate cortex of blindsight patients when a visual stimulus was presented, although changes did occur in extrastriate cortex.

If blindsight relies on a visual pathway used in normal vision, albeit seriously damaged, the implication is that blindsight should be like very poor normal vision. The apparent dissociation between the abilities of blindsight patients and their reports of awareness may be explained in terms of a change in their willingness to report that they have seen a stimulus – not a surprising change given their knowledge that they have a serious visual impairment. It is, however, possible to disentangle the effects of such biases from the underlying visual sensitivity. The results of such experiments

indicate that, for normal subjects presented with stimuli near the limits of visual ability, there is no difference between mechanisms which serve conscious report and those which serve the ‘forced-choice’ discrimination tasks typically used in assessing blindsight. A similar comparison in a blindsight patient showed quite different properties for conscious report and forced-choice discrimination, indicating behaviorally that blindsight is not simply near-threshold normal vision.

Ingenuous experiments have been devised which show that monkeys with unilateral visual cortex lesions treat stimuli in their ‘blind’ and normal visual fields quite differently, even though they are quite capable of making behavioral responses to those blind-field stimuli. The monkeys were first trained to point at visual targets presented in either their blind or normal hemifields and the minimum brightness contrast required was measured for each hemifield. The target contrasts were then adjusted so that they easily exceeded these thresholds for the rest of the experiment. The monkeys now learned a new task in which they had to make different responses depending on whether one or two stimuli were presented. They performed accurately when both stimuli were presented in the intact hemifield. However, when two targets were presented but one of them fell in the lesioned hemifield, the animals made the ‘one target’ response. They behaved as if they had seen only one target even though the target they ignored was easily bright enough for them to point at accurately.

There is no question that these monkeys had no spared cortex – striate cortex was surgically removed and the completeness of the damage verified at the end of the experiment. Unless one accepts that there are fundamental differences in the anatomy of vision and awareness between monkeys and man, these results suggest that blindsight cannot rely on spared striate cortex.

Blindsight and Awareness

Although blindsight is the dissociation between awareness of visual stimuli and the ability to respond appropriately to them, it is not the case that blindsight subjects are unaware of all visual stimuli presented in their scotoma. We have already seen that injuries to the occipital cortex leave patients able to report conscious experience of vigorously moving stimuli, as Riddoch discovered at the end of the First World War. Blindsight subjects also report some experience of rapidly moving stimuli or stimuli with sudden onsets or offsets. It is not clear whether these experiences are anything like

visual sensations. Blindsight subjects differ in the descriptions they give of these experiences, ranging from a feeling that the response they are making is not quite a guess, to descriptions of movement being like a black hand moving across a black background.

Some authors have argued that the fact that blindsight subjects sometimes have an experience induced by visual stimuli, even if it is quite dissimilar to a normal visual experience, invalidates the contention that visual processing and visual consciousness are dissociated in blindsight. Weiskrantz has suggested that blindsight be divided into two subtypes:

- Type 1 blindsight conforms to the 'classical' definition and is residual visual function in the absence of any acknowledged awareness.
- Type 2 blindsight is defined as residual vision accompanied by an acknowledged experience of events in the blind field but in the absence of acknowledged 'seeing'.

It can be hard to draw broad conclusions about the nature of awareness from type 2 blindsight, as distinguishing between visual and nonvisual experience involves a difficult subjective decision about the nature of experience. Interesting results have, however, been obtained by comparing brain activation in blindsight patient when they do and do not report this type 2 nonvisual experience. These results suggest that frontal areas of the brain are activated during the experience of knowing but not seeing, whereas subcortical structures are primarily active during trials in which there is no report of experience whatsoever.

It is important to point out that the distinction between type 1 and type 2 blindsight is not based on performance. It is possible to show that the ability to perform a task and awareness of the stimuli involved are quite dissociated in type 1 blindsight. For example, as task difficulty is varied the performance of blindsight subjects can increase from chance to being near 100 percent correct without any change in their reported absence of awareness. With appropriate stimuli the dissociation in blindsight between awareness and performance remains unequivocal.

