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## *The Status of Blindsight*

*Near-threshold vision, islands of cortex  
and the Riddoch phenomenon*

**Abstract:** *In this introductory paper, we assess the current status of blindsight — the phenomenon in which patients with damage to their primary visual cortex retain the ability to detect, discriminate and localize visual stimuli presented in areas of their visual field in which they report that they are subjectively blind. Blindsight has garnered a great deal of interest and critical research, in part because of its important implications for the philosophy of mind. We briefly consider why this is so, and then go on to examine three empirical questions which have fuelled challenges to the validity of blindsight as a distinct neuropsychological phenomenon. First, is blindsight simply degraded normal vision? Second, does blindsight depend on undamaged areas of primary visual cortex? Third, does evidence that blindsight patients are aware of moving stimuli undermine the apparent dissociation between access to visual information and visual experience in blindsight? In the course of the review we introduce the four other papers on blindsight in this issue. We conclude that, although patients with primary visual cortex damage may indeed perceive moving stimuli, there is still good evidence for a dissociation between access to information and phenomenal experience which cannot be accounted for in terms of degraded normal vision or undamaged primary visual cortex.*

### **Introduction**

‘Blindsight’ is a term coined by Weiskrantz for the phenomenon in which subjects with lesions confined to the primary visual cortex retain the ability to detect, discriminate and localize visual stimuli presented in their blind fields despite a denial of acknowledged awareness (Sanders *et al.*, 1974). This dissociation between the phenomenal experience of stimuli and the ability to access information conveyed by those stimuli when required to do so, for example in a two-alternative forced choice procedure, has proved to be of considerable interest to philosophers of mind, experi-

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mental psychologists and neuroscientists (see e.g. Block, 1995; Weiskrantz, 1997; Crick & Koch, 1998). Four further papers in this issue of the *Journal of Consciousness Studies* tackle current issues in blindsight research, both empirical and philosophical. In this paper we consider some of the factors which have made the study of blindsight such an active area of research and introduce the other contributions to this issue which deal with some of these topics more specifically.

Blindsight has captured the interest of philosophers because the apparent dissociation between access to visual information and visual experience in blindsight clearly has implications for our understanding of 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. Many philosophers have attempted to resolve the issue of the nature or existence of qualia (a technical term first used by Lewis [1929] referring to the introspectible qualities of experience, such as the experience of redness, bitterness and so on) by conducting thought experiments about ‘zombies’ — beings whose behaviour is indistinguishable from that of real people, but who are supposed to have no experience at all of the world in which they are behaving (see, for example, the *Journal of Consciousness Studies*, 2 (4) [1995], for a fascinating series of papers on philosophical zombies). The crux of thought experiments about zombies is often a *reductio ad absurdum*, purporting to show that presupposing the existence of zombies leads to some paradoxical difference between our behaviour and that of zombies (e.g. the inability of zombies to develop a language for describing mental states), so, as zombies and beings with qualia differ behaviourally, zombies who lack qualia and yet are indistinguishable from us behaviourally 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. The notion that blindsight is a real example of a kind of vision-limited zombiehood is therefore enticing to philosophers. It is, however, mainly used as a stepping stone in further arguments about zombies who have residual visual abilities far exceeding those of real blindsight patients. Holt’s (1999) paper ‘The use of blindsight in debates about qualia’ in this issue of the *Journal of Consciousness Studies* examines this approach, but emphasizes the usefulness of blindsight itself, rather than analogies derived from it, in debates about qualia. Holt’s aim is not to explain what qualia are, but to tackle the more fundamental question of their existence. He contrasts the abilities of real blindsight patients with those of zombies and shows that by taking account of the limitations of residual vision in blindsight many paradoxes can be avoided.

It was originally assumed that the residual visual function in blindsight was mediated by pathways which bypassed the damaged striate cortex, either subcortically via the superior colliculus or by direct projections from the dorsal lateral geniculate nucleus and inferior pulvinar to the cortex beyond the damaged striate area (see e.g. Stoerig and Cowey, 1997). Both the phenomenon itself and its supposed anatomical bases have been challenged over the years (Campion *et al.*, 1983; Fendrich *et al.*, 1992; Zeki and ffytche, 1998). The main thrusts of these criticisms have been (a) that blindsight is not qualitatively different from degraded normal vision and (b) that blindsight may still be mediated by undamaged portions of the striate cortex rather than by distinct pathways which bypass it. Although these criticisms are independent of one another it is really the combination of the two which has the greatest potential

impact on the extent to which blindsight can inform us about the nature and bases of consciousness. Blindsight has garnered such interest precisely because it was not thought simply to be a complete but degraded form of ‘normal’ vision, but rather that the failure of the primary visual pathway through striate cortex exposed parallel pathways of visual processing which, although only capable of supporting simple forms of residual function, did so without engaging consciousness.