Residual Abilities in Blindsight

The early work of Weiskrantz with patient DB showed that a range of visual functions were spared in blindsight. Since then some controversial new claims have been made about the abilities of blindsight patients. First we shall look at some uncontroversial findings.

There is little doubt that blindsight patients can localize single bright or dark visual targets in their blind fields. Similarly, they can discriminate when such targets appear in a task where the subject is required to indicate in which of two time intervals a target is presented (a temporal two-alternate forced choice task). There is evidence from a number of sources that blindsight patients retain some ability to discriminate the color of stimuli presented in their blind fields, although they are impaired in comparison with normal subjects. Blindsight patients can also detect the presence of a pattern of alternating bright and dark stripes even if the average brightness of the pattern does not differ from the background. Their ability to detect these patterns is much poorer than normal in their blind field – they are unable to detect very fine or faint patterns of stripes. The ability to discriminate between stimuli composed of lines with different orientations is also preserved, albeit in a severely impaired guise and with some variations between patients. GY, for example, can discriminate the orientation of single lines but not patches of stripes. His performance becomes poorer than normal as the lines get shorter than 10 degrees of visual angle.

The ability to discriminate the orientation of lines is one of the basic building blocks of form perception. The extent to which blindsight patients can discriminate between complex forms is, however, a vexed question. Weiskrantz found that his patient DB could discriminate reliably between circles and crosses. As he showed, however, this discrimination may be based on discrimination of differences in the components of these shapes, such as the orientation of the line segments that make them up, rather than discrimination of the shapes *per se*. This is borne out by findings that blindsight subjects fail to discriminate between different shapes constructed from the same line segments, for example equilateral triangles with the point either at the top or at the bottom (Δ versus ∇) and are poor at discriminating between rectangles differing in the ratio of side lengths but not orientation.

Early results indicated that form discrimination is absent or severely impaired in blindsight. More recent studies which have tested form-processing abilities indirectly appear to tell a different story. Studies of the manual responses of blindsight subjects to objects placed wholly or partially in their blind fields indicate that shape, orientation, or size properties which could not elicit appropriate verbal or forced choice discriminations nevertheless influenced hand movement and grasp. Other studies have sought to identify whether shapes presented in the blind field influence subsequent responses to

stimuli presented to the conscious good field. Although a number of groups have apparently failed to find any such effects, there have been at least two reports of positive results. In one case words presented to the blind field were reported to influence the interpretation of ambiguous words in the good field. For example, if the word 'money' was presented to the blind field then the subject was more likely to describe the word 'bank' in the good field as a financial institution than as the edge of a river. Unfortunately, relatively few short ambiguous words could be used in this study and so the result, which is of great interest given the weakness of simple form processing in blindsight, is based on relatively few observations. Further evidence derives from a study in which the similarity between the shape of stimuli (in this case single letters) presented in the blind and good fields influenced reaction time to the good field stimulus in a letter discrimination task. The most dramatic evidence supporting the existence of complex shape discrimination without awareness comes from a study on the perception of emotion in blindsight. The blindsight patient GY correctly attributed one of four emotions (happiness, sadness, fear, or anger) to video clips presented to his blind field of an actress expressing one of these emotions.

What are we to make of the apparent contradiction between the limited shape-processing abilities indicated by studies of simple geometric shapes and the abilities necessary to make the complex discriminations required in order to be influenced by letters, words, and facial emotions presented in the blind field? One possibility is that most of the latter tasks did not involve the subject in responding directly to the stimulus in the blind field. By assessing blind-field shape-processing through its effects on seen targets, subjects are relieved of the problem of making decisions about stimuli they do not believe they can see. Perhaps removing the conflict for the subjects between their conscious blindness and the demands of a task in which they must respond to stimuli they cannot see uncovers abilities hidden in direct tasks. It may also be the case that certain properties of stimuli and methods of response are mediated by specialized neural circuits. Perhaps the processing of emotion is of such basic evolutionary importance that facial cues to emotion are processed by systems independent of the brain's general shape identification system. These are open questions; at present there is insufficient evidence to come to a firm conclusion about why and whether blindsight subjects can discriminate complex shapes without awareness.