### **Is Blindsight Simply Degraded Normal Vision?**

The simplest explanation of blindsight is that it is poor normal vision. The apparent dissociation between performance and awareness is explained in terms of a change in subjects’ willingness to report that they have seen a stimulus — not a surprising change given a serious visual impairment. This bias against reporting awareness of stimuli affects free response tasks such as clinical perimetry, but cannot influence two-alternate forced choice tasks which inherently control for bias. Measures of subjects’ willingness to report stimuli (their response bias) and their ability to discriminate the presence of a stimulus from its absence can be dissociated in signal detection experiments where changes in a subject’s likelihood of making a positive response given the presence of a stimulus are analysed. Azzopardi and Cowey (1997) conducted an elegant study in which they measured discrimination ability independently from bias by categorizing responses both according to their correctness and the subjects’ rating of their confidence in these judgements. They compared normal subjects with the extensively studied blindsight subject GY, who suffered damage to visual cortex in a road accident as a child. Azzopardi and Cowey conducted these analyses both for a free-response task in which the subject had to determine whether or not a stimulus had been presented on each trial and a two-alternate forced choice task in which he had to determine in which of two intervals a stimulus was presented on each trial. The free-response task is subject to response bias since a subject who is unwilling to respond positively is likely to perform more poorly than one whose ability to discriminate the presence of a stimulus is just as good but who divides his responses equally between positive and negative. Response bias cannot affect performance in the two alternate forced choice task since the number of trials in which the stimulus is presented in the first interval equals that in which the stimulus is presented in the second interval. Azzopardi and Cowey showed that GY’s discrimination sensitivity differed significantly between the free-response and the forced choice tasks. There were, however, no differences between the sensitivities of the normal control subjects performing the same tasks with stimuli near the limits of their discrimination ability. These results can be interpreted as showing that the mechanisms which subservise conscious report and forced-choice discrimination of near threshold stimuli in normal subjects are the same whereas the mechanisms which subservise forced-choice discrimination and conscious report in GY differ — hence GY’s blindsight is not simply near-threshold normal vision. Azzopardi and Cowey were unable to compare GY’s blind field performance with his own normally sighted hemifield. In a recent paper Kentridge *et al.* (1999) made such a comparison. Although we were not conducting a signal detection experiment we were able to compare reports of awareness and performance in a spatial two alternate forced choice task between GY’s blind field and his good field with near-threshold stimuli. We found conditions in which he was more

likely to report awareness in his good than his blind field while his discrimination performance was better in the blind than good field. In short, when we attempted to equate the likelihood of GY reporting experience in his good and blind field by using near-threshold contrast stimuli in his good field we still found a marked difference between discrimination performance in his good and blind fields. This was all the more striking since his blind field performance was better than his good field performance even though we had not used good field stimuli of a sufficiently low contrast to reduce his awareness in the good field to the level he reported from his blind field.

### **The Anatomy of Blindsight**

The second question to address is whether blindsight relies on undamaged parts of the main visual pathway which passes through the striate cortex or whether there are separate pathways which bypass striate cortex and still support residual visual functions without engaging consciousness. Fendrich *et al.* (1992) reported findings which suggested that small spared regions of cortex surrounded by damage may indeed underlie blindsight. They tested a subject who has suffered an extensive lesion to the striate cortex. Brain imaging revealed that an apparently undamaged area survived within this lesion. Fendrich *et al.* used a technique in which the location of a stimulus on a screen is yoked to a subjects' eye-position in order to precisely present stimuli at a fixed retinal location no matter how the subject moves his eyes. Using such stabilized images they were able to show that their subject's ability to discriminate the presence of a stimulus in a two-alternate forced choice task was well above chance in a small area within his blind area or scotoma. The subject nevertheless denied conscious experience of visual stimuli within his scotoma (although he occasionally has a sense the 'something happened there'). The retention of visual ability accompanied by denial of conscious experience following damage to the striate cortex is, of course, consistent with the classic definition of blindsight. This subject (and others like him, Wessinger *et al.*, 1997) therefore possesses blindsight, albeit only in a very small area of his visual field. This is all very well, but how can such spared cortex explain the apparently extensive region of blindsight found in GY and other blindsight subjects? One possibility is that they may have a number of 'islands' of spared cortex which they use to detect stimuli over a wide area. The patchiness of their blindsight would not be revealed in most studies because random movements of their eyes would often fortuitously bring unstabilized stimuli into a region of the retina which activated an island of spared cortex. Kentridge *et al.* (1997) tested this explanation using an image stabilization method with the subject GY. If GY's blindsight were mediated by islands of spared cortex then we expected to find that only a few of a set of arbitrarily selected locations within his blind field would support residual vision once stimuli were stabilized against eye-movements. Instead we found that his detection of appropriate stimuli in all but one rather peripheral location, out of the nine we tested in his upper visual field and horizontal midline, was significantly above chance. His performance in his lower visual field, remained poor. This suggests to us that an explanation of GY's blindsight in terms of islands of spared cortex is untenable. The lack of blindsight in the lower visual field may possibly be explained by damage extending beyond striate cortex into extrastriate regions. On their own these results do not rule

out mediation by some kind of diffuse sparing of striate cortex. Recent neuroimaging experiments by Stoerig *et al.* (1998) suggest otherwise. They found no changes of activity during functional magnetic resonance imaging of the damaged visual cortex of a blindsight patient after presentation of visual stimuli whereas changes did occur in extrastriate cortex. Despite the fact that they used a range of analytic methods to detect activity changes with abnormal time-courses, they failed to find evidence for islands of sparing, yet the spatial resolution of their images was sufficient to resolve stimulus dependent changes of extrastriate activity in volumes of the order of 5 mm<sup>3</sup>. This suggests that although Fendrich *et al.* (1992) did indeed find blindsight in an island of spared striate cortex, spared striate cortex is unlikely to account for blindsight found in subjects with extensive regions of residual vision such as GY, DB, the patient described in the original Weiskrantz (1986) monograph on blindsight, and FS, the subject of the Stoerig *et al.* (1998) study.

Most discussions of the anatomy of blindsight concentrate on the question of why blindsight patients retain some visual function. In this issue of the *Journal of Consciousness Studies*, Marzi (1999) and Pribram (1999) address different but equally important questions. Marzi's paper is entitled 'Why is blindsight blind?' Reviewing data from experiments in which an unseen or unattended stimulus influences reaction time to a second target in heminaopic and parietal neglect patients as well as normal subjects Marzi distinguishes between sub-cortical processing which cannot give rise to conscious experience and cortical processing which *may* give rise to such experience. Pribram's paper considers the properties of cortical processes which give rise to consciousness. His central thesis is that the processes which code the contexts in which experiences occur differ from those which organize the contents of experience. He distinguishes between these levels both in terms of their physical bases and, using neuropsychological examples, in terms of their psychological concomitants. Both of these papers, together with the evidence we outline above, suggest that *awareness* of the simple visual properties discriminable by blindsight subjects, such as contrast and location, depend on intact striate cortex, whereas their *detection* and *discrimination* do not. The story for other stimuli may, however, be more complicated.

### **Awareness in Blindsight and the Riddoch Phenomenon**

Since the original description of blindsight it has been found that some blindsight subjects report a form of conscious experience associated with the presentation of visual stimuli which move or flicker rapidly in intensity (Weiskrantz *et al.* 1995). This adds a complication to the question of whether blindsight is something other than degraded vision mediated by an only partially damaged striate cortex. We must now add the possibility that all of blindsight can be explained in terms of a second well known form of residual vision following brain damage first described by Riddoch (1917). In the 'Riddoch Phenomenon' patients who suffered damage to striate cortex were completely unable to discriminate or detect stationary stimuli but they could reliably detect moving stimuli. This residual ability was, however, always accompanied by conscious experience of motion. In their study of awareness and motion discrimination in GY, Zeki and ffytche (1998) note that GY's performance and awareness vary greatly among blocks of trials. In some blocks of trials GY reports no awareness yet his performance is better than chance while in others he