The basis of another residual ability in blindsight is also controversial. The ability to detect the direction or speed of moving stimuli has been studied in blindsight for many years, and there is good evidence that such discriminations can be made both with and without an accompanying experience (rapidly moving high-contrast stimuli are particularly likely to elicit reports of awareness). There are, however, two ways in which motion can be inferred from the stimuli typically used in these experiments. One of these is not strictly a matter of motion perception. One can infer the direction and speed of a moving dot or line by noting its position at one instant and comparing this with its position some time later. Unfortunately there are stimuli with which such a positional comparison method will not work. For example, one can construct a stimulus comprising many dots, in which each dot is displayed for only a short time before it disappears and another dot appears at a different place. If each of these dots moves in a different direction, but on average the dots move more in one direction than any other, then a normal observer will easily be able to report the average direction and speed of the pattern (this is an example of a random dot kinematogram). In some experiments (but not all) the blindsight subject GY failed to discriminate the direction of motion when stimuli which precluded the use of position comparison were used. He could, however, still distinguish moving from stationary stimuli. It is therefore not safe to assume that motion processing is fully preserved in blindsight, even though some forms of motion can be discriminated by blindsight patients.

Some recent studies indicate that residual abilities in the blind field can be modulated by processes of alerting and spatially selective attention. GY's ability to perform a spatial localization task is enhanced if the visual stimuli are immediately preceded by an auditory warning. He is also faster at responding to a visual target if it appears in the location indicated by a preceding cue. This effect can be found even when the cue itself is also presented in the blind field. Such results may have profound implications for our understanding of the relationship between consciousness and attention.

RELEVANCE OF BLINDSIGHT FOR CONSCIOUSNESS AND COGNITIVE SCIENCE

For years consciousness was a taboo word in psychology. If blindsight has done one thing for

psychology and cognitive science it is to make the scientific study of consciousness respectable once again. As well as providing insights about the relationship between processing visual stimuli and visual awareness, blindsight offers some insight into the modularity of psychological processes and the extent to which apparently complex processes can, in fact, occur essentially automatically, without awareness.

Blindsight is often used in philosophical arguments about the nature of consciousness. In particular, the apparent dissociation between access to visual information and visual experience in blindsight has been used to explore the role, and even existence, of experiences as something distinct from the properties of stimuli in the outside world, our knowledge of them, and our responses to them. One of the attractions of blindsight to philosophers is that it appears to offer a real, albeit partial, example of a favorite of the philosophical thought experiment – the zombie.

The philosophical zombie is a being whose behavior is indistinguishable from that of real people, but who is supposed to have no inner experience at all of the world in which it is behaving. These inner experiences are often referred to as 'qualia'. Thought experiments about zombies sometimes hinge on a *reductio ad absurdum*, purporting to show that presupposing the existence of zombies leads to some paradoxical difference between our observation of the world of real people and that of zombies. It is argued that if zombies and beings with inner experience differ behaviorally, zombies who lack inner experience and yet are indistinguishable from us behaviorally must be an impossibility. Since the only difference between ourselves and zombies is the presence or absence of qualia and zombies cannot exist, then qualia have no explanatory power and hence no existence outside an individual's mind. On the other hand, one might argue that inner experiences are real (it makes sense to discuss them, as I am doing here, for example); perhaps they just do not have causal consequences for the physical world. (See **Zombies**)

Blindsight appears to make zombiehood concrete. Blindsight has been used to argue that inner mental states are real and correspond to physical,

that is neural, states. In fact blindsight adds an extra twist to zombiehood – well-tested blindsight subjects come to know consciously that they respond appropriately to visual stimuli even though they do not know what they see and have no inner experience of seeing. Of course, it might be the case that blindsight people do have inner experiences, it is just that they do not know they have them. Unfortunately, many of these arguments are weakened when they either ignore some abilities of real blindsight people or use thought experiments which go far beyond the actual abilities of blindsight subjects. Philosophical consideration of the real properties of blindsight (as opposed to those of nonexistent super-blindsighters) does suggest that inner experiences are real, can be investigated scientifically, and make a difference in the real world, even if they do not solve the problem of telling us what such inner experiences are and why they feel the way they do.

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