reports high levels of awareness, not always accompanied by good performance. Zeki and ffytche argue that when many blocks are averaged then GY's ability to discriminate the direction in which a stimulus is moving correlates with his degree of awareness as stimulus salience is changed. In general then, GY's residual motion detection resembles the Riddoch Phenomenon. They claim that, while it is true that in some blocks GY's performance is consistent with the definition of blindsight, this is a transitory phenomenon. They further claim that when awareness is reported by blindsight patients it is characterized by descriptions of visual experience. In other words, when blindsight patients are aware that they are detecting visual stimuli this awareness is a phenomenal visual experience rather than a 'feeling of knowing' which might be derived from monitoring secondary responses of the brain or body. Attempting to distinguish between these alternatives by interpreting the introspections of blindsight subjects is far from ideal. As Zeki and ffytche point out, GY changes the way he describes his introspections according to the type of description he thinks is expected — he himself says that sometimes he describes his experience in terms that he thinks a sighted person would understand.

Morland's (1999) paper in this issue of the *Journal of Consciousness Studies* reports new data on the relationship between the conscious experience of motion and its processing in the blind field. Instead of simply asking for an indication of whether GY is aware of a stimulus or not, Morland asked him to adjust the speed of a stimulus presented in his good field with one being simultaneously presented in his blind field. This method of asking GY to match between intact and blind fields is novel in blindsight research. GY's ability to match speeds was excellent, suggesting that the conscious process involved in his adjustment of stimulus speed in his good field had access to stimulus speed information from his blind field. It clearly begs application to other residual abilities such as discrimination of colour, luminance, orientation and so on. It is not obvious, however, that matching *per se* requires conscious experience of stimuli in the blind field. Once again, we must trust GY's report that he says he is conscious of the blind-field motion stimulus. It would be very interesting if there was a correlation between GY's ability to match stimulus properties between good and blind fields and his report of awareness of those properties. A correspondence of psychophysical dissociations between matching and forced choice performance and dissociations between reported awareness and forced choice performance would be well worth searching for.

Given the evidence that some stimuli *can* give rise to awareness in blindsight subjects two further questions present themselves: 'Is residual vision after striate cortex damage ever consistently unaccompanied by awareness?' and 'Is awareness of visual stimuli following striate cortex damage a phenomenal experience or a feeling of knowing?'

Studies using a commentary paradigm in which the subject formally reports on their awareness in addition to performing a discrimination task have tended to concentrate on motion perception, as moving stimuli often elicit awareness in blindsight subjects. There is disagreement over whether performance in these tasks is ever dissociated from awareness. These disagreements may be accounted for by the fact that motion is processed by at least two brain regions, one of which responds to quickly moving stimuli and one of which responds to slowly moving ones (see e.g. Gegenfurtner and Hawken, 1996). The division between 'slow' and 'fast' for these systems

is a speed of about 4 cycles per second for moving sinusoidal grating stimuli. Both Weiskrantz *et al.* (1995) and Zeki and ffytche (1998) used patches of light with gaussian profile rather than repeating sinusoidal patterns as stimuli. Equating the speed of motion of a gaussian blob with that of a sinusoidal grating is not straightforward; even so, there is still an apparent difference between GY's responses to fast and slow motion in these studies. Weiskrantz *et al.* (1995) reported that GY maintained high levels of performance in the absence of awareness for low velocity stimuli (moving at between 2.5 and 7.5 degrees per second). As noted above, Zeki and ffytche found that the dissociation between awareness and performance in motion discrimination was variable in their study. They argued that this variability undermined the concept of blindsight as a distinct phenomenon. It should, however, be noted, that the bulk of their data are derived from trials using stimuli moving at 15 degrees per second and faster, speeds at which Weiskrantz *et al.* (1995) also report high levels of awareness and hence do not claim that GY is demonstrating blindsight. Zeki and ffytche do report data from 3 blocks each of 50 trials using stimuli moving slowly at 2 degrees per second. GY reported no awareness during these blocks, his discrimination performance varied from chance (50%) on one block to 60% and 70% on the others. In fact, taken together, GY's performance on these blocks is significantly better than chance (the two-tailed binomial probability of obtaining 90 correct responses from 150 trials purely by chance is 0.011). Benson *et al.* (1998) did use sinusoidal grating in their study of motion discrimination with GY, however, none of their stimuli would produce large activation of the slow motion system since the minimum velocity used is of the order of 5 cycles per second.

We have used the commentary key paradigm in studies using stationary stimuli, albeit flashing ones, with GY and found that these stimuli can also give rise to reports of awareness when the stimuli are of sufficiently high contrast (Kentridge *et al.*, 1997; 1999). With low contrast stimuli, GY's discrimination performance is significantly above chance even though he reports awareness on extremely few trials (e.g. in the low contrast condition in Kentridge *et al.* (1999), GY's 2-alternate forced choice target location discrimination was correct on 253 of 320 trials (79%), yet he reported awareness on only 13 trials (4%). Our experience is that GY's performance does vary in the months between visits to our laboratory and between testing blocks during a visit; we do not, however, believe this accounts for the dissociation we find between performance and awareness. The data just described were collected over 4 separate test blocks. We did find considerable variability between his level of reported awareness (0%, 1.25%, 3.75% and 12.5%) while his performance remained relatively constant and well above chance (70%, 84%, 81% and 81%). In fact, if all of the trials where awareness was reported were removed, GY's performance in each individual block would still be well above chance (the maximum 2-tail binomial probability of obtaining any individual block performance by chance after removal of aware trials is less than 0.0005; the probability of obtaining this performance over all four blocks is vanishingly small (of the order of  $10^{-18}$ )). This is clear evidence that residual visual function without awareness is a real phenomenon, even if the case for such a dissociation specifically in motion discrimination is less than clear.

The alternative answers to our final question 'Is awareness of visual stimuli following striate cortex damage a phenomenal experience or a feeling of knowing?' lead to different predictions about the interaction between awareness and performance. Of

course, in either case, one would expect performance and awareness to covary as stimulus characteristics are varied over a limited range. An additional manipulation which suggests that awareness in blindsight is not visual was studied by Benson *et al.* (1998). They asked GY both to report his awareness and to make a direction discrimination on a series of stimuli. They compared the results when GY had to make the awareness report before the discrimination on each trial with those when he had to make the discrimination first and then report awareness. If awareness and discrimination are mediated by separate systems then attending to one may degrade a subject's ability to retrieve information from the other. On the other hand, if discrimination performance and awareness are all part of the same visual response, then attention is being directed to the same system no matter in which order responses are required. The fact that Benson *et al.* found that when asked to report awareness first, GY showed poorer discrimination, but higher levels of awareness than when he was asked to report direction of motion first, suggests that the GY is not directly aware of the information on which he bases his discriminations. Neuroimaging provides further evidence for separate systems mediating awareness and residual vision. Sahraie *et al.* (1997) compared the changes in brain-activity revealed by fMRI when GY either did or did not report awareness whilst making a motion direction discrimination. They found that different areas were activated during aware and unaware responses, 'aware' responses being associated with activation of the dorsolateral prefrontal cortex, whereas 'unaware' responses were associated with subcortical activity, notably in the superior colliculus.

### Conclusion

In conclusion, the crucial issue which determines whether blindsight is philosophically interesting is whether it is mediated by a pathway which conveys visual information without engaging consciousness. The first challenge to this position is the question of whether blindsight is distinguishable from normal vision near the threshold of discrimination. A number of studies indicate that blindsight is qualitatively different from near-threshold vision. The second challenge concerns the issue of whether blindsight is mediated by complete pathways which bypass the striate cortex and do not give rise to consciousness, even in normal observers, or whether it is mediated by spared tissue in the damaged pathway passing through the striate cortex. Studies using stabilized perimetry and neuroimaging both indicate that islands of spared cortex are unlikely to mediate blindsight in cases where it is spatially extensive. These studies cannot conclusively rule out mediation by some partial but diffuse damage; however, even in this case one might expect to detect some striate activation in imaging studies where none was found. A complication arises when the response of blindsight subjects to moving stimuli is considered. It may be the case that moving stimuli give rise to conscious visual experience in the absence of striate cortex. It does not, however, appear credible that all blindsight is based on this conscious experience. There is also evidence, from both behavioural and neuroimaging studies, which suggests that the basis of awareness and the basis for residual visual function in blindsight differ. We conclude that the status of blindsight as an example of the dissociability between access to visual information and phenomenal consciousness remains intact.

